Partitioning of resources: the evolutionary genetics of sexual conflict over resource acquisition and allocation

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Abstract
Fitness depends on both the resources that individuals acquire and the allocation of those resources to traits that influence survival and reproduction. Optimal resource allocation differs between females and males as a consequence of their fundamentally different reproductive strategies. However, because most traits have a common genetic basis between the sexes, conflicting selection between the sexes over resource allocation can constrain the evolution of optimal allocation within each sex, and generate trade-offs for fitness between them (i.e. ‘sexual antagonism’ or ‘intralocus sexual conflict’). The theory of resource acquisition and allocation provides an influential framework for linking genetic variation in acquisition and allocation to empirical evidence of trade-offs between distinct life-history traits. However, these models have not considered the emergence of trade-offs within the context of sexual dimorphism, where they are expected to be particularly common. Here, we extend acquisition–allocation theory and develop a quantitative genetic framework for predicting genetically based trade-offs between life-history traits within sexes and between female and male fitness. Our models demonstrate that empirically measurable evidence of sexually antagonistic fitness variation should depend upon three interacting factors that may vary between populations: (1) the genetic variances and between-sex covariances for resource acquisition and allocation traits, (2) condition-dependent expression of resource allocation traits and (3) sex differences in selection on the allocation of resource to different fitness components.

Introduction

In order to spend on one side, nature is forced to economize on the other side
(Darwin, 1859, quoting Goethe)

Evolutionary life-history theory predicts that the limited resources that are available to each individual (e.g. energy, nutrients) will naturally give rise to trade-offs in performance between competing bodily functions. Well-adapted organisms must allocate a finite resource pool to different traits in a manner that optimizes total fitness (Stearns, 1992). The different strategies of resource allocation broadly define each species’ life cycle, for example, differentiating species along a life-history gradient from low-reproduction/high-survival species to high-reproduction/low-survival species. Moreover, intraspecific variation in resource acquisition and allocation generate trade-offs between traits that compete for the same resources within individuals. Abundant variation in both resource acquisition ability and resource allocation ‘decisions’ (e.g. Dudycha & Lynch, 2005; Bleu et al., 2013) can directly influence empirical evidence of trade-offs, including negative correlations between the expression of different fitness components (van Noordwijk & de Jong, 1986; Metcalf, 2016).

The most fundamental life-history trade-offs involve major life-history traits that are closely linked to fitness, such as development, growth, reproduction and survival (somatic maintenance). Such trade-offs may occur
among competing traits that are expressed within the same life stage (e.g. fecundity vs. immune response, Lazzaro et al., 2008), among different life stages (e.g. early life reproduction vs. longevity, Harshman & Zera, 2007), or between different environments (e.g. differential fitness of mites on different hosts, Agrawal, 2000). Although there is no question that fundamental trade-offs between competing traits exist, the problem is rather how to identify them. The genetic architecture underlying competing traits is central to the dynamics of adaptive phenotypic evolution (Lynch & Walsh, 1998; Roff & Fairbairn, 2007), yet the relative quantities of additive genetic variation for acquisition and allocation can hinder the detection of trade-offs (Houle, 1991). On the phenotypic level, defining and empirically measuring the relevant limiting resource (common currency) is, in most cases, not trivial.

The scope for genetically based trade-offs in fitness and performance are expected to further expand in species with distinct sexes. Due to the fundamentally different reproductive strategies that females and males employ in achieving high overall fitness (a consequence of anisogamy; Parker, 2006), selection naturally favours the evolution of different phenotypes and life-history strategies for each sex (Arnqvist & Rowe, 2005). However, the genetic basis of most traits is shared between the sexes (Poissant et al., 2010), and as such, the sexes are fundamentally constrained in evolving towards their distinct phenotypic optima. The existence of positive genetic covariances between male and female allocation levels to homologous traits, together with sex-specific selection on allocation, constitutes a form of sexual antagonism, or intralocus sexual conflict (IASC), in which alleles that improve fitness of one sex reduce it in the other (Bonduriansky & Chenoweth, 2009). The evolution of sexual dimorphism can eventually resolve IASC, yet strong genetic correlations between the sexes can make this process exceedingly slow, leading to persistent IASC over long stretches of evolutionary time (Lande, 1980: Bedhomme & Chippindale, 2007; Pennell & Morrow, 2013).

Empirical evidence for IASC has accumulated steadily during the past decade, although it is perhaps less prevalent than initially predicted by theory (Lande, 1980; Conallon & Hall, 2016). IASC is expected to generate a negative genetic correlation for fitness between females and males of a population (Bonduriansky & Chenoweth, 2009) – a prediction that has now been tested within several populations and environmental contexts (e.g. Chippindale et al., 2001; Pischetta & Chippindale, 2006; Brommer et al., 2007; Foerster et al., 2007; Zajitschek et al., 2007; Poissant et al., 2008; Berger et al., 2014; Punzalan et al., 2014). Evidence for IASC from these studies is mixed, with some revealing the characteristic negative genetic correlation between female and male fitness (as predicted under IASC), and others showing a positive fitness correlation between the sexes. Direct estimates of sex-specific selection gradients on homologous male and female traits tell a similar story. IASC is implied when the direction of phenotypic selection differs between the sexes, as most quantitative traits exhibit a strong and positive between-sex additive genetic correlation (Poissant et al., 2010; Griffin et al., 2013). Cox & Calsbeek (2009) compiled a large dataset of sex-specific selection gradients and identified sexual antagonistic selection (i.e. opposing sex-specific selection gradients with magnitudes larger than 0.1, including statistically significant and nonsignificant gradients) in only 17% of cases where data were available. This suggests that IASC, while present in several traits and animal populations, might generally be moderate to rare in wild animal populations.

The variable evidence for IASC among populations and traits raises interesting, yet currently unresolved questions. In particular, what are the potential biological factors that might influence the strength of empirical signals of IASC? What are the genetic, ecological and evolutionary circumstances that are likely to yield the strongest signals of IASC, by way of selection gradient estimates, or through measures of the additive genetic correlation for fitness between the sexes? One possible factor that has received recent attention is the role of environmental change in resolving IASC. Although the process of adaptation may often generate sexual antagonism (Conallon & Clark, 2014), continuous change in environmental conditions can leave both sexes perpetually maladapted, and thereby maintain the alignment of directional selection between the sexes. This possibility has recently received support from theory (Conallon, 2015; Conallon & Hall, 2016) and experiments (e.g. Long et al., 2012; Berger et al., 2014).

We explore another (nonmutually exclusive) possibility: that trade-offs between the sexes are at least partially masked by developmental processes that convert genetic variation in resource acquisition and allocation to traits that are subject to sex-specific selection. Our model is inspired by acquisition–allocation models for life-history evolution (beginning with the seminal model of van Noordwijk & de Jong, 1986), which provide much insight into the trade-offs that we see (and those that we do not) among the traits and life-history stages that are expressed by all individuals of a population (see Metcalf, 2016). This theory has, however, largely neglected the role of sex differences and the potential for selection and intersexual genetic correlations to impact trade-offs that emerge within and between individuals of each sex (Cox, 2015). For example, current theory ignores the fact that fitness landscapes differ between sexes, that the genetic architecture of acquisition and allocation rates might be sexually dimorphic and that acquisition and allocation traits likely covary within individuals (see Descamps et al.,
2016), and in a sexually dimorphic manner (see Rowe 
& Houle, 1996). The latter is implied by widespread 
condition-dependent expression of traits that are 
involved in reproduction, particularly traits that are 
elaborately expressed in males.

Here, we develop a quantitative genetic framework 
of resource acquisition and allocation that takes the 
correlated genetic architecture of males and females 
into account, and includes the joint effects of sexually 
dimorphic rates of acquisition and allocation and the 
divergent optima of female and male phenotypes. Our 
models thus allow for both resource-based and genetic 
constraints within individuals, between individuals and, 
explicitly, between individuals of different sexes. The 
models allow us to identify specific quantitative genetic 
scenarios of within- and between-sex correlations in 
an acquisition and allocation that amplify or dampen 
empirical signals of fitness trade-offs between the sexes. 
We focus throughout on evaluating the effects of 
sex-specific genetic (co)variance in acquisition and allo-
cation on the sign and magnitude of the genetic covari-
ance between male and female fitness. In addition, we 
ask whether condition dependence should facilitate or 
impe the detection of intersexual genetic trade-offs. 
Lastly, we ask how the sex-specific genetic basis of allo-
cation and acquisition will influence patterns of genetic 
covariance – within and between the sexes – between 
individual life-history traits.

Materials and methods

Theoretical background of the ‘Y-model’ framework

The Y-model of resource acquisition and allocation is a 
remarkably simple, yet powerful and influential, frame-
work for linking the partitioning of resources, and the 
relative variation in resource acquisition and allocation, 
to the covariances between traits that compete for 
acquired resources (van Noordwijk & de Jong, 1986). 
In its basic form, the Y-model rests upon two assump-
tions about the relationship between resource acquisi-
tion, resource allocation and fitness. First, it assumes 
that individuals vary in their ability to acquire and con-
vert external resources into an internal pool of ener-
gic resources and that fitness increases with resource 
acquisition ability (Houle, 1991). Thus, alleles that 
increase resource acquisition (e.g. through increased 
nutrient components, increased total energetic value of 
dietary intake and/or high rates of conversion of raw 
resources into useful metabolites) are always beneficial 
in the absence of any pleiotropic side effects on other 
traits (see Wilson, 2014).

Second, the models assume that acquired resources are 
competitively allocated to two or more traits and that 
individuals vary in the resource allocation ‘decisions’ that 
they use during development. In contrast to 
aquisition, increased allocation to one trait (which 
improves expression, function or performance of the 
trait) necessarily depletes the amount of resources 
available to other traits (thereby reducing expression, 
function or performance of the other traits). This natu-
rally generates trade-offs between traits that make use 
of the same set of resources. Optimal allocation with 
respect to fitness must therefore strike a balance 
between the benefits of allocating to each trait or fit-
ness component, as this comes at an expense to others. 
Trade-offs between competing allocation traits are 
empirically detectable through negative covariances (or 
correlations) between the traits. The key insight from 
the Y-model is that covariances between traits can be 
positive even when the traits trade-off against one 
other (van Noordwijk & de Jong, 1986). Positive covari-
ances are particularly likely when population variation 
in resource acquisition is large relative to the magni-
tude of variability in resource allocation.

The Y-model has been exceptionally successful at 
answering some of the most vexing questions in life-his-
tory evolution (Zera & Harshman, 2001; Metcalf, 2016). 
When formalized in a population genetic framework, 
this model can also account for trade-offs between traits 
by way of pleiotropic gene actions (Houle, 1991). Trade-
offs between traits may be masked if a much larger 
fraction of loci within a genome influences acquisition 
relative to allocation, as is often assumed to be the case 
(Houle, 1991). Such masking may arise when variability 
in acquisition is maintained under mutation–selection 
balance alone (Houle, 1991). Positive correlations 
between traits are perhaps even more likely in contexts 
involving large arrays of fitness components (i.e. beyond 
a single pair of traits) (Charlesworth, 1990).

More complex models of resource acquisition and 
allocation that incorporate multiple traits with compet-
ing demands, ecological context-dependent priority 
rules for their allocation, hierarchical allocation trees 
and genotype-by-environment interactions have all 
been developed (de Laguerie et al., 1991; Boggs, 1992; 
de Jong, 1993; Worley et al., 2003; Robinson & Beck-
eman, 2013). For example, if acquired resources are allo-
cated hierarchically to multiple traits, high variation in 
allocation towards the top of the hierarchy (e.g. 
between branches affecting reproduction vs. survival) 
can result in positive covariances between traits further 
down (e.g. between components of reproduction; de 
Jong, 1993; Worley et al., 2003).

Intersexual genetic covariance for fitness in the 
absence of condition dependence

Despite extensive development of Y-models in the con-
text of life-history trade-offs, we currently lack a model 
that takes into account sex differences in: (1) selection, 
(2) genetic variability of acquisition and allocation and 
(3) the relative expression of traits that trade-off under 
competitive allocation. To address this gap, we first
extend the basic quantitative genetic framework of van Noordwijk & de Jong (1986) to a population comprised of separate sexes. We assume that there are two major traits that are expressed by both sexes and that influence fitness in a sex-specific manner. We assume that acquisition and allocation underlying these two traits vary randomly among individuals of the population, and potentially in a sex-specific manner (i.e. there may be sexual dimorphism in the means and variances of acquisition and/or allocation, with imperfect genetic correlations between the sexes for each). For simplicity, we initially assume (following van Noordwijk & de Jong, 1986) that acquisition and allocation are independently distributed among individuals. In the context of sex differences, this assumption equates to the absence of condition-dependent expression (condition sensu Rowe & Houle, 1996). We subsequently relax this assumption, further below, by allowing for condition-dependent expression as a function of individual acquisition.

Following Houle (1991), we model sex-specific fitness as a function of background genetic quality ($R_j$ in the $j$th sex), which is directly related to resource acquisition, and allocation to each of the two focal traits (where $P_j$ represents the allocation rate to one of the traits, and $1 - P_j$ represents allocation to the other). Fitness in the $j$th sex is:

$$W_j = R_j^{k_j} \exp \left( - \frac{(P_j - \theta_j)^2}{2\omega_j^2} \right)$$

where $k_j$ is a shape parameter that determines the scaling between acquisition and fitness, $\theta_j$ is the optimal allocation rate, and $\omega_j$ is the intensity of stabilizing selection on allocation (for a visual illustration of the effects of $k$ and $\omega$ on $W$, see Fig. 1a,b). For analytical convenience, we first convert fitness to a logarithmic scale, and then express the resulting function in terms of the marginal means and variances of the distribution of sex-specific acquisition and allocation (i.e. by way of a Taylor series expansion around the means of the marginal distributions). This leads to the following approximation of $\ln[W]$ that is now linear with respect to $R_j$ and $P_j$:

![Fig. 1](image-url) Effects of the relationship between acquisition ($R$) and fitness ($W$), determined by $k$ (a), and of the strength of stabilizing selection on allocation ($\omega$) (b) on acquisition-dependent fitness, and conceptual overview of a two-sex resource acquisition and allocation model (c). In (a) and (b), other parameters are set to $P = 0.1$, $\theta = 0.9$, and either to (a) $\omega = 1$, or to (b) $k = 1$. In (c), unidirectional arrows indicate causal effects and bidirectional arrows indicate genetic covariances between components. $E\emptyset$ stands for a standardized environment, experienced by males and females; we assume throughout that environmental variance in $R$ and $P$ is controlled.
is defined as:

\[ \ln(W_j) \approx k_j \ln(R_j) - \frac{(P_j - \theta_j)^2}{2\sigma^2} + \frac{(R_j - \overline{R}_j)^2}{\overline{R}_j} + \frac{(P_j - \overline{P}_j)(\theta_j - \overline{P}_j)}{\sigma^2}, \]

where \( \overline{R}_j \) and \( \overline{P}_j \) represent averages for the jth sex. The approximation works best in the limit where the variances of the variables are small and symmetrical (e.g. normally distributed). In practice, eqn (1) holds up well under a much broader range of conditions (see below).

**Intersexual genetic covariance for fitness under condition-dependent expression**

The simplest version of our model assumes that there is no condition dependence of allocation strategies. In this case, covariances between acquisition and allocation are all zero (i.e.: \( \text{cov}(R_j, P_j) = \text{cov}(R_m, P_m) = \text{cov}(R_m, P_l) = 0 \)). We subsequently relax the assumption of independence of allocation and acquisition, and allow for the more realistic scenario of condition-dependent allocation, where the ability to acquire resources and convert them into usable units (i.e. acquisition) covaries with allocation (Robinson & Beckerman, 2013; Descamps et al., 2016). Condition dependence is pervasive (Hill, 2011), particularly among sexually selected traits (Tomkins et al., 2004; Bonduriansky, 2007).

Condition-dependent allocation – if adaptive – implies that the optimal allocation strategy of a given sex is also a function of acquisition. Thus, to introduce condition dependence into our models, we must define the sex-specific allocation strategies and the sex-specific allocation optima as functions of acquisition within that sex. To define the relationship between acquisition and allocation, let sex-specific acquisition follow a normal distribution: \( R_j \sim N(\overline{R}_j, \text{var}(R_j) = G_{Rj}) \), where \( G_{Rj} \) represents the additive genetic variance in acquisition within the jth sex. We approximate sex-specific allocation as a linear function of acquisition: \( P_j = y_j + \eta_j(R_j - \overline{R}_j) \), where \( \eta_j \) represents the strength of condition dependence (\( \eta_j > 0 \) implies that the relative allocation of resources to trait 1 increases with condition; larger values of \( |\eta_j| \) represent stronger condition dependence), and \( y_j \) is a normally distributed random variable that is influenced by the residual genetic variability in allocation that is not explained by genetic variability in acquisition (thus, \( y_j \sim N(\overline{P}_j, \text{var}(y_j) = G_{yj}) \), where \( G_{yj} \) is the additive genetic variance for residual allocation; \( \text{cov}(R_j, y_j) = 0 \)). As \( P_j \) is still bounded between 0 and 1, each individual’s allocation strategy is defined as:

\[
P_j = \begin{cases} 
  \eta_j(R_j - \overline{R}_j) & \text{for } y_j + \eta_j(R_j - \overline{R}_j) \in (0, 1) \\
  1 & \text{for } y_j + \eta_j(R_j - \overline{R}_j) > 1 \\
  0 & \text{for } y_j + \eta_j(R_j - \overline{R}_j) < 0 
\end{cases}
\]

When the distribution of \( P_j \) is entirely within the range, \( 0 < y_j + \eta_j(R_j - \overline{R}_j) < 1 \), as when are \( \eta_j \) and \( \eta_m \) small, then the marginal distribution of allocation in sex \( j \) can be approximated as normal with \( P_j \sim N(\overline{P}_j, \text{var}(P_j) = G_{yj}^2 + \eta_j^2G_{Rj}) \). We use this approximation in deriv-}

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subsequently explore the influence of condition dependence on intersexual genetic correlations for fitness. Finally, we explore how sexual dimorphism in the genetic basis of acquisition and allocation traits should influence patterns of genetic covariance within and between the sexes for homologous and nonhomologous traits, including life-history components.

**Intersexual genetic covariance for fitness in the absence of condition dependence**

Using eqn (1), we can calculate the genetic covariance between sexes for log fitness in the absence of condition-dependent effects on individual and optimal allocation. In this case, acquisition and allocation are independent (cov(R_m, P_m) = cov(R_f, P_f) = cov(R_m, P_f) = cov(R_f, P_m) = 0) and optimal allocation is fixed across individuals (θ_m and θ_f are constants). The fitness covariance between the sexes reduces to:

\[
cov[\ln(W_f), \ln(W_m)] \approx \frac{k_f k_m \text{cov}(R_f, R_m)}{R_f R_m} + \frac{(\theta_f - \mu_f)(\theta_m - \mu_m)\text{cov}(P_f, P_m)}{(\sigma_f^2 \sigma_m^2)^{\frac{1}{2}}},
\]

which compares well against exact computer simulations (see Appendix S1). A conceptual overview of the functional architecture of parameters within eqn (4) is shown in Fig. 1c.

We can now ask what biological conditions facilitate detection of a negative fitness covariance between sexes—an unequivocal signal of IASC. We initially assume that the covariance between male and female acquisition, cov(R_f, R_m), is positive (we relax the assumption further below). A positive acquisition covariance is plausible when genetic variation that improves foraging and metabolism in females also improves these traits in males—a scenario that is likely when the sexes feed on and process the same basic food resources. With cov(R_f, R_m) > 0, the first term of eqn (4) is always positive, and thus, always elevates the covariance of fitness between the sexes. The magnitude of the first term is influenced by its two subcomponents. First, it increases with k_f k_m, which represents the sensitivity of male and female fitness to acquisition; increased sensitivity to acquisition (i.e., larger values of k_i) leads to higher fitness covariance between the sexes. Second, the first term of eqn (4) also increases with cov(R_f, R_m)/(R_f R_m), a standardized measure of covariance in acquisition between the sexes (note the qualitative similarity between this covariance metric and the square of the coefficient of variation from probability theory).

The second term of eqn (4) can be positive or negative, depending on the displacement of each sex away from its optimum (i.e., mean allocation, P_f*, relative to optimal allocation, θ_f), and the covariance in allocation between the sexes, cov(P_f, P_m). There are three scenarios of biological interest:

1. **At least one of the sexes is at its optimum.** When mean allocation rates in at least one sex correspond to its optimum (mean P_f = θ_f and/or mean and P_m = θ_m), then the second term of eqn (4) is zero. Therefore, provided cov(R_f, R_m) > 0, the overall fitness covariance between sexes must be positive (i.e., no signal of sexual conflict; Fig. 2a).

2. **Sexes differ in their allocation strategy.** If mean allocation rates in at least one sex differ from its optimum (i.e., P_f = θ_f and/or mean and P_m = θ_m), then the second term of eqn (4) can be positive or negative, depending on the magnitude of intersexual fitness covariances. Heat maps with contour lines of the magnitude of intersexual fitness covariances, cov[ln(W_f), ln(W_m)], with blue (and minus signs) representing negative and red positive values. Optimal allocation rates are identical for males and for females with θ_m = θ_f = 0.5. Same mean allocation rates in the sexes P_f* = P_m* = 0.5 (a), sexual dimorphic mean allocation rates with P_f* = 0.1 and P_m* = 0.9 (b), located on different sides of the optimal allocation rate (var(R) = var(P) = 0.1 for a and b), and, similar to b, except higher variance of allocation compared to acquisition (var(P) = 0.1, var(R) = 0.01, var(P)/var(R) = 10) (c). For all plots, P_m* = P_f* = θ_f = θ_m = k_f = k_m = 1.
Each sex is displaced in the same direction from its allocation optimum. With concordant directional selection on allocation between the sexes (i.e. \((\theta_f - \overline{P}_f)(\theta_m - \overline{P}_m) > 0\)), the total fitness covariance between sexes is always positive provided male and female acquisition and allocation rates are each positively correlated as well (\(\text{cov}(R_f, R_m), \text{cov}(P_f, P_m) > 0\)). If intersex covariance for allocation is negative (\(\text{cov}(P_f, P_m) < 0\)), fitness covariance between the sexes can be negative, provided the covariance between female and male acquisition rates is sufficiently small, or negative (Fig. S1).

Each sex is displaced in opposite directions from its allocation optimum. In this case, selection on allocation is sexually antagonistic (i.e. \((\theta_f - \overline{P}_f)(\theta_m - \overline{P}_m) < 0\)). The second term of eqn (4) becomes positive, provided the intersex covariance for allocation is positive (\(\text{cov}(P_f, P_m) > 0\)). This leads to a reduction of the total fitness covariance between the sexes, or – if the magnitude of the second term is sufficiently large – a negative covariance for fitness (Fig. 2b). The second term of eqn (4) becomes positive, and thereby constrains any positive fitness covariance for allocation (see above). Condition dependence in allocation has no impact on fitness covariance between the sexes. Equation (5) reveals two sequences of condition dependence on intersexual covariances for fitness. Under condition dependence, the intersexual fitness covariance becomes:

\[
\text{cov}(\ln(W_f), \ln(W_m)) \approx \left[ \frac{k_f}{R_f} \left( \overline{P}_f - \overline{P}_f \right) \left( \theta_f - \xi_f \right) \right] \frac{\text{var} \left( \ln(P_f) \right)}{\text{var}(R_f)} \\
+ \left[ \frac{k_m}{R_m} \left( \overline{P}_m - \overline{P}_m \right) \left( \theta_m - \xi_m \right) \right] \text{cov}(R_f, R_m) \\
+ \left( \frac{P_m - \overline{P}_m}{\text{var}(y_m)} \right) \text{cov}(y_f, y_m) \left( \frac{\text{var}(y_f)}{\text{var}(R_f)} \right)^2
\]

(5)

where \(\overline{P}_j\) is the allocation optimum for an individual with average acquisition and \(\text{cov}(y_f, y_m)\) refers to the residual between-sex covariance for allocation (i.e. for the fraction of variation in allocation that is independent of acquisition, see above). Condition dependence parameters, \(\eta_j\) and \(\xi_j\), describe the degree and direction of condition dependence in individual and optimal allocation in the \(j\)th sex, respectively. Sums within each square bracket will be positive as long as \(\eta_j\) and \(\xi_j\) are sufficiently small, as we assume below.

Note the correspondence between the individual terms of eqns (4) and (5); the second term remains unchanged (\(\text{cov}(y_f, y_m)\) in eqn (5) is analogous to \(\text{cov}(P_f, P_m)\) from eqn (4)). Effects of condition dependence emerge within the first term, where they modify the contribution of acquisition to the overall covariance in fitness between the sexes. Equation (5) reveals two general consequences of condition dependence for the between-sex additive genetic covariance for fitness. First, when individual and optimal allocation strategies shift in parallel with one another (\(\eta_j = \xi_j\) in each sex, \(i.e.\) when condition-dependent trait expression perfectly tracks condition-dependent optimal allocation), condition dependence has no impact on fitness covariance (in such cases, eqn (5) effectively reduces to eqn (4)). Second, when trait allocation imperfectly tracks optimal allocation (\(\eta_j \neq \xi_j\)), condition dependence is capable of elevating or decreasing the genetic covariance between the sexes, although the magnitude of such effects will
be small as long as condition-dependent allocation patterns are adaptive such that differences between $\eta_j$ and $c_j$ are small.

Although condition-dependent effects can, in principle, increase or decrease the fitness covariance between the sexes, we are particularly interested in whether or not condition dependence should typically serve to mask or to reveal signals of IASC in the presence of sexually antagonistic selection over allocation. Consider, then, an idealized scenario of symmetrical sexual conflict over allocation, where $(\bar{y}_j - \bar{y}_m)/\sigma^2 = (\bar{T}_m - T_m)/\omega^2_m$. For simplicity, further assume that the sexes exhibit similar patterns of condition dependence ($\eta_j = \eta_m$; $c_j = c_m$), equal mean resource acquisition ($\bar{R}_j = \bar{R}_m$) and equal sensitivities of fitness to acquisition ($k_j = k_m$). In this case, eqn (5) simplifies to:

$$\text{cov} \left( \text{ln} \left( W_j \right), \text{ln} \left( W_m \right) \right) \approx \left( k_j \right)^2 \left( \frac{1}{\bar{R}_j} \right)^2 \text{cov} \left( y_j, y_m \right).$$

(6)

Equation (6) shows that condition-dependent expression will – if anything – amplify signals of sexual conflict by reducing the capacity of variation in acquisition to mask effects of sexual antagonistic selection over allocation.

**Covariances between competing traits, within and between the sexes**

Variation in acquisition and allocation, and their intersexual covariances, ultimately determines the covariance between female and male fitness. On the other hand, it is rarely experimentally tractable to measure acquisition and allocation, and instead most research focuses on measuring variances and covariances of traits that are likely to compete for competitively allocated resources. We therefore examined how intersexual correlations of acquisition and allocation (corr($R_m$, $R_f$) and corr($P_m$, $P_f$)) influenced the within- and between-sex covariances between two competing traits, $T_1$ and $T_2$. For simplicity, we present cases under the assumption of no condition-dependent allocation (i.e. independence between acquisition and allocation). The following results also assume that marginal distributions of acquisition and allocation are the same within each sex. Although there is currently little data to validate or reject this assumption, we note that the genetic variances of most quantitative traits are, on average, similar between the sexes (Wyman & Rowe, 2014).

Under the stated assumptions, the within-sex covariance between traits $T_1$ and $T_2$ (Appendix S1) is:

$$\text{cov}(T_{1j}, T_{2j}) = \bar{T}_j \left[ 1 - \bar{P}_j \right] \text{var}(R) - \left[ \bar{R}_j^2 + \text{var}(R) \right] \text{var}(P),$$

(7)

which is analogous to the classic result of van Noordwijk & de Jong (1986). The between-sex covariance for nonhomologous traits is:

$$\text{cov}(T_{1m}, T_{2f}) = \text{cov}(T_{1f}, T_{2m}) = \bar{T}_1 \left[ 1 - \bar{P}_f \right] \text{var}(R) - \left[ \bar{R}_1^2 + \text{var}(R) \right] \text{var}(P),$$

(8)

where genetic correlations are defined using $\text{corr}(x, y) = \text{cov}(x, y)/[\text{var}(x) \text{var}(y)]^{1/2}$. We contrast eqs. (7) and (8) in the following sections, focusing on how between-sex covariances for acquisition and allocation impact signals of trade-offs between different traits that are expressed within the same sex (eqn 7) or between different sexes (eqn 8). We specifically focus on three idealized scenarios of the between-sex covariances in acquisition and allocation: (1) between-sex genetic correlations for acquisition and allocation are both strong; (2) acquisition is strongly correlated and allocation is weakly correlated; and (3) allocation is strongly correlated and acquisition is weakly correlated.

**Strong intersexual genetic correlations for acquisition and allocation**

First, consider the case where the intersexual correlations in acquisition and allocation are both strong and positive. In the limit of perfect correlations between the sexes (corr($R_m$, $R_f$), corr($P_m$, $P_f$) $\to 1$), between-sex covariance between the two nonhomologous traits (eqn 8) reduces to:

$$\text{cov}(T_{1m}, T_{2f}) = \text{cov}(T_{1f}, T_{2m}) = \bar{T}_1 \left[ 1 - \bar{P}_f \right] \text{var}(R) - \left[ \bar{R}_1^2 + \text{var}(R) \right] \text{var}(P),$$

which is identical to the within-sex covariance between competing traits (cov($T_{1p}$, $T_{2p}$) = cov($T_{1m}$, $T_{2m}$) = cov($T_{1f}$, $T_{2m}$); Appendix S1). Thus, when the genetic basis of acquisition and allocation is identical between the sexes (i.e. marginal distributions are the same and the sexes are perfectly correlated for $R$ and $P$), then within-sex covariances between competing traits (as originally derived by van Noordwijk & de Jong, 1986) are equal to the between-sex covariances for the same pair of traits.

**Strong intersexual genetic correlation in acquisition, with uncorrelated male and female allocation**

Suppose the intersexual correlation between acquisition rates is strong and positive, but allocation rates are weakly correlated. In the limit of a perfect correlation in the former and no correlation in the latter (corr ($R_m$, $R_f$) = 1 - corr($P_m$, $P_f$) $\to 1$), the between-sex covariance for the two traits reduces to:

$$\text{cov}(T_{1m}, T_{2f}) = \text{cov}(T_{1f}, T_{2m}) = \bar{T}_1 \left[ 1 - \bar{P}_f \right] \text{var}(R).$$

From this, it is clear (relative to eqn 7) that the between-sex covariance for two competing traits will be...
greater than the within-sex covariance between the same pair of traits, provided nonzero variances in acquisition and allocation (\(\text{var}(P), \text{var}(R) > 0\)). It also follows that the between-sex covariance between competing traits will be positive (\(\text{cov}(T_{1m}, T_{2g})\), \(\text{cov}(T_{1g}, T_{2m}) > 0\)), proportional to the variance in allocation (\(\text{var}(R)\)), and maximized at intermediate values of resource allocation (i.e. \(P = 0.5\)).

**Uncorrelated male and female acquisition, with strong intersexual correlation in allocation**

If there is no intersexual correlation for acquisition (\(\text{corr}(R_{m}, R_{f}) \approx 0\)) and a tight, positive correlation for allocation (\(\text{corr}(P_{m}, P_{f}) \approx 1\)), then eqn (8) reduces to:

\[
\text{cov}(T_{1m}, T_{2f}) = \text{cov}(T_{1f}, T_{2m}) = -\rho^2 \text{var}(P)
\]

Thus, the between-sex covariance for competing traits will always be negative, and less than the within-sex covariance (i.e. as long as \(\text{var}(P), \text{var}(R) > 0\)). The magnitude of the negative covariance is proportional to \(\text{var}(P)\), and increases exponentially with mean resource acquisition.

The idealized scenarios above suggest that patterns of (within- and between-sex) covariance between traits that compete for shared resources should provide information about the extent to which the genetic architectures of acquisition and allocation are shared between the sexes. Studies that estimate genetic variances and covariances for multiple fitness components (e.g. survival vs. fecundity or mating success) should therefore shed light on the genetic basis – shared vs. unshared – of female and male traits. There are only a handful of studies that have estimated intra- and intersexual genetic variances and covariances for multiple fitness components (Lewis et al., 2011; Gosden et al., 2012; Stearns et al., 2012), but measures of error for point estimates are lacking. We therefore evaluated the qualitative results of our models against intra- and intersexual genetic covariances estimated in Zajitschek et al. (2007; Table 1), who conducted a paternal full-sib/half-sib breeding experiment and reported additive genetic correlations between development time, adult body size, lifespan and multiple reproductive traits (male calling effort and female fecundity) in the Australian black field cricket (Teleogryllus commodus). We focus below on covariances between four traits that are likely to trade-off against each other: development time and body size, early and late reproduction, early reproduction and lifespan, and total reproduction and lifespan.

In many insect species, optimal development time, adult size and reproductive schedule differ between the sexes (Fairbairn et al., 2008). In the example presented here, these traits were positively genetically correlated, and intersexual fitness covariance was found to be strong and positive as well (Zajitschek et al., 2007) – a sign that sexual conflict was not strong in the tested population. The covariances between male and female development time and adult body size, and between male and female early and late reproduction, conform to theoretical expectations for the case where both acquisition and allocation had strong positive correlations between the sexes (\(\text{corr}(R_{m}, R_{f}) \approx 1\) and \(\text{corr}(P_{m}, P_{f}) \approx 1\); Table 1). Such a scenario corresponds with conditions leading to a positive intersexual covariance for fitness, including a masking of signals of sexual conflict over allocation (Fig. 2). It is therefore unsurprising that the fitness covariance between the sexes, as measured in this study, was indeed positive.

### Discussion

Our aim in this study is to formally define conditions of resource acquisition and allocation that promote – and

---

**Table 1**  Inter- and intrasexual genetic covariances based on data from Zajitschek et al. (2007). First value (on top) for a trait combination gives the additive genetic intrasexual covariances (\(\text{cov}_G\) ± SE, with covariances above the diagonal representing within-female estimates, and below diagonal within-male estimates. Covariances that conform to \(\text{corr}(R_{m}, R_{f}) \approx 1\) and \(\text{corr}(P_{m}, P_{f}) \approx 1\) (first scenario in main text) are given in bold.

<table>
<thead>
<tr>
<th>Male</th>
<th>Development time</th>
<th>Adult size</th>
<th>Early reproduction</th>
<th>Late reproduction</th>
<th>Total reproduction</th>
<th>Adult lifespan</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Development time</td>
<td>0.049 ± 0.026</td>
<td>0.037 ± 0.022</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adult size</td>
<td>0.043 ± 0.019</td>
<td>0.056 ± 0.022</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early reproduction</td>
<td>0.006 ± 0.019</td>
<td>0.024 ± 0.020</td>
<td>-0.021 ± 0.016</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Late reproduction</td>
<td>0.008 ± 0.024</td>
<td>0.047 ± 0.024</td>
<td>-0.028 ± 0.017</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total reproduction</td>
<td>-0.004 ± 0.013</td>
<td>-0.025 ± 0.014</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adult lifespan</td>
<td>0.027 ± 0.016</td>
<td>0.023 ± 0.015</td>
<td>0.024 ± 0.014</td>
<td>0.017 ± 0.014</td>
<td></td>
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</tr>
</tbody>
</table>
those that inhibit – the detectability of sexual conflict over fitness (‘intralocus sexual conflict’ or IASC). Our framework links sex-specific genetic effects of resource acquisition and allocation to sex-specific fitness, and takes into account the potentially shared genetic architectures of male and female traits. We show that a shared genetic basis of female and male resource acquisition, leading to a positive intersexual covariance for acquisition (cov($R_f$, $R_m$) > 0), should often mask signals of sexual conflict over resource allocation. Condition dependence of trait expression can – in some cases – allow sexual conflict over allocation to be uncovered more easily.

Conflict over resource acquisition itself is perhaps the most likely scenario leading to a detectable signal of IASC (i.e. estimates of fitness covariance are more likely to be negative when cov($R_f$, $R_m$) < 0). Recent data suggest that such scenarios are indeed plausible. In the red-spotted newt (Notophthalmus viridescens), for example, males and females experience divergent selection on feeding morphology and resource use in a manner that may impact sex-specific resource acquisition ability (De Lisle & Rowe, 2015). Male newts were found to feed more on planktonic microcrustacea, whereas females feed more on benthic invertebrates, leading to sexually antagonistic selection over feeding morphology. Instances of sexual dimorphism in traits that affect feeding ecology are found in many other taxa (Darwin, 1871; Shine, 1989), and these may reflect past or ongoing sexual conflict over resource acquisition.

We have emphasized the potentially more common case where there is no sexual conflict over acquisition itself (cov($R_f$, $R_m$) > 0), but rather, that conflict emerges over the allocation of acquired resources. Under these circumstances, we found that IASC is least detectable when the genetic basis of acquisition ability is strongly aligned between the sexes. Potential empirical evidence for this case has been found in the fly Drosophila serrata, where intersexual fitness correlations could be positive or negative for flies reared in different nutritional environments (Delcourt et al., 2009; Punzalan et al., 2014). Intersexual fitness covariances are expected to be strong and positive when a population that is adapted to one specific type of environment (e.g. a diet) is tested in a different environment (e.g. on a diet that might be much more difficult for both sexes to process and digest (Lande, 1980; Punzalan et al., 2014)). Changes in the environment may amplify the contribution of shared genetic variance in acquisition to total variation in fitness, resulting in strong between-sex correlations for both acquisition ability and fitness.

Environmental perturbations have the potential to increase or decrease fitness covariances between the sexes by way of shifts in optimal allocation that correspond to changes in environmental conditions. Abrupt environmental changes may in some cases lead to displacements of males and females from their optimal allocation rates, and thereby enhance or reveal new signals of IASC when the sexes exhibit different orientations of displacement from their allocation optima (e.g. females under-allocating and males over-allocating to a given trait). Conversely, greater similarity of displacements of each sex away from their allocation optima should elevate the intersexual fitness covariance and mask signals of sexual conflict. Susceptibility of the between-sex fitness covariance to variable environmental conditions can potentially account for mixed empirical results regarding the pervasiveness of IASC (see Introduction). Although this general possibility has been raised elsewhere (Long et al., 2012; Berger et al., 2014; Connallon & Clark, 2014; Connallon & Hall, 2016), the results presented here provide an additional rationale for it. These results also justify further study into the role of genetic correlations and environmental change in the manifestation of evolutionary constraints. Methods for sophisticated environmental manipulations that are directly pertinent to resource intake (for diet manipulations see, for example, the ‘Geometric Framework of Nutrition’ approach, Raubenheimer et al., 2009) can be used in the future to improve empirical estimates of acquisition and optimal allocation in each sex.

Whether the aim is to directly study the effect of environmental conditions on signals of IASC, or to simply control for environmental variation in experiments that estimate intersexual fitness covariances, it is worth considering how specific details of the hierarchical process of allocation might influence the development of male and female traits. Firstly, defining biological traits (anatomical, physiological or behavioural) that directly compete for resources remains a daunting task. Although fundamental trade-offs between life-history traits can be intuitive (e.g. between reproduction and survival), their molecular and physiological basis is often unclear (Zera & Harshman, 2001; Flatt & Heyland, 2011). One way to tackle this empirical problem is to integrate functional, physiological and molecular signalling information into models of resource allocation trade-offs. Although such calls for cross-disciplinary studies are far from new (e.g. Zera & Harshman, 2001), it remains a desirable goal. Future studies could potentially take advantage of well-characterized physiological systems or methods that quantify the fates of specific resources (e.g. nutrients) during allocation to specific traits (e.g. using isotopic radiotracing: O’Brien et al., 2008; Boggs, 1997) to gain insights into the allocation strategies that each sex employs during development. Secondly, given that more than two traits typically compete for acquired resources, an extension of our framework to accommodate higher-dimensional networks of competing traits could widen the scope of possible applications. As we mentioned earlier, if more
than two traits are genetically linked through competitive allocation, genetic covariances between some pairs of traits within an allocation network might not appear to trade-off (Charlesworth, 1990). Whether genetic covariances between the sexes might alter these predictions remains an open question.

Condition-dependent expression is an important attribute of exaggerated sexually selected traits. Genetic variation in the expression of sexually selected traits is thought to represent genetic variation in condition (‘genic capture’), and may serve as a mechanism to resolve sexual conflict, if this process is sex-limited (Bonduriansky & Rowe, 2005; Bonduriansky, 2007). Recent theory demonstrates that positive or negative covariance between acquisition and allocation can elevate the likelihood of detecting trade-offs between different traits expressed by individuals (van Noordwijk & de Jong, 1986; Descamps et al., 2016). Our results show that condition dependence is unlikely to have a large effect on the detectability of IASC because it has a small effect on the genetic covariance between female and male fitness. But whether condition dependence matters in this context ultimately depends upon the effect of condition on both the actual and optimal allocation rates of individuals in the population. Widespread empirical patterns of condition-dependent trait expression imply that optimal allocation rates are also condition dependent. For example, high-condition individuals may allocate a larger fraction of energy towards reproduction than low-condition individuals do because the optimal allocation strategies differ between the two classes of individuals. In the context of our model, the effect of condition dependence on the intrasexual covariance for fitness will be modest when individual and optimal allocation rates shift in parallel with changes in condition, as they are expected to do when condition dependence is adaptive.

Our model illustrates how high-quality quantitative genetic data on sex-specific allocation and acquisition can be used to predict between-sex covariances for fitness and between pairs of competing traits or fitness components. If we want to understand how sexual traits and life histories evolve, we need to better understand the sex-specific genetic architecture of trade-offs: measurable as the within- and between-sex genetic variance-covariance matrix for resource acquisition and trait allocation. Such data, when coupled with models like the one presented here, provide a potentially fruitful framework for understanding the empirical signals of trade-offs that arise within and between the individuals of a population.

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References


**Supporting information**

Additional Supporting Information may be found online in the supporting information tab for this article:

**Appendix S1** Intersexual genetic correlations for fitness (no condition-dependence).

**Appendix S2** Intersexual genetic covariance for fitness under condition-dependence.

**Appendix S3** Within- and between-sex covariances between traits.

**Figure S1** Effect of allocation-acquisition variance ratio when the sex-specific direction of selection on allocation rates is aligned (compare to Fig 2 in main text).

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SUPPLEMENT

Partitioning of resources: the evolutionary genetics of sexual conflict over resource acquisition and allocation

Please find below, in the following order:

Appendix 1.
Appendix 2.
Appendix 3.
Supplementary material.
Appendix 1. Intersexual genetic correlations for fitness (no condition-dependence)

In the simplest model, with no condition-dependence, we approximate the logarithm of individual fitness as:

\[
\ln(W_j) = k_j \ln(R_j) - \frac{(P_j - \theta_j)^2}{2\sigma_j^2}
\]

\[
= k_j \ln(R_j) - \frac{(R_j - \theta_j)^2}{2\sigma_j^2} + (R_j - \overline{R}) \frac{\partial \ln(W_j)}{\partial R_j} \bigg|_{R_j = \overline{R}, P_j = \overline{P}_j} + (P_j - \overline{P}) \frac{\partial \ln(W_j)}{\partial P_j} \bigg|_{R_j = \overline{R}, P_j = \overline{P}_j} + O\left(\left(R_j - \overline{R}\right)^2, \left(P_j - \overline{P}\right)^2\right)
\]

\[
= k_j \ln(R_j) - \frac{(P_j - \theta_j)^2}{2\sigma_j^2} + \frac{(R_j - \overline{R})}{R_j} k_j + \frac{(P_j - \overline{P})}{\overline{P}_j} (\theta_j - \overline{P}_j)
\]

The approximation was then used to obtain the intersexual fitness covariance:

\[
\text{cov}[\ln(W_j), \ln(W_m)] = \text{cov}\left(\frac{R_j k_f}{R_f} + \frac{P_j (\theta_j - \overline{P}_j)}{\overline{P}_j}, \frac{R_m k_m}{R_m} + \frac{P_m (\theta_m - \overline{P}_m)}{\overline{P}_m}\right)
\]

\[
= \frac{k_j k_m \text{cov}(R_f, R_m)}{R_f R_m} + \frac{(\theta_m - \overline{P}_m)(\theta_j - \overline{P}_j) \text{cov}(P_f, P_m)}{\overline{P}_j^2 R_m^2 \sigma_j^2} + \frac{\left(\omega_m \omega_j\right)^2}{R_f R_m \sigma_j^2 \sigma_m^2} + \frac{k_j (\theta_m - \overline{P}_m) \text{cov}(R_f, P_m)}{R_f \sigma_j^2} + \frac{(\theta_j - \overline{P}_j) k_m \text{cov}(P_f, R_m)}{R_m \sigma_m^2}
\]

In the absence of condition dependence, \(\text{cov}(R_f, P_m) = \text{cov}(R_m, P_f) = 0\), and the above equation simplifies to eq. (4) in the main text.

To validate the Taylor Series approximation of the covariance between male and female fitness (eq. (4)), we carried out simulations to calculate exact covariances. We generated random individuals by sampled \(R_f\) and \(R_m\) from a bivariate normal distribution (our simulations covered the parameter space of \(\text{cor}(R_m, R_f)\) between 0 and 1), and \(P_f\) and \(P_m\) from either a Beta distribution (with \(\alpha = \beta = 3\), giving a symmetric PDF around mean = 0.5), or a multivariate normal distribution. For Beta-distributed \(P_f\), sex-specific optimal phenotypic values, \(\theta_j\), were set to be: (1) identical between the sexes (\(\theta_m = \theta_f = 0.5\); corresponding to low intersexual conflict when \(P_f\) and \(P_m\) have similar distributions); (2) of intermediate divergence between the sexes (\(\theta_m = 0.25, \theta_f = 0.75\); intermediate conflict); or (3) strongly divergent (\(\theta_m = 0.1, \theta_f = 0.9\); corresponding to strong intersexual conflict). For \(P_f\) sampled from a Normal distribution, we manipulated values that were sampled outside of the plausible range (randomly sampled values of \(P_f < 0\) were set to zero; values of
$P_j > 1$ were set to one, and we kept $\theta_j$ constant at 0.5. We calculated the exact correlation between male and female fitness, and compared it to results using the covariance approximation (equation (4)).

A1a. Approximation of the correlation between male and female (log-) fitness using Beta distributed allocation rates.

For all plots: $\bar{R}_m = \bar{R}_f = 1; \omega_m = \omega_f = 1; k_m = k_f = 1; \alpha = \beta = 3$. 
A1b. Approximation of the correlation between male and female (log-) fitness using normally distributed allocation rates. For all plots: $R_m = R_f = 1$; $\omega_m = \omega_f = 1$; $k_m = k_f = 1$.

Appendix 2. Intersexual genetic covariance for fitness under condition-dependence

Supposing that the optimum and the allocation strategies for all individuals in the population fall within the interval $(0, 1)$, then individual allocation and optimal allocation can be modelled as simple functions of individual acquisition:

\[ P_j = y_j + \eta_j \left( R_j - R \right) \]

and

\[ \theta_j = \bar{\theta}_j + c_j \left( R_j - R \right) \]

We substitute these expressions into the general equation for log fitness, and then approximate using a Taylor Series expansion near the average acquisition and allocation, we obtain:
\[
\ln(W_j) = k_j \ln(R_j) - \frac{(y_j - \bar{\eta}_j + (\eta_j - c_j)(R_j - \bar{R}_j))^2}{2\omega_j^2} = k_j \ln(\bar{R}_j) - \left(\frac{(\bar{P}_j - \bar{\eta}_j)^2}{2\omega_j^2} + (\bar{R}_j - \bar{R}_j)\right) \left[\frac{k_j}{\bar{R}_j} - \frac{2(\bar{P}_j - \bar{\eta}_j)(\eta_j - c_j)}{2\omega_j^2}\right] - \frac{(y_j - \bar{P}_j)(\bar{P}_j - \bar{\eta}_j)}{\omega_j}
\]

Consequently, the covariance between the sexes becomes:

\[
\text{cov}\left[\ln(W_j), \ln(W_m)\right] = \left[\frac{k_j}{\bar{R}_f} + \frac{(\bar{\eta}_f - \bar{P}_f)(\eta_j - c_j)}{\omega_j^2}\right] \left[\frac{k_m}{\bar{R}_m} + \frac{(\bar{\eta}_m - \bar{P}_m)(\eta_m - c_m)}{\omega_m^2}\right] \text{cov}(R_j, R_m) + \frac{(\bar{P}_m - \bar{\eta}_m)(\bar{P}_j - \bar{\eta}_j)\text{cov}(y_j, y_m)}{\left(\omega_m\omega_j\right)^2}
\]

The final result corresponds to eq. (5) of the main text.

**Appendix 3. Within- and between-sex covariances between traits**

To describe the genetic architecture of resource-based trade-offs between two traits, we can formulate variances and intra- and intersexual covariances of traits \(T_1\) and \(T_2\) as follows. As in the main text, traits are simple functions of individual acquisition and allocation; in the \(f\)th sex, \(T_{2f} = R_fP_j\) and \(T_{2j} = R_j(1 - P_j)\). Under the assumption of independence between acquisition and allocation, it is straightforward to calculate all variances and pairwise covariances between traits and sexes:

\[
\begin{align*}
\text{var}(T_{1f}) &= E(P_j)^2\text{var}(R_j) + [E(R_j)^2 + \text{var}(R_j)]\text{var}(P_j) \\
\text{var}(T_{2f}) &= (1 - E(P_j))^2\text{var}(R_j) + [E(R_j)^2 + \text{var}(R_j)]\text{var}(P_j) \\
\text{cov}(T_{1f}, T_{2j}) &= E(P_j)(1 - E(P_j))\text{var}(R_j) - [E(R_j)^2 + \text{var}(R_j)]\text{var}(P_j) \\
\text{cov}(T_{1m}, T_{1j}) &= E(P_m)E(P_j)\text{cov}(R_m, R_j) + [E(R_m)E(R_j) + \text{cov}(R_m, R_j)]\text{cov}(P_m, P_j) \\
\text{cov}(T_{2m}, T_{2j}) &= (1 - E(P_m))(1 - E(P_j))\text{cov}(R_m, R_j) + [E(R_m)E(R_j) + \text{cov}(R_m, R_j)]\text{cov}(P_m, P_j) \\
\text{cov}(T_{1m}, T_{2j}) &= E(P_m)(1 - E(P_j))\text{cov}(R_m, R_j) - [E(R_m)E(R_j) + \text{cov}(R_m, R_j)]\text{cov}(P_m, P_j) \\
\text{cov}(T_{2m}, T_{1j}) &= E(P_j)(1 - E(P_m))\text{cov}(R_m, R_j) - [E(R_m)E(R_j) + \text{cov}(R_m, R_j)]\text{cov}(P_m, P_j)
\end{align*}
\]
**Figure S1.** Effect of allocation-acquisition variance ratio when the sex-specific direction of selection on allocation rates is aligned (compare to Fig 2 in main text). The ratio between variation in allocation and in acquisition rate is set (A) to 0.5 (var(P) = 0.05, var(R) = 0.1), (B) to 1 (var(P) = 0.1, var(R) = 0.1), and (C) to 10 (var(P) = 1.0, var(R) = 0.1). Optimal and mean allocation rates are identical for males and for females with $\theta_f = \theta_m = 0.5$, and $P_f = P_m = 0.1$. Blue (and minus signs) represents negative, and red positive values of intersexual fitness covariance.