The 'sexual selection hypothesis' for the origin of aposematism

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Abstract

The evolution of aposematism, in which prey exhibit conspicuous signals indicating the presence of anti-predator defenses, is puzzling. Before predators learn to associate the signal with defense, increased visibility makes the conspicuous prey highly vulnerable to predation. Although several hypotheses have been proposed to explain the evolution of aposematism, they often assume that only predators recognize these signals. Yet, many studies show that aposematism signals can also be involved in mate choice. Here, we demonstrate that some aposematic signals may have originally evolved as mating signals driven by sexual selection. In this study, we analyze a mathematical model to explore how sexual selection can drive the evolution of aposematism. We thereby identify key features of this 'sexual selection hypothesis' for the origin of aposematism to be tested with empirical data. Our results show that the evolution of conspicuous signals through sexual selection increases the visibility of prey to predators and thus predation pressure. This, in turn, promotes the evolution of defense mechanisms, ultimately leading to aposematism when predators learn to associate the signal with defense. Additionally, we show that when sexual selection drives the evolution of aposematism, it often results in sexual dimorphism in both signaling and defense traits. These findings call for further research into the role of sexual selection in the evolution of aposematism.

1 Introduction

Many organisms display conspicuous 'warning' signals that warn predators of the presence of a defense mechanism, such as toxins or spines. This anti-predator strategy, known as aposematism, is taxonomically widespread, as it has been documented in plants (Lev-Yadun, 2009, 2024), molluscs (Cortesi and Cheney, 2010), insects (Lindstedt et al., 2017), reptiles (Toledo et al., 2011; Maan and Cummings, 2012), birds (Hedley and Caro, 2022) and mammals (Stankowich et al., 2011; Howell et al., 2021). The initial evolution of aposematism has been widely debated, as it may seem paradoxical at first glance, for at least two reasons. First, a new conspicuous signal evolving from a cryptic trait should be counter-selected because it increases the risk of individuals being detected by predators. Second, a new conspicuous signal is initially rare in the population and offers little protection to the prey since predators are unlikely to have already learned to recognize it as a warning signal. Despite this apparent paradox, many theories have shown that a mutant displaying a warning signal can be favored in a population of cryptic prey, thus explaining the initial evolution of aposematism. Notably, all the underlying mechanisms, detailed below, are based on the assumption that prey are initially defended. This is likely to be the case when defenses increase the prey's chances of surviving predator attacks (Leimar et al., 1986), with the resulting benefit outweighing the costs of maintaining these defenses.

The initial evolution of aposematism may be promoted by kin selection (Fisher, 1930; Benson, 1971; Waldman and Adler, 1979; Gittleman and Harvey, 1980; Harvey et al., 1982; Leimar et al., 1986; Broom et al., 2006; Scaramangas and Broom, 2022; Scaramangas et al., 2023). When a defended individual displaying a warning signal is attacked by a predator, the predator may learn to avoid the signal. This learning process reduces predation on the individual's relatives, provided they are likely to be nearby and display the same warning signal. Therefore, for kin selection to favor the initial evolution of aposematism, a predator attacking a prey individual must be likely to encounter the individual's relatives. This is the case for a fine-scale population structure, such as that caused by family grouping or low dispersal (Mallet and Singer, 2008).

Other theories have shown that the initial evolution of aposematism may be based on individual selection rather than kin selection. A defended individual may benefit from displaying a warning signal if it survives a first attack by a predator, leading the predator to avoid attacking it in future encounters (Järvi et al., 1981; Wiklund and Järvi, 1982; Sillén-Tullberg and Bryant, 1983; Sillén-Tullberg, 1985; Tullrot, 1994). A defended individual displaying a warning signal may also have higher fitness than cryptic individuals simply because

of the protection provided by aposematism (Servedio, 2000; Guilford, 1985). This protection provided by aposematism occurs when predators have learned to avoid the warning signal by attacking other defended prey displaying the signal. Since this process relies on the warning signal being sufficiently common in the prey population, it cannot explain the evolution of aposematism from a single mutant displaying a warning signal (Servedio, 2000). Nonetheless, a new warning signal could nevertheless become sufficiently common to be favored because of stochasticity (Mallet and Singer, 2008) or other aspects of predator psychology, such as dietary conservatism or neophobia (Lee et al., 2010; Aubier and Sherratt, 2015). It should be noted that the different mechanisms explaining the emergence of aposematism are not mutually exclusive and may operate in combination.

Most of the mechanisms explaining the initial evolution of aposematism rely on the assumption that warning signals are only recognized by predators. However, warning signals can also serve as the basis for mate choice (Rojas et al., 2018), as shown by numerous empirical studies (e.g., Reynolds and Fitzpatrick, 2007; Maan and Cummings, 2008, 2009; Nokelainen et al., 2012; Finkbeiner et al., 2014b; Jiggins et al., 2001; Estrada and Jiggins, 2008; Gordon et al., 2015; Chouteau et al., 2017). Such mate choice can generate sexual selection, i.e., selection through differential mating success, that is known to have favored the evolution of colorful signals (Hill, 1991; Stuart–Fox and Ord, 2004; Olsson et al., 2013; Maan and Sefc, 2013; Dale et al., 2015). It has therefore been proposed that some warning signals may have evolved initially as sexual signals (Mallet and Singer, 2008; Ruxton et al., 2004). According to this scenario, sexual selection may eventually lead to aposematism: when prey are defended, predators may associate these sexual signals favored by sexual selection with the prey's defenses to avoid attacking them.

While many mathematical models have studied the effects of sexual selection on the diversity of warning signals (Tazzyman and Iwasa, 2010; Boussens-Dumon and Llaurens, 2021; Ponkshe and Endler, 2022; Yeager and Penacchio, 2023; Maisonneuve et al., 2021, 2023), none has investigated the role of sexual selection in the initial evolution of aposematism. Yet, sexual selection favoring signal evolution should impact the evolution of aposematism by affecting the co-evolutionary dynamics between defense and signal (as in Leimar et al., 1986; Broom et al., 2008, in a case without sexual selection). Indeed, the evolution of a conspicuous sexual signal increases predation pressure, which should favor defense evolution. In turn, defense evolution increases the efficiency of predator learning, which should reduce predation pressure and thus affect signal and defense evolution.

In this study, we develop a mathematical model to investigate the coevolution of signal and defense, and

aim to examine the impact of sexual selection on the evolution of aposematism. Our results indicate that sexual selection can favor the initial evolution of aposematism, even when prey originally had no defense against predators. Assuming that female is the choosy sex, we also show that the evolution of aposematism through sexual selection is likely to result in strong sexual dimorphism in signal and defense, with a more pronounced effect on defense.

2 Model

We consider a sexually reproducing haploid population with a balanced sex ratio. Each individual expresses a trait $s_\bullet$, which determines the intensity of a signal it displays (note that, here and hereafter, the symbol $\bullet$ refers to the trait of a focal individual). Such a signal may take the form of a colorful pattern or exaggerated ornamentation. If an individual does not display a signal, then $s_\bullet = 0$. We assume that the signal can be recognized by both predators and mate-seeking females. Each individual also expresses a trait $d_\bullet$, which determines the individual's defense level, i.e., the efficiency of the defense mechanism in increasing the prey's survival probability in the event of a predator attack. For example, individuals may be poisonous, have an unpalatable taste, or may protect themselves by stinging. If an individual is completely harmless to predators, then $d_\bullet = 0$. We also assume that predators decide whether to attack prey based on their previous experiences, and that they may learn to associate the signal with defense, so that they avoid attacking prey displaying the signal.

We consider a Wright-Fisher model with a constant population size N that we assume to be large enough to ignore stochastic effects. We assume the following life cycle: (1) individuals survive or die depending on predation and the intrinsic costs associated with their traits; (2) females choose which males to mate with based on their signal; (3) mating pairs reproduce equally and offspring are produced; and (4) adults die so that generations do not overlap.

We investigate whether the combined action of sexual selection and viability selection can favor the evolution of signal and defense in a population, leading to predators to avoid attacking prey displaying the signal. In the following, we first describe how individual traits ($s_\bullet$ and $d_\bullet$) affect different components of individual fitness, including production costs, predation and sexual selection (sections 2.1 to 2.3). We then describe how these components together determine the fitness associated with a phenotype, depending on whether traits

are sex-independent or sex-dependent (section 2.4). Finally, we explain how we analyze the co-evolutionary dynamic in the model (section 2.5).

2.1 Production costs

Individuals allocate a part of their metabolism to the production of signal and defense. This allocation may occur at the expense of other biological functions (*e.g.*, tissue growth, thermoregulation, antioxidant defense). In line with empirical evidence (*e.g.*, Bryant and Julkunentiitto, 1995; Grill, 1999; Rigby and Jokela, 2000; Zalucki et al., 2001; Marak et al., 2003; Ojala et al., 2007; Lindstedt et al., 2010; Blount et al., 2012, 2023), we therefore assume that the production of the signal or defense induce constitutive costs. In our model, the fertility of an individual with traits $s_\bullet$ and $d_\bullet$ is proportional to

$$\mathcal{C}(s_\bullet, d_\bullet) = \exp\left[-\gamma_{\mathrm{cs1}}\, s_\bullet - \gamma_{\mathrm{cs2}}\, s_\bullet^2\right] \times \exp\left[-\gamma_{\mathrm{cd1}}\, d_\bullet - \gamma_{\mathrm{cd2}}\, d_\bullet^2\right], \tag{1}$$

which decreases as the individual's traits $s_\bullet$ and $d_\bullet$ increase. The first (resp. second) exponential accounts for the production costs associated with the signal (resp. defense). The parameters γ_{cs1} and γ_{cs2} (resp. γ_{cd1} and γ_{cd2}) tune the strength of the linear and quadratic production costs associated with the signal (resp. defense).

2.2 Predation

2.2.1 Prey detection by the predator

We assume that prey encounter predators at a constant rate p. After an encounter, the predator can detect the prey, depending on the intensity $s_\bullet$ of the prey's signal. The detection probability P_{D} increases with the individual's signal intensity $s_\bullet$ and is given by

$$P_{\mathrm{D}}(s_\bullet) = P_{\mathrm{D0}} + (1 - P_{\mathrm{D0}})\,(1 - \exp\left[-\beta_{\mathrm{D}}\, s_\bullet\right]), \tag{2}$$

where P_{D0} is the probability that a predator will detect a prey without signal (i.e., a prey with trait $s_\bullet = 0$) and β_{D} quantifies the extent to which the signal intensity increases the probability of detection.

2.2.2 Attack of the prey by the predator

If a predator detects a prey, it chooses whether or not to attack. A predator chooses to attack based on the prey's signal and its own previous experiences. We assume that the predator notices the prey's signal with probability

$$P_{\mathrm{N}}(s_\bullet) = 1 - \exp\left[-\beta_{\mathrm{N}}\, s_\bullet\right], \tag{3}$$

which increases with the intensity $s_\bullet$ of the prey's signal. The parameter β_{N} quantifies how much the signal intensity increases the probability of the signal being noticed. If the predator does not notice the signal (with probability $1 - P_{\mathrm{N}}(s_\bullet)$), it always attacks the prey. By contrast, if the predator notices the signal (with probability $P_{\mathrm{N}}(s_\bullet)$), it will only attack the prey if it has not learned to associate the prey's signal with defense, i.e., if it does not yet consider the prey's signal to be a warning signal.

The resulting attack probability on a prey with a signal intensity $s_\bullet$ is then given by

$$P_{\mathrm{A}}(s_\bullet) = (1 - P_{\mathrm{N}}(s_\bullet)) + P_{\mathrm{N}}(s_\bullet)\,(1 - \hat{\theta}), \tag{4}$$

where $\hat{\theta}$ denotes the proportion of experienced predators that have learned to associate the prey's signal with defense and thus avoid attacking signaling prey. To derive the expression for the proportion of experienced predators $\hat{\theta}$, we make assumptions about predator avoidance learning, as explained in the next subsection.

2.2.3 Predator avoidance learning

Following Ferreira and Marcon (2014), we assume that predators learn to avoid prey displaying the signal but also forget at rate λ. We assume that after attacking prey for which the predator has noticed the signal, the predator learns to avoid prey displaying the signal with probability $P_{\mathrm{L}}(d_\bullet)$. In line with empirical evidence (*e.g.*, Lindström et al., 1997; Skelhorn and Rowe, 2006b), the avoidance learning probability $P_{\mathrm{L}}(d_\bullet)$ increases with the defense level $d_\bullet$ of the prey, so that

$$P_{\mathrm{L}}(d_\bullet) = 1 - \exp\left[-\beta_{\mathrm{L}}\, d_\bullet\right], \tag{5}$$

where β_{L} modulates the effect of the prey defense level $d_\bullet$ on predator avoidance learning. Note that after attacking a defenseless prey (i.e., a prey with $d_\bullet = 0$), the predator has no negative experience and does not

learn to avoid signaling prey ($P_{\mathrm{L}}(0) = 0$).

We assume that the prey population is much larger than the predator population, and that the proportion of experienced predators reaches its equilibrium value before predation significantly changes the trait distribution in the prey population. Under these assumptions, the proportion of experienced predators at equilibrium is given by

$$\hat{\theta} = \frac{\int_{(s_\bullet, d_\bullet)} p P_{\mathrm{D}}(s_\bullet) P_{\mathrm{N}}(s_\bullet) P_{\mathrm{L}}(d_\bullet) f(s_\bullet, d_\bullet)\, \mathrm{d}s_\bullet\, \mathrm{d}d_\bullet}{\lambda + \int_{(s_\bullet, d_\bullet)} p P_{\mathrm{D}}(s_\bullet) P_{\mathrm{N}}(s_\bullet) P_{\mathrm{L}}(d_\bullet) f(s_\bullet, d_\bullet)\, \mathrm{d}s_\bullet\, \mathrm{d}d_\bullet}, \tag{6}$$

where f is the trait distribution in the prey population (see Appendix A1 for more details on the analytical derivations).

Eq. (6) shows that a balance between learning and forgetting determines the equilibrium proportion of experienced predators $\hat{\theta}$. Indeed, the proportion of experienced predators $\hat{\theta}$ decreases with the forgetting rate λ. In contrast, the proportion of experienced predators $\hat{\theta}$ is high when predators attack prey and notice their signal, and when they efficiently learn to associate the prey signal with defense. Overall, $\hat{\theta}$ depends on the prey's signal and defense. In other words, signal and defense within the prey population together determine predator avoidance learning, as shown empirically (Halpin et al., 2008a,b).

2.2.4 Prey escape

As detailed above, predators attack the prey they encounter with probability $P_{\mathrm{A}}(s_\bullet)$. We assume that the prey can escape an attack with a probability $P_{\mathrm{E}}(d_\bullet)$ that increases with the prey's defense level $d_\bullet$ (in line with empirical studies; Wiklund and Järvi, 1982; Skelhorn and Rowe, 2006a,c) so that

$$P_{\mathrm{E}}(d_\bullet) = 1 - \exp\left[-\beta_{\mathrm{E}}\, d_\bullet\right], \tag{7}$$

where β_{E} quantifies how the defense level increases the probability of prey escape. We assume that if the prey does not succeed in escaping, it gets killed by the predator.

2.2.5 Overall predation pressure

Overall, the fertility of an individual with traits $s_\bullet$ and $d_\bullet$ is proportional to

$$\mathcal{P}(s_\bullet, d_\bullet) = 1 - p\, P_{\mathrm{D}}(s_\bullet) P_{\mathrm{A}}(s_\bullet)(1 - P_{\mathrm{E}}(d_\bullet)), \tag{8}$$

which captures the effect of predation on survival. The prey survival is low (low $\mathcal{P}(s_\bullet, d_\bullet)$) when the prey is likely to be killed by predators (high $p\, P_{\mathrm{D}}(s_\bullet) P_{\mathrm{A}}(s_\bullet)(1 - P_{\mathrm{E}}(d_\bullet))$).

2.3 Sexual selection

The surviving adults come together to mate. We consider a panmictic population, where each individual encounters others with equal probability. We assume that all females mate equally. We also assume that females are the choosy sex and have the same mating preference, so that they prefer males with the most intense signal. The reproductive success of a male with signal intensity $s_\bullet$ is therefore given by the following sigmoid function

$$\mathcal{R}(s_\bullet) = \frac{1}{1 + \exp\left[-\rho(s_\bullet - s_{\mathrm{m}})\right]}, \tag{9}$$

where ρ describes the strength of female preference, and s_{m} is the mean male signal in the population. The reproductive success of a focal male, ranging from 0 to 1, increases as the intensity of his signal exceeds the population mean.

2.4 Fitness

2.4.1 Individual fitness

The reproductive output f_{m} and f_{f} of males and females with traits $s_\bullet$ and $d_\bullet$ are then given by

$$f_{\mathrm{m}}(s_\bullet, d_\bullet) = k\, \mathcal{C}(s_\bullet, d_\bullet) \mathcal{P}(s_\bullet, d_\bullet) \mathcal{R}(s_\bullet), \tag{10}$$

$$f_{\mathrm{f}}(s_\bullet, d_\bullet) = k\, \mathcal{C}(s_\bullet, d_\bullet) \mathcal{P}(s_\bullet, d_\bullet), \tag{11}$$

where $k > 0$ is a constant arbitrarily defined so that the mean fertilities of males and females in the population are greater than one.

The fitnesses w_{m} and w_{f} of males and females with traits $s_\bullet$ and $d_\bullet$ are given by

$$w_{\mathrm{m}}(s_\bullet, d_\bullet) = \frac{f_{\mathrm{m}}(s_\bullet, d_\bullet)}{\overline{f}_{\mathrm{m}}}, \tag{12}$$

$$w_{\mathrm{f}}(s_\bullet, d_\bullet) = \frac{f_{\mathrm{f}}(s_\bullet, d_\bullet)}{\overline{f}_{\mathrm{f}}}, \tag{13}$$

where $\overline{f}_{\mathrm{m}}$ and $\overline{f}_{\mathrm{f}}$ are the mean fertilities of males and females in the population, respectively.

2.4.2 Phenotypic fitness

The evolutionary dynamics hinge on the fitness of individuals, characterized by their signaling and defense traits, and therefore depend on whether the traits expressed in females and males are encoded by the same genes or by different genes. We thus consider two versions of the model that differ in how we define the phenotypic space: a model with sex-independent traits where the population is necessarily sexually monomorphic, and a model with sex-dependent traits where the population can be sexually dimorphic.

Model with sex-independent traits. In this model, the signal and the defense of an individual does not depend on whether the individual is a male or a female. Each individual is characterized by a phenotypic vector $\mathbf{x}_\bullet = (s_\bullet, d_\bullet)$. Since the sex ratio is assumed to be balanced, the fitness associated with phenotype $\mathbf{x}_\bullet$ is given by:

$$w(\mathbf{x}_\bullet) = \frac{1}{2} w_{\mathrm{f}}(s_\bullet, d_\bullet) + \frac{1}{2} w_{\mathrm{m}}(s_\bullet, d_\bullet). \tag{14}$$

Model with sex-dependent traits. In this model, the signal and the defense of an individual depends on whether the individual is a male or a female. Each individual is characterized a phenotypic vector $\mathbf{x}_\bullet = (s_{\mathrm{f}\bullet}, d_{\mathrm{f}\bullet}, s_{\mathrm{m}\bullet}, d_{\mathrm{m}\bullet})$. If the individual displaying phenotype $\mathbf{x}_\bullet$ is female (resp. male), it expresses the traits $s_{\mathrm{f}\bullet}$ and $d_{\mathrm{f}\bullet}$ (resp. $s_{\mathrm{m}\bullet}$ and $d_{\mathrm{m}\bullet}$). The fitness associated with the phenotype $\mathbf{x}_\bullet$ is then given by:

$$w(\mathbf{x}_\bullet) = \frac{1}{2} w_{\mathrm{f}}(s_{\mathrm{f}\bullet}, d_{\mathrm{f}\bullet}) + \frac{1}{2} w_{\mathrm{m}}(s_{\mathrm{m}\bullet}, d_{\mathrm{m}\bullet}). \tag{15}$$

2.5 Model analyses

2.5.1 Invasion analyses

We assume that mutations are rare and have little effect on phenotype. Based on this assumption, the phenotypic evolutionary dynamic can be inferred from the invasion of rare mutants into a resident population (Champagnat et al., 2006; Mullon and Lehmann, 2019).

We denote $w_{\text{inv}}(\mathbf{x}_\bullet, \mathbf{x})$ the invasion fitness of a mutant, i.e., the fitness of a rare mutant with phenotype $\mathbf{x}_\bullet$ in a population with phenotype $\mathbf{x}$. The invasion fitness of a mutant with phenotype $\mathbf{x}_\bullet$ in a resident population with phenotype $\mathbf{x}$ (with $\mathbf{x} = (s, d)$ or $\mathbf{x} = (s_{\text{f}}, d_{\text{f}}, s_{\text{m}}, d_{\text{m}})$ depending on whether we consider sex-independent or sex-dependent traits) is given by

$$w_{\text{inv}}(\mathbf{x}_\bullet, \mathbf{x}) = w(\mathbf{x}_\bullet). \tag{16}$$

Note that $w(\mathbf{x}_\bullet)$, defined in equations 14 and 15, depends on the resident phenotype $\mathbf{x}$ because the proportion of experienced predators $\hat{\theta}$ actually depends on the resident phenotype $\mathbf{x}$ in the prey population (see Appendix A2).

We assume that mutations independently affect each trait. The change in phenotypic traits per unit of time can then be approximated by

$$\Delta\mathbf{x} = N\,\mu\,\sigma^2\,\mathbf{g}(\mathbf{x}), \tag{17}$$

where μ is the mutation rate, σ^2 is the variance in mutational effects and $\mathbf{g}(\mathbf{x})$ is the selection gradient (Dieckmann and Law, 1996; Champagnat et al., 2006; Metz, 2011; Ripa and Dieckmann, 2013). The selection gradient is defined by

$$\mathbf{g}(\mathbf{x}) = \left(\frac{\partial w_{\text{inv}}(\mathbf{x}_\bullet, \mathbf{x})}{\partial a_\bullet}\right)_{a \in T}, \tag{18}$$

where $T = \{s, d\}$ or $T = \{s_{\text{m}}, s_{\text{f}}, d_{\text{m}}, d_{\text{f}}\}$ depending on whether we consider sex-independent or sex-dependent traits. The selection gradient describes the direction of evolutionary changes within the phenotypic space. The expressions of the selection gradients in models with sex-independent or sex-dependent traits are shown in Appendix A3.

2.5.2 Assumptions on the ancestral population

We consider that the ancestral population has reached a state of evolutionary equilibrium, with sexual selection playing no role. In the absence of sexual selection, we assume that signaling is deleterious and that the ancestral population is therefore not aposematic (see Appendix A5). Sexual selection on the signal then occurs, disrupting this equilibrium and affecting the evolution of signaling and defense.

In a first analysis, we assume that the ancestral population is not defended at its evolutionary equilibrium, that is, the selection gradients for defenses within this ancestral population are negative ($g_d(\mathbf{0}) < 0$, $g_{d_\mathrm{f}}(\mathbf{0}) < 0$ and $g_{d_\mathrm{m}}(\mathbf{0}) < 0$; see eqs. (A8), (A10) and (A12), and note that this occurs under the same conditions for both sex-independent and sex-dependent traits). This means that, ancestrally, selection inhibits the evolution of defense, which is a condition that prevents the evolution of aposematism. In this 'worst-case scenario', we investigate whether sexual selection can enable the initial evolution of aposematism through the evolution of signaling and defense in the prey. We analytically determine the conditions required for the emergence of signal and defense from the expression of the selection gradients. We then numerically estimate the phenotype $\mathbf{x}^*$ reached at evolutionary equilibrium when sexual selection takes place. At this evolutionary equilibrium, we assess the degree of aposematism as the proportion of experienced predators $\hat{\theta}$ that avoid attacking prey displaying the signal.

In a second analysis, we take a more general approach and consider that defense may evolve in the ancestral population even without sexual selection. As in the previous analysis, we numerically estimate the phenotype reached at evolutionary equilibrium. But this time, we choose parameter values at random (see details of the procedure in Appendix A4) and assess whether sexual selection correlates with the evolution of signal and defense (i.e., with aposematism).

3 Results

3.1 Evolution of aposematism in ancestrally defenseless populations

3.1.1 Sexual selection and predation jointly promote the evolution of aposematism

We consider the model with sex-independent traits, in which the population is assumed to be sexually monomorphic. As explained above, we assume that when there is no sexual selection on the signal, prey defense is counter-selected; therefore, the population is ancestrally defenseless. We show that the condition for the initial evolution of the signal (that is, in a population characterized by $s = 0$ and $d = 0$) is then

$$\underbrace{\frac{\rho}{4}}_{\substack{\text{benefit associated}\\ \text{with increased male}\\ \text{reproductive success}}} > \underbrace{\gamma_{\text{cs1}}}_{\substack{\text{constitutive cost}\\ \text{associated with}\\ \text{displaying signal}}} + \underbrace{\frac{1}{P^{\circ}(0,0)} p\, P'_{\text{D}}(0)}_{\substack{\text{cost associated with}\\ \text{increased detectability}\\ \text{by predators}}}, \tag{19}$$

where $P^{\circ}(s, d)$ is the probability that a resident prey is not killed by a predator when traits s and d are expressed in the population (see Eq. (A7)) and $P'_{\text{D}}(s) = \beta_{\text{D}}(1 - P_{\text{D}}(s))$. See Appendix A6.1 for details on the derivation.

Eq. (19) shows that the costs inhibiting signal evolution are twofold: displaying the signal comes with constitutive costs and increases detectability by predators. These costs may prevent the initial evolution of the signal in the population (*e.g.*, as in Fig. 1a). Nonetheless, Eq. (19) also reveals that the signal can be favored by sexual selection if females sufficiently prefer it. Indeed, a high reproductive success of signaling males (high ρ) can offset the costs associated with signaling, favoring signal evolution (*e.g.*, as in Fig. 1b-c).

The evolution of the signal is associated with an increase in the probability of prey being killed by predators (via an increase in detectability). This affects selection on prey defense. Indeed, the condition for the initial evolution of defense once the signal has evolved (that is, in a population characterized by $s > 0$ and $d = 0$) is

$$\underbrace{\frac{1}{P^{\circ}(s,0)} p P_{\text{D}}(s) P'_{\text{E}}(0)}_{\substack{\text{benefit associated with}\\ \text{increased escape probability}}} > \underbrace{\gamma_{\text{cd1}}}_{\substack{\text{constitutive cost associated}\\ \text{with having a defense}}}, \tag{20}$$

where $P'_{\text{E}}(d) = \beta_{\text{E}}(1 - P_{\text{E}}(d))$. See Appendix A6.2 for details on the derivation.

Eq. (20) shows that defense can evolve despite its constitutive cost because it enables prey to escape predators.

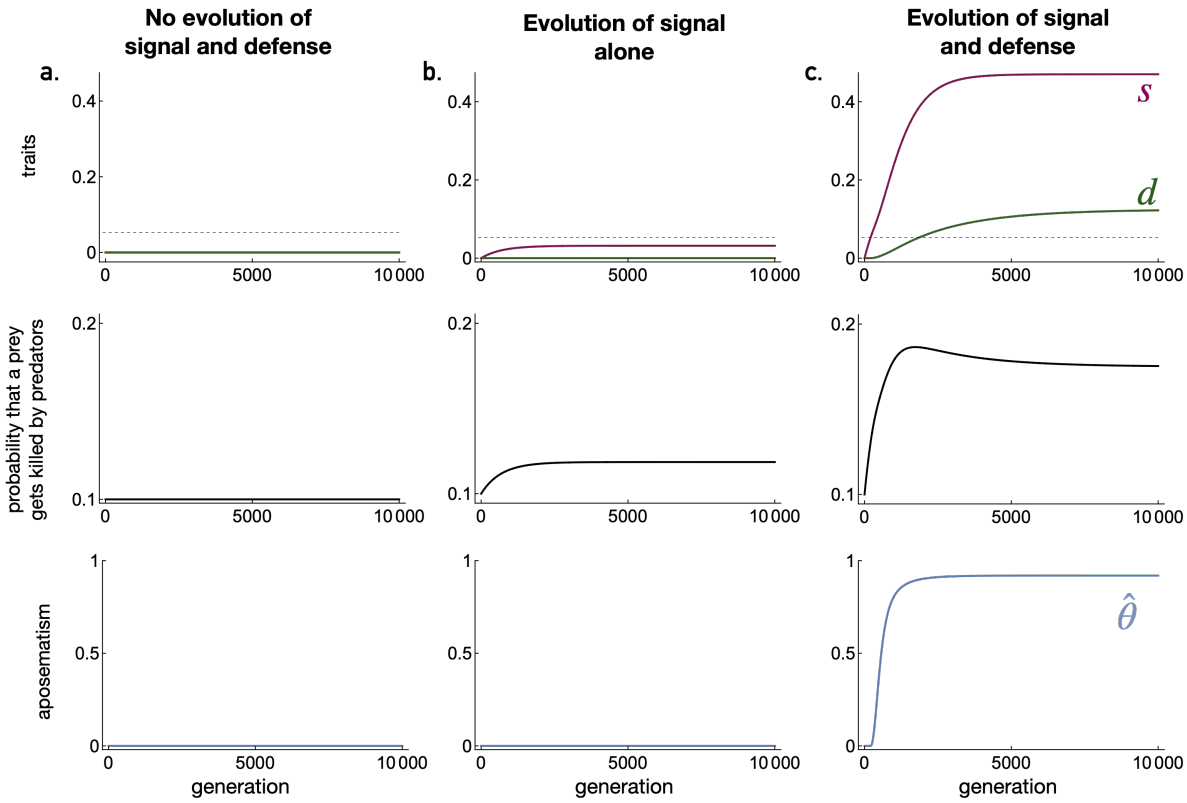

Figure 1: **The evolution of signal and defense in the model with sex-independent traits.** Temporal dynamics under three different scenarios of mean signal intensity (s) and defense level (d), the probability that a prey gets killed by predators, and aposematism quantified as the proportion of experienced predators. **a.** When females express weak preference for male signal ($\rho = 2.25$), costs associated with signal prevent its evolution. **b.** When females express moderate preference for male signal ($\rho = 3.25$), sexual selection promotes the evolution of signal despite the associated costs. Nevertheless, the signal does not become intense and does not increase predation pressure enough for defense to be favored. **c.** When females express strong preference for male signal ($\rho = 4.25$), sexual selection promotes the evolution of an intense signal. The evolution of an intense signal increases predation pressure and promotes the evolution of defense against predators. In all panels, the dashed line represents the threshold value outlined in Eq. (21); defense can initially evolve when the intensity of the signal in the population has reached this threshold value (as occurs in panel **c.**, but not in panels **a.-b.**). Parameter values are: $\gamma_{\text{cs1}} = 0.1$, $\gamma_{\text{cs2}} = 1$, $\gamma_{\text{cd1}} = 0.15$, $\gamma_{\text{cd2}} = 0.25$, $\beta_{\text{D}} = 1.5$, $\beta_{\text{N}} = 1$, $\beta_{\text{E}} = 1$, $\beta_{\text{L}} = 100$, $\lambda = 0.001$, $P_{\text{D0}} = 0.2$ $p = 0.5$.

Defense is particularly beneficial when the predation pressure is high (high $p\,P_{\text{D}}(s)$), including when prey are highly detectable (high $P_{\text{D}}(s)$). As the evolution of the signal through sexual selection increases prey detectability and therefore predation pressure, it favors the initial evolution of defense. We show that the intensity of the signal must exceed a threshold value $\tilde{s}$ for predation pressure to be high enough to favor defense evolution (see Appendix A6.1 for derivation):

$$\tilde{s} = \frac{1}{\beta_{\text{D}}} \log \left[\frac{p(\beta_{\text{E}} + \gamma_{\text{cd1}})(1 - P_{\text{D0}})}{p(\beta_{\text{E}} + \gamma_{\text{cd1}}) - \gamma_{\text{cd1}}} \right]. \tag{21}$$

When signal intensity remains below this threshold, there is no defense evolution (*e.g.*, as in Fig. 1a-b). In

contrast, when signal intensity exceeds this threshold, predation pressure becomes sufficiently high to favor the initial evolution of defense (*e.g.*, as in Fig. 1c).

As signaling and defense evolve, predators may learn to avoid prey that display the signal; the prey's signal is considered as a warning signal by experienced predators, and aposematism has thus evolved (*e.g.*, as in Fig. 1c). The evolution of prey defense, in turn, influences the selective pressures acting on the signal. To investigate this feedback on signal evolution, let us consider the selection gradient on the signal in a population characterized by $s > 0$ and $d > 0$:

$$\begin{aligned} g_{\mathrm{s}}(\mathbf{x}) = {} & \overbrace{\frac{\rho}{4}}^{\substack{\text{benefit associated}\\ \text{with increased male}\\ \text{reproductive success}}} - \overbrace{(\gamma_{\mathrm{cs1}} + 2\,s\,\gamma_{\mathrm{cs2}})}^{\substack{\text{constitutive costs}\\ \text{associated with}\\ \text{displaying signal}}} \\ & - \underbrace{\frac{pP_{\mathrm{A}}(s,d)(1-P_{\mathrm{E}}(d))\beta_{\mathrm{D}}(1-P_{\mathrm{D}}(s))}{P^{\circ}(s,d)}}_{\substack{\text{cost associated with}\\ \text{increased detectability}\\ \text{by predators}}} + \underbrace{\frac{pP_{\mathrm{D}}(s)(1-P_{\mathrm{E}}(d))}{P^{\circ}(s,d)}\,\hat{\theta}(s,d)P'_{\mathrm{N}}(s)}_{\substack{\text{benefit of being considered}\\ \text{as defended by}\\ \text{experienced predators}}}, \end{aligned} \tag{22}$$

where $P'_{\mathrm{N}}(s) = \beta_{\mathrm{N}}\,(1 - P_{\mathrm{N}}(s))$ (see Eq. (A6)). The selection gradient $g_{\mathrm{s}}(\mathbf{x})$ indicates the direction of selection on signal intensity. For instance, a more intense signal is favored when this selection gradient is positive.

The first three terms of Eq. (22) are analogous to the terms found in Eq. (19) describing the condition for the initial evolution of signaling. In contrast, the last term of Eq. (22) is equal to zero when $d = 0$ and is therefore not found in Eq. (19). It shows that once signaling and defense have both evolved, aposematism promotes the evolution of a more intense signal. This is because signal intensity increases the chance of the signal being noticed and avoided by experienced predators that consider the signal as a warning signal. As signal intensity increases, aposematism strengthens, reducing the probability of prey being killed by predators (*e.g.*, as in Fig. 1c). This benefit is high when most predators are experienced and avoid attacking prey that display the signal (high $\hat{\theta}(s,d)$), and when signal intensity greatly increases the probability of predators noticing the signal (high $P'_{\mathrm{N}}(s)$). The proportion $\hat{\theta}(s,d)$ of experienced predators that consider the signal as a warning signal is given by

$$\hat{\theta}(s,d) = \frac{p\,P_{\mathrm{D}}(s)P_{\mathrm{N}}(s)P_{\mathrm{L}}(d)}{\lambda + p\,P_{\mathrm{D}}(s)P_{\mathrm{N}}(s)P_{\mathrm{L}}(d)}. \tag{23}$$

See Appendix A2 for details on the derivation. The proportion of experienced predators is high when predators frequently attack prey and notice their signal (high $p\,P_{\mathrm{D}}(s)P_{\mathrm{N}}(s)$), when they efficiently learn to

associate the signal with defense (high $P_{\mathrm{L}}(d)$), and when they have a low forgetting rate (low λ).

To further study the impact of aposematism on predation pressure, we investigate the effects of predator forgetting rate λ on traits and the probability of prey being killed by predators at evolutionary equilibrium. When the predator forgetting rate is low, the proportion of experienced predators becomes high (Fig. 2), corresponding to a case with strong aposematism. As described earlier, we find that such strong aposematism leads to a low probability of prey being killed by predators (Fig. 2). However, sensitivity analyses indicate that, on average, the predator forgetting rate has a very weak effect on the probability of a prey being killed by a predator (see Tab. A1). This is because protection provided by aposematism favors the evolution of a signal with high intensity, which increases predation pressure, partially offsetting the benefits of aposematism.

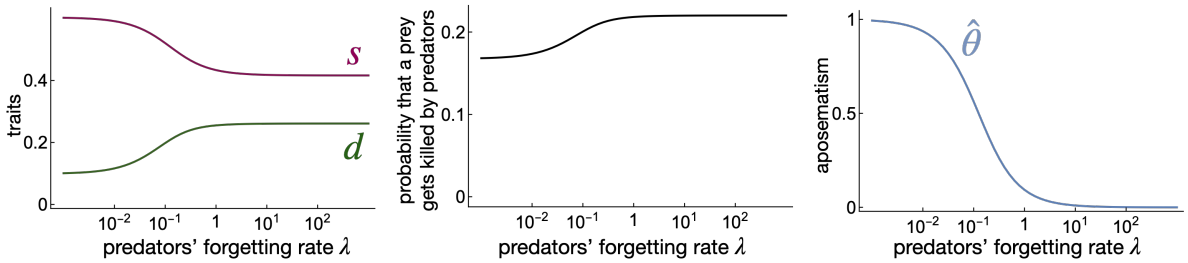

Figure 2: **Impact of predators' forgetting rate on the evolution of signal and defense in the model with sex-independent traits.** Mean signal intensity s and defense level d, probability that a prey gets killed by predators, and aposematism quantified as the proportion of experienced predators, depending on the predators' forgetting rate λ. Parameter values are: $\rho = 5$, $\gamma_{\mathrm{cs1}} = 0.1$, $\gamma_{\mathrm{cs2}} = 1$, $\gamma_{\mathrm{cd1}} = 0.15$, $\gamma_{\mathrm{cd2}} = 0.25$, $\beta_{\mathrm{D}} = 1.5$, $\beta_{\mathrm{N}} = 1$, $\beta_{\mathrm{E}} = 1$, $\beta_{\mathrm{L}} = 100$, $P_{\mathrm{D0}} = 0.2$ $p = 0.5$.

3.1.2 The evolution of aposematism results in sexual dimorphism in signaling and defense

In the previous section, we demonstrated that the interplay between sexual selection and predation can favor the evolution of aposematism. However, we assumed that males and females necessarily express the same signal intensity and defense level, so that sexual monomorphism is maintained in the population. Since sexual selection only affects the reproductive success of males in our model, we now investigate how aposematism can evolve in a population that may be sexually dimorphic. We consider an extreme case where males and females express different signaling and defense traits that are encoded by different genes. In this context, the female signal is not under sexual selection.

We show that the condition for the initial evolution of signal and defense in males (that is, in a population characterized by $s_{\mathrm{f}} = 0$, $d_{\mathrm{f}} = 0$, $s_{\mathrm{m}} = 0$ and $d_{\mathrm{m}} = 0$, and then by $s_{\mathrm{f}} = 0$, $d_{\mathrm{f}} = 0$, $s_{\mathrm{m}} > 0$ and $d_{\mathrm{m}} = 0$) is qualitatively similar to that for the initial evolution of the signal and defense in the previous model with

sex-independent traits (see Appendix A7.1).

Once signal and defense have evolved in males, the condition for the initial evolution of female signal (that is, in a population characterized by $s_\mathrm{f} = 0$, $d_\mathrm{f} = 0$, $s_\mathrm{m} > 0$ and $d_\mathrm{m} > 0$) is

$$\overbrace{\frac{pP_{\mathrm{D}0}}{P^{\circ}(0,0)}\,\hat{\theta}(0,0,s_\mathrm{m},d_\mathrm{m})P'_\mathrm{N}(0)}^{\substack{\text{benefit of being considered}\\ \text{as defended by}\\ \text{experienced predators}}} > \overbrace{(\gamma_{\mathrm{cs}1} + 2\,s\,\gamma_{\mathrm{cs}2})}^{\substack{\text{constitutive costs}\\ \text{associated with}\\ \text{displaying signal}}} + \overbrace{\frac{p\beta_\mathrm{D}(1-P_{\mathrm{D}0})}{P^{\circ}(0,0)}}^{\substack{\text{cost associated with}\\ \text{increased detectability}\\ \text{by predators}}}\,, \tag{24}$$

where

$$\hat{\theta}(s_\mathrm{f},d_\mathrm{f},s_\mathrm{m},d_\mathrm{m}) = \frac{\frac{1}{2}p\,\left[P_\mathrm{D}(s_\mathrm{f})P_\mathrm{N}(s_\mathrm{f})P_\mathrm{L}(d_\mathrm{f}) + P_\mathrm{D}(s_\mathrm{m})P_\mathrm{N}(s_\mathrm{m})P_\mathrm{L}(d_\mathrm{m})\right]}{\lambda + \frac{1}{2}p\,\left[P_\mathrm{D}(s_\mathrm{f})P_\mathrm{N}(s_\mathrm{f})P_\mathrm{L}(d_\mathrm{f}) + P_\mathrm{D}(s_\mathrm{m})P_\mathrm{N}(s_\mathrm{m})P_\mathrm{L}(d_\mathrm{m})\right]}, \tag{25}$$

is the proportion of experienced predators when the prey population is characterized by the phenotypic vector $(s_\mathrm{f}, d_\mathrm{f}, s_\mathrm{m}, d_\mathrm{m})$.

Eq. (24) indicates that despite the constitutive costs and the increased predation risk associated with increased detectability, female signaling can be beneficial when there is aposematism mediated by signaling and defense in males. Here, aposematism allows signaling females to be identified as defended by predators, even if they are defenseless. If this benefit is substantial, female signal can evolve (*e.g.*, as in Fig. 3). This is the case, for instance, when predators have a low forgetting rate leading to a high proportion of experienced predators (Fig. 4). Interestingly, we observe a high degree of sexual dimorphism in signal at evolutionary equilibrium (Fig. 4). This is due to sexual selection, which results in the intensity of the male signal being greater than that of the female signal.

We next examine whether female defense can evolve once signaling (in both males and females) and male defense have evolved. We show in Appendix A7.3 that female defense cannot evolve if the population is ancestrally defenseless. Remember that we assume that in the absence of signaling, the constitutive cost of defense exceeds the benefit associated with increased escape probability. Yet, the evolution of female signal reduces predation pressure on females by allowing them to benefit from aposematism (Fig. 3). This further diminishes the benefit of female defense (the increase in escape probability), inhibiting its evolution. As a result, the population displays sexual dimorphism in defense, with females having no defense at all.

Our findings indicate that the evolution of aposematism through sexual selection on male signal should be associated with sexual dimorphism in both signaling and defense. Here, we assume that male and female traits are encoded by entirely separate genes. We also assume that sexual selection affects only the evolution

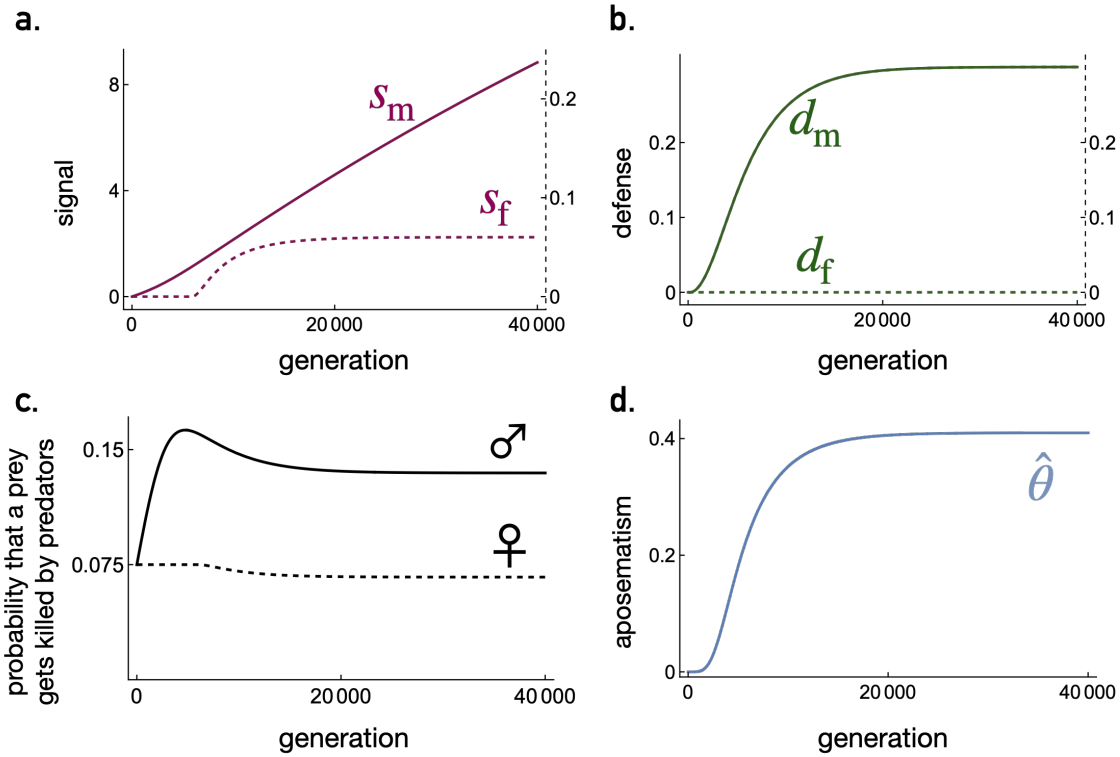

Figure 3: **The evolution of signal and defense in males and females in the model with sex-dependent traits.** Temporal dynamics of **a.** mean male and female signal intensity, **b.** mean male and female defense level, **c.** the probability that a male or female prey gets killed by predators, and **d.** aposematism quantified as the proportion of experienced predators. Parameter values are: $\rho = 1.2$, $\gamma_{\mathrm{cs1}} = 0.04$, $\gamma_{\mathrm{cs2}} = 0.01$, $\gamma_{\mathrm{cd1}} = 0.08$, $\gamma_{\mathrm{cd2}} = 0.1$, $\beta_{\mathrm{D}} = 1.25$, $\beta_{\mathrm{N}} = 17.5$, $\beta_{\mathrm{E}} = 0.9$, $\beta_{\mathrm{L}} = 0.5$, $\lambda = 0.03$, $P_{\mathrm{D0}} = 0.25$ $p = 0.3$.

of male signal. Sexual dimorphism may therefore be less pronounced in natural populations where these assumptions are unmet. Nonetheless, in populations where sexual selection is at the origin of aposematism, our model predicts sexual dimorphism in both signaling and defense, at least to some extent.

3.2 Evolution of aposematism in defended and defenseless populations

We now investigate the evolution of aposematism by sexual selection, without making any restricting assumptions about the ancestral population. We consider the possibility that defense may be favored before sexual selection occurs, so that prey are ancestrally defended. As in the previous analysis, we consider sex-dependent traits, but this time we conduct a sensitivity analysis to assess the impact of the different parameters on aposematism and sexual dimorphism. More specifically, we randomly choose parameter values within a given range (see Appendix A4 for details) and then determine numerically trait values at evolutionary equilibrium. At this equilibrium, we compute the level of aposematism ($\hat{\theta}$) along with measures of

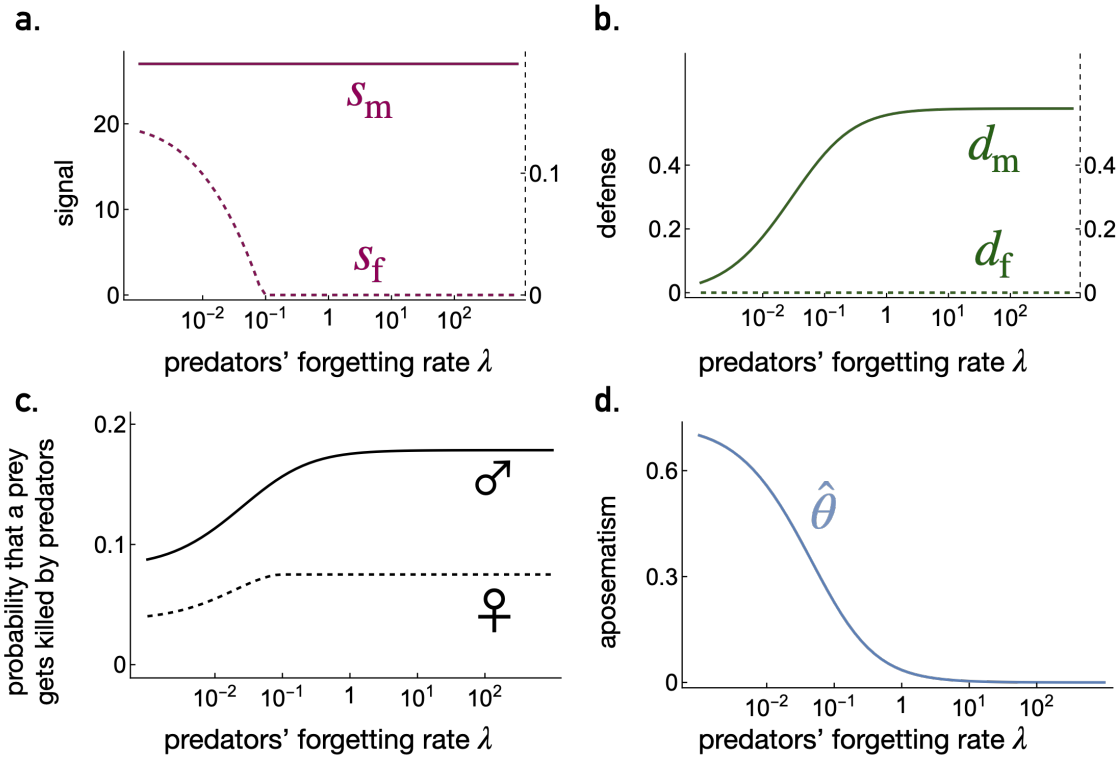

Figure 4: **Impact of predators' forgetting rate on the evolution of signal and defense in males and females in the model with sex-dependent traits a.** Mean male and female signal intensity, **b.** mean male and female defense level, **c.** the probabilities that a male and a female get killed by predators, and **d.** aposematism quantified as the proportion of experienced predators, depending on the predators' forgetting rate λ. Parameter values are: $\rho = 1$, $\gamma_{\mathrm{cs1}} = 0.002$, $\gamma_{\mathrm{cs2}} = 0.1$, $\gamma_{\mathrm{cd1}} = 0.015$, $\gamma_{\mathrm{cd2}} = 0.001$, $\beta_{\mathrm{D}} = 0.1$, $\beta_{\mathrm{N}} = 10$, $\beta_{\mathrm{E}} = 0.05$, $\beta_{\mathrm{L}} = 10$, $P_{\mathrm{D0}} = 0.15$ $p = 1$.

sexual dimorphism in both signal and defense (δ_{s} and δ_{d}; see Appendix A8 for details).

Based on 1,000,000 simulations with different combinations of parameter values, we find that strong sexual selection is associated with high levels of aposematism (see Fig. 5a; Pearson correlation coefficient: $r_{\rho,\hat{\theta}} \approx 0.025$). This finding confirms our previous result, which suggests that sexual selection can promote the evolution of aposematism. Furthermore, we find that the evolution of a high level of aposematism is correlated with a high level of sexual dimorphism in both signal and defense (see Fig. 5b and 5c; $r_{\hat{\theta},\delta_{\mathrm{s}}} \approx 0.088$ and $r_{\hat{\theta},\delta_{\mathrm{d}}} \approx 0.16$). Note that the correlation between the level of aposematism and the level of sexual dimorphism in defense is particularly strong, as expected given our previous model analysis. Altogether, this reinforces our previous findings that aposematism evolving through sexual selection should be associated with sexual dimorphism in both signal and defense.

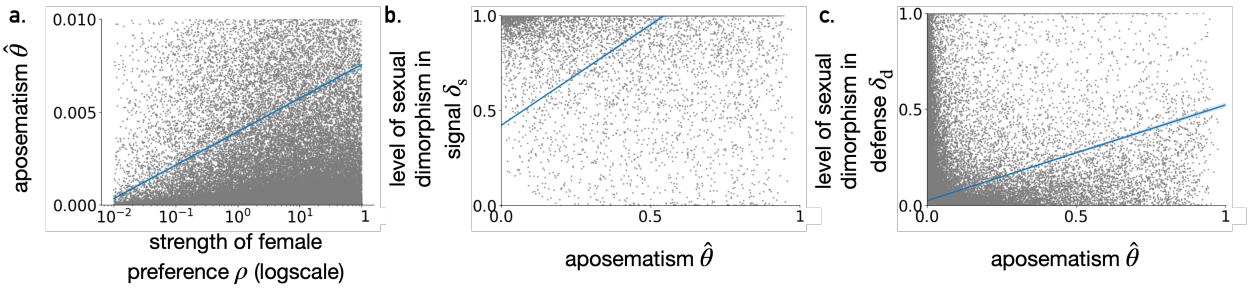

Figure 5: **The impact of female preference on the evolution of aposematism and its role in the evolution of sexual dimorphism.** Each dot represents the outcome of a numerical simulation at evolutionary equilibrium. Simulations were performed using a randomly selected combination of parameter values according to the procedure described in section A4. We represent: **a.** the proportion of experimented predators $\hat{\theta}$ depending on the strength of female preference ρ, **b.** $\hat{\theta}$ depending on the level of sexual dimorphism in signal δ_s and **c.** $\hat{\theta}$ depending on the level of sexual dimorphism in defense δ_d. Blue lines indicate the fit of a linear regression model generated using the *lmplot* function from the *seaborn* package in Python 3.12.

4 Discussion

We showed that sexual selection can drive the evolution of aposematism, even when prey do not initially have a defense mechanism. Sexual selection can favor the evolution of a signal, which increases prey detectability. This, in turn, increases predation pressure, favoring the evolution of a defense mechanism. When prey display a signal and are defended, predators may then learn to associate the signal with defense and avoid attacking these prey, making the prey signal a 'warning' signal. This evolutionary process at the origin of aposematism can be called the 'sexual selection hypothesis' for the origin of aposematism.

In addition to demonstrating that sexual selection can lead to the evolution of aposematism, our study highlights key features that can arise when sexual selection is at play. This could help identify empirical cases of aposematism in nature that support the 'sexual selection hypothesis' for the origin of aposematism. We show that under sexual selection, the evolution of the signal can precede the evolution of defense (but note that sexual selection can also favor the evolution of aposematism in a defended prey population). It may be possible to investigate whether this has occurred in some empirical cases by reconstructing the ancestral state (*e.g.*, Wang and Shaffer, 2008; Forthman and Weirauch, 2018). The evolution of the signal before that of the defense may be peculiar to cases of exaptation, where the aposematic signal has evolved under selective pressures unrelated to aposematism. The involvement of sexual selection and the initial evolution of aposematic signals as sexual signals would constitute a likely evolutionary path to aposematism in these empirical cases. It should be noted that other evolutionary pathways implying exaptation are possible. In particular, a warning signal may also evolve initially as a signal indicating social dominance, thus preventing

costly conflicts. Indeed, some badges of status can be colorful (e.g., Pryke et al., 2002; Murphy et al., 2009), although dominance is usually indicated by the color black (Kenyon and Martin, 2023).

Another key feature that arises under the 'sexual selection hypothesis' for the origin of aposematism is high sexual dimorphism in signaling and defense. We showed that when sexual selection acts mostly in males, males have a stronger signal and defense than females at evolutionary equilibrium. The opposite outcome is expected to occur: when sexual selection acts mostly in females, females may have a stronger signal and defense than males at evolutionary equilibrium. Empirical evidence shows that there is sexual dimorphism in signaling in some aposematic populations (Klein and de Araújo, 2013; Rojas and Endler, 2013; Maan and Cummings, 2009; Palacios-Rodríguez et al., 2022), which could indicate that aposematism was favored by sexual selection in these empirical cases. In contrast, when there is no sexual dimorphism in aposematic signal (e.g., Betancourth-Cundar and Palacios-Rodriguez, 2022), aposematism may have been favored by processes not involving sexual selection. It is important to note that the observation of sexual dimorphism is not a reliable proof supporting the 'sexual selection hypothesis' for the origin of aposematism. Aposematism may first evolve through processes not involving sexual selection (*e.g.*, via kin selection in structured populations) and the aposematic signal may have been later used as a sexual signal, leading to sexual dimorphism. Likewise, an aposematic signal may have evolved initially as a sexual signal, but sexual dimorphism may have been lost later as sexual selection was no longer at play.

In our model with sex-dependent traits, we made several simplifying assumptions that favor sexual dimorphism. We expect sexual dimorphism to be less pronounced when these assumptions are not met. We assume a simple genetic architecture where male and female traits have a totally independent genetic basis, whereas this is not necessarily the case in nature. We also assume that only females express mating preferences, yet many studies report male mate choice in aposematic species (Finkbeiner et al., 2014a; Hausmann et al., 2021; Kuo et al., 2024, e.g.,). Finally, we have not taken into account the fact that selective pressures other than sexual selection can act differently on male and female traits, and can determine the intensity of sexual dimorphism. For instance, females and males may have different escape abilities, impacting their predation rate. If females are more predated than males (as shown in a butterfly species; Ohsaki, 1995), this should increase the advantage of females to express the aposematic signal, reducing the intensity of sexual dimorphism in signaling. Similarly, defense and signaling may not be as costly for males as for females (e.g., Blount et al., 2023), which should impact sexual dimorphism. Finally, other selective forces independent of sex (*e.g.* kin selection) could promote the initial evolution of aposematism at the same time as sexual

selection, thus reducing the intensity of sexual dimorphism.

In our models, we have assumed that all females express the same mating preference for signaling males. Yet, it has been shown that mating preferences can be genetically encoded and subject to evolution (e.g., in *Heliconius* butterflies; Rossi et al., 2024). If mating preferences evolve, the strength of the resulting sexual selection will determine the initial evolution of signaling, as we have shown that it must outweigh the intrinsic costs for signaling to be favored. Once signaling and defense have both evolved and resulted in aposematism, we do not expect sexual selection to vanish because the evolutionary forces affecting mating preferences should be maintained. For instance, if mating preference is favored as a result of signaling indicating a high viability (under the 'good-gene hypothesis'), this should still be the case once aposematism has evolved. Whether this occurs for all types of mating preference that lead to sexual selection should be further investigated using theoretical models.

Overall, our theoretical models add support to the idea that sexual selection can play a role in the initial evolution of aposematic signals. We have shown that sexual selection due to mating preferences can favor the initial evolution of signaling, which in turn increases the predation rate and favors the evolution of defense. This exaptation scenario, characterized by a change in the 'function' of the signaling trait during evolution, constitutes a likely evolutionary pathway for the evolution of some aposematic signals. Some indirect empirical evidence is likely to confirm the 'sexual selection hypothesis' for the origin of aposematism in some aposematic species. Indeed, we have shown that the evolution of signaling before defense, and a high level of sexual dimorphism are expected when sexual selection comes into play in the evolution of aposematism. More generally, our study should stimulate further research on other evolutionary hypotheses for the origin of aposematism involving exaptation.

5 Data Availability

Codes are available online at github.com/Ludovic-Maisonneuve/sexual_selection_origin_of_aposematism

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Appendix to "The 'sexual selection hypothesis' for the origin of aposematism"

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A1 Predator avoidance learning

This section presents how we model the learning dynamics of predators, following Ferreira and Marcon (2014). Initially, predators are naive and attack any prey they encounter. As explained in the main text, predators encounter prey at a rate of p. When a predator encounters a prey, it detects and attacks it with a probability $P_{\mathrm{D}}(s_\bullet)$ depending on the prey's signal intensity $s_\bullet$ (see Eq. (2)). When a naive predator attacks a prey, it notices the signal with probability $P_{\mathrm{N}}(s_\bullet)$ (see Eq. (3))). If it does notice the signal, it may learn to avoid attacking signaling prey with probability $P_{\mathrm{L}}(d_\bullet)$, depending on the prey's defense level $d_\bullet$. In line with empirical evidence, we assume that the avoidance learning probability $P_{\mathrm{L}}(d_\bullet)$ increases with the prey's defense level $d_\bullet$ (*e.g.* Lindström et al. (1997); Skelhorn and Rowe (2006)), so that

$$P_{\mathrm{L}}(d_\bullet) = 1 - \exp\left[-\beta_{\mathrm{L}}\, d_\bullet\right], \tag{A1}$$

where β_{L} modulates the effect of the prey's defense level $d_\bullet$ on avoidance learning. We assume that once predators have learned to avoid attacking signaling prey, they will not attack prey when they notice prey's signal.

We consider a large population of predators and denote θ the proportion of experienced predators that have learned to avoid attacking signaling prey. We assume that predators forget at a rate of λ. The dynamics of θ through time is given by the following differential equation:

$$\dot{\theta} = (1-\theta)\, p \int_{(s_\bullet, d_\bullet)} P_{\mathrm{D}}(s_\bullet) P_{\mathrm{N}}(s_\bullet) P_{\mathrm{L}}(d_\bullet) f(s_\bullet, d_\bullet)\, \mathrm{d}s_\bullet\, \mathrm{d}d_\bullet - \lambda\theta, \tag{A2}$$

where f is the distribution of traits in the prey population.

We assume that the prey population is much larger than the predator population. As a result, the proportion θ of experienced predators reaches its equilibrium value $\hat{\theta}$ before predation significantly alters the distribution of traits in prey. To investigate the evolutionary dynamics in prey, we then consider the proportion of experienced predators at equilibrium (see Eq. 4). This equilibrium value is given by

$$\hat{\theta} = \frac{p \int_{(s_\bullet, d_\bullet)} P_{\mathrm{D}}(s_\bullet) P_{\mathrm{N}}(s_\bullet) P_{\mathrm{L}}(d_\bullet) f(s_\bullet, d_\bullet)\, \mathrm{d}s_\bullet\, \mathrm{d}d_\bullet}{\lambda + p \int_{(s_\bullet, d_\bullet)} P_{\mathrm{D}}(s_\bullet) P_{\mathrm{N}}(s_\bullet) P_{\mathrm{L}}(d_\bullet) f(s_\bullet, d_\bullet)\, \mathrm{d}s_\bullet\, \mathrm{d}d_\bullet}. \tag{A3}$$

A2 Proportion of experienced predators in the invasion analyses

In the case of a sexually monomorphic population where all prey have the same signal intensity s and defense level d so that $f(s_\bullet, d_\bullet) = \delta\left((s_\bullet, d_\bullet) - (s, d)\right)$, where δ is the Dirac function (which is zero everywhere except at (0,0), where it is infinitely high, and the integral of which over the entire phenotypic space is equal to one), the proportion of experienced predators at equilibrium becomes:

$$\hat{\theta} = \frac{p\, P_{\mathrm{D}}(s) P_{\mathrm{N}}(s) P_{\mathrm{L}}(d)}{\lambda + p\, P_{\mathrm{D}}(s) P_{\mathrm{N}}(s) P_{\mathrm{L}}(d)}. \tag{A4}$$

In the case of a sexually dimorphic population where all females (resp. males) have the same signal intensity s_{f} (resp. s_{m}) and defense level d_{f} (resp. d_{m}), the proportion of experienced predators at equilibrium becomes:

$$\hat{\theta} = \frac{p\, \left(P_{\mathrm{D}}(s_{\mathrm{f}}) P_{\mathrm{N}}(s_{\mathrm{f}}) P_{\mathrm{L}}(d_{\mathrm{f}}) + P_{\mathrm{D}}(s_{\mathrm{m}}) P_{\mathrm{N}}(s_{\mathrm{m}}) P_{\mathrm{L}}(d_{\mathrm{m}})\right)}{2\lambda + p\, \left(P_{\mathrm{D}}(s_{\mathrm{f}}) P_{\mathrm{N}}(s_{\mathrm{f}}) P_{\mathrm{L}}(d_{\mathrm{f}}) + P_{\mathrm{D}}(s_{\mathrm{m}}) P_{\mathrm{N}}(s_{\mathrm{m}}) P_{\mathrm{L}}(d_{\mathrm{m}})\right)}. \tag{A5}$$

A3 Selection gradients

A3.1 Model with sex-independent traits

From Eq. (18), we determine the selection gradient on each trait. The selection gradient on the signal is given by

$$\begin{aligned} g_{\mathrm{s}}(\mathbf{x}) &= \frac{\rho}{4} - \gamma_{\mathrm{cs1}} - 2\,s\,\gamma_{\mathrm{cs2}} \\ &\quad - \frac{p}{P^{\circ}(s,d)}\,\left[P'_{\mathrm{D}}(s)(1-P_{\mathrm{E}}(d))P_{\mathrm{A}}(s)\,+P'_{\mathrm{A}}(s)P_{\mathrm{D}}(s)(1-P_{\mathrm{E}}(d))\right], \end{aligned} \tag{A6}$$

where

$$P^{\circ}(s,d) = 1 - p + p\left(1 - P_{\mathrm{D}}(s) + P_{\mathrm{D}}(s)(1 - P_{\mathrm{A}}(s) + P_{\mathrm{A}}(s)P_{\mathrm{E}}(d))\right), \tag{A7}$$

is the probability that a resident prey will not be killed by a predator, $P'_{\mathrm{D}}(s) = \beta_{\mathrm{D}}(1 - P_{\mathrm{D}}(s))$ and $P'_{\mathrm{A}}(s) = -\beta_{\mathrm{N}}(1 - P_{\mathrm{N}}(s))\hat{\theta}$ with the expression of $\hat{\theta}$ given in Eq. (A4).

The selection gradient on the defense is given by

$$g_{\mathrm{d}}(\mathbf{x}) = -\gamma_{\mathrm{cd1}} - 2\,d\,\gamma_{\mathrm{cd2}} + \frac{p}{P^{\circ}(s,d)}\,P_{\mathrm{D}}(s)P_{\mathrm{A}}(s)P'_{\mathrm{E}}(d), \tag{A8}$$

where $P'_{\mathrm{E}}(d) = \beta_{\mathrm{E}}(1 - P_{\mathrm{E}}(d))$.

A3.2 Model with sex-dependent traits

From Eq. (18), we determine the selection gradient on each trait, just like with model with sex-independent traits. The selection gradient on the female signal is given by

$$\begin{aligned} g_{s_{\mathrm{f}}}(\mathbf{x}) &= \frac{1}{2}\Big(- \gamma_{\mathrm{cs1}} - 2\,s_{\mathrm{f}}\,\gamma_{\mathrm{cs2}} \\ &\quad - \frac{p}{P^{\circ}(s_{\mathrm{f}},d_{\mathrm{f}})}\,\left[P'_{\mathrm{D}}(s_{\mathrm{f}})(1-P_{\mathrm{E}}(d_{\mathrm{f}}))P_{\mathrm{A}}(s_{\mathrm{f}})\,+P'_{\mathrm{A}}(s_{\mathrm{f}})P_{\mathrm{D}}(s_{\mathrm{f}})(1-P_{\mathrm{E}}(d_{\mathrm{f}}))\right]\Big), \end{aligned} \tag{A9}$$

where $P'_{\mathrm{A}}(s_{\mathrm{f}}) = -\beta_{\mathrm{N}}(1 - P_{\mathrm{N}}(s_{\mathrm{f}}))\hat{\theta}$ with the expression of $\hat{\theta}$ given in Eq. (A5).

The selection gradient on the female defense is given by

$$g_{d_{\mathrm{f}}}(\mathbf{x}) = \frac{1}{2}\Big(- \gamma_{\mathrm{cd1}} - 2\,d_{\mathrm{f}}\,\gamma_{\mathrm{cd2}} + \frac{p}{P^{\circ}(s_{\mathrm{f}},d_{\mathrm{f}})}\,\left[P_{\mathrm{D}}(s_{\mathrm{f}})P_{\mathrm{A}}(s_{\mathrm{f}})P'_{\mathrm{E}}(d_{\mathrm{f}})\right]\Big). \tag{A10}$$

The selection gradient on the male signal is given by

$$g_{s_\mathrm{m}}(\mathbf{x}) = \frac{\rho}{4} + \frac{1}{2}\Big(- \gamma_{\mathrm{cs1}} - 2\, s_\mathrm{m}\, \gamma_{\mathrm{cs2}} + \frac{p}{P^\circ(s_\mathrm{m}, d_\mathrm{m})}\, [P'_\mathrm{D}(s_\mathrm{m})(1 - P_\mathrm{E}(d_\mathrm{m}))P_\mathrm{A}(s_\mathrm{m}) \, - P'_\mathrm{A}(s_\mathrm{m})P_\mathrm{D}(s_\mathrm{m})(1 - P_\mathrm{E}(d_\mathrm{m}))]\Big). \tag{A11}$$

Finally, the selection gradient on the male defense is given by

$$g_{d_\mathrm{m}}(\mathbf{x}) = \frac{1}{2}\Big(- \gamma_{\mathrm{cd1}} - 2\, d_\mathrm{m}\, \gamma_{\mathrm{cd2}} + \frac{p}{P^\circ(s_\mathrm{m}, d_\mathrm{m})}\, [P_\mathrm{D}(s_\mathrm{m})P_\mathrm{A}(s_\mathrm{m})P'_\mathrm{E}(d_\mathrm{m})]\Big). \tag{A12}$$

A4 Sensitivity analyses

In this section, we describe the sensitivity analyses we conduct to evaluate the robustness of our analytical results. We carry out the sensitivity analyses for the models with sex-independent or sex-dependent traits. We begin by randomly selecting the values of the parameters (see below for details on the range of parameter values). For each combination of parameter values drawn at random, we numerically determine the corresponding evolutionary equilibrium. We assume that the population initially displays no signal and no defense ($s = d = 0$ or $s_\mathrm{f} = d_\mathrm{f} = s_\mathrm{m} = d_\mathrm{m} = 0$, depending on the model). We then use Eq. (17) with $N\mu\sigma^2 = 0.0001$ to model the evolution of mean trait values through generations. During the simulations, we constrain the mean trait values to ensure that they remain within the phenotypic space. We run the simulations until the norm of the changes in mean trait values falls below a threshold of 10^{-6}. This condition ensures that the simulations have reached a state close to equilibrium, where subsequent changes in mean trait values are negligible.

As mentioned above, all parameter values are randomly drawn within a given range. The parameter P_{D0} is randomly drawn from a uniform distribution within the range $[0, 1]$. This ensures that all values within this interval are equally likely to be drawn. The parameter p is chosen using a uniform distribution restricted to the range $[0, 0.5]$. This specific range helps prevent the predator encounter rate from becoming excessively high, which could otherwise result in negative fitness values for some individuals (since in some cases $\mathcal{P}(s_\bullet, d_\bullet)$ given in Eq. (8) can be negative). For the remaining parameters ($\gamma_{\mathrm{cs1}}, \gamma_{\mathrm{cs2}}, \gamma_{\mathrm{cd1}}, \gamma_{\mathrm{cd2}}, \beta_\mathrm{D}, \beta_\mathrm{N}, \beta_\mathrm{L}, \beta_\mathrm{E}, \lambda, \rho$), we generate random values by drawing u from a uniform distribution within $[-3, 3]$ and then computing 10^u. The use of exponential transformations ensures that parameter values span several orders of magnitude,

enabling in-depth exploration of the parameter space.

To analyze the simulated data, we compute the Pearson correlation coefficient between parameters and variables (see Tables A1 and A2). We compute the mean absolute correlation between parameter values in the dataset comprising 1,000,000 simulation outputs. If there was an infinite number of replicates, this correlation should be zero because the parameters are drawn independently of each other. Any correlation between parameters and variables whose absolute value is not one order of magnitude greater than the mean absolute correlation between parameters is considered not to be significant. Indeed, we cannot distinguish whether the correlation is due to the parameters affecting the variables at evolutionary equilibrium or to correlations between parameters in the dataset.

A5 Selection on the signal in the ancestral population

We assume that the ancestral population has reached a state of evolutionary equilibrium, with sexual selection playing no role ($\rho = 0$). Here, we show that in the ancestral population the signal is always counter-selected.

The selection gradient on the signal describes whether a higher signal intensity is favored by natural selection. In the model with sex-independent traits, the selection gradient on the signal is initially equal to

$$g_s(\mathbf{0}) = -\gamma_{\mathrm{cs1}} - \frac{p}{P^{\circ}(0,0)}\, P'_{\mathrm{D}}(0). \tag{A13}$$

This is obtained by substituting $s = d = 0$ and $\rho = 0$ into Eq. (A6). Since $g_s(\mathbf{0})$ is always negative, the signal is always counter-selected.

In the model with sex-dependent traits, the selection gradients on female and male signals are initially equal to

$$g_{s_{\mathrm{f}}}(\mathbf{0}) = g_{s_{\mathrm{m}}}(\mathbf{0}) = -\frac{1}{2}\Big(\gamma_{\mathrm{cs1}} + \frac{p}{P^{\circ}(0,0)}\, P'_{\mathrm{D}}(0)\Big), \tag{A14}$$

This is obtained by substituting $s_{\mathrm{f}} = d_{\mathrm{f}} = s_{\mathrm{m}} = d_{\mathrm{m}} = 0$ and $\rho = 0$ into Eqs. (A9) and (A11). Since $g_{s_{\mathrm{f}}}(\mathbf{0})$ and $g_{s_{\mathrm{m}}}(\mathbf{0})$ are always negative, the male and female signals are always counter-selected.

A6 Initial evolution in the model with sex-independent traits

A6.1 Initial evolution of the signal

Here we derive Eq. (19) of the main text. This gives the condition for the initial evolution of the signal in a population characterized by $s = 0$ and $d = 0$ in the model with sex-independent traits. The signal can initially evolve if

$$g_s(\mathbf{0}) > 0. \tag{A15}$$

By substituting the expression of $g_s(\mathbf{0})$ (obtained by substituting $s = d = 0$ into Eq. (A6)) into the condition (A15), we get

$$\frac{\rho}{4} > \gamma_{\mathrm{cs1}} + \frac{p}{P^{\circ}(0,0)} P'_{\mathrm{D}}(0)(1 - P_{\mathrm{E}}(0))P_{\mathrm{A}}(0), \tag{A16}$$

which is Eq. (19) in the main text.

A6.2 Initial evolution of the defense

Here we derive Eq. (20) of the main text. This gives the condition for the initial evolution of the defense in a population characterized by $s > 0$ and $d = 0$ in the model with sex-independent traits. The defense can initially evolve if

$$g_{\mathrm{d}}(\mathbf{x})|_{d=0} > 0. \tag{A17}$$

By substituting the expression of $g_{\mathrm{d}}(\mathbf{x})|_{d=0}$ (obtained by substituting $d = 0$ into Eq. (A8)) into the condition (A17), we get

$$\frac{1}{P^{\circ}(s,0)} p P_{\mathrm{D}}(s) P'_{\mathrm{E}}(0) > \gamma_{\mathrm{cd1}}, \tag{A18}$$

which is Eq. (19) in the main text.

From Eq. (A18), we derive Eq. (21) of the main text, which gives the threshold value $\tilde{s}$ that signal intensity must exceed for predation pressure to be high enough to favor defense evolution. By substituting the expression of $P_{\mathrm{D}}(s)$ (obtained by substituting $s_{\bullet} = s$ into Eq. (2)) into Eq. (A18) and isolating s, we get the condition

$$s > \frac{1}{\beta_{\mathrm{D}}} \log \left[\frac{p(\beta_{\mathrm{E}} + \gamma_{\mathrm{cd1}})(1 - P_{\mathrm{D0}})}{p(\beta_{\mathrm{E}} + \gamma_{\mathrm{cd1}}) - \gamma_{\mathrm{cd1}}} \right]. \tag{A19}$$

The threshold value is therefore

$$\tilde{s} = \frac{1}{\beta_{\mathrm{D}}} \log \left[\frac{p(\beta_{\mathrm{E}} + \gamma_{\mathrm{cd1}})(1 - P_{\mathrm{D0}})}{p(\beta_{\mathrm{E}} + \gamma_{\mathrm{cd1}}) - \gamma_{\mathrm{cd1}}} \right], \tag{A20}$$

which is Eq. (21) in the main text.

A7 Initial evolution in the model with sex-dependent traits

A7.1 Initial evolution of male signal and defense

Here, we derive the condition for the initial evolution of male signal and defense in the model with sex-dependent traits. In a population characterized by $s_{\mathrm{f}} = 0$, $d_{\mathrm{f}} = 0$, $s_{\mathrm{m}} = 0$ and $d_{\mathrm{m}} = 0$, the male signal can initially evolve if

$$g_{s_{\mathrm{m}}}(\mathbf{0}) > 0. \tag{A21}$$

By substituting the expression of $g_{s_{\mathrm{m}}}(\mathbf{0})$ (obtained by substituting $s_{\mathrm{f}} = d_{\mathrm{f}} = s_{\mathrm{m}} = d_{\mathrm{m}} = 0$ into Eq. (A11)) into the condition (A21), we get

$$\frac{\rho}{2} > \gamma_{\mathrm{cs1}} + \frac{p}{P^{\circ}(0,0)} P'_{\mathrm{D}}(0)(1 - P_{\mathrm{E}}(0))P_{\mathrm{A}}(0). \tag{A22}$$

After the initial evolution of male signal (*i.e.*, in a population characterized by $s_{\mathrm{f}} = 0$, $d_{\mathrm{f}} = 0$, $s_{\mathrm{m}} > 0$ and $d_{\mathrm{m}} = 0$) the male defense can initially evolve if

$$g_{d_{\mathrm{m}}}(\mathbf{x})|_{(d_{\mathrm{f}}, s_{\mathrm{m}}, d_{\mathrm{m}})=(0,0,0)} > 0. \tag{A23}$$

By substituting the expression of $g_{d_{\mathrm{m}}}(\mathbf{x})|_{(d_{\mathrm{f}}, s_{\mathrm{m}}, d_{\mathrm{m}})=(0,0,0)}$ (obtained by substituting $d_{\mathrm{f}} = 0$, $s_{\mathrm{m}} = 0$ and $d_{\mathrm{m}} = 0$ into eq. (A12)) into the condition (A23), we get

$$\frac{1}{P^{\circ}(s,0)} p P_{\mathrm{D}}(s) P'_{\mathrm{E}}(0) > \gamma_{\mathrm{cd1}}. \tag{A24}$$

A7.2 Initial evolution of the female signal

Here, we derive Eq. (19) of the main text, which gives the condition for the initial evolution of female signal in a population characterized by $s_\mathrm{f} = 0$, $d_\mathrm{f} = 0$, $s_\mathrm{m} > 0$ and $d_\mathrm{m} > 0$. The female signal can initially evolve if

$$g_{s_\mathrm{f}}(\mathbf{x})|_{(s_\mathrm{f},d_\mathrm{f})=(0,0)} > 0. \tag{A25}$$

By substituting the expression of $g_{s_\mathrm{f}}(\mathbf{x})|_{(s_\mathrm{f},d_\mathrm{f})=(0,0)}$ (obtained by substituting $s_\mathrm{f} = d_\mathrm{f} = 0$ into Eq. (A9)) into the condition (A25), we get

$$\frac{pP_\mathrm{D0}}{P^\circ(0,0)}\,\hat{\theta}(0,0,s_\mathrm{m},d_\mathrm{m})P'_\mathrm{N}(0) > (\gamma_\mathrm{cs1} + 2\,s\,\gamma_\mathrm{cs2}) + \frac{p\beta_\mathrm{D}(1-P_\mathrm{D0})}{P^\circ(0,0)}, \tag{A26}$$

which is Eq. (24) in the main text.

A7.3 Initial evolution of the female defense

Here, we prove that female defense can never evolve in an ancestrally defenseless population, even when the other traits have evolved. Once male signal, male defense and female signal have evolved (*i.e.*, in a population characterized by $s_\mathrm{f} > 0$, $d_\mathrm{f} = 0$, $s_\mathrm{m} > 0$ and $d_\mathrm{m} > 0$; note that s_f is not necessarily at equilibrium), the female defense cannot evolve if

$$g_{d_\mathrm{f}}(\mathbf{x})|_{d_\mathrm{f}=0} < 0. \tag{A27}$$

To demonstrate that the condition in Eq. (A27) always holds, we first need to establish that $P_\mathrm{A}(s_\mathrm{f})P_\mathrm{D}(s_\mathrm{f}) < P_\mathrm{A}(0)P_\mathrm{D}(0) = P_\mathrm{D0}$ and that $P^\circ(s_\mathrm{f},0) > P^\circ(0,0)$. This is due to the impact of the female signal on the predation pressure on females. We assume that the male traits reached their evolutionary equilibrium before female signal began to evolve. Throughout the evolution of a more intense female signal, natural selection consistently favors an increase in female signal intensity. In other words, we have

$$\forall s'_\mathrm{f} \in [0, s_\mathrm{f}), \quad g_{s_\mathrm{f}}(\mathbf{x})|_{(s_\mathrm{f},d_\mathrm{f})=(s'_\mathrm{f},0)} > 0, \tag{A28}$$

where s'_f takes all the past values of the variable s_f.

By substituting the expression of the selection gradient on female signal (obtained by substituting $s_\mathrm{f} = s'_\mathrm{f}$

and $d_{\mathrm{f}} = 0$ into Eq (A9)), this is equivalent to the condition:

$$\begin{aligned} &\forall s'_{\mathrm{f}} \in [0, s_{\mathrm{f}}), \\ &\quad -\gamma_{\mathrm{cs1}} - 2\, s'_{\mathrm{f}}\, \gamma_{\mathrm{cs2}} - \frac{p}{P^{\circ}(s'_{\mathrm{f}}, 0)}\, [P'_{\mathrm{D}}(s'_{\mathrm{f}}) P_{\mathrm{A}}(s'_{\mathrm{f}}) \, + P'_{\mathrm{A}}(s'_{\mathrm{f}}) P_{\mathrm{D}}(s'_{\mathrm{f}})] > 0. \end{aligned} \tag{A29}$$

This entails that throughout the evolution of a more intense female signal, we have

$$\forall s'_{\mathrm{f}} \in [0, s_{\mathrm{f}}), \quad \overbrace{P'_{\mathrm{D}}(s'_{\mathrm{f}}) P_{\mathrm{A}}(s'_{\mathrm{f}}) \, + P'_{\mathrm{A}}(s'_{\mathrm{f}}) P_{\mathrm{D}}(s'_{\mathrm{f}})}^{=[P_{\mathrm{D}}(s'_{\mathrm{f}}) P_{\mathrm{A}}(s'_{\mathrm{f}})]'} < 0. \tag{A30}$$

This means that the product $P_{\mathrm{D}}(s'_{\mathrm{f}}) P_{\mathrm{A}}(s'_{\mathrm{f}})$ decreases as the female signal evolves, which implies that $P_{\mathrm{A}}(s_{\mathrm{f}}) P_{\mathrm{D}}(s_{\mathrm{f}}) < P_{\mathrm{A}}(0) P_{\mathrm{D}}(0)$, with $P_{\mathrm{A}}(0) P_{\mathrm{D}}(0) = P_{\mathrm{D0}}$.

Moreover, we have

$$\begin{aligned} P^{\circ}(s_{\mathrm{f}}, 0) &= 1 - p + p\,(1 - P_{\mathrm{D}}(s_{\mathrm{f}}) + P_{\mathrm{D}}(s_{\mathrm{f}})(1 - P_{\mathrm{A}}(s_{\mathrm{f}}))\,), \\ &= 1 - p + p\,(1 - P_{\mathrm{A}}(s_{\mathrm{f}}) P_{\mathrm{D}}(s_{\mathrm{f}}))\,. \end{aligned} \tag{A31}$$

Because $P_{\mathrm{A}}(s_{\mathrm{f}}) P_{\mathrm{D}}(s_{\mathrm{f}}) < P_{\mathrm{D0}}$, we have

$$P^{\circ}(s_{\mathrm{f}}, 0) > 1 - p + p\,(1 - P_{\mathrm{D0}}) = P^{\circ}(0, 0). \tag{A32}$$

Using $P_{\mathrm{A}}(s_{\mathrm{f}}) P_{\mathrm{D}}(s_{\mathrm{f}}) < P_{\mathrm{D0}}$, $P^{\circ}(s_{\mathrm{f}}, 0) > P^{\circ}(0, 0)$ and the expression for $g_{d_{\mathrm{f}}}(\mathbf{x})$ from Eq. (A10), we can show that

$$g_{d_{\mathrm{f}}}(\mathbf{x})|_{d_{\mathrm{f}}=0} < \frac{1}{2}\Big(-\gamma_{\mathrm{cd1}} + \frac{p}{P^{\circ}(0, 0)} P_{\mathrm{D0}} P'_{\mathrm{E}}(0)\Big), \tag{A33}$$

and therefore

$$g_{d_{\mathrm{f}}}(\mathbf{x})|_{d_{\mathrm{f}}=0} < \, g_{d_{\mathrm{f}}}(\mathbf{x})|_{(s_{\mathrm{f}}, d_{\mathrm{f}}, s_{\mathrm{m}}, s_{\mathrm{f}})=\mathbf{0}}\,. \tag{A34}$$

If the population is ancestrally defenseless, we have $g_{d_{\mathrm{f}}}(\mathbf{x})|_{(s_{\mathrm{f}}, d_{\mathrm{f}}, s_{\mathrm{m}}, s_{\mathrm{f}})=\mathbf{0}} < 0$, which implies that

$$g_{d_{\mathrm{f}}}(\mathbf{x})|_{d_{\mathrm{f}}=0} < 0. \tag{A35}$$

This demonstrates that female defense can never evolve in an ancestrally defenseless population, even when

the other traits have evolved.

A8 Expressions of the levels of sexual dimorphism

In the model with sex-dependent traits, we express the level of sexual dimorphism in signal as

$$\delta_{\mathrm{s}} = \frac{|s_{\mathrm{f}} - s_{\mathrm{m}}|}{\max(s_{\mathrm{f}}, s_{\mathrm{m}})}, \tag{A36}$$

and the level of sexual dimorphism in defense as

$$\delta_{\mathrm{d}} = \frac{|d_{\mathrm{f}} - d_{\mathrm{m}}|}{\max(d_{\mathrm{f}}, d_{\mathrm{m}})}. \tag{A37}$$

References

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	s	d	$\hat{\theta}$	$1 - P^{\circ}(s, d)$
γ_{cs1}	0.00020	-0.00490*	-0.04309*	-0.01929*
γ_{cs2}	-0.00425*	-0.00202*	-0.01961*	-0.01097*
γ_{cd1}	-0.00052	-0.14267*	-0.04854*	0.11339*
γ_{cd2}	-0.00078	-0.12387*	-0.02486*	0.05118*
β_{D}	-0.00188*	0.00851*	-0.00042	0.02644*
β_{N}	0.00115*	-0.00378*	0.06000*	-0.00288*
β_{L}	-0.00041	-0.00357*	0.06775*	-0.00076
β_{E}	0.00011	0.01169*	0.05248*	-0.24516*
λ	-0.00061	0.00495*	-0.04954*	-0.00015
ρ	0.00034	0.00571*	0.03474*	0.02506*
P_{D0}	0.00081	0.12458*	0.04203*	0.51258*
p	0.00032	0.13848*	0.0533*0	0.55004*

Table A1: Results of the sensitivity analyses performed using the model with sex-independent traits. Pearson correlation coefficient between the different parameters and variables using the dataset produced as described in the section A4 with 1,000,000 combinations of randomly selected parameter values. The mean absolute correlation between parameters is equal to 0.00078. The symbol * indicates the correlation coefficients whose absolute values are an order of magnitude greater than this mean absolute correlation between parameters, and which we therefore consider significant. This implies that we assume these parameters are likely to affect these variables at evolutionary equilibrium. For the other correlations (without the symbol *), we cannot distinguish whether the correlation is due to the parameters affecting the variables at evolutionary equilibrium, or due to the correlations between the parameters in the dataset.

	s_{f}	d_{f}	s_{m}	d_{m}	δ_{s}	δ_{d}	$\hat{\theta}$	$1-P^{\circ}(s_{\mathrm{m}},d_{\mathrm{m}})$	$1-P^{\circ}(s_{\mathrm{f}},d_{\mathrm{f}})$
γ_{cs1}	-0.01760*	0.00114*	-0.07623*	-0.00629*	-0.32320*	-0.07926*	-0.04257*	-0.03026*	-0.00072
γ_{cs2}	-0.01547*	0.00061	-0.07545*	-0.00321*	-0.00095	-0.03445*	-0.01943*	-0.01432*	0.00028
γ_{cd1}	-0.01632*	-0.13941*	-0.00219*	-0.14216*	-0.00569*	-0.09417*	-0.04824*	0.11482*	0.10912*
γ_{cd2}	-0.00473*	-0.12122*	-0.00305*	-0.12353*	-0.00452*	0.00797*	-0.02474*	0.05232*	0.05006*
β_{D}	-0.01281*	0.00089	-0.03415*	0.01188*	-0.12922*	0.07497*	0.00101*	0.04155*	0.00100*
β_{N}	0.01812*	-0.00478*	-0.00135*	-0.00653*	-0.00122*	0.00458*	0.06413*	-0.00020	0.00030
β_{L}	0.02728*	-0.00010	-0.00016	-0.00148*	-0.00270*	0.00552*	0.07602*	-0.00107*	-0.00010
λ	0.00121*	0.01307*	0.00613*	0.01088*	0.01942*	0.07939*	0.05012*	-0.24985*	-0.23973*
β_{E}	-0.01741*	-0.00001	-0.00079	0.00149*	0.00079	-0.00078	-0.04975*	0.00122*	0.00049
ρ	0.00300*	0.00016	-0.02754*	0.00713*	0.40570*	0.08593*	0.02484*	0.02835*	-0.00140*
P_{D0}	0.03142*	0.13959*	0.01513*	0.12129*	0.04096*	-0.09651*	0.04267*	0.48720*	0.54761*
p	0.02745*	0.13971*	-0.01362*	0.14026*	-0.04426*	0.02708*	0.05541*	0.55323*	0.54767*

Table A2: Results of the sensitivity analyses performed using the model with sex-dependent traits. Pearson correlation coefficient between the different parameters and variables using the dataset produced as described in the section A4 with 1,000,000 combinations of randomly selected parameter values. The mean absolute correlation between parameters is equal to 0.00072. The symbol * indicates the correlation coefficients whose absolute values are an order of magnitude greater than this mean absolute correlation between parameters, and which we therefore consider significant. This implies that we assume these parameters are likely to affect these variables at evolutionary equilibrium. For the other correlations (without the symbol *), we cannot distinguish whether the correlation is due to the parameters affecting the variables at evolutionary equilibrium, or due to the correlations between the parameters in the dataset.