NATUR
WISSENSCHAFTEN AUFSÄTZE

Parasites and Carotenoid-based Signal Intensity: How General Should the Relationship Be?

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Evidence that selection by parasites maintains heritable variation in sexually selected signals (Hamilton-Zuk model) has proved equivocal. Bright individuals do not always have fewer parasites in intraspecific comparisons. Because the lymphocyte-based defence system and the production of some colors used in sexual signaling require carotenoids, we consider a trade-off between defence against parasites and sexual signals. The nature and the sign of the covariance between defence and signal brightness can vary. Depending on carotenoid availability and allocation, and the type of sexual signal, various relationships between parasite load and signal intensity are expected.

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The connection between sexual selection [12] and parasites was first formalized by Hamilton and Zuk [19]. They showed that frequency-dependent selection from rapidly evolving parasites and diseases can maintain fitness-related heritable variability for traits under directional sexual selection. Therefore, female mate choice based on male secondary sexual characteristics that signal health, and thereby resistance against prevalent parasites, can evolve continually. Their model provides a mechanism for both the maintenance of variability for traits that reveal good genes, and for the evolution of female choice for handicaps [63, 64] under the following premises (as summarized in [36, 44]): (1) the expression of secondary sexual characteristics in an individual depends on its general health and vigor; (2) parasites have a negative influence on host health and vigor and thereby on the expression of secondary sexual characteristics; (3) hosts and parasites are involved in a coevolutionary arms race, implying heritable variation in parasite resistance, and (4) females prefer mates with fewer parasites.

A number of predictions for the relationships among parasite burden, signal intensity, and female choice both within and among species have been generated from the hypothesis of Hamilton and Zuk [19]. Across species, male signal intensity, e.g., plumage brightness, should correlate positively with parasite load because reliable signals of parasite resistance acquire higher adaptive value. Within species, those males with lower parasite loads will produce more intense signals. Females, by choosing the most intense signals, will acquire appropriate resistance genes against prevalent parasites for their offspring.

To date, empirical tests of these predictions have yielded equivocal results both across ([19, 43, 54, 55]; but see [8, 45]) and within species (see Table 1). In the studies reported to date, females usually chose bright over dull males and generally chose those with fewer

Table 1. Studies investigating the relationships among secondary sexual signals, parasite load, and female choice. We chose only those examples in which secondary sexual signals were presumably based on carotenoid pigments. Where the evidence supports the hypothesis that females prefer bright males, and bright males have fewer parasites, or that females prefer males with fewer parasites, we entered a "+" in the table. A "0" indicates that no relationship was found, and a "−" indicates the opposite relationship to the stated one

	Parasites	Females prefer bright males	Bright males have fewer parasites	Females prefer males with fewer parasites	Ref.
Fish					
Guppy		−			[13]
Guppy		+			[25]
Guppy	*Gyrodactylus*			+	[31]
Guppy	*Camallanus*	+			[32]
Stickleback	*Ichthyopthirius*	+	+	+	[35]
Guppy	*Gyrodactylus*	+	+	+	[26]
Guppy		+			[39]
Stickleback	Trematodes	0	0	0	[14]
Stickleback	*Crepidostomum* cestodes		0		[16]
Stickleback	*Schistocephalus* cestodes		+		[16]
Stickleback	*Diphyllobothrium* cestodes		−		[16]
Reptiles					
Whiptail lizard	*Haemogregarines*		−		[50]
Whiptail lizard	*Plasmodium*		−		[46]
Broad-headed skink		0			[11]
Sand lizard	*Ixodes*		0		[41]
Birds					
House finch		+			[20]
Red jungle fowl		+			[65]
Red-winged blackbird	*Haemoproteus* *Plasmodium*		+	0	[56]
Sage grouse	*Haemoproteus*			0	[18]
Ring-necked pheasant	*Eimeria*			+	[23]
Red jungle fowl	*Ascaridia*	+	+	+	[66]
House finch		+			[21]
Zebra finch	Lice		−		[6]
Sage grouse	*Plasmodium*			+	[29]
Sage grouse	Haematozoa Coccidia			+	[52]
American goldfinch		+			[28]
Red-winged blackbirds	Microfilaria		+		[58]
Red-winged blackbird	*Leucocytozoon* *Haemoproteus* *Plasmodium* Tapeworm, flukes		0		[58]
Red-winged blackbird	*Leucocytozoon* *Haemoproteus* *Plasmodium* Tapeworm, flukes, mites, Microfilaria		0		[58]
Zebra finch		0			[9]
House finch		0			[22]
European kestrel		+			[42]
Redpoll	*Leucocytozoon* *Haemoproteus* *Trypanosoma*		0		[51]
Ring-necked pheasant	*Heterakis*			+	[24]

over those with more parasites. The relationship between color or signal brightness and parasite loads in natural populations is much less clear, however. Field-caught individuals with the brightest coloration within a species do not always have the lowest parasite burdens. In contrast, however, where parasite loads have been manipulated experimentally, infection causes the expected reduction in signal intensity and in female preference [26, 35, 66]. Results from intraspecific studies that examine the relationship between parasite burden, color or signal intensity, and female choice (or some subset thereof) are summarized in Table 1.

We present a line of reasoning that explains why the relationship between parasite burden and coloration may not be clear within natural populations. This thereby suggests that the prediction that males with lower parasite loads will generally produce more intense signals may only hold under certain restrictive situations. Male brightness need not represent low parasite loads alone. Many male signals involving color are based on carotenoid pigments that cannot be endogenously synthesized, but must be acquired from food [47]. Signals that are used in female choice have been shown to be based on dietary carotenoids in both fish [5, 32] and birds [4, 20], so attractive signals also demonstrate the ability to acquire and sequester carotenoids from a diet that is often carotenoid-poor [13]. Males, however, may need their carotenoids for more than just putting on a good show for the females. Carotenoids are essential precursors for immune function, enhancing B and T lymphocyte production [2]. Therefore the interaction between current parasite burden and colorful signals that reveal information about health and vigor is a complex one, in part mediated by carotenoid metabolism [33] and hormone production [15, 59, 60], and may be especially complex when carotenoid acquisition involves exposure to parasites [16]. Androgens, especially testosterone, stimulate the production of carotenoid-based signals [27, 61, 62] possibly by expropriating carotenoids and thereby reducing the availability of this limiting resource for immune defence. This could, in part, explain the immunosuppressant effect of androgen hormones (for a recent review see [15]). In addition, parasite infection has been shown to reduce the strength of carotenoid-based colors ([66] and references therein), possibly because parasites consume resources themselves or a parasite infection causes carotenoids to be drawn away from signals in favor of the immune defence system.

Since the production of sexual ornamentation appears to involve limited resources such as carotenoids for which other tissues including the immune system compete [33], an individual is faced with a trade-off; it can use its carotenoids for defence or for signaling. Life history theory provides the tools for examining such trade-offs and shows that, under different conditions and constraints, very different phenotypic outcomes can be expected [53]. For example, if resource acquisition and allocation pattern both vary, a trade-off can be expressed as a positive or a negative phenotypic correlation [40].

Here we examine the interplay of resource (carotenoid) acquisition ability, allocation pattern, and parasite infection rates. Individuals may vary in their access to or ability to incorporate carotenoids and in their allocation pattern, i.e., how much resource is shunted into signal and how much into defence. These two variables will affect the strength of the secondary sexual signal males can produce. By examining the relationships between acquisition and allocation of carotenoids with parasite exposure, we investigate the different possible interactions between sexual signals and parasite loads. Parasite load indicates the number of infections contracted or the number of parasites actually carried by an individual, which will, in turn, be a product of parasite exposure and the defence system.

We construct a verbal model with the following assumptions:

1. Parasites negatively affect host phenotype, reducing signal intensity or health, and possibly causing a shift in the allocation of carotenoid resources to defence versus signal.
2. Variation in immune defence and hence in susceptibility to parasites depends solely on resource (carotenoid) allocation strategy or acquisition ability. Thus we are not considering genetic variation in susceptibility due to resistance genes not involved with the carotenoid metabolism and carotenoid-based defence system.
3. Allocation decisions may be of two types: fixed or continuous. Fixed decisions involve the production of a signal phenotype only once for a given mating season. One example is the production of nuptial plumage during a molt that may occur long before mate choice. Continuous decisions are made before and during the mate choice process. They involve production of pigmented skin such as a cock's comb, the red throat and belly of a stickleback, or the smearing of carotenoid-rich secretions on a patch of strategically placed white feathers [48]. This type of signal must be maintained or renewed by additional input of carotenoids.

The Allocation Decision Is Made Once: Nuptial Plumage or the Redpoll's Rump

For fixed allocation decisions it is useful to view two extreme situations [40]. In the first, hereafter referred

to as Case 1 (Fig. 1 a), resource acquisition ability varies but allocation pattern varies little in the population. Thus some individuals have absolutely more resource, some less, but all allocate a similar proportion to defence versus signal. At the opposite extreme, Case 2 (Fig. 1 b), a similar amount of resource is available to all, but allocation pattern varies, with different individuals putting more or less into defence versus signal.

Completely different relationships between parasite load and signal result from these two very basic patterns. Under similar parasite exposure for all individuals, Case 1 will yield a negative relationship (Fig. 2 a). The brightest colored individuals will have the fewest parasites because they had more resource to devote to both signal and defence. The dullest will have the most parasites, having had little resource for either signal or

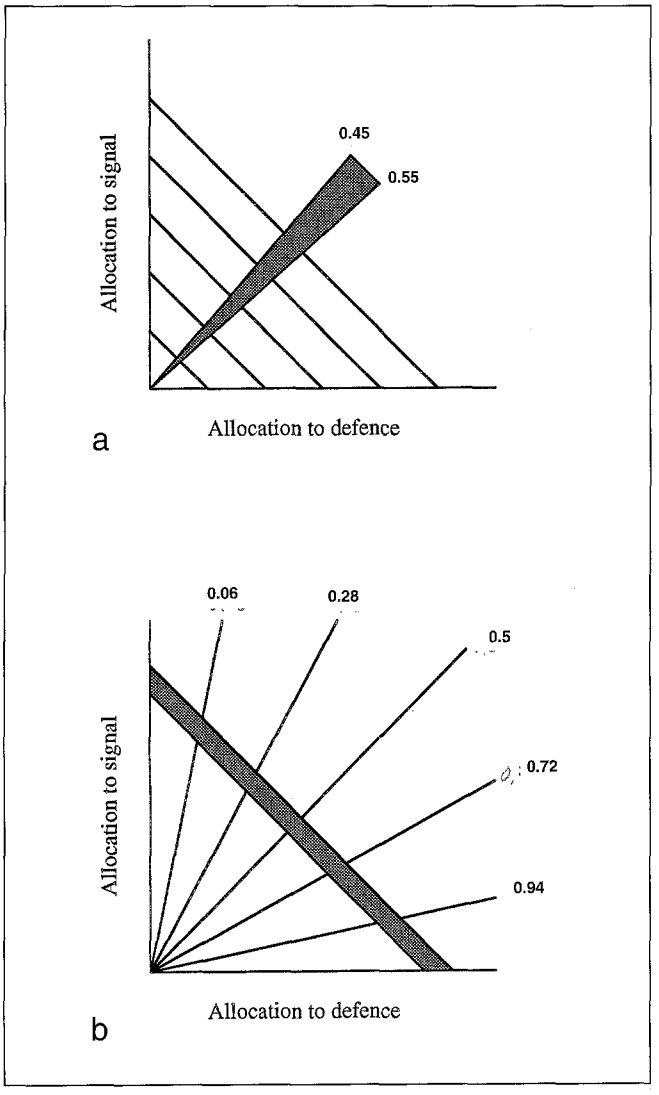

Fig. 1. Two extreme cases of variation in resource availability and allocation pattern. Different resource availabilities are represented by parallel lines of slope −1, different allocation patterns by lines radiating from the origin. The proportional allocation to defence is written at the end of each line. a) (Case 1) Resource availability varies and all individuals allocate approximately half of their resource to defence and half to signal. b) (Case 2) Resource availability varies little and allocation patterns vary greatly from allocation almost entirely to defence to allocation almost entirely to signal

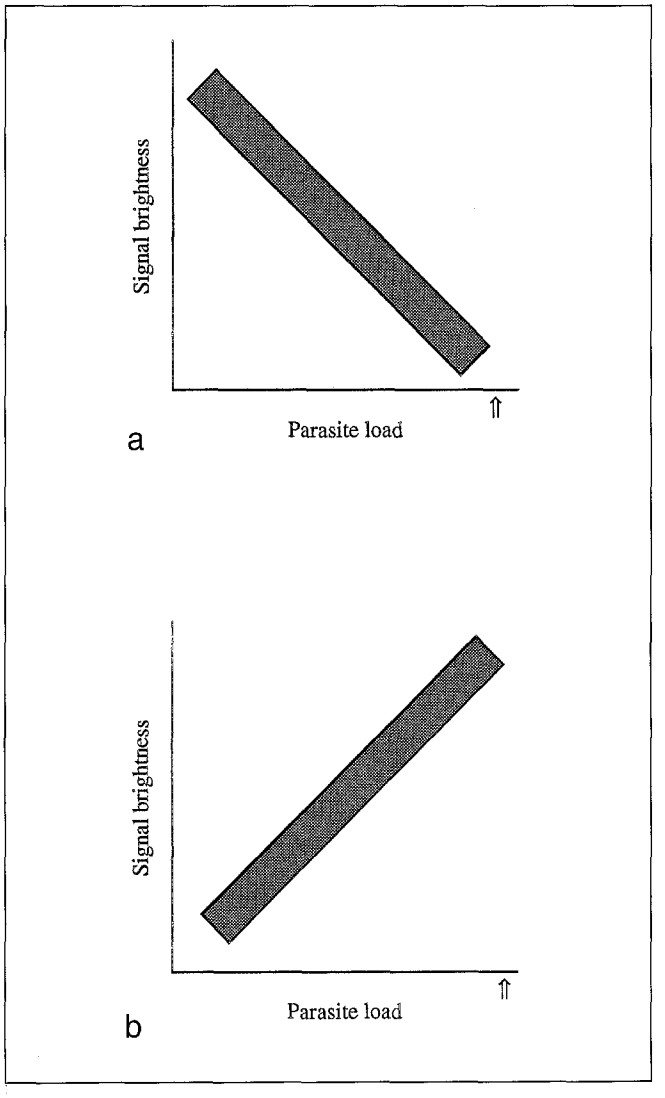

Fig. 2. The relationship between parasite load and signal brightness under the conditions of constant parasite pressure and variation in resource availability and allocation outlined in Fig. 1. Here we consider an arbitrary maximum in parasite load that an organism can tolerate (⇑) and assume that variation in signal covaries with variation in parasite load. a) (Case 1) There is a negative relationship between signal brightness and parasite load because those individuals with abundant resource produce bright signals and have good defence against parasites. b) (Case 2) A positive relationship between parasite load and signal brightness is evident because individuals that invested heavily in their signals could allocate less to defence

defence. Case 2 will lead to the opposite relationship (Fig. 2b). The brightest individuals are those that allocated most of their resource to signal. They, therefore, will have little available for defence and will thus be more susceptible to parasites. Those that shunt more resource into their immune defence will be more resistant to parasites, but at the cost of their signals.

Parasite exposure may not be constant, but rather may vary idependently of either resource availability or allocation strategy. Observed combinations of parasite load and signal brightness will then lie in two different triangular-shaped areas (Fig. 3). In both cases, relatively unprotected individuals may have lower parasite loads than their immunocompetence would predict, simply because they encounter few or no parasites in their environment [16].

In the situations outlined above, we consider an individual with a fixed capital of resource that can be allocated to one function or another, and parasites are encountered after the allocation decision has been made. If parasites are part of the environment during the production of the signal, they may affect the phenotype by reducing the amount of resource avialable for individuals with set allocation strategies. Reduction in resource availability will cause a reduction in signal intensity and a concomitant increase in parasite load. However, it will not affect the sign of the covariance between signal strength and parasite load found for Case 1. For Case 2, two situations are possible. Parasites may reduce the resources available for both functions as above, superimposing variation in resource availability on the variation in allocation. When both these factors vary, the outcome of parasite challenge will depend on the amount and allocation pattern of the resource. Reducing available resources will reduce the variation in signal strength expressed in the population, but the positive relationship between signal and parasite load will still hold. Alternatively, individuals may have flexible allocation patterns that respond to infection. If, with exposure to parasites, individuals modify their allocation strategy in favor of defence, the same phenomenon will be observed – variation in signal strength will be reduced but the positive relationship will stand. Both situations will produce the same phenotypic effect.

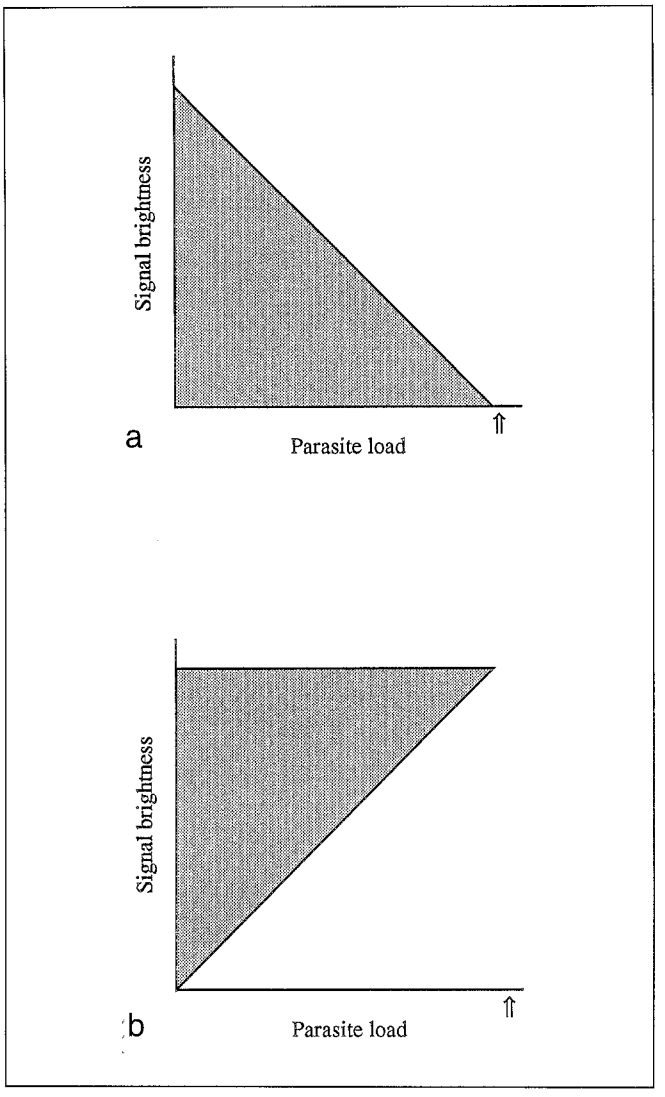

Fig. 3. The relationship between parasite load and signal brightness under varying parasite exposure with resource availability and allocation variation as outlined in Fig. 1 and maximal tolerable parasite load (⇑). Triangular relationships should be observed because parasite load represents differences in parasite exposure that vary independently of immunocompetence. Some individuals that in (a) (Case 1) had little resource to allocate to either signal or defence or in (b) (Case 2) allocated most of their resource to signal rather than defence may still have low parasite loads because they were exposed to few parasites

Continuous Allocation: the Belly of the Stickleback

If the signal under consideration is one that needs progressive input of resource, it is best to view allocation in the form of an allocation tree of repeated consecutive decisions [30]. A completely determined pattern of allocation, with a fixed proportion of resources always going to a particular function, will give rise to the same pattern as that observed with Case 1 fixed allocation. With a flexible allocation strategy, the outcome of this allocation tree will depend on the priority of either sink, signal or defence, at each decision node.

We consider the following scenario: individuals may vary in how they prioritize their sinks at each node of the decision tree. Some individuals may give higher

priority to signal than do others. High investment in signal is driven by sexual selection for mating advantage, but may involve higher risk of parasitization and less control of parasite replication rates during an infection. Similar to arguments about conspicuous sexually selected handicaps and predation [1], selection through mating advantage for bright signals should be balanced by mortality or morbidity selection via parasites. Thus optimal allocation strategies that differ for different parasite regimes should evolve. In addition, low allocation to defence may incur costs such as an individual's ability to invest subsequently into signal. High parasite loads could restrict the amount of resource available to maintain signals or could cause a shift in allocation pattern by placing more demand on the defence system (see above). The important variables in parasite regime are: parasite replication rates relative to the duration of the mating season during which signal must be maintained through continuing allocation decisions, and the costs in survival to subsequent seasons depending on the allocation strategy [49].

For parasites with slow replication rates relative to the period over which signal is maintained, there may be little immediate cost to starving the defence system in favor of signal. Parasite levels dictated by allocation decisions do not influence signal maintenance. This situation reflects that of one-time allocation discussed above in terms of nuptial plumage. In this case, for a single mating season, the more invested in signal, the brighter the signal, the higher the mating advantage, but also the higher the potential parasite load. If, on the other hand, parasites replicate rapidly and the signal must be maintained over a long mating season, high investment in signal at any allocation node may result in the immediate inability to allocate much or anything to signal at the next node.

In general, the higher the parasite exposure, the less resource can be allocated to the maintenance of signal. Therefore, for signals that must be maintained by continued input of resources throughout the breeding season, parasites will limit signal, either by placing more demands on the defence system or by restricting resource availability. Such renewable signals therefore give more reliable information about parasitization status than do the signal phenotypes that are produced once per breeding season. They are also less prone to cheating, because overgenerous allocation to signal at one allocation node will be rectified at the next if parasites are present.

Although this argumentation suggests that, within a species, the more parasites, the less bright will be the signal – exactly the predictions from the Hamilton and Zuk [19] model – flexible allocation strategies may allow for the control of parasite loads over a large range of parasite exposures. Within the controllable range, increasing parasite exposure may draw more resource to the defence system. Little variation in parasite load will then result if individuals can control their parasite populations (Fig. 4). Signal intensity will vary, however, as more resource is appropriated from signal (Fig. 4). Variation in signal intensity will therefore not be accompanied by variation in parasite load over the controllable range of parasite exposure. When parasite exposure increases beyond that which can be controlled, parasite burden will increase without associated variation in signal intensity (Fig. 4).

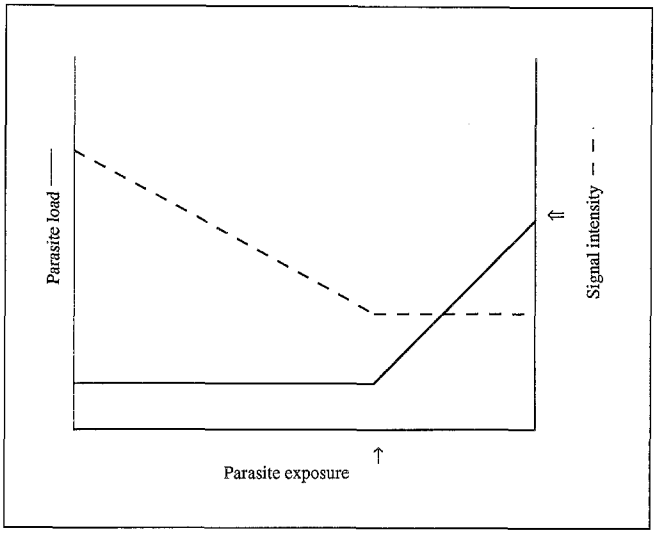

Fig. 4. The relationships between parasite exposure and both parasite load and signal brightness with repeated consecutive allocation decisions. Over a range of increasing parasite exposure, individuals may be able to control infections (parasite load –) by allocating increasing amounts of resource to defence, leading to a decrease in signal intensity (---). Only at the level of parasite exposure when all available carotenoid resources are allocated to defence (↑) does increased parasite exposure lead to increased parasite load. Therefore over the controllable range of parasite exposure signal intensity will vary without associated variation in parasite load. At higher levels of parasite exposure, parasite load will rise to the maximal tolerable (⇑) but signal intensity, already at a minimum, will not change

Discussion

This simple illustration of different conditions of resource availability and allocation patterns reveals that any relationship between parasite burden and the intensity of a sexually selected trait based on carotenoid pigments is possible. For one-time allocation decisions such as those involved with the production of nuptial plumage in birds, the sign of the relationship between parasite load as a function of immune defence and

plumage brightness will depend on resource availability and the allocation strategy. Variation in resource availability with invariant allocation strategies will result in a negative relationship between parasite load and plumage brightness – the relationship predicted by the Hamilton and Zuk hypothesis [19]. Variation in allocation with little variation in resource availability will result in the opposite relationship.

With continuous allocation to a signal that requires maintenance, such as the color of a stickleback's belly and throat, the relationship will depend on the relative priority of the sinks, signal or defence, at each decision node, and the replication rates of parasites relative to the duration of signal maintenance. Parasites with slow replication rates will produce relationships qualitatively similar to those predicted for one-time allocation decisions. Rapidly replicating parasites may generate the negative relationship between signal intensity and parasite burden predicted by the Hamilton-Zuk model. However, if organisms allocate their resources progressively to control their parasite burdens below a critical level, then over the range of parasite exposures controllable by immune response, no relationship between parasite burden and signal intensity is to be expected. Signal intensity will vary with the parasite challenge experienced, but this will not translate into actual parasite load.

Such considerations may explain the inconsistency of the findings reported in Table 1. We suggest some modifications of the Hamilton-Zuk model to clarify its predictions. The following factors should be considered:

What sort of signal is being measured, one with fixed or continuous allocation?

With fixed allocation decisions made once per breeding season, such as the color of nuptial plumage in birds, the nature and sign of the relationship between parasite load and signal intensity will depend on variation in parasite encounter rates in natural populations and whether resource availability or acquisition ability versus resource allocation strategy differs among individuals. In fact, any relationship between parasite burden and signal intensity might be expected in nature.

With continuous allocation to a renewable signal where parasites can influence the allocation pattern or the resource available for signal production, signal intensity may vary in the absence of variation in parasite load over the range of parasites that can be controlled by the defence system. At higher parasite pressure, parasite load may vary without variation in signal intensity. How both signal and allocation strategy will evolve should depend on the generation time of the parasites relative to the time scale over which the signal is produced. Several questions involving the nature of the signal should be addressed with theoretical models, for example: what generation times of parasites compared to the timing and nature of signal phenotype (one-time versus continuous allocation within a breeding season) will generate the coevolutionary cycles that can maintain additive genetic variability for signals? Do signals that require maintenance through continuing investment throughout the breeding season tend to be more honest depictions of a male's quality and those generated only once per breeding season more open to cheating?

Which of the two factors, resource acquisition ability or allocation strategy, varies in natural populations?

If allocation strategies do not vary within populations, variation in signal intensity and resource invested into defence will depend only on the amount of resource available to individuals. Habitats may vary in their ability to provide carotenoids. Individuals may vary in their ability in finding, metabolizing, and sequestering this resource. Under these conditions, signal intensity will covary negatively with parasite load, and a bright display will reveal a male's quality as forager, territory holder, or his metabolic superiority. On the other hand, allocation strategies may vary among individuals, in which case a bright signal may represent a poorly provisioned defence system.

A useful first approach would be to determine if different individuals respond differently to a controlled amount of dietary carotenoid. Kodric-Brown [32] measured the difference in coloration in split sib-ships of guppies reared with and without carotenoids in their diets. The treatment groups differed significantly, but the possibility of an interaction between sib-ships and treatment was not addressed. If some families were more, others less, efficient at converting environmentally available carotenoids into signal, this could suggest one of two things. Either there are genetic differences in the ability to acquire this resource, or there are differences in allocation patterns, with some families allocating more to signal, others more to the other demands for carotenoid pigments, such as the immune system. Which of these two mechanisms is operating could be resolved with an experiment. The immunocompetence of similar split sib-ships reared on carotenoid-enriched or reduced diets should be compared along with their color response. If individuals simply vary in their ability to acquire this resource, those that show higher response in pigmentation should also

show higher titers of lymphocytes and immune defence. If individuals vary in their allocation patterns, the opposite relationship should be evident.

How do parasites influence allocation to defence versus signal?

Parasites may simply reduce the resource levels available to both functions. Parasites have been shown to reduce energy intake by altering metabolism and food utilization efficiency [10, 38]. In addition, the presence of parasites can impose direct energetic costs [3]. Alternatively, the presence of parasites can lead to altered allocation patterns [17], shifting the strategy from reproductive effort, including signal maintenance, in favor of defence. Experimental exposure to parasites has been shown to reduce the intensity of sexual signals [26, 35, 66]. These experiments did not measure concomitant changes in immune response, so it is not clear whether the reduction in signal intensity was due to allocation to defence and away from signal, or reduction in overall resource availability.

Are we looking at the right parasites?

Another explanation for the lack of relationship between parasite load and signal could be that field surveys look at the wrong kind of parasites. As Read [44] pointed out, the predictions of the Hamilton-Zuk model [19] will only hold if parasite-induced negative effects on hosts occur at parasite intensities commonly encountered in the field. Investigations of hematozoa in red-winged blackbirds [56], brown-headed cowbirds [57], and whiptail lizards [50], and of trematodes in sticklebacks [14] revealed no difference between parasitized and unparasitized individuals in condition or, in the birds, in survival. Parasite infections below damaging levels will not produce coevolutionary cycling and may not adversely affect signal expression. Further complications arise when individuals expose themselves to parasites while acquiring carotenoids, as is the case for sticklebacks feeding on carotenoid-rich copepods that are the intermediate hosts for a number of cestode parasites [16].

The type of signal observed will determine the type of parasite that should be investigated. Rapidly multiplying parasites, with replication rates short relative to the duration of the mating season during which the signal must be maintained, will have the most impact on signals that require maintenance by continued input of pigments. However, if immune defence has priority over signal, signal intensity will vary far more than will parasite load over the levels of parasitism that can be controlled by the immune system, whereas at higher parasite exposure parasite load will vary without variation in signal. If defence against parasites involves varying investment to the lymphocyte-based immune system, measures of plasma lymphocyte titers might reveal important information about parasite pressure and immune demand. Field surveys that measure plasma lymphocyte levels as well as parasite numbers [49] could provide useful information about the interplay between signal, defence, and parasites.

Comparisons of populations that vary in their length of breeding season or number of expected breeding seasons could also illuminate the effect of relative generation times of parasites and duration of signaling in the hosts. Similarly, comparisons among host species that vary in these traits could be revealing. The more iteroparous a species or population relative to its parasite fauna, the more important is a functional defence system, therefore the less elaborate should be the signal if it is produced at the cost of the immune system. We therefore suggest that individuals of semelparous species or populations will be able to expend more resource on signal than on defence. Similarly, populations or species that harbor parasites with low replication rates or long relative generation times can invest more into signal than can those whose parasites, through rapid replication, will suppress their ability to maintain signals. In other words, defence must be mounted against those parasites for which the costs of parasitization are paid immediately in reduced reproductive success.

This discussion addresses only that variation in immune defence that depends on resource (carotenoid) allocation and acquisition ability (see p. 115). We are dealing therefore with environmentally induced variation in lymphocyte production and the lymphocyte-based defence system. This variation can, of course, have a genetic basis since ability to acquire and metabolize carotenoids, and the pattern of allocation to competing resource sinks may have a genetic basis. However, this variation may be superimposed upon intrinsic genetic resistance to certain parasites. Individuals with a better resistance profile would need to allocate less to their defence system to attain a given level of parasite control. If parasites influence either the allocation strategy or the amount of resource available for signal and defence, individuals with more or better resistance alleles would be able to produce more intense signals under similar parasite exposure and with similar amounts of resource. Further theoretical investigations are required to examine the interaction between the lymphocyte-based defence system that competes with other physiological functions such as signal production and alleles for resistance against parasites.

The set of relationships between carotenoid-based signals and parasite loads that we outline here will not hold for all types of male signals. In fact, where females chose less parasitized over more parasitized males in choice trials, the traits upon which they based their choice were signals that reflect current health, such as intrasexual aggression [56, 58], lek attendance ([29]; but see [18]), or the ability to perform strenuous courtship displays [7, 23, 31, 34]. Such signals should show a negative relationship with parasite load and are thereby signals less prone to cheating. Indeed, cheating, in the form of investing all available resources into signal, thereby weakening immune defence, may be untenable where additional cues besides color are used in mate choice. Having beautiful plumage but having to be nailed to the perch (Monty Python's Flying Circus [37]) to remain upright is not likely to contribute to mating success.

The development and refinement of these ideas was stimulated by discussions with Erika Bucheli, Markus Frischknecht, Donna Holmes, Alfred Köpf, Roj Lowe, and Katerina Pirounakis. The following people read and commented on an earlier draft of this manuscript: Malte Andersson, Anders Pape Møller, Jamie Reaser, Paul Schmid-Hempel and three anonymous reviewers. Support for the authors during the course of this work was provided by Swiss Nationalfonds Grants 31-33638.92 (to J.A.S.) and 5001-035217 (to P. Schmid-Hempel, for A.W.).

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