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CHAPTER 17

The genetic architecture of sexual dimorphism: the potential roles of genomic imprinting and condition-dependence

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17.1 Introduction: the puzzle of sexual dimorphism

Differences between the sexes in morphology, physiology, behavior, and life history are ubiquitous, but we still have much to learn about how genomes produce strikingly different phenotypes in different sexes. In this chapter, I explore some emerging research that promises to enrich our understanding of the evolution and genetic architecture of sexually dimorphic traits. Building on the ideas and findings outlined in Section 17.1, I consider the potential roles of two genetic mechanisms, genomic imprinting and condition-dependence. I outline recent advances in theory and empirical knowledge that link these phenomena to sexual dimorphism, and suggest some questions for future investigation.

17.1.1 Sex-specific selection

The fundamental dimorphism in gamete size (anisogamy) is thought to engender the contrasting reproductive strategies of females and males that, in turn, select for sexual dimorphism. Competition among the numerous, tiny male gametes for access to a limited number of large, resource-rich female gametes selects for the 'primary' sex differentiation of the reproductive system, and ultimately leads to elaborate and often spectacular forms of male–male competition for access to females, mediated by 'secondary' sexual traits such as weapons, signals, clasping devices, or specialized sensory adaptations. Sex-specific reproductive strategies also typically comprise life-history traits such as growth rates, reproductive schedules, and aging rates. Thus, numerous male traits are subject to sexual selection, whereas the homologous traits of females are not. We still know very little about sex-specific selection (but see Preziosi and Fairbairn 2000; Chenoweth and Blows 2005). Nonetheless, it is clear that sex-specific selection favors the independent evolution of the sexes towards sex-specific (sexually dimorphic) phenotypic optima. As illustrated by previous chapters in this volume, sexual size dimorphism, where one sex expresses a trait such as an appendage, or the entire body, at a greater size than the other sex, characterizes many sexually homologous traits, including some spectacularly exaggerated secondary sexual traits.

17.1.2 Intersexual genetic correlations and intralocus sexual conflict

The puzzle of sexual dimorphism, first clearly formulated by Fisher (1930a, 1931), reflects the fact that sex-specific selection acts on genes that are expressed in both sexes and transmitted from mother to son and from father to daughter (Mendelian genes). Trait expression in females and males is therefore typically subject to an intersexual genetic correlation which can impede the

evolution of sexual dimorphism (Lande 1980a, 1987; and see Chapters 1, 16, and 18 in this volume). Sexual selection on male traits can cause correlated evolution in homologous female traits, displacing the female phenotype from the optimum for viability and fecundity. Likewise, viability and fecundity selection on female traits can constrain the evolutionary response of homologous male traits. Consequently, genes subject to sex-specific selection may have sexually antagonistic fitness effects, increasing fitness when expressed in one sex, but reducing fitness when expressed in the other sex (Rice 1984; Rice and Chippindale 2001; Chapter 18). Genes with sexually antagonistic fitness effects have been documented in the fly *Drosophila melanogaster* (Chippindale *et al*. 2001; Rand *et al*. 2001; Chapter 18) and the cricket *Allonemobius socius* (Fedorka and Mousseau 2004). Such genes contribute to intralocus sexual conflict, a deviation from the optimal genotype at a locus as a result of a different pattern of selection on the same locus in the opposite sex, manifested in sub-optimal expression of traits affected by loci under sex-specific selection. Although the most intense intralocus sexual conflict is associated with sexually antagonistic selection (i.e. selection acting in opposite directions in females and males), some degree of intralocus sexual conflict is likely under any pattern of sex-specific selection (see Section 17.2).

An autosomal-Mendelian genetic architecture may be regarded as the genetic null model and primitive condition for most sexually homologous traits. This genetic architecture generates a high intersexual genetic correlation (see Lande 1987; Chapter 1). If, in one sex, such a trait is displaced from its viability optimum by sexual selection, then the genes affecting the expression of this trait will have sexually antagonistic fitness effects and generate intralocus sexual conflict. Lande (1980a, 1987) argued that sex-specific selection would ultimately lead to a reduction of the intersexual genetic correlation, allowing sexual dimorphism to evolve more rapidly. Although this prediction remains controversial (see Reeve and Fairbairn 2001; Fairbairn and Roff 2006), it is supported by recent evidence, from a fly, a bug, a moss, and a gynodioecious strawberry, showing that the more dimorphic traits within species tend to exhibit lower intersexual genetic correlations than traits with little or no dimorphism (Ashman 2003; Bonduriansky and Rowe 2005a; McDaniel 2005; Chapter 9). The most parsimonious explanation for this pattern is that traits that are (or have been) subject to intense intralocus sexual conflict evolve modifications to autosomal-Mendelian genetic architecture, and these genomic adaptations reduce the intersexual genetic correlation and facilitate the evolution of sexual dimorphism. But what are these genomic adaptations?

17.1.3 Genomic adaptations to intralocus sexual conflict

Genomic modifications that may reduce intralocus sexual conflict and facilitate the evolution of sexual dimorphism can be separated into two non-exclusive categories: those based on sex-linked segregation of loci located on the sex chromosomes, and those based on sex-limited epistasis. The former may reduce intersexual genetic correlations because sex chromosomes exhibit sex-dependent dosage. Fisher (1930a, 1930b, 1931) recognized that the Y chromosome is absent from female genomes, so any male-benefit genes on that chromosome will be expressed in males only. Consequently, Y-linked genes do not contribute to intersexual genetic correlations or intralocus sexual conflict (see Roldan and Gomendio 1999). However, Rice and Chippindale (2002) argued that the small, degenerate Y chromosome is unlikely to accommodate a sufficient number of genes to substantially mitigate intralocus sexual conflict. The much larger X chromosome is present in double dose in females, relative to males (although dosage compensation or X inactivation may reduce or eliminate this dosage difference). Rice (1984) showed that sex-linked segregation may facilitate the evolution of sexually antagonistic genes on the X chromosome. Nonetheless, because the X chromosome is expressed in both sexes, X-linked genes are not immune from intralocus sexual conflict (Rice 1984). It remains unclear whether sexually antagonistic genes are disproportionately located on the sex chromosomes (see Reinhold 1998; Roldan and Gomendio 1999; Lindholm and Breden 2002; Parisi

et al. 2003; Fitzpatrick 2004). However, because the sex chromosomes (in species that have them) are necessarily the basis of all other differences between the sexes, sex-linked segregation is likely to play an important role in genomic adaptation to intralocus sexual conflict.

Sex-linked genes may serve as 'switches' for mechanisms of sex-limited epistasis (gene interaction patterns that occur in one sex only) that mitigate intralocus sexual conflict. One mechanism of sex-limited epistasis that may be of considerable importance is the duplication and sex limitation of autosomal loci (Rice and Chippindale 2002; Proulx and Phillips 2006; Chapter 16). If an autosomal locus carrying a male-benefit gene is duplicated, and one of the resulting loci subsequently evolves male-limited expression (under the control of a sex-linked genetic switch), the intersexual genetic correlation will be reduced, and this locus will evolve towards the male optimum, unopposed by antagonistic selection on females. Sexually antagonistic selection may similarly favor sex-limited expression of some alleles at autosomal loci (Rhen 2000; Chapter 16; and see Montgomery *et al*. 1996; Chase *et al*. 2005, for empirical examples). A long-recognized form of sex-limited epistasis is the role of sex-hormones in sexual differentiation (Jost *et al*. 1973; Chapter 16). Hormonal control of sexual dimorphism is well known in mammals (Renfree 1992; Renfree *et al*. 2001) and insects (Stern and Emlen 1999; Emlen *et al*. 2006). However, sex-limited epistasis can also take the form of 'direct' (i.e. not hormone-mediated) genetic control of sexual differentiation. Direct genetic control can involve both sex-linked and autosomal genes, and has been observed in mammals (O *et al*. 1988; Glickman *et al*. 2005; Chase *et al*. 2005) and insects (Kopp *et al*. 2000). Sexual dimorphism may often reflect a combination of hormonal and direct genetic mechanisms (see Emlen *et al*. 2006). We still know very little about variation among traits and taxa in the genetic architecture of sexual dimorphism, and it is likely that novel mechanisms await discovery.

In the following sections, I examine two other forms of sex-limited epistasis that may contribute to the reduction of genomic conflict and the evolution of sexual dimorphism: genomic imprinting and condition-dependence of sexually selected traits. These genomic adaptations are expected to be particularly relevant for secondary sexual traits (which can include overall body size), rather than primary sexual traits, for two reasons. First, secondary sexual traits are thought to undergo rapid (co)evolutionary cycles (Gavrilets and Hayashi 2006), and hence may be of relatively recent origin and characterized by abundant genetic variation. As I explain below, sufficient genetic variation is a key precondition for the evolution of genomic imprinting via intralocus sexual conflict. Second, such traits are expected to evolve strongly condition-dependent expression (Rowe and Houle 1996).

17.2 Genomic imprinting

17.2.1 Intralocus sexual conflict and the evolution of offspring–parent resemblance

For traits under sex-specific selection, individuals benefit by minimizing resemblance to their opposite-sex parent because the opposite-sex parent is likely to transmit low-fitness genes for such traits (Day and Bonduriansky 2004). Intralocus sexual conflict may thus be unavoidable when traits inherited from both parents are under sex-specific selection. Since both sex-specific selection and intersexual inheritance characterize many autosomal and X-linked loci, intralocus sexual conflict is likely to beset much of the genome, and impose substantial fitness costs (Chapter 18).

To illustrate this, consider a hypothetical sexually selected male trait, elongated antennae used by male flies as a signal in courtship (see Bonduriansky and Rowe 2003 for an empirical example). To become a father, a male must normally succeed in sexual competition, and his performance will depend, in part, on the length of his antennae, so fathers will have longer antennae than the average for all males. In contrast, sexual selection does not act on antenna length in females, so mothers will not have longer antennae than females that fail to breed. Assuming that the phenotypic variation reflects additive genetic variation, fathers will be more likely to transmit genes for long antennae than mothers will. Consequently, on average, a male offspring will be more

likely to inherit a high male-fitness allele from its father than from its mother. This will be true even for a locus with male-limited expression, because females will shelter and pass on alleles that would have been eliminated by sexual selection had they been expressed in a male. Thus, for male secondary sexual traits, selection should favor mechanisms that diminish a male's resemblance to its mother, or, more formally, reduce the expression of maternally inherited alleles and weaken maternal heritability. For traits under sexually antagonistic selection, selection will also favor reduced expression of paternally inherited alleles in female offspring. But how could such inheritance patterns evolve?

Unequal expression of maternally and paternally inherited alleles, resulting in differential resemblance to the mother and father, can result from genomic imprinting. The DNA at an imprinted locus receives an epigenetic label (an imprint) in either eggs or sperm: in mammals, the imprint consists of cytosine methylation, but other taxa employ different molecular mechanisms (see Lloyd 2000). Imprints are retained in the zygote, and replicated through somatic cell divisions. They affect the rate of transcription, resulting in differential expression of alleles inherited from the mother and father and, often, the complete silencing of genes from one parent in some tissues. However, the imprint is removed in the germ line, and reapplied during gametogenesis in one sex. In a quantitative genetic analysis, genomic imprinting is manifested as unequal heritability through the mother and father (Spencer 2002). For example, silencing of maternally inherited alleles results in reduced maternal heritability for traits affected by the imprinted locus. Genomic imprinting has been identified in diverse organisms, including mammals (de Koning *et al*. 2000; Moore 2001; Suzuki *et al*. 2005), *Drosophila* (Lloyd *et al*. 1999; Lloyd 2000) and other insects (Herrick and Seger 1999), and plants (Alleman and Doctor 2000). Indeed, it appears that cytosine methylation is frequently applied to novel chromosomal regions as a means of silencing the promoters of selfish transposable elements (Bestor 2003), so it is not unreasonable to expect novel imprinted regions to evolve in response to intralocus sexual conflict.

A simple genetic model can illustrate selection on genomic imprinting under intralocus sexual conflict (Figure 17.1). Imagine a trait under sexual selection in males. The trait phenotype is determined by an autosomal locus with two alleles: p increases trait size and q decreases trait size. These alleles have additive phenotypic effects, so that heterozygous phenotypes are intermediate between those of the two homozygotes. Sexual selection favors large trait size in males so that, on average, pp males are most successful and qq males least successful. For simplicity, assume that females' probability of breeding is not strongly affected by this locus (corresponding to male-limited expression, or weak stabilizing selection in females). However, directional selection on the male trait is maintained through infusion of q alleles by mutation and gene flow. Now imagine a modifier locus where an 'imprinter' allele arises, silencing maternally inherited alleles at the trait locus (maternal silencer). The imprinter allele will alter the phenotypes of heterozygotes (denoted hereafter with the paternally inherited allele

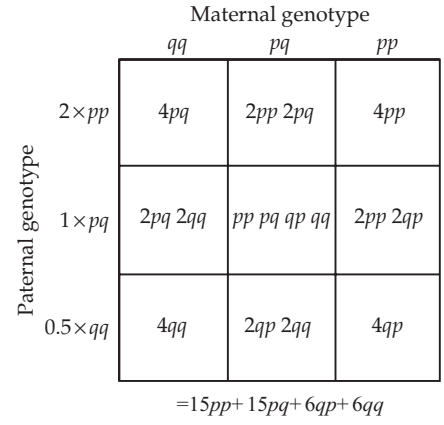

Figure 17.1 A genetic model illustrating selection on genomic imprinting (with maternal silencing) of a trait under sexual selection in males (see text). The relative fitness of each paternal genotype is given by the factors to the left (with fitness of pq and qp fathers averaged, for simplicity), whereas maternal fitness is assumed to be unaffected by this locus. Offspring genotypes are shown for each possible cross (for heterozygotes, the paternally inherited allele is shown first). The tally at the bottom shows that, because males are more likely to pass on p alleles to their offspring, pq heterozygotes (whose fitness is increased by imprinting) are more common than qp heterozygotes (whose fitness is reduced by imprinting).

indicated first): *pq* males will express the same phenotype as *pp* homozygotes, whereas *qp* males will express the same phenotype as *qq* homozygotes. Net positive selection on the imprinter allele occurs because sexual selection weeds out males carrying *q* alleles, insuring that *pq* genotypes are more common than *qp* genotypes in offspring (Figure 17.1). The imprinter allele should therefore increase in frequency, and simulations for this two-locus system show that this is indeed what happens, provided that genetic variation is maintained (Day and Bonduriansky 2004).

Similar logic applies under sexually antagonistic selection. If selection is stronger on males than on females (but genetic variation persists), then breeding males will be more likely to carry and transmit *p* alleles, whereas breeding females will tend to transmit *q* alleles. In this case, a maternal silencer will decrease the fitness of female offspring, but this cost will be more than offset by the benefit to male offspring. Thus, as shown in Figure 17.2, an imprinter allele that silences the maternally inherited trait allele will increase in frequency if selection acts more strongly on males (Day and Bonduriansky 2004).

In the above examples, as in well-studied imprinted genes like *igf2* and *igf2r* of humans (Moore 2001), alleles from parents of a particular sex are silenced in all offspring. However, under sexually antagonistic selection, this type of imprinting would be costly to one sex. Indeed, if sexually antagonistic selection is equally strong on both sexes (balanced), a situation envisioned by Lande (1980a) as an intermediate stage in the evolution of sexual dimorphism where intralocus sexual conflict is most intense, then the net cost of maternal silencing to females will completely negate the net benefit to males. In such cases, selection would favor a more complex form of imprinting where male offspring silence maternally inherited alleles and female offspring silence paternally inherited alleles (Day and Bonduriansky 2004). Such sexually dimorphic imprinting would benefit both sexes. Moreover, unlike conventional imprinting, sexually dimorphic imprinting would reduce the intersexual genetic correlation (Bonduriansky and Rowe 2005a).

Note that the strength of selection for genomic imprinting reflects the magnitude of additive genetic variation for the imprinted trait. This suggests that genomic imprinting may be especially likely to evolve in traits with recently acquired secondary sexual functions, where genetic variation is abundant. Imprinting may also evolve when gene flow between populations with differing optima for sexually selected traits prevents these populations from attaining their local optima, or when genetic variation is maintained by balanced sexually antagonistic selection.

The intralocus sexual-conflict model (Day and Bonduriansky 2004) provides a more general alternative to previous theories of the evolution of genomic imprinting under sexual conflict. The most widely accepted of these, the parental-conflict theory (Moore and Haig 1991; Moore 2001), proposes that genomic imprinting evolves because paternally inherited alleles in embryos are more selfish towards their mother than are maternally

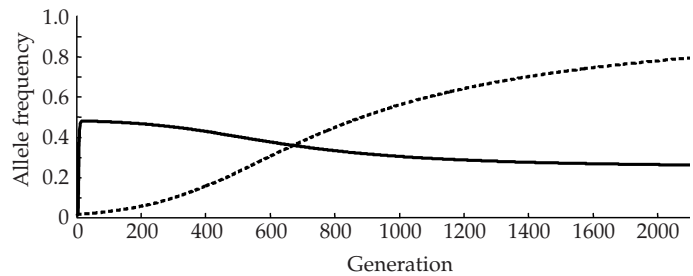

Figure 17.2 A simulation illustrating the evolution of genomic imprinting (with maternal silencing) for a secondary sexual trait subject to sexually antagonistic selection (see text). A male-benefit allele (*p* allele, solid line) that increases trait size occurs at a frequency of 0.01 at generation 0. An imprinter allele (dashed line) that silences maternally inherited alleles at the trait locus also has an initial frequency of 0.01. In each generation, 1% of *p* alleles are replaced by *q* alleles (which reduce trait size) as a result of mutation and gene flow, resulting in stronger selection on males than on females.

inherited alleles, and applies to traits involved in the extraction of maternal resources by offspring in species with polyandry and maternal provisioning after fertilization. The X-linked genomic imprinting theory (Iwasa and Pomiankowski 1999) shows that genomic imprinting can result in differential levels of expression of X-linked genes in males and females, so that imprinting of X-linked genes with sexually dimorphic expression optima is favored by selection. This theory is applicable to X-linked (or Z-linked) genes. In contrast, the intralocus sexual conflict model is potentially applicable to any trait under sex-specific selection in any species. Distinguishing between these theories, which predict imprinting in different sets of genes, is a problem of both theoretical and medical importance, given that genomic imprinting is directly implicated in the development of cancer and other disorders (Horsthemke 1997; Jirtle 1999).

17.2.2 Sexual dimorphism via genomic imprinting

Curiously, in addition to reducing the intersexual genetic correlation and mitigating intralocus sexual conflict, the sexually dimorphic form of genomic imprinting produces phenotypic sexual dimorphism in the imprinted trait when that trait is subject to sexually antagonistic selection (Day and Bonduriansky 2004). This phenotypic effect simply reflects unequal probabilities of inheriting sexually antagonistic alleles from the mother and father. In the sexually antagonistic selection example outlined above, fathers disproportionately transmit p alleles to their offspring, whereas mothers disproportionately transmit q alleles. Because, under sexually dimorphic imprinting, individuals only express alleles inherited from the same-sex parent, males are more likely than females to express p alleles, which produce a greater mean trait size (Figure 17.3). Thus, in theory, sexual dimorphism can result from a combination of sexually antagonistic selection and sexually dimorphic genomic imprinting.

17.2.3 Empirical evidence

The intralocus sexual-conflict model (Day and Bonduriansky 2004) predicts a role for genomic imprinting in the genetic architecture of sexually

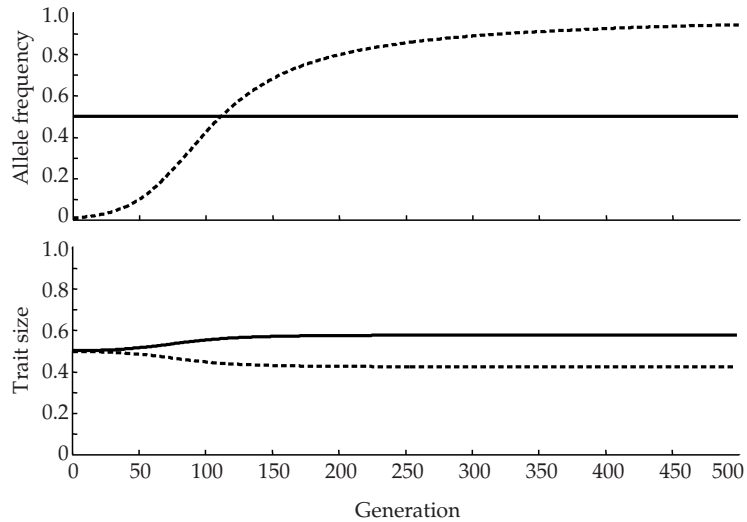

Figure 17.3 A simulation illustrating the evolution of a sexually dimorphic imprinter allele that causes silencing of maternally inherited alleles in males and paternally inherited alleles in females (top panel), and resulting evolution of phenotypic sexual dimorphism in the imprinted trait (bottom panel). The imprinter allele (top panel, dashed line) occurs at a frequency of 0.01 at generation 0. The imprinter allele modifies the expression of a secondary sexual trait under balanced sexually antagonistic selection. The male-benefit p allele (top panel, solid line) is present at a frequency of 0.5 in generation 0, and there is no mutation or gene flow. The bottom plot shows the evolution of the secondary sexual trait phenotype, assuming that a p allele contributes a phenotypic value of 1, and a q allele contributes a phenotypic value of 0. As the imprinter allele increases in frequency, the mean trait sizes of males (bottom panel, solid line) and females (bottom panel, dashed line) diverge.

dimorphic traits. Bonduriansky and Rowe (2005a) tested this prediction by comparing maternal and paternal heritabilities of sexually dimorphic body-shape components in the piophilid fly *Prochyliza xanthostoma*. As predicted, sexual traits tended to exhibit stronger heritabilities through the father than through the mother, whereas non-sexual traits tended to exhibit more similar heritabilities through both parents. Notably, the most strongly dimorphic sexual trait was heritable only through the same-sex parent, a pattern consistent with sexually dimorphic imprinting. Similar patterns have been reported previously (see Bonduriansky and Rowe 2005a). However, directional differences between maternal and paternal heritabilities do not provide unequivocal evidence of genomic imprinting. Further studies, using quantitative genetic and molecular techniques, are needed to test for genomic imprinting of autosomal loci affecting sexually selected traits. It may also be possible to use artificial sexually antagonistic selection as a direct test of the theory.

17.3 Condition-dependence

17.3.1 Coevolution of condition-dependence and sexual dimorphism

In many species, striking variation among males in the degree of 'exaggeration' of their secondary sexual traits appears to reflect variation in individual condition (see Cotton *et al*. 2004b). This suggests a conceptual link between condition-dependence and sexual dimorphism: if sexual dimorphism reflects mean trait exaggeration in males, relative to females, then sexual dimorphism is a function of condition-dependence. Yet, the association between condition-dependence and sexual dimorphism has received little attention. In this section, I argue that condition-dependence may play a key role in the evolution and genetic architecture of sexual dimorphism.

According to theory, condition-dependence evolves because it allows individuals to optimize the trade-off between viability and secondary sexual trait expression (see Nur and Hasson 1984; Rowe and Houle 1996). Condition reflects the quantity of metabolic resources available to an individual and the efficiency with which it can convert those resources into fitness. Thus, condition depends on both the quality of genes affecting resource acquisition and utilization, and the availability of resources in the ambient environment. Genetic variation in condition is difficult to quantify, and remains poorly understood (Hunt *et al*. 2004). In contrast, numerous experimental manipulations have confirmed the importance of environmental effects on condition, showing that individuals exposed to more abundant dietary resources are more vigorous and typically exhibit more exaggerated secondary sexual traits (Emlen 1997; Cotton *et al*. 2004a; Bonduriansky and Rowe 2005b; Bonduriansky 2007). The magnitude of such environmental treatment effects reflects the strength of condition-dependence.

Life-history theory predicts that condition-dependence and sexual dimorphism will coevolve (Bonduriansky and Rowe 2005b; Bonduriansky 2007). Because sexual selection displaces male traits from their viability-selected optima, the degree of trait exaggeration in males, relative to the viability-selected optimum approximated by the female phenotype, should reflect both the degree of sexual dimorphism and the viability costs of trait expression for males. These viability costs, in turn, favor the evolution of condition-dependence. In other words, all else being equal, the (mean) degree of trait exaggeration in males should also reflect the strength of condition-dependence that is favored by selection.

The coevolution of condition-dependence and sexual dimorphism may result in a common genetic and developmental basis (i.e. a positive genetic correlation) for these traits. Empirically, it also predicts phenotypic covariation among traits between condition-dependence and sexual dimorphism. If an organism possesses a suite of traits targeted to varying degrees by sexual selection, then the more dimorphic traits, which are targeted most directly by sexual selection, should also exhibit stronger condition-dependence. As predicted, the magnitude of condition-dependence of body-shape components is positively correlated with the degree of sexual dimorphism in the piophilid fly *P. xanthostoma* (Bonduriansky and Rowe 2005b) and the neriid fly *Telostylinus angusticollis* (Bonduriansky 2007).

These studies support the prediction that condition-dependence coevolves with sexual dimorphism, although tests using other taxa and other types of secondary sexual traits (e.g. genitalic traits, which tend to exhibit low phenotypic variances) are needed to assess the generality of this pattern. The prediction can also be tested interspecifically: species with greater dimorphism in the size of a trait (or overall body size) should exhibit stronger condition-dependence.

17.3.2 Intralocus sexual conflict and genic capture

Rowe and Houle (1996) hypothesized that directional sexual selection on a trait drives the evolution of condition-dependence via "genic capture", a form of epistasis linking the expression of the targeted trait with variation at numerous loci affecting the efficiency of resource acquisition and allocation. Their model is based on the implicit assumption that genic capture can evolve in a sex-limited manner (i.e. in males only, without a correlated evolutionary response in females). This assumption reflects the idea that sexual selection favors the evolution of condition-dependent expression in male secondary sexual traits, but not in the homologous traits of females (Figure 17.4).

If genic capture represents a form of sex-limited epistasis, as assumed by Rowe and Houle (1996), then the evolution of condition-dependence in secondary sexual male traits may reduce intersexual genetic correlations and facilitate the (co)evolution of sexual dimorphism in those traits (see Section 17.1.3). The capture of variation at numerous loci affecting resource acquisition and allocation efficiency by male secondary sexual traits can result in dramatic phenotypic differences between females and high-condition males. However, because the modifying effects of these genes (loci L_a–L_e in Figure 17.4) are assumed to be male-limited, they are expected to contribute little to intralocus sexual conflict. Nonetheless, even under genic capture, it is likely that some non-condition-dependent genes (such as L_f in Figure 17.4) will still affect trait expression in both sexes, exacerbating intralocus sexual conflict. Interestingly, covariation among traits between the strength of condition-dependence and the degree of sexual dimorphism (see Section 17.3.1) suggests that traits can evolve varying degrees of genic capture, corresponding to the intensity of intralocus sexual conflict.

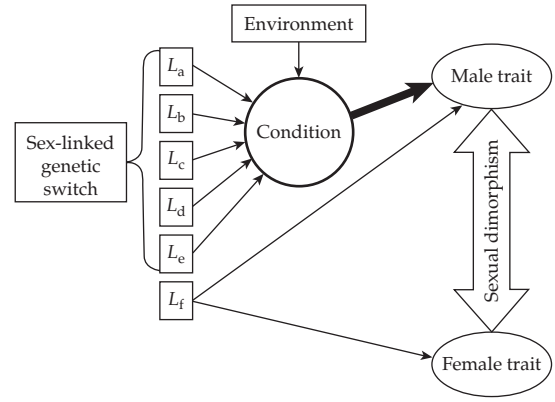

Figure 17.4 The genic-capture model postulates a form of sex-limited epistasis that has the potential to mitigate intralocus sexual conflict and facilitate the evolution of sexual dimorphism. Ancestrally, trait expression is affected by the same locus in both sexes (locus L_f) , and is independent of condition. Once the trait assumes a function in male sexual competition, the genic-capture model predicts that male trait expression will come to reflect variation at numerous loci affecting the efficiency of resource acquisition and allocation (loci L_a–L_e) and, thus, the genetic component of condition. Male trait expression will also reflect the availability of resources in the ambient environment. In contrast, it is assumed that female trait expression will remain independent of condition, and unaffected by genic capture. The sex-limited nature of genic capture requires some form of sex-linked genetic switch to activate this epistatic architecture in males.

However, if the evolution of condition-dependence in male traits results in correlated responses in females, then condition-dependence might exacerbate intralocus sexual conflict by imposing two distinct costs on females: over-allocation of resources to the homologues of male secondary sexual traits, and over-expression of those traits relative to the female optimum. Whether the evolution of condition-dependence is likely to mitigate or exacerbate intralocus sexual conflict thus depends on the intersexual genetic correlation for condition-dependence. The magnitude of this genetic correlation may reflect the degree of sex-limitation of the genetic switch that "activates" genic capture (see Figure 17.4). The assumption of sex limitation presupposes a genetic correlation of zero. However, an analysis of reaction norms for sexually dimorphic body shape components in the fly *P. xanthostoma*

suggested otherwise: although male traits were more strongly condition-dependent than homologous female traits, a positive correlation between the sexes for condition-dependence strength was observed (Bonduriansky and Rowe 2005b).

17.3.3 The genetic architecture of condition-dependent sexual dimorphism

The theory and empirical observations outlined above suggest that condition-dependence and sexual dimorphism may have a common genetic and developmental basis and, thus, may be regarded as distinct pleiotropic consequences of the same genes. These findings point to a need to integrate genetic and evolutionary models of condition-dependence and sexual dimorphism. Although considerable thought has been given to the genetic architecture of sexual dimorphism (see Section 17.1.3), current models fail to account for the condition-dependent expression of sexually dimorphic secondary sexual traits. Conversely, theory on the genetic architecture of condition-dependence (Rowe and Houle 1996; Tomkins *et al*. 2004) fails to address its sex-specific nature, and the assumption that genic capture can evolve in a sex-limited manner has received little theoretical or empirical examination. This gap calls for models that can account for the wholly or partially sex-limited effects of resource acquisition and allocation genes and environmental factors on the expression of secondary sexual traits, as well as variation among traits in the strength of condition-dependence (see Bonduriansky and Rowe 2005b; Bonduriansky 2007).

17.4 Summary

In Section 17.2, I outline new theory showing that genomic imprinting may play a role in the genetic architecture of sexually dimorphic traits, and that it can contribute to the expression of a sexually dimorphic phenotype (Day and Bonduriansky 2004). Because sex-specific selection results in an elevated risk of inheriting low-fitness alleles from the opposite-sex parent in one or both sexes, an imprinter gene that causes the silencing of those alleles will be favored by selection. In traits under sexual selection in males, the silencing of maternally inherited alleles is predicted. However, under sexually antagonistic selection, greater fitness advantage results from a hypothetical form of imprinting whereby males silence maternally inherited alleles and females silence paternally inherited alleles. Interestingly, this form of imprinting, in conjunction with sexually antagonistic selection, is sufficient to produce a sexually dimorphic trait phenotype. The theory is tentatively supported by empirical evidence (Bonduriansky and Rowe 2005a).

In Section 17.3, I consider the potential role of condition-dependence in the evolution and genetic architecture of sexual dimorphism. Condition-dependent expression of secondary sexual traits allows individual males to optimize the trade-off between viability and reproductive rate. Life-history theory predicts that sexual dimorphism and condition-dependence will coevolve because the degree of exaggeration of male traits by sexual selection (i.e. the magnitude of sexual dimorphism) reflects the viability costs of trait expression and, therefore, the benefits of condition-dependence. This prediction is supported by positive covariation of sexual dimorphism and condition-dependence among morphological traits (Bonduriansky and Rowe 2005b; Bonduriansky 2007). Besides the hypothesized fitness benefits to males, however, I argue that condition-dependence may also contribute to the reduction of intersexual genetic correlations, thus benefiting both sexes by mitigating intralocus sexual conflict and facilitating the evolution of sexual dimorphism.

17.5 Suggested readings

Bonduriansky, R. (2007) The evolution of condition dependent sexual dimorphism. *American Naturalist* **169**, 9–19.

Bonduriansky, R. and Rowe, L. (2005) Intralocus sexual conflict and the genetic architecture of sexually dimorphic traits in *Prochyliza xanthostoma* (Diptera: Piophilidae). *Evolution* **59**, 1965–1975.

Bonduriansky, R. and Rowe, L. (2005) Sexual selection, genetic architecture, and the condition dependence of body shape in the sexually dimorphic fly, *Prochyliza xanthostoma* (Diptera: Piophilidae). *Evolution* **59**, 138–151.

Day, T. and Bonduriansky, R. (2004) Intralocus sexual conflict can drive the evolution of genomic imprinting. *Genetics* **167**, 1537–1546.