Reproductive compensation

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Abstract

The reproductive compensation hypothesis says that individuals constrained by ecological or social forces to reproduce with partners they do not prefer compensate for likely offspring viability deficits. The reproductive compensation hypothesis assumes that (i) pathogens and parasites evolve more rapidly than their hosts, (ii) mate preferences predict variation in health and viability of offspring, (iii) social and ecological factors keep some individuals from mating with their preferred partners (some are constrained to mate with partners they do not prefer), (iv) all individuals may be induced to compensate, so that (v) variation in compensation is due to environmental and developmental factors affecting between-individual abilities to express compensatory mechanisms. Selection favouring compensation may act through variation in prezygotic physiological mechanisms, zygotic mechanisms, or parental care to eggs or young that enhance offspring health, increasing the likelihood that some offspring survive to reproductive age, often at a survival cost to the parents. Compensation may be through increased number of eggs laid or offspring born, a compensatory effort working during a single reproductive bout that sometimes will match the number of offspring surviving to reproductive age produced by unconstrained parents during the same bout. The reproductive compensation hypothesis therefore predicts trade-offs in components of fitness for breeders, such that parents constrained to mating with a nonpreferred partner, but who compensate sometimes match their current productivity (number of offspring at reproductive age) to unconstrained parents (those breeding with their preferred partners), and, when all else is equal, die faster than unconstrained parents. The reproductive compensation hypothesis emphasizes that reproductive competition is not just between constrained and unconstrained individuals, but also among constrained individuals who do and do not compensate. The reproductive compensation hypothesis may thus explain previously unexplained between-population and within-population, between-individual variation in reproductive success, survival, physiology and behaviour.

Introduction

But if you try sometimes you might find, You get what you need

The Rolling Stones

What do individuals do when they are constrained to reproduce with nonpreferred partners? If constrained or

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coerced reproduction leads to lower fitness than reproduction with preferred partners, whenever heritable variation (in genes, epigenetic elements, other developmental programmes, etc.) exists, selection will result in individuals sensitive to fitness outcomes and able to flexibly resist mating and reproduction with nonpreferred partners. But, there are situations in which reproduction with nonpreferred partners is the only option (Gowaty, 1997). The question then becomes, what can individuals – constrained to mate with partners they do not prefer – do, if anything, to make up likely fitness deficits compared with those individuals mating

with individuals they do prefer? One possibility is that constrained individuals attempt to compensate for predictable fitness deficits in their offspring (Foerster et al., 2003; Gowaty, 2003; Bluhm & Gowaty, 2004a; Navara et al., 2006; Byers & Waits, 2006; Anderson et al., 2007; Gowaty et al., 2007).

Thinking about some of the ways reproduction may occur under social constraints on female mate preferences inspired the reproductive compensation hypothesis (Gowaty, 1996, 2003; Gowaty & Buschhaus, 1998), but it is important to keep in mind that reproductive compensation can be induced by ecological constraints on mating with one's best (preferred) partner for offspring viability, such as dispersal limitation and demographic stochasticity, not just social constraints. Known social constraints acting on males and females include male–male competitive contests, and in some species in which females are invulnerable to social coercion, female mate choice may limit males' options for mating partners. Sexually coercive constraints limit females access to partners, but also males' and include infanticide (Hrdy, 1974, 1977, 1979, 1981), forced copulation (Brownmiller, 1975; Cheng et al., 1982, 1983; McKinney et al., 1983; Gowaty & Buschhaus, 1998), male aggression against females (Smuts, 1992, 1995; Smuts & Smuts, 1993), male mate-guarding (Dickemann, 1979a,b, 1981; Gowaty, 1996), sperm plugs and peptides that decrease females' re-mating tendencies (Chapman et al., 1994; Chapman & Partridge, 1996; Rice, 1996).

The reproductive compensation hypothesis emphasizes how selection might act not just among constrained and not constrained individuals, but between constrained individuals who can and cannot trade-off components of fitness to flexibly adjust physiology and behaviour in ways that enhance the future survival of their offspring. The reproductive compensation hypothesis is sexually symmetric, predicting that both sexes of parent are sensitive to, can assess, and respond flexibly to environmental and social conditions, i.e. both sexes may compensate. This paper contains a brief review of the assumptions of the reproductive compensation hypothesis, first presented in Gowaty (1996, 2003); Gowaty & Buschhaus (1998), and Gowaty et al. (2007). It also contains a quantitative description of components of fitness essential for understanding the reproductive compensation hypothesis, a comparison with the differential allocation hypothesis, and a discussion of the range of predictions of the reproductive compensation hypothesis with a focus on tests that depend on components of fitness. Last, I include a discussion of the question, 'Can compensation evolve?'

The assumptions

The assumptions of the reproductive compensation hypothesis include: (i) The most ubiquitous and persistent threats to offspring health are pathogens and parasites (Van Valen, 1973). (ii) Variation in offspring viability favours mate preferences in both sexes (Bonduriansky, 2001; Gowaty & Hubbell, 2005). (iii) Impediments (ecological and social) to reproduction with one's best partner exist (see references above). (iv) Mechanisms of compensation evolve rapidly to fixation, so that all individuals may compensate and (v) variation in expressed mechanisms of compensation is due primarily to environmental and developmental factors affecting individual ability to express compensatory mechanisms.

The Red Queen's challenge to parents, mate preferences and constraints on mate preferences

As the metaphor of the Red Queen stresses, pathogens evolve more rapidly than their hosts. This means that the pathogens that attack the parental generation may not be the same pathogens challenging the health of offspring. As Hamilton & Zuk (1982) said, if host–parasite cycles of evolutionary response ''are very short, then trying to choose mates for the 'right' genes for resistance is a perverse task'' (p. 384). In this case it would be unlikely that elaborate traits in potential fathers signalled 'good genes' for offspring health. If variation in the underlying genetic components of offspring viability favour mate preferences as the reproductive compensation hypothesis assumes, there is unlikely to be a single best male, better for all females than any other male, because females vary, and like males, contribute to the genetics, (as well as the epigenetics, ecology, development and culture) of offspring viability. If indicator traits show the true health status of the signaller, they may be honest indicators of an individuals' ability to provide direct benefits to females and to their offspring, but still say little about the genetics and development of offspring immune systems that must be an important component of offspring viability – unless there is little or no evolution of pathogens between parental and offspring generations. However, if offspring viability is influenced by alleles that work against offspring generation pathogens, the current health status of a potential mate may not predict the health of offspring. In such cases, it is possible that 'honest' indicators manipulate or dazzle choosers, exploiting their pre-existing sensory biases (West-Eberhard, 1979, 1984), so that choosers make reproductive decisions that may not favour offspring health. Thus, showy and elaborate traits could be simultaneously honest about some components of fitness (e.g. the breeders' current health and/or probability of survival), but may provide limited or no information relevant to other components of fitness. This perspective emphasizes that there may be other effects of host–pathogen arms races on individuals' reproductive decisions besides the evolution of elaborate traits in males. Of central importance here is that the perennial evolutionary arms races between hosts and their pathogens challenge parents anew in every generation.
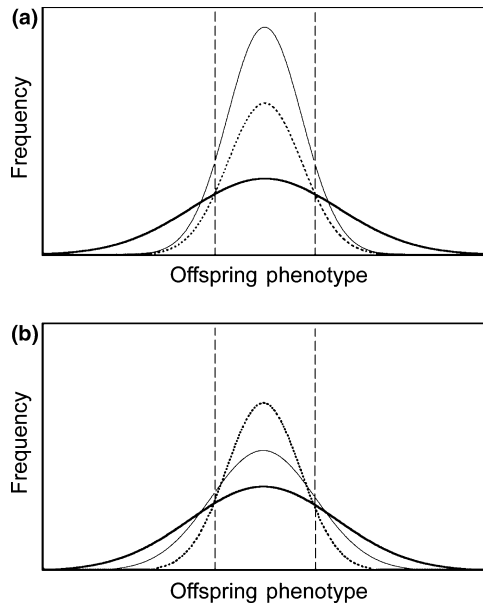
Fig. 1 Cartoon models of the Red Queen's challenge to parents and reproductive compensation by (a) increasing fecundity (the number of offspring born or eggs laid) and (b) increasing variance in offspring phenotypes without increasing fecundity. Assuming that pathogens evolve more rapidly than their hosts, that mate preferences predict viability of offspring, and that ecological and social constraints keep some individuals from mating with their preferred partners, the wide, dark curve represents offspring phenotypes produced by unconstrained parents. The peaked areas of the curves in the centre of the graphs between the vertical, dotted lines represent the offspring phenotypes parents are most likely to produce, i.e. offspring phenotypes likely to resist the pathogens and parasites of the parental generation. The areas under the curves in the tails of the distributions represent those phenotypes likely to resist the novel pathogens and parasites in the offspring generation. The dotted curve represents the offspring phenotypes produced when parents are constrained to reproduction with partners they do not prefer. The narrow, solid line represents the offspring phenotypes produced when constrained parents attempt to compensate by increasing the number of eggs laid or offspring born (a) or by increasing the variance in offspring phenotypes they produce (b). When constrained parents increase fecundity as in (a), the area under the curve in the tails of the distribution would be increased, enhancing the probability that some of their offspring are able to resist offspring generation pathogens. When constrained parents increase the variance in offspring phenotypes without increasing fecundity, perhaps through mating with more than one partner, the area under the curve in the tails of the distribution would be increased, enhancing the probability that some of their offspring are able to resist offspring generation pathogens.

In Fig. 1a,b, the dark solid curves are cartoons of an adaptive response to the Red Queen's challenge to parents. These high-variance curves are frequency distributions of the pathogen-fighting phenotypes unconstrained parents produce. The phenotypes likely to work against the pathogens in the parental generation are between the vertical dashed lines. However, these phenotypes will

be no more, and, more often, less effective against offspring generation pathogens. This will be so if pathogens differentially evolve offensive mechanisms against the most common defensive phenotypes in the parental generation. The biggest challenge parents may face, therefore, is to produce the rare phenotypes that are likely to work against the modified pathogens in the offspring generation. Rare phenotypes in Fig. 1a,b are in the tails of the distribution of offspring phenotypes parents can produce (those outside the vertical dashed lines).

Red Queen logic led Brown (1997) to argue that selection favours female mate preferences for heterozygosity in males and Wedekind & Furi (1997) and Wedekind *et al.* (1995) to argue that selection favours female mate preferences for complementary (dissimilar) immunogenes, an argument Penn & Potts (1999) formalized for vertebrates in their focus on the major histocompatibility complex (MHC). The mechanisms of mate preference posited by Brown, Wedekind, Penn and Potts are consistent with the idea that mate preferences are favoured by offspring viability selection, perhaps based on compatibility between mates (Ryan & Altmann, 2001). The focus here then is not on the cues associated with mate preferences, but on the fitness consequences of reproducing under social constraints on the free expression of preferences for both sexes. This analysis assumes that offspring viability favours mate preferences by both sexes independent of patterns of parental investment (Hubbell & Johnson, 1987; Ryan & Altmann, 2001; Gowaty *et al.*, 2003; Gowaty & Hubbell, 2005). I also assume that offspring viability selection works in invertebrates not just vertebrates and can favour mate preferences mediated not just through MHC variation, but also through variation in innate immunity.

Against this backdrop, the question becomes, what can individuals of either sex do when ecological or social forces constrain them to reproduction with partners that are nonoptimal for offspring viability? Constrained parents may attempt to compensate for offspring viability deficits by flexibly adjusting the number of offspring born or eggs laid, i.e. fecundity (Fig. 1a) to affect other fitness components (Tables 1 and 2), or physiology and behaviour (Table 3) in attempts to compensate for relative deficits in offspring viability.

Note that I am using the term 'constrained' to refer to the constraint of mating with a nonpreferred partner, specifically when mate preferences – evaluated in the absence of within sex interactions or between-sex coercion (so that sexual selection and sexual conflict are controlled) – predict variation in offspring viability.

Reproductive compensation via enhanced fecundity

Because the most radical prediction of the reproductive compensation hypothesis is that mothers constrained to reproduction with partners they do not prefer increase fecundity (the solid light line in Fig. 1a), I emphasize

Component of fitness	Symbol	Definition
Offspring viability	V	Fraction of offspring born or eggs laid, F, surviving to time α
Offspring age of first reproduction	α	Time from birth or hatch to first reproduction, i.e. age at first reproduction
Breeder fertility	B	Number of bouts of reproduction: > 1 for iteroparous species
Breeder fecundity	F	Number of offspring born or eggs laid per B
Breeder productivty	N	Number of offspring that survive to reproductive age per B
Breeder time between reproductive bouts	I	Time interval between Bs
Breeder survival probability	S	Likelihood of continued survival

Table 1 Definitions and symbols for components of fitness for breeders and offspring.

Table 2 Variation in components of fitness predicted by the hypothesis of reproductive compensation and differential allocation (*sensu* Burley, 1988).

Fitness component	Reproductive compensation (constrained to mate with partners individuals do not prefer, *c*, vs. nonconstrained, *nc*, mating with partners individuals do prefer)		Differential allocation (mated with attractive, *a*, or unattractive, *ua*, partners)	
	Females	Males	Females	Males
V, offspring viability	Vnc > Vc	Vnc > Vc	No prediction	
N, parent productivity	Nnc > Nc	Nnc > Nc	Na > Nua	Na > Nua
S, parent probability of survival	Snc > Sc	Snc > Sc	Sa < Sua	Sa < Sua
M, parent number of mates	Mnc > Mc	Mnc > Mc	Ma < Mua	Ma < Mua
F, parent fecundity (num offspring born or eggs laid)	Fnc < Fc		Fa > Fua	
SP, sperm ejaculated		SPnc < SPc		SPa > SPua
B, parent fertility (num reproductive bouts)	Bnc < Bc	Bnc < Bc	Ba > Bua	Ba > Bua
I, parent interval between reproductive bouts	Inc > Ic	Inc > Ic	Ia < Iua	Ia < Iua
α offspring age at first reproduction	anc > ac	anc > ac	No prediction	No prediction

Table 3 The reproductive compensation hypothesis predictions about physiology and behaviour when individuals do and do not make reproductive decisions under constraints. '*c*' indicates 'constrained' and '*nc*' indicates 'nonconstrained'.

Timing	Morphological, physiological, and behavioural traits of breeders and parental effects on offspring	Predictions of the reproductive compensation hypothesis
Pre-mating	Aggressive	c > nc
	Dominant	c > nc
	Most exaggerated 'indicator' traits	c > nc
	Multiple mating ('profligacy', extra-pair copulations)	c > nc
Pre-zygote	Number of sperm per ejaculate (males)	c > nc
	Number of eggs ovulated (females)	c > nc
Post-zygote	Intensity of brooding or fanning eggs	c > nc
	Food provisioning	c > nc
	Immune elements contributed through nursing or other provisioning	c > nc
	Parental contributions to eggs, foetuses, or offspring of hormones or peptides facilitating 'puberty acceleration'	c > nc

discussion of compensation via fecundity in this section and discuss compensation by other means (Table 1 and 3) further on. The discussion that follows is an operationalized discussion of the trade-offs in components of fitness associated with compensation by an increase in fecundity.

Define offspring viability, *V*, as the fraction of individuals born or eggs laid (*F* or fecundity) that survive to reproductive age, (*N* or productivity) (see Table 1 for a list of fitness components). The reproductive compensation hypothesis says that within- and between-population variation in *V* arises most reliably because of within- and between-population variation (1) in the degree of constraints on parents' reproductive decisions and (2) in the rate of pathogen evolution. Breeder productivity, *N* (the number of offspring that survive to reproductive age, α, see Table 1), is the product of breeder fecundity, *F* and offspring viability, *V*. Here variation in *V* is a component of offspring fitness (Anderson *et al.*, 2007) that, through its effects on *N*, acts as a selection pressure on parents.

Under pathogen pressure, as the degree of constraint on individual reproductive options increases, *V* declines. This causes selection on constrained parents to compensate for

lower N by increasing F (Fig. 1a, light solid curve), other fitness components (Table 2), or offspring and/or parent physiology and behaviour (Table 3) related to offspring health (Fig. 1b). Increased parental fecundity increases the likelihood that some of the offspring express rare phenotypes that may confer better resistance against offspring generation pathogens (Fig. 1a, solid light curve). Parents may compensate by increasing fecundity or similarly the number of bouts of reproduction (B or fertility, Table 1), because as the number of offspring produced increases, the number likely to express rare phenotypes that may work best against offspring genera-tion pathogens also increases, a simple mean to variance argument (Fig. 1a, light solid curve). Note that the number of offspring born or eggs laid, F, is expected to be higher for constrained breeders who do compensate (Fig. 1a, light solid line) compared with constrained breeders who do not compensate (Fig. 1a, dashed line) and also compared with unconstrained breeders (Fig. 1a, high variance, dark solid line). Note that although the phenotypic offspring vari-ance of the dotted curve (constrained, but noncompen-sating) and the light solid curve (constrained and compensating) are the same, because the compensation curve is higher (higher fecundity), there are numerically more offspring in the tails. The phenotypic variance has to be the same because there is no change in the parental genotypes, just in the number of offspring that they have. Thus in a comparison of constrained parents, those that compensate will produce absolutely more offspring than those who do not.

For equivalent fertility, unconstrained breeders will expose more phenotypic variation among their offspring for defence against evolving pathogens than will con-strained breeders (Fig. 1a). However, if constrained breeders increase their fertility (B, the number of bouts of reproduction), they can potentially expose more variation in offspring phenotypes for pathogen resis-tance. Increased F increases the probability that at least some offspring of constrained breeders will be resistant to evolving pathogens (Fig. 1a). This effect is compensatory because it reduces the difference in the productivity of parents constrained to breed with partners they do not prefer (Nc) and the productivity of parents not constrained (Nnc). (In the following the notation, 'c' indicates constrained to mating with partners nonpre-ferred and 'nc' indicates not constrained, so that individ-uals mate with those they prefer).

It is worth emphasizing that even production of more offspring with the parental phenotypes is a compensatory benefit in response to lower survival of these offspring. The compensatory benefit accrues over the entire distribution of offspring phenotypes that parents produce, not just in the tails of the distribution, because unless constrained parents adaptively control the distribution of offspring phenotypes so that their variance is increased (Fig. 1b) they will produce more compensatory offspring between the dotted vertical lines in the middle of the distribution

than in the tails of the distribution. This might often be the major benefit of compensating. Figure 1b is a cartoon model showing a distribution of offspring phenotypes unconstrained parents produce (dark solid line as in Fig. 1a) along with the distribution of phenotypes con-strained parents might produce if they compensate by adaptive modification of the variance in offspring pheno-types (light solid line). Here, the compensatory effect is not through increasing numbers throughout the distribution of offspring phenotypes, but through adaptive distribution of offspring numbers into the tails of the distribution, a variance increasing effect. Mating with more than one partner may be a compensatory mechanism to achieve adaptive modification of variance in offspring phenotypes.

Two conditions induce compensation

Compensation can be induced by variation in constraints on mate preferences that predict offspring viability or by changes in the pathogen environment alone.

When no constraints on the expression of mate prefer-ences exist (i.e. no stochastic effects, no ecological or dispersal limitations, no sexual selection and no sexual coercion), the mean strength of pathogen challenge alone will be a primary determinant of breeder productivity, N. If pathogen challenge increases during a given breeder's lifetime, mean offspring viability, V, would decline. In response, all breeders with appropriate developmental flexibility should attempt to compensate. As with individ-uals under social or ecological constraints on their mate preferences, individuals who respond to pathogen chal-lenge can increase productivity, N, by increasing fecun-dity, F, which may expose low-frequency variation among progeny in the genetic and phenotypic elements likely to work against offspring generation pathogens (Fig. 1). Or, they might compensate in other ways (Table 3).

Whether compensation is induced by variation in constraints on mate preferences or from pathogenic change, increasing F and/or other compensatory adjust-ments are expected to be costly to breeders, so that breeders who compensate may incur costs, which should be obvious in terms of declines in breeder survivorship, S (Table 1). Elsewhere we model the social-selective conditions under which a quantitative trait for reproduc-tive compensation can evolve (P. A. Gowaty & S. P. Hubbell, unpublished data). Under a large range of selective scenarios, even when breeder survival costs are quite high, compensation evolves (see below). Moore *et al.* (2003) and Anderson *et al.* (2007) reported survival costs of compensation respectively in *Nauphoeta cinera* and *Drosophila pseudoobscura*.

The offspring viability assumption

The assumption that $Vc < Vnc$ is met in a wide variety of species including flies (Anderson *et al.*, 2007), mice (Drickamer *et al.*, 2000, 2003), pronghorn (Byers &

Waits, 2006), ducks (Bluhm & Gowaty, 2004b) and other species (see review in Gowaty *et al.*, 2007). For compensation to be successful, compensatory efforts need only increase productivity, N, of constrained breeders that do compensate above constrained breeders that do not compensate. When constrained breeders compensate, they may rarely succeed in achieving perfect compensation (i.e. $Nc = Nnc$) partly because of the costs of compensation. Compensation would, nevertheless, be favoured whenever there is variation among constrained individuals in their abilities to compensate. Thus, the bar is set in two places for constrained individuals: first, in comparison to unconstrained competitors and, second, in relation to variation in the ability of other constrained individuals to compensate.

Because V, offspring viability, is operationally defined here as N/F, there are nine possible relationships between fecundity, F, and productivity, N characterizing Vc and Vnc (Fig. 2). In Fig. 2, panels c, d, and f show that $Vnc > Vc$ and therefore, only these relationships between F and N for constrained and unconstrained individuals meet the reproductive compensation hypothesis assumption that $Vc < Vnc$. Figure 2c shows $Fnc = Fc$, but $Nnc > Nc$ so that although $Vc < Vnc$, there exists no evidence of attempted compensation via increased F. However, Fig. 2d and e indicate $Fc > Fnc$, consistent with attempts to compensate for $Vc < Vnc$. Figure 2d shows completely successful compensation via increased F, because $Nc = Nnc$. Figure 2f shows unsuccessful compensation because despite $Fc > Fnc$, $Nc < Nnc$.

Observation of relationships in Fig. 2a,b,e,h and i would indicate that the assumption of the reproductive compensation hypothesis is not met. This is because variation in F and N is such that $Vnc < Vc$.

The discussion above emphasizes what is expected if mothers compensate by increasing fecundity, but other compensatory mechanisms (discussed below) are possible. The selection differential, $Nnc > Nc$ will favour compensatory fecundity, fertility, behaviour or physiology (Tables 2 and 3) such that the differences in productivity, Nnc and Nc, are minimized relative to existing pathogen pressures. Even when compensation is completely successful via increased F (Fig. 2d), bouts of reproduction, B, longer intervals between reproductive bouts, I (Table 3) or via various types of parental care, so that $Nc = Nnc$, the costs of compensation for $Vnc < Vc$ often will result in lower survival, S, of breeders reproducing under constraints, i.e. $Snc > Sc$. This should be so even when $Bnc = Bc$ and even when breeder fecundity is $Fnc = Fc$. The predicted relationship between $Snc > Sc$ is not simply due to higher Fc but due to the costs of attempts to compensate for lower Vc, which could occur through fecundity compensation or another compensation mechanism altogether (see below).

Compensation is unlikely when variation in developmental flexibility is low

The reproductive compensation hypothesis does not predict fecundity variation in highly inbred pairs in which genetic and developmental variation are low or nonexistent (Meagher *et al.*, 2000); it does predict compensation via increased fecundity whenever latent variation can be uncovered by increasing the number of offspring produced. Of course, inbred pairs may compensate by other means (Table 3) including, but not limited to, increased B, number of bouts of reproduction, or shorter I, intervals between bouts of reproduction (Tables 1 and 2).

Pre-copulation compensatory mechanisms

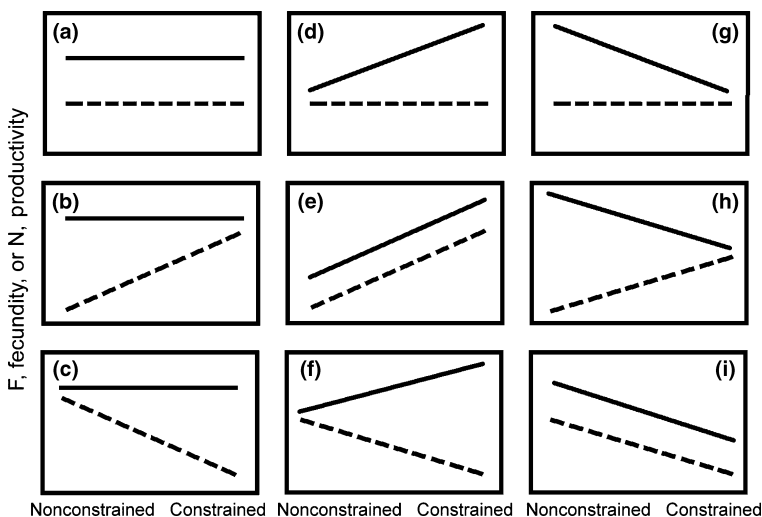Males may compensate by increasing access to females through enhanced within-sex aggression, dominance

Fig. 2 All possible combinations of fecundity, F (solid lines), and productivity (number of offspring reaching reproductive age), N (dashed lines), when breeders are nonconstrained, reproducing with partners they prefer, and constrained, reproducing with partners they do not prefer. Offspring viability, N/F, is the same for constrained and unconstrained in (a, e, and i). N/F is greater in constrained than nonconstrained in (b, g, and h). Thus, (a, e, i, b, g, and f) fail to meet the assumption of the reproductive compensation hypothesis that offspring viability is greater for nonconstrained breeders. This assumption is met in panels (c, d, and f). However, compensation is fully successful only in panel (d) in which N is the same for nonconstrained and constrained individuals.

behaviour and their rate of attempted reproduction with multiple females (Table 2). Similarly, females may compensate by increasing their numbers of extra-pair mates to enhance their access to alleles favourable to enhanced offspring viability (Foerster *et al.*, 2003).

Exaggerated male dominance or aggression, or exaggerated indicator traits may be compensatory for likely $Vc < Vnc$. This possibility allows predictions about which males within a population are most likely to be aggressive, dominant or fancy (Moore *et al.*, 2002) and profligate (Table 3). If the reproductive compensation hypothesis is correct, in genetically structured populations, males least likely to be preferred (those that are perhaps most similar to the females at immune coding genes) are more likely to express compensatory within-sex aggression and dominance behaviour than males more likely to be preferred.

Compensatory mechanisms during copulation

The reproductive compensation hypothesis predicts that males copulating under constraints with partners they do not prefer ejaculate more sperm than males copulating with fewer or no constraints with partners they do prefer (Table 3). Larger ejaculates would increase the number of and, thus, the variation among male gametic haplotypes available to females to sort among. Kim *et al.* (2005) showed that male *D. pseudoobscura* ejaculate more sperm when mating with females they do not prefer and with females who do not prefer them. The hypothesis predicts males copulating under constraints with females they do not prefer will contribute more peptides or nutrients in their ejaculates that will enhance immunity of zygotes and that they will do this flexibly in response to encountered circumstances. Likewise, the reproductive compensation hypothesis predicts that females physiologically select among the sperm of males they do not prefer, so as to increase the likelihood that resulting zygotes will have a higher probability of survival to reproductive age than they otherwise would. It predicts that females mating under constraints with males they do not prefer will produce more oocytes to increase the likelihood of rarer haplotypes in eggs available for fertilization. It predicts that females and males contribute more nutritive, immunity-enhancing resources to the cells and tissues supporting their gametes and resulting zygotes when they are constrained to reproduction with partners they do not prefer than with partners they do prefer.

Post-zygotic compensatory mechanisms, and contrasts with the differential allocation hypothesis

Parents of either sex may attempt to compensate for lower offspring viability, *V,* by increasing types of parental care that mitigate the effects of pathogens on their offspring. The reproductive compensation hypo-

thesis predicts that constrained mothers will lay bigger eggs, as found in ducks (Bluhm & Gowaty, 2004a) or nurse their offspring longer than unconstrained mothers, as found in pronghorn (Byers & Waits, 2006). It predicts that constrained mammal mothers will contribute more antibodies over longer periods to dependent offspring than those reproducing with preferred partners. It predicts, all else equal, that constrained avian parents more reliably incubate eggs than unconstrained parents, whenever thermal stress to embryos is associated with increased susceptibility to disease in embryos. For frogs and fish that aerate egg masses to control against fungal contamination, all else equal, the reproductive compensation hypothesis predicts that constrained parents aerate egg masses more than unconstrained parents. The reproductive compensation hypothesis predicts that constrained parents may manipulate their offspring through 'parental effects' in ways that reduce the age of first reproduction relative to the offspring of unconstrained parents. Given that the host–pathogens arms races are renewed in each generation, a parental effect to lower the age of first reproduction of their offspring from constrained matings would enhance the likelihood that they would have grand-offspring.

In contrast to the reproductive compensation hypothesis, the differential allocation hypothesis (Burley, 1988) predicts greater parental allocations when individuals reproduce with relatively attractive partners. This hypothesis says that in socially monogamous species, selection would favour individuals that flexibly adjust their allocations to offspring as a function of the relative attractiveness of each of the parents, with the less attractive parent allocating more parental effort than the more attractive parent. Sheldon (2000) extended this idea to species without bi-parental care, and predicted that females allocate more resources to the offspring of attractive males. Whether the predictions of this hypothesis contrast with predictions of the reproductive compensation hypothesis depends on whether consensus attractive individuals are also individually preferred (Table 4) and on whether consensus attractiveness predicts offspring viability (see discussion above in relation to Hamilton & Zuk's (1982) hypothesis).

If attractive individuals are those that opposite sex individuals display to the most in arenas containing more than one of each sex as in Cunningham & Russell (2001), it is possible that 'attractive' is not the same as a preferred partner in an arena that eliminates intra-sexual interactions and inter-sexual coercion to measure individual preferences as in Bluhm & Gowaty, (2004a,b). If offspring viability is equal or lower when females mate with consensus attractive partners rather than consensus unattractive partners, one could speculate that 'attractive traits' exploit chooser sensory biases so that choosers make reproductive decisions that fail to enhance offspring viability or that group level attractiveness is manipulated by intrasexual interactions or coercive

Table 4 Similar and contrasting predictions about F or other trait such as increased egg size from the differential allocation hypothesis and the reproductive compensation hypothesis, when the differences in methods for evaluating consensus attractiveness and individual preferences are taken into account.

Choosers' partners*	Differential allocation hypothesis	Reproductive compensation hypothesis
C-A and I-NC	+	−
C-A and I-C	+	+
C-UA and I-NC	−	−
C-UA and I-C	−	+

As elsewhere in this paper, those mated with partners they individually prefer are non-constrained, (i.e. I-NC) and those mated with partners they individually do not prefer are constrained (i.e. I-C). Individuals mated with consensus attractive partners is indicated by C-A; those mated with consensus unattractive partners by C-UA.

signalling; if this happens, females mated to 'attractive' partners might be constrained, mating with partners they do not individually prefer. Then, the predictions of the two hypotheses would match, e.g. both would predict enhanced number of eggs laid or offspring born, F, or enhanced care, for example (Table 4). If consensus attractive partners have offspring of higher viability than consensus unattractive individuals; however, the predictions are contrasting, with the differential allocation hypothesis predicting higher F for females mated with consensus attractive males and the reproductive compensation hypothesis predicting higher F for females mated with males they do not individually prefer. Tests of these two hypotheses depend on the offspring viability assumption, and before comparative tests of these two hypotheses are carried out, the association with offspring viability of consensus attractiveness and individual preferences should be evaluated.

It is important in this context to emphasize the meaning of 'attractive' and 'constrained to mate with a partner one does not prefer'. In the reproductive compensation hypothesis preference is about 'self-referential preferences' (Ryan & Altmann, 2001); explicitly, this means that there is unlikely to be a best male that all females prefer or a best female that all males prefer. In contrast, the differential allocation hypothesis (Burley, 1988) depends on there being males and females that are attractive to most opposite sex individuals, i.e. consensus attractive. Thus, whether these two hypotheses always make contrasting predictions (Table 2) depends on the special assumptions of the compensation hypothesis, and the definitions and methods for evaluating 'individual preferences' and 'consensus attractiveness'.

Do unconstrained breeders compensate?

Unconstrained breeders will compensate whenever there is an increase in pathogenic challenge relative to background conditions. Thus, the reproductive compensation hypothesis predicts within population variation in F as a function of temporal variation in pathogen exposure, even when social or ecological constraints on mate preferences are absent. So, when pathogenic challenge is changing relatively rapidly, unconstrained breeders might compensate.

However, when pathogen challenge is not changing more rapidly than usual between parents and offspring generations, constraints on the expression of mating preferences theoretically determine which parents compensate. So that when some breed with partners they do not prefer, whereas some breed with partners they do prefer, the reproductive compensation hypothesis says that individuals adjust F so that N for constrained breeders might approximate N for unconstrained breeders (Fig. 2c). However, except for the compensatory effects against pathogens described in the preceding paragraph, the theory does not predict compensation by unconstrained breeders. This is because compensation is likely to impose costs that lower parental survival. When constrained breeders differ in resource availability, one might expect that compensation would be expressed more readily for those with high access to resources because the effects of compensation on parental survival could be reduced depending on the expressed mechanism of compensation. Furthermore, because compensation by constrained breeders should rarely increase N above that for nonconstrained breeders (see for example, Anderson *et al.*, 2007), the reproductive compensation hypothesis says that N of unconstrained breeders is the fitness component that constrained compensating individuals attempt to match.

Sexual selection among constrained and nonconstrained breeders favours compensation

Many investigators consider variation in offspring viability to result only in natural selection. When compensation is in response only to differential pathogen challenge, this argument would hold. However, the argument that offspring viability selection is only natural selection ignores the fact that variation in offspring viability also occurs in response to variation in social constraints on reproductive decision-making. Within-population, within-sex variation in individual abilities to remain in control of their own reproductive decisions (e.g. with whom they mate) also results in reproductive success variation and fits the Darwinian definition of sexual selection – for females as well as males. For example, whenever offspring viability is less for constrained than unconstrained females, reproductive competition among females may occur through fitness effects from variation in individuals' vulnerabilities to coercion (Gowaty, 1996). This perspective suggests that offspring viability and other lineage effects, usually not considered in models of sexual selection, may, in fact, be important

to understanding the full range of mechanisms of within-sex reproductive competition (sexual selection).

Testing the reproductive compensation hypothesis

Whenever $Vnc \leq Vc$, the main assumption of the reproductive compensation hypothesis is not met, so that the reproductive compensation hypothesis would not apply.

Whenever $Vnc > Vc$, the main assumption of the reproductive compensation hypothesis is met, so that it would be reasonable to seek evidence of reproductive compensation. If $Fnc < Fc$, the prediction of attempted compensatory reproduction via fecundity enhancement of constrained individuals would be fulfilled. However, only if $Nnc = Nc$ would reproductive compensation be fully successful. Whenever constrained individuals provision more than unconstrained (e.g. as in pronghorn, Byers & Waits, 2006), the prediction of attempted compensation via enhanced provisioning would be fulfilled. Again, however, only if $Nnc = Nc$ would reproductive compensation be fully successful.

Thus, the contest is most intense not between unconstrained and constrained breeders, but between constrained breeders who do (Fig. 1a,b, solid light curve) or do not (Fig. 1a,b, dashed curve) compensate or those who compensate relatively more and less efficiently than others.

Evaluation of F or N alone would be inadequate for evaluation of the hypothesis (Fig. 2). In lab studies, to evaluate variation in V, i.e. offspring viability for breeders in which constraints vary, investigators often compare the effect of being with preferred and nonpreferred partners. As ornaments in one sex may manipulate the behaviour of the other sex, one must control the possibility that honest indicators of health nevertheless may manipulate the opposite sex into mating with partners that are nonoptimal for the viability of offspring. To do this, one might evaluate offspring viability against preferences determined at random with respect to phenotypic traits in the discriminated sex as in Drickamer *et al.* (2000), Bluhm & Gowaty (2004a), Moore *et al.* (2003) and Anderson *et al.* (2007). Field tests of these ideas, such as Byers & Waits (2006) will require a large amount of information on individual breeders, mate status and their offspring. In most cases, in the field it is not clear how to measure the expression of mate preferences free of the presence of confounding factors such as male–male combat, female–female interactions and mechanisms of male control and female resistance. When choices are manipulated or coerced, choices may not reflect preferences: individuals may have paired under multiple constraints, including ecological and developmental ones. All these potential factors make judging which are the constrained pairings in the wild difficult. Despite this difficulty, evaluation of predicted relationships in mean offspring V and mean breeder F and N between populations differing in pathogen pressures is possible. However, such studies must then take into account the likelihood that within populations some individuals may, and others may not, be reproducing under further constraints and may be compensating.

Testing for compensation via trade-offs in fitness components of adults and their offspring seems the most comprehensive way to test compensation. Anderson *et al.* (2007) used a demographic, components-of-fitness approach to fitness variation in constrained and unconstrained individuals. Replication of their methodology in studies of other species will be valuable in evaluating the general ability of the reproductive compensation hypothesis to explain usually unexplained variation in components of fitness (Table 2).

Testing for compensation via parental effects on physiology and behaviour will be more difficult. Although making and testing predictions of egg-size variation or nursing duration are relatively easy to imagine and carry out, what is needed is a hierarchical set of predictions about parental effects, perhaps ranked in terms of their costs to compensating parents. Since the relative cost – either in calories or fitness – of different compensatory mechanisms is entirely unknown, this is a question for the future.

Within-population, between-sex variation in the ability to compensate is likely affected by individuals' intrinsic metabolic rate, the efficiency with which individuals exploit resources and their access to extrinsic resources, which probably interacts with the ability of individuals to simultaneously compensate and withstand the pathogenic challenges they face (P. A. Gowaty & S. P. Hubbell, unpublished data). Variation among females in their abilities to avoid coercion will be a productive avenue for future research on the costs and benefits of compensation.

Reproductive compensation can evolve

Is it reasonable to assume, given appropriate access to resources, that all individuals can compensate? This is a question related to the evolution of developmental plasticity, flexibility and induced responses (West-Eberhard, 2003; Joblonka & Lamb, 2005) and the costs of plasticity (Scheiner & Berrigan, 1998; Relyea, 2002; Bize *et al.*, 2004; Steiner & Van Buskirk, 2008). P. A. Gowaty & S. P. Hubbell (unpublished data) have answered this question theoretically using a simple quantitative approach, which takes into account not only genetic influences, but environmental, developmental and social influences on compensation, as well as stochastic influences on fitness variances. In this model, the compensatory response is an increase in the number of eggs laid or offspring born, F. Compensation, which is composed of sensitivity to inducing stimuli, assessment of fitness outcomes from mating with alternative potential partners and responsiveness to individual assessments and to inducing stimuli, is a metric trait influenced by 20 loci and 40 alleles, and the strength of compensation is

dependent on the degree of similarity in 'immuno-genes' of parents (how similar and dissimilar they are) and the dose of compensation alleles. When access to resources is held constant as we assume in the model, higher doses of compensation alleles allow individuals to compensate more. The degree of required compensation is set by the difference in shared immuno-genes and the maximum difference possible. Thus, when individuals have identical access to resources, being able to compensate more means that individuals have better assessments of fitness outcomes, finer sensitivity to inducing stimuli and more efficient physiological and behaviour mechanisms of compensation. The individual-based model of fitness variation of constrained individuals that do and do not compensate shows that compensation can evolve to fixation (all individuals are able to compensate) relatively rapidly under high costs and relatively small benefits. In the model each individual breeder had an immuno-genotype. The more variable an individuals' immuno-genotype the higher was its probability of survival. An individual's immuno-genotype when combined with the immuno-genotype of any mate, influenced the probability of survival of offspring they produced (there were also stochastic effects on survival). When individuals mated, compensation was induced relative to the maximum possible percent shared parental immuno-genes in the population, and the degree of compensation was a function of the number of shared parental immuno-genes so that those mating pairs with more immuno-genes in common had offspring with a lower probability of survival than pairs with dissimilar immuno-genes. Using the model, we specifically evaluated the ability of compensating individuals to increase fecundity, when breeders differed in immuno-genes, which determined their vulnerability to current pathogens, though holding resource availability constant. We consider our model conservative because compensation would be expected to evolve more rapidly when resources vary among individuals, as they do in nature. The cost of compensation was allowed to vary due to intrinsic (e.g. age, size, previous breeding experience) differences among individuals. Even under high costs of compensation (extracted in terms of breeders' survival probability to the next bout of reproduction), compensation always evolved to fixation relatively rapidly, within 200–400 generations.

So, what traits evolve when compensation evolves? As in other flexibly expressed traits, for individuals to be able to successfully compensate, they must to be sensitive and responsive to inducing stimuli. Further, because reproductive compensation assumes comparisons of individual fitness outcomes, individuals must be able to assess likely fitness outcomes of mating with alternative potential mates (no consciousness implied) as well as the likely fitness of offspring they would produce; i.e. relative to the offspring of other breeders in the population (see similar discussion in Gowaty & Hubbell, 2005; unpublished data). Therefore, traits associated with sensitivity and assessment of inducing stimuli, and responsiveness to stimuli must evolve in order for compensation to evolve.

Thus, because in theory compensation evolves rapidly even when costs are high and benefits relatively low, in this paper, my assumption that all individuals in a population may compensate, is reasonable. If this is the case in nature, it means that the most important sources of variation in expressed compensation and perhaps the use of alternative compensatory mechanisms depends on (1) the level of compensation that would approach productivity (number of offspring that survive to reproductive age) of individuals mating with partners they prefer and (2) access to resources, which could affect the level of individual responses and the extractive costs to survival to the next reproductive bout.

Acknowledgments

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