Published in final edited form as:

Annu Rev Entomol. 2010 ; 55: 227–245. doi:10.1146/annurev-ento-112408-085500.

Sex Differences in Phenotypic Plasticity Affect Variation in Sexual Size Dimorphism in Insects: From Physiology to Evolution

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Abstract

Males and females of nearly all animals differ in their body size, a phenomenon called sexual size dimorphism (SSD). The degree and direction of SSD vary considerably among taxa, including among populations within species. A considerable amount of this variation is due to sex differences in body size plasticity. We examine how variation in these sex differences is generated by exploring sex differences in plasticity in growth rate and development time and the physiological regulation of these differences (e.g., sex differences in regulation by the endocrine system). We explore adaptive hypotheses proposed to explain sex differences in plasticity, including those that predict that plasticity will be lowest for traits under strong selection (adaptive canalization) or greatest for traits under strong directional selection (condition dependence), but few studies have tested these hypotheses. Studies that combine proximate and ultimate mechanisms offer great promise for understanding variation in SSD and sex differences in body size plasticity in insects.

Keywords

sexual dimorphism; body size; sexual selection; development; Rensch's rule

Introduction

Sexual size dimorphism (SSD), a difference in body size between males and females, is a widespread phenomenon in plants and animals (50, 52). Both the direction and magnitude of SSD vary considerably among species and among populations within species (20, 21). Much

Disclosure Statement: The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

of this variation is genetically based and likely due to variation in selection, primarily sexual selection, among species/populations (50, 51). However, recent studies have shown that a considerable amount of intraspecific variation in SSD may be due to differences in phenotypic plasticity between males and females (51, 114, 115, 126). In this review, we examine how sex differences in phenotypic plasticity affect patterns of SSD in insects. We explore recent advancements in the development and physiology underlying sex differences in plasticity, the sources of selection producing these sex differences, and the consequences of sex differences in plasticity for patterns of SSD observed in nature.

The evolution of SSD likely arises from counteracting sources of selection acting in concert, creating stabilizing selection on body size that differs between the sexes (5, 14–16, 52, 96, 106). Fecundity selection on females and sexual selection on males are likely the major sources of selection favoring larger size in insects. Larger males often have increased mating success due to male-male competition or female choice (3) and may increase female fecundity via larger nuptial gifts (61). Larger females generally have greater fecundity and often produce larger offspring (32, 60, 72, 95). These forms of selection favoring large body size are counteracted by a variety of sources of selection favoring small size (14). For example, reaching a large body size often requires longer development, which can extend the period during which individuals are exposed to sources of mortality (11, 100). Alternatively, individuals can grow larger by increasing growth rate, but this carries similar costs; faster growth is energetically costly and increases the risk of starvation and predation (66, 68). Variation in the degree of balance between these sources of selection favoring large versus small size likely explains much of the variation in SSD among taxa and among environments.

The evolution of SSD is slowed by genetic (97), phylogenetic (26), developmental (8), and/or physiological (93) constraints. For example, empirical studies have shown that heritabilities are similar between the sexes and that the between-sex genetic correlations (r_G) are near 1.0 (because males and females share the same genes that control growth and development) (79, 98, 102). The evolution of SSD should thus be constrained even under strong selection. However, sex-biased gene expression can overcome genetic constraints (8). Slow evolutionary divergence of the sexes means that substantial time is required to evolve sex differences that are different from that of a species' ancestor, even in the presence of strong directional selection, creating phylogenetically constrained patterns of SSD (49). In addition, energetic or mechanical constraints can set limitations on morphology and behavior (14, 99, 107), preventing populations from achieving the pattern of dimorphism (i.e., male-biased versus female-biased SSD) predicted from measurements of selection.

Little is known about the proximate mechanisms that generate SSD, particularly in invertebrates (8). The variation in SSD that occurs at the adult stage requires the sexes to differ in (*a*) their size at hatching, (*b*) their rate of growth, (*c*) the duration of their growth period (8, 47), and/or (*d*) size-dependent survival (114). Few studies have examined sex differences in size at hatching but, of those that have, none has found any difference in hatching size between the sexes in insects (47 and references therein). In many insects, SSD is produced by sex differences in growth rate (19), duration of the growth period (19, 46, 47, 121, 123), or through a combination of both (24, 44). In ectotherms, growth and

development are strongly dependent on environmental variables such as diet quantity/quality or temperature (4, 6, 7, 12, 34, 35, 117), inducing considerable phenotypic plasticity (a change in an organism's phenotype in response to a change in the environment) in body size. Recent studies have shown that plasticity in body size can differ quite markedly between sexes, generating intraspecific variation in SSD (e.g., among populations inhabiting different environments; 114, 126). Thus, understanding the evolution of sex differences in phenotypic plasticity in growth and development is critical to interpreting observed variation in SSD and the evolution of this variation (51, 126). Studies that combine underlying proximate mechanisms of the developmental processes producing adult SSD with ultimate patterns are lacking, but are essential to understanding the evolution of SSD (8).

Here we review the diversity of patterns of SSD observed in animals, with a focus on insects. We then examine the role of phenotypic plasticity in generating variation in SSD within and among species. Finally, we focus on the proximate and ultimate mechanisms that generate sex differences in phenotypic plasticity and how they affect the evolution of SSD.

Variation in Sexual Dimorphism in Body Size

Variation Among Taxa

SSD varies substantially among the higherorder taxa. Mammals and birds often exhibit considerable male-biased SSD (80, 119), whereas invertebrates and many poikilothermic vertebrates primarily show female-biased SSD (20, 28, 47, 78, 126). A vast majority of insect species exhibit female-biased SSD: 72–95% of species within each order exhibit female-biased SSD (Table 1). An exception are the Odonata, of which only 27% of species show femalebiased SSD, with most species, primarily damselflies (Zygoptera), exhibiting male-biased or no SSD (Table 1). Fecundity selection is believed to be driving the female-biased SSD that is found in most insect orders (72). In contrast, sexual selection mediated by territoriality favors large males in many odonates (108). However, most studies on SSD emphasize studying sexual selection, which generally favors large size; few studies examine the sources of selection that may favor small size (but see References 29, 75, 83, and 84). This limits our current understanding of the diversity of SSD found among higher-order taxa.

SSD also varies considerably among insect species within higher-order taxa. Rensch's rule (2, 19, 31, 49, 50, 55, 118, 134), the widely observed pattern in animals that male body size varies more among related species (and thus presumably evolves faster) than does female body size, is supported for some insect groups but not others (20, 50, 71). It is unclear why this occurs, but sexual selection—selection for large male body size via contest competition or female choice versus selection for small body size for agility—explains considerable variation in Rensch's rule in flying organisms (birds and odonates; 108, 119). Other than these types of comparative studies, detailed case studies of closely related species, especially of species that do not fit the standard pattern of SSD of their particular taxonomic group, are needed to provide insights into how variation in SSD evolves among closely related species.

Variation Among Populations Within Species

The degree of SSD, and sometimes the direction, varies among populations within species (20, 21, 116). Such variation could be genetically based, i.e., due to natural selection or random genetic drift. However, much of this variation observed in field studies may be caused by phenotypic plasticity (51). For example, several studies have found latitudinal and/or altitudinal clines in SSD (18, 21, 116); interestingly, in the majority of animal taxa male size varies more with latitude than does female size (21). However, most biogeographic studies of body size and SSD are based on field samples. As a result, it is often not possible to distinguish between phenotypic plasticity in response to ecological and environmental variables from evolutionary responses to climatic or other variables that covary with latitude or altitude. Unfortunately, common garden studies needed to disentangle the effects of genetic differentiation from phenotypic plasticity in geographic patterns of SSD are lacking. Sex differences in body size plasticity have only recently been recognized as an important contributor to variation in SSD (51, 114, 115, 126), but sufficient data now exist to warrant a detailed review examining the importance of these sex differences in plasticity as a contributor to variation in SSD.

Phenotypic Plasticity in Body Size in Insects

Phenotypic plasticity is a nearly universal characteristic of all organisms (17, 91, 117, 127, 135); almost all behavioral, physiological, morphological, and life-history traits of animals are affected by the environmental conditions in which those traits are expressed (17). Although plasticity is often nonadaptive (38, 65, 133), such as when food limitation impedes development, much of the plasticity induced during the immature stage in growth traits (body size, growth rate, and development time) is probably adaptive (91), e.g., the specific response by which growth is affected by food limitation is molded by selection.

Phenotypic plasticity in body size is induced by a number of ecological and environmental variables (17, 117, 127), of which diet quality and developmental temperature are arguably the two most important (34, 117). In general, ectothermic animals (including insects) mature at a larger size when raised at lower temperatures, a phenomenon known as the temperature-size rule (6, 76). Ectotherms raised on lower-quality diets generally mature at a smaller size (12, 34). In addition, the length of the growing season, predators, humidity, and photoperiod can induce considerable plasticity in size (17, 91). Most plasticity research focuses on examining how these environmental variables affect body size and other traits in isolation of each other. Yet, recent studies have shown that environmental variables can interact during development to produce complex patterns in plasticity (117, 138). More important for this review is that the sexes commonly respond differently to environmental variation, and that interactions between environmental variables have different effects on the sexes, affecting patterns of SSD (37).

Sex Differences in Phenotypic Plasticity and Their Consequences for Variation in SSD

Few studies have examined sex-specific plasticity in size in the context of the evolution of SSD, although many studies of plasticity present data on both males and females. Teder & Tammaru (126) surveyed the literature for such studies. We updated their dataset with

additional studies to examine how sex differences in plasticity in response to a variety of ecological and environmental variables affect patterns of SSD in insects. In nearly all studies, individuals were exposed to environmental treatments throughout all or nearly all stages of juvenile development (only a few studies manipulated treatments for shorter time periods, such as for pathogen infection, which occurs only in certain larval stages). Body size was assessed in pupae or newly emerged adults. Because the direction and even the degree of SSD can depend on which measure of body size is used (see sidebar, Measuring Body Size and Sexual Size Dimorphism), we separated data on sex-specific plasticity in body mass from sex-specific plasticity in morphological measures of size (e.g., body length).

In general, there are considerable sex differences in body size plasticity. Females are more often the most plastic sex ($\beta < 1$ for ~62% of species, pooling all environmental variables; $\chi^2 = 10.91$, $P < 0.01$; Table 2) and mean plasticity is greater in females than in males, when body mass is the measure of size ($t = 3.32$, $P < 0.01$; Table 2). In contrast, there are no general sex differences in plasticity when other measures of size are used (Table 2). However, the general patterns appear to vary with the source of environmental variation. When partitioning the data on plasticity in body mass according to the type of environmental manipulation, plasticity differed between the sexes only when density, competition, diet quantity, or diet quality was manipulated. This suggests that diet (quantity and quality) is likely more significant for producing sex-specific plasticity in nature, but the number of studies manipulating other environmental variables is too small to generalize.

Aside from these general patterns observed in our meta-analysis, the three most significant observations are that (*a*) females commonly respond to environmental variability differently than males do; (*b*) the degree to which females respond differently than males varies substantially among species, and sometimes even among populations, in both magnitude and direction; and (*c*) this sex difference in plasticity varies not only among environmental conditions but also along the range of specific environmental variables. These last two points are illustrated by studies with seed beetles. Stillwell & Fox (114) (Figure 1a) examined sex differences in plasticity over a range of temperatures in the seed-feeding beetle, *Callosobruchus maculatus*. Males showed much greater plasticity in body size than females when reared at temperatures ranging between 20 and 25°C, whereas females showed greater plasticity than males when reared at temperatures between 30 and 35°C (Figure 1a). In a related seedfeeding beetle (*Stator limbatus*), whether female body size is more or less plastic than the male's depends on the study population examined (115). Complexity of results, such as those found for seed beetles, may be the norm in nature.

This variation in sex-specific plasticity along the range of temperatures experienced by beetles, and among temperatures, generates complex body size and SSD patterns across time in response to seasonality and across space due to climatic variation and population differentiation. For example, the variance in plasticity along the range of temperatures experienced by *C. maculatus* has large effects on patterns of SSD (Figure 1b), so that populations experiencing different temperatures in nature will exhibit large differences in SSD even if there is no genetic differentiation in size among populations. Such sex differences in plasticity probably play a role in generating the geographic variation in SSD

observed for many animals (21). Using a common-garden experiment, Fairbairn (51) tested whether geographic variation in SSD of the water strider *Aquarius remigis* was due to genetic differences among populations or due to a sex difference in phenotypic plasticity of body size. Most of the geographic variation in SSD was produced by sex differences in plasticity (51). We suspect that this result—that plasticity explains much of the interpopulation variation in SSD observed in nature—will be common.

The implications of large sex differences in plasticity are not limited to understanding variation in SSD within species; they could explain variation in SSD among species. This is because strong environmental effects on body size and SSD mean that no species will have one single characteristic estimate of SSD. Also, because congeneric species are often allopatric, and thus encounter different environmental conditions, differences in plasticity almost always are confounded by differences in environmental experiences. Even when sympatric, related species often differ in diet or other aspects of their niche, which can affect males and females differently, generating differences in SSD. Because SSD varies over space and time within species, SSD estimates used in comparative studies may not be representative of the genetic difference in size between sexes (126), particularly if only a few individuals or one population of a species is used. Species-level estimates must therefore consider variation inbody size across time and space.

Environmental effects on SSD can have profound implications for studies that examine evolutionary patterns of dimorphism. For example, Rensch's rule is often examined by plotting male size on female size using reduced major axis (RMA) regression; Rensch's rule is supported if the slope is >1 but not if the slope is <1 (50, 55). The assumption underlying such analyses is that variation in these slopes reflects genetic differentiation among populations: Either males or females are evolving more quickly. However, as discussed above, these slopes can be environment dependent (Figure 1a). Environmental effects are not as likely to affect comparisons among species collected within common environments, but for species compared across geographic areas or different seasons, or feeding on different diets, sex differences in plasticity can affect RMA slopes and thus tests of Rensch's rule.

In brief, sex differences in plasticity are common and likely to have major effects on observed patterns of SSD in nature. Despite this, we have a poor understanding of the degree to which the sexes differ in plasticity, the physiological basis for these differences in plasticity, and the sex differences in selection that produce this variation.

Development and Physiology of Sex Differences in Phenotypic Plasticity

Few studies have examined the proximate mechanisms that create SSD, much less the proximate causes of sex differences in plasticity (8, 114). Although the proximate mechanisms that produce adult SSD in vertebrates are beginning to be understood (8), little is known about the mechanisms that produce SSD or sex differences in body size plasticity in invertebrates. However, knowing the proximate mechanisms that produce SSD is important in addressing ultimate patterns of SSD: Analyses of proximate mechanisms can reveal developmental constraints on juvenile development patterns (123). Understanding the

proximate basis of SSD may thus help to identify the most likely directions of evolutionary change.

Developmental mechanisms—Like SSD, sex differences in body size plasticity must be achieved through a sex difference in hatching size, growth rate, and/or the duration of the growth period. However, the degree to which sex differences in plasticity of development time and growth rate contribute to sex differences in plasticity of body size varies among species. For example, Stillwell & Fox (114) showed that a temperature-induced sex difference in plasticity of body mass of *C. maculatus* is produced by a sex difference in plasticity of growth rate and not development time. However, in the butterfly *Lycaena tityrus*, a combination of temperature-induced sex differences in development time, growth rate, and pupal mass loss results in sex differences in body mass (58). Likewise, in the yellow dung fly, *Scathophaga stercoraria*, adult body size plasticity differences are generated by a combination of growth and development time plasticity (128). A sex difference in plasticity of development time appears to explain sex differences in body mass plasticity in *Lycaena hippothoe* (59). These inconsistent results indicate that many further studies are needed to determine if sex differences in development time plasticity and/or growth rate plasticity are more important in producing sex-specific plasticity in body size.

One unique way of prolonging development in one sex and achieving large size is to add one or more supernumerary juvenile instars (1, 45–47). Esperk et al. (47) showed that sex differences in the number of instars between males and females are common in insects, and that those species with a sex difference in in-star number exhibit a higher than average level of female-biased SSD. Furthermore, larval instar number can also vary within the sexes, such that plasticity in the number of instars could produce sex differences in plasticity in body size (47, 64). For example, in the grasshopper *Chorthippus brunneus*, females tend to produce a supernumerary instar when raised on a highquality diet and when raised at high temperature, allowing them to be considerably larger than males (70, 139). However, female-biased SSD and sex differences in body size plasticity occur in many species that have a fixed instar number and the same number of instars in both sexes (e.g., many Lepidoptera; 123). Supernumerary instars are thus not necessary to generate SSD in insects.

Because most research on SSD focuses on adult size, little is known about when the sexes start to diverge in body size during development (123). In vertebrates, males and females are often identical in size during early development, but undergo rapid developmental changes later in life to become sexually dimorphic as adults (8). In invertebrates, there is some evidence that SSD does not occur until later in development (46–48, 53, 74, 108, 141), whereas other studies show SSD is present early in development (1, 10, 120, 123). Although sex differences in size plasticity can be present during the late larval stage, it can be a poor predictor of adult sexspecific plasticity (74). Previous studies on the ontogeny of SSD are limited because most examined only one or a few life stages. Studies that examine the ontogeny of SSD from hatching to adult emergence are sorely needed.

Sex differences in body size and body size plasticity could develop through physiological and/or behavioral differences between males and females. For instance, growth rate differences could result if the sexes differ in rates of food consumption, efficiency of

conversion of food into larval mass, assimilation rates (digestibility), or metabolic rates. Recent studies have shown that female butterflies and beetles achieve larger size because they consume more food per day than males do and because females have a higher conversion efficiency of food into body mass (74, 141). In *L. tityrus*, females are larger than males and this degree of SSD is greatest at low temperature (74). However, larval mass differed little at low temperature, whereas males were larger than females at high temperature (74). The adult sexspecific plasticity was generated because males appear to have lost a greater amount of mass compared with females during metamorphosis at low temperature, whereas the mass loss difference between sexes was much smaller at high temperature. Thus, the difference in plasticity between sexes in this species appears to be due simply to a sex difference in plasticity in mass loss during metamorphosis. We need more such studies that measure a multitude of behavioral and physiological variables to understand what is generating sex differences in plasticity, particularly studies that manipulate diet quantity/quality because most sex-specific plasticity in size is generated by variation in diet.

Physiological mechanisms—Variation in growth and development, and thus the degree of adult SSD, is likely regulated by hormonal differences between males and females. In insects, the evolution of body size and polyphenic development (i.e., discrete alternative morphologies such as in queen and worker ants) are under strong hormonal control (30, 86–88, 136, 137). In particular, hormones play an essential role in regulating body size plasticity (34, 113). Four hormones are primarily responsible for controlling growth and development in insects: Insulin, juvenile hormone (JH), prothoracicotropic hormone (PTTH), and ecdysone (30, 33–35, 39, 41, 87, 88, 110). Recent progress has shown how three of these hormones (see below) regulate body size in the tobacco hornworm, *Manduca sexta*, a model system for studying insect physiology (30, 33–35, 87, 88). These same hormones also regulate development time in insects (35, 36).

As in other insects, adult *M. sexta* do not grow. The size that a larva attains at the time of metamorphosis almost completely defines the body size of the adult insect (34). In *M. sexta*, 90% of the increase in mass occurs during this last instar, a period during development in which there is a close causal association between somatic growth and the timing of endocrine events that induce the onset of metamorphosis (89, 90). Secretion of PTTH and ecdysteroids are inhibited by the presence of JH (89, 103). The circulating level of JH is high during the first few days of the instar but drops sharply when the larva reaches a specific critical weight (89). Attainment of a critical weight causes the corpora allata (CA), the glands that synthesize and secrete JH, to switch off. About a day later, JH esterase accumulates in the hemolymph and enhances the rate of JH degradation (25). A few days after passing the critical weight, JH is fully cleared from the hemolymph and secretion of PTTH and ecdysteroids is disinhibited (89, 103). Secretion of PTTH occurs during the first photoperiodic gate (a well-studied window of time that recurs during the same time each day) that follows after the clearance of JH (129, 130). The time period between when the critical weight is reached and when PTTH and ecdysteroids are secreted is known as the interval to cessation of growth (ICG) (35, 88). The release of PTTH triggers the secretion of ecdysteroids, which causes the larvae to stop feeding and induces the commitment to pupate

and, a few days later, the metamorphic molt (86, 101). The final size of the larva is thus determined by how the critical weight and the ICG interact with the growth rate of the larva (33–36, 87, 88).

The interaction among three physiological factors—growth rate (which includes initial larval size), the critical weight, and the ICG (which includes the photoperiodic gate for PTTH release)—thus explains 95% of the variation in body size and development time in *M. sexta* as well as plasticity in body size and development time. Growth rate exhibits plasticity in response to both rearing diet and rearing temperature, but the ICG exhibits plasticity only in response to temperature, whereas the critical weight exhibits plasticity only in response to diet (34, 35). Because these three physiological factors explain nearly all variation in body size plasticity, it is likely that they play a major role in generating sex-specific reaction norms in insects. However, it is currently not known whether males and females differ in their critical weight and ICG, or whether the response of these variables to environmental variation differs between sexes.

In the forest tent caterpillar, *Malacosoma disstria*, a female-biased SSD occurs because females have a higher threshold size—which is different from, but nonetheless a good predictor of the critical weight—than males (48). Because critical weight is directly linked to JH production, the sexes should differ in their JH titers. Although no study has measured all three physiological variables that explain most of the variation in body size in the context of SSD, prior work does indicate that JH production may differ between sexes in *M. sexta*. Bhaskaran et al. (13) demonstrated that in the last larval instar JH production stops in males, but not in females. In contrast, Baker et al. (9) detected no sex differences in the degree or timing of JH, JH esterase, and ecysteroid titers in *M. sexta*. However, they did note there is a prepupal burst of JH in which females have considerably higher titers of JH than males, and that ecdysteroid titers of females did not decline completely following its peak in the fifth instar. In addition, medium to high levels of the JH analog methoprene applied to the final instar of stalk-eyed flies results in a reduction or even a complete switch in the direction of SSD (from male-biased SSD to female-biased SSD) (63). Furthermore, several studies have shown that JH is important in creating dimorphism in beetle horns (42, 109). The results of these studies suggest that JH may play an important role in producing adult SSD, but the inconsistent results among studies indicate that much more work is needed before we will have an understanding of how and even if JH plays a role in producing SSD.

Another important insect hormone that controls body size and which could affect SSD is insulin (39, 41, 110). The insulin pathway controls growth, and thus body size, by sensing nutritional conditions and regulating the growth of organs by controlling cell proliferation and protein synthesis (41, 110). JH and ecdysone also interact with the insulin signaling pathway; for example, suppressing the insulin signaling pathway results in reduced ecdysone levels, which produces an increase in adult body size (41). In addition, the insulin signaling pathway is involved in the production of dimorphism in beetle horns (43). Also, the insulin signaling pathway is sensitive to variation in nutrition (43, 110), making it a likely candidate for controlling sex-specific plasticity of body size (as most sex-specific plasticity is due to variation in nutrition) (Table 2). To our knowledge, no study has investigated the potential role of the insulin signaling pathway or its interaction with JH and

ecdysone in producing adult SSD and sex differences in plasticity of size, although such investigations seem warranted given the overwhelming evidence of this pathway's involvement in controlling insect body size.

Ultimately, variation in SSD and sex-specific body size plasticity must be controlled by the genetic architecture underlying body size. Because most genes that control growth and development are nearly identical in both sexes, how the sexes achieve such variation in SSD is puzzling (8). One obvious way the sexes can overcome this constraint is through sex-biased gene expression, which could play a role in the evolution of SSD in vertebrates (56, 82). How SSD is produced at the molecular level in invertebrates is poorly understood, but a few studies offer some interesting insights. In the fruit fly, *Drosophila melanogaster*, Horabin (73) demonstrated that the *Sex-lethal* (*Sxl*) gene, a sex-determination master regulator, interacts with another autosomal gene to produce femalebiased SSD; mutations in the *Sxl* gene produce animals that are male-like in body size. Likewise, mutations in the mRNA *transformer* in females result in animals that appear as males but are the same size as females. Thus, sexdetermining genes could regulate autosomal genes to produce variation in SSD in insects.

Additional evidence suggests that the regulation of body size may be sex-linked in Lepidoptera. Sperling (112) showed that many of the genes that account for species differences (including body size) in Lepidoptera are sex-linked. In *M. sexta*, the diminutive black larval mutant (bl^{-}) is the result of a significantly lower JH titer during a crucial period of determination of larval pigmentation (131). Animals that are homozygous for bl^{-} are 37% smaller than the wild-type (bl^{+}/bl^{+}) larvae and have a 20% lower growth rate (105). The *bl* gene is sex-linked and the effect on larval pigmentation is recessive (62, 105). Subsequent studies have shown that the bl^{-} mutation causes an overall decrease in JH synthesis, resulting in low titers of JH during much of larval life (77, 92, 105). The bl^{-} mutation also apparently causes a greater sensitivity of the CA to inhibitory factors circulating in the hemolymph (69, 77). Thus, the *bl* gene appears to affect the titer of JH via regulatory control of its secretion, possibly by means of an allatostatic (inhibitory) hormone (140). It is reasonable to assume that the bl^{-} mutant has a lower critical weight and ICG, which could account for the smaller body size of bl^{-}/bl^{-} mutants compared with wild-type individuals. These two factors (ICG and the critical weight) may have a direct connection to the diminished JH titer of the mutant such that when JH titers are low the mutant will take less time to clear JH from the hemolymph, producing a shortened ICG in mutant larvae. A lower critical weight could be due to the increased sensitivity of the CA to inhibitory factors (69, 77), which could cause a premature cessation of JH secretion. These insights offer a promising glimpse into how genetic and physiological mechanisms may regulate SSD in insects, but studies that address SSD per se are needed to fully understand the mechanisms by which SSD is produced.

Evolution of Sex Differences in Phenotypic Plasticity

Although research on sex differences in body size plasticity is relatively new, several adaptive hypotheses have been proposed to explain why selection acts differently on males and females and how this selection might generate variation in SSD among environments

within species. These hypotheses fall into two categories: Those that predict plasticity to be smallest for traits under the strongest selection (adaptive canalization hypothesis) (51) and those that predict plasticity to be greatest for traits under the strongest selection (condition dependence hypothesis) (22, 23, 27, 94, 104). Among tests of the adaptive canalization hypothesis, some researchers predict that traits under the strongest directional selection should be most canalized (least plastic) (51), but others (including us) predict that plasticity should be least for traits under the strongest stabilizing selection. These ideas were initially developed to explain variation in canalization among traits within one sex—stabilizing selection should favor canalization, and thus reduced plasticity (compared to the average trait in an organism), of morphological traits (such as genitalia) that must match between males and females (40), since deviation from the average phenotype can have substantial negative fitness consequences (85, 125)—but the argument applies equally to other traits (including body size) and to differences between sexes (57). In contrast, the condition dependence hypothesis predicts that traits under the strongest directional selection will exhibit greater sensitivity to environmental conditions, and thus be the most plastic, relative to other traits (22, 23, 27). This hypothesis was proposed to explain reproductive investment into sexually selected traits—individuals are expected to invest as many resources as possible into sexually selected traits, with the phenotype thus highly dependent on resource availability or other environmental conditions—but the hypothesis is equally applicable to any trait, including body size, under strong directional selection. Both classes of hypotheses thus make different predictions about which types of traits should be most plastic and how plasticity of those traits should compare to plasticity of an average trait.

Few studies have explicitly tested any of these hypotheses. Most tests of the condition dependence hypothesis examine exaggerated secondary sexual traits of males (22, 27) and the results generally support the hypothesis. Fewer studies have tested the adaptive canalization hypothesis. In water striders (*Aquarius remigis*), male genital length is under strong directional selection and is less plastic in response to temperature variation than are other traits less closely related to fitness in males or females, a result interpreted as consistent with the adaptive canalization hypothesis (51). Other tests of the adaptive canalization hypothesis have explicitly predicted that plasticity should be least for the sex under strong stabilizing selection (57) and likewise claim support for this hypothesis. However, any explicit test of the hypotheses must compare plasticity in body size not just between the sexes but also with other traits under weaker selection. For example, sex differences in the magnitude of stabilizing versus directional selection possibly explain sex-specific reaction norms in capital breeding moths; in many species, females are under stronger directional selection to be large compared with males (122, 124, 127), and females are more plastic than males (120, 127). This result can be consistent with either the adaptive canalization hypothesis (male size, which is under greater stabilizing selection, is less plastic than female size) or the condition dependence hypothesis (female size, which is under the strongest directional selection, is more plastic); distinguishing between these hypotheses requires us to determine which sex evolved increased plasticity in body size relative to other traits.

A problem for tests of the adaptive hypotheses for sex-specific plasticity is the observation that sex differences in plasticity, and how plasticity varies among traits, are dependent on the specific environmental variables and the range of conditions examined (Figure 1). Complexity of results, such as those found for seed beetles, may be the norm whether the plasticity is adaptive or not; for example, if canalization is possible under favorable conditions but not under periods of stress when developmental and physiological constraints become more important (127). Unfortunately, very few detailed analyses of environment and sex-dependent selection have been performed, and there have been none in species for which phenotypic plasticity in body size (or other size-related traits) has been compared between the sexes.

Selection need not directly act on body size (or morphology) to affect patterns of sexspecific plasticity in size and morphological traits (97). Selection on traits correlated with body size, either positively (such as nuptial gifts) or negatively (i.e., traits that trade-off with size, such as development time), can vary between the sexes and favor increased or decreased plasticity of one sex over the other. Perhaps the best studied of these indirect sources of selection on body size is selection favoring early emergence (protandry) or synchronized emergence of males (67 and references therein). In these species, delaying emergence, or missing an emergence window, may have a much greater effect on male fitness than does emerging at a small size. However, whether this imposes indirect stabilizing selection on male size (due to selection for synchronized emergence) or strong indirect directional selection for small size (e.g., due to selection for protandry) probably varies considerably among ecological contexts. Thus, predicting whether selection will favor increased or decreased plasticity in males relative to females requires detailed analyses of environment and sex-dependent selection on body size and size-related traits. These studies need to be followed by common garden studies of sex differences in phenotypic plasticity to test whether, and to what degree, sex differences in plasticity are adaptive.

Conclusions and Future Directions

There is considerable variation in the degree of SSD within species of insects. Much of this variation is likely due to sex differences in body size plasticity. Little is currently known about the evolution and development of sex differences in phenotypic plasticity, but recent research is beginning to unravel the complex physiology underlying, and the selection producing, these sex differences. We have identified several possible developmental and physiological mechanisms by which adult SSD and sex differences in plasticity in body size can arise, but these mechanisms are poorly understood in the context of SSD. SSD is likely regulated by the endocrine system, although few studies have specifically examined sex differences in the role the endocrine system plays in growth, much less the role it plays in creating sex differences in plasticity.

Several hypotheses have been proposed to explain the evolution of sex differences in plasticity. These hypotheses make different predictions regarding which traits should be more or less phenotypically plastic, but few studies have explicitly tested them. The dearth of experimental tests of these hypotheses results partly because the specific predictions are trait specific and require a detailed understanding of how the relationship between fitness

and phenotype differs between males and females and among environments. The greatest advances in our understanding of sex-specific plasticity are thus likely to come from detailed studies that quantify the specific sources of selection that affect body size in different environments.

Acknowledgments

We thank T. Esperk, D. Fairbairn, K. Fischer, B. Helm, M. Herron, J. Jandt, R. Palli, K. Potter, and T. Tammaru for comments on a previous version of the manuscript. R.C.S. was supported by a Postdoctoral Excellence in Research and Teaching fellowship through NIH Training Grant #1 K12 GM00708 to the Center for Insect Science, University of Arizona. W.U.B. was supported by the University of Zürich and the Swiss National Fund. T.T. was supported by the Estonian Science Foundation (grants 6619 and 7522), the Estonian Ministry of Education and Science (targeted financing project SF0180122s08), and the European Union through the European Regional Development Fund (Center for Excellence FIBIR). C.W.F. was supported by the Kentucky Agricultural Experiment Station at the University of Kentucky.

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Glossary

Sexual size dimorphism (SSD)	a difference in body size between males and females
Phenotypic plasticity	the production of different phenotypes by a single genotype in response to different environmental conditions
Stabilizing selection	selection favoring intermediate phenotypes; i.e., an intermediate phenotype(s) has the highest fitness
Directional selection	selection for a larger or smaller value of a phenotype than the current population mean
Rensch's rule	male body size varies more among populations/species, or evolutionarily diverges faster, than does female body size
RMA	reduced major axis regression
JH	juvenile hormone
PTTH	prothoracicotropic hormone
CA	corpora allata
ICG	interval to cessation of growth
Canalization	a trait becomes developmentally buffered against environmental variation such that a genotype exhibits little phenotypic variation in response to variation in environmental conditions

Measuring Body Size and Sexual Size Dimorphism

Estimates of SSD depend on what measure of body size is used. Body size is commonly assessed using body mass and a variety of other traits (52). Estimates of body mass can vary substantially throughout life for capital breeders, which do not feed as adults and acquire all resources for growth, development, and maintenance during the immature stage and thus lose mass throughout life. For income breeders (which continue to acquire resources as adults), body mass is dependent on how often an individual feeds. Mass will thus be highly variable and a poor measure of size and SSD in field-collected animals. Morphological measures of size are less variable in an adult and generally insensitive to nutritional status. Morphological measures are thus commonly used as a proxy for body size. More importantly, patterns of SSD will vary among traits. For example, in the aradid bug *Aradus cinnamomeus*, estimates of SSD range from the two sexes being monomorphictofemales being 50% larger than males depending on the trait measured (132). Studies can mitigate this problem by measuring SSD from multiple traits and by computing a single measure of size, e.g., using principal component analysis.

Summary Points

1. SSD varies considerably among species of insects and among populations within species.
2. Much of the variation in SSD may be due to sex differences in body size plasticity in response to climatic or ecological variables.
3. Variation in SSD and sex differences in body size plasticity arise through a variety of developmental and physiological mechanisms, but how these mechanisms differ between males and females remains largely unstudied.
4. Adaptive hypotheses, including the adaptive canalization hypothesis and the condition dependence hypothesis, have been proposed to explain the evolution of sex differences in body size plasticity. These generally focus on the degree to which variation in plasticity is generated by stabilizing versus directional selection on body size, but few studies have tested these hypotheses.
5. Studies that address both the proximate and ultimate mechanisms by which SSD and sex differences in body size plasticity are produced will lead to a better understanding of the diversity of SSD observed in nature.

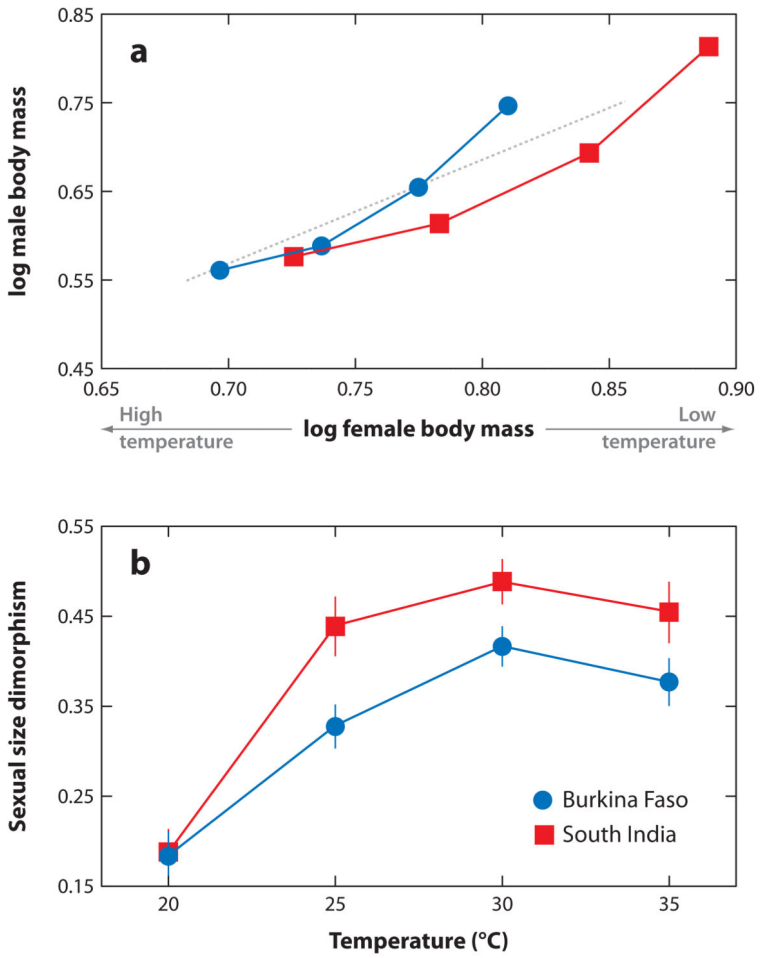

Figure 1.

(*a*) A reduced major axis regression of log male body size on log female body size of two populations (Burkina Faso and South India) of the seed-feeding beetle, *Callosobruchus maculatus*, raised at four different temperatures. The two lines connect the temperature treatments for each population. The dashed gray line indicates a slope equal to 1.0. Note that at high temperatures (30 and 35°C) the slope is <1, whereas at low temperatures (20 and 25°C) the slope is >1. Panel *a* is redrawn from figure 4 in Reference 114. (*b*) The effect of rearing temperature on sexual size dimorphism of *C. maculatus*. Sexual size dimorphism was calculated using the Lovich and Gibbons (81) sexual dimorphism index (SDI), in which sexual dimorphism is estimated as (mean female size/mean male size) −1, arbitrarily made positive when females are the larger sex and negative when males are the larger sex. The SDI was calculated separately for each family and then averaged across families. Panel *b* is redrawn from figure 1 in Reference 114.

Table 1

The frequency of male-biased versus female-biased sexual size dimorphism (SSD) in some major taxa of insects

Order	Male-biased SSD (%)	Female-biased SSD (%)	Monomorphic[c] (%)	Sample size (number of species)
Coleoptera	9	72	19	69
Diptera	11	86	3	37
Hemiptera	10	80	10	10
Hymenoptera	4	84	12	25
Odonata	46	27	27	149
Orthoptera	4	95	2	1508
Lepidoptera	6	73	21	48
All pooled	7	88	5	1846[a,b,d]

[a] Body size was measured differently for many studies. Many measurements were on a linear scale, but if body mass was measured, it was cube-root transformed to make it comparable to other measures of size.

[b] SSD was calculated for all species as (size of the larger sex divided by the size of the smaller sex) −1 and was made positive when females were the larger sex and negative when males were the larger sex (81).

[c] Monomorphic species are those that showed less than ∼2% difference in size between the sexes.

[d] References are available upon request from W.U. Blanckenhorn.

Table 2

Sex-specific phenotypic plasticity in body mass and other measures of body size of insects

Sex-specific phenotypic plasticity in body mass						
	Which sex is more plastic[c,d,e,g]			Average degree of plasticity (CV among environments)[f,g]		
Environmental variable	Females (number of species with RMA <1)	Males (number of species with RMA >1)	χ^2	Females	Males	*t*
Larval density/larval competition/diet quantity	18 (72.0%)	7 (28.0%)	4.84[a]	16.0%	12.2%	3.42[b]
Pathogenic infection	3 (50%)	3 (50%)	0.00	6.9%	7.2%	0.34
Temperature	15 (65.2%)	8 (34.8%)	2.13	11.2%	10.6%	0.55
Photoperiod	1 (16.7%)	5 (83.3%)	2.67	8.6%	10.7%	2.18
Diet quality	83 (61.9%)	51 (39.1%)	7.64[b]	12.5%	11.5%	2.47[a]
All pooled	120 (61.9%)	74 (39.1%)	10.91[b]	12.5%	11.3%	3.32[b]

Sex-specific phenotypic plasticity in other measures of body size						
	Which sex is more plastic[c,d,e,g]			Average degree of plasticity (CV among environments)[f,g]		
Environmental variable	Females (number of species with RMA <1)	Males (number of species with RMA >1)	χ^2	Females	Males	*t*
Larval density/larval competition/diet quantity	8 (47.1%)	9 (52.9%)	0.06	5.4%	5.1%	0.59
Temperature	3 (37.5%)	5 (62.5%)	0.50	3.2%	3.1%	0.12
Diet quality	22 (53.7%)	19 (46.3%)	0.22	6.2%	5.9%	0.59
All pooled	33 (50.0%)	33 (50.0%)	0.00	5.6%	5.4%	0.79

[a] $P < 0.05$.

[b] $P < 0.01$.

[c] χ^2 is the test for whether females are more frequently the most plastic sex, and *t* is for the test of whether mean plasticity is greater in females than in males.

[d] To compare plasticity in body size, we regressed log(male trait) on log(female trait) (females on the *x*-axis and males on the*y*-axis).

[e] Because there is error in both the axes, reduced major axis (RMA) regression was used rather than ordinary least-squares regression (111). RMA slopes <1 indicate more variation in female size than in male size, whereas RMA slopes >1 indicate the opposite.

[f] For each species, the Coefficient of Variation (CV) of average body size was calculated across different environmental treatments for each sex. The average of these CVs are presented for each environmental variable.

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[g] References are available upon request from T. Teder.