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Growing apart: an ontogenetic perspective on the evolution of sexual size dimorphism

Alexander V. Badyaev

Sexes play different roles in reproduction and the adaptive significance of the often remarkably distinct morphologies of adult males and females is documented frequently. Yet, in most vertebrates, the sexes are nearly identical in morphology during early development and undergo highly divergent growth to achieve different adult sizes. The mechanisms that enable the virtually genetically identical sexes to have such divergent growth are not well understood. Of special interest are the constraints that a shared gene pool imposes on sex-specific modifications of growth and the ways that males and females overcome these constraints in response to divergent selection pressures. Recent studies show that the rapid evolution of sex-specific developmental regulators and modifiers can produce sexual dimorphism in size whilst maintaining the integrity of the developmental program that is shared between the sexes.

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Because of their different roles in reproduction, males and females are often under selection that favors their divergent morphological appearance (Fig. 1). Yet, sexes share most of the genes that control basic aspects of growth, and sex-biased expression of these shared genes during development is required to accomplish adult sexual-size dimorphism (SSD) [1]. At each developmental stage, discordance between the realized expression of genes in both sexes and the degree of sex-biased expression of genes that is favored by selection on each sex sets the stage for intersexual ontogenetic conflict (Fig. 2a) [2]. This conflict is most pronounced in complex traits, such as body size, that require prolonged and coordinated development, and where the evolution of sex-specific expression is likely to be slow.

Indeed, numerous empirical studies have documented the lack of sex-biased genetic variance in many size-related traits ([3] and references therein), and standard quantitative genetics theory (Box 1) suggests that the gene pool that sexes share prevents or slows adaptive morphological evolution substantially in each sex even in response to strong selection (Box 1; Table 1). Yet, numerous empirical examples of a rapid change in SSD in response to the environment (Table 2) show that the apparent constraints imposed by a shared gene pool can be mitigated, at least temporarily, by a variety of mechanisms.

The inconsistencies between theory and empirical observations (Box 1) highlight a major gap in our studies of SSD: existing approaches ignore the developmental aspects of its evolution. The exclusive focus on population-level variation in SSD of fully

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Fig. 1. Sexual-size dimorphism is common among many animal species. (a) northern elephant seals *Mirounga angustirostris*, (b) wild turkeys *Meleagris gallopavo*, (c) bison *Bison bison*, (d) impalas *Aespyceros melampus*, (e) olive baboons *Papio cynocephalus anubis*, (f) northern fur seals *Callorhinus ursinus*. These species show some of the highest sexual dimorphism in size and mass in vertebrates, yet neonate males and females are identical in size in each species. Reproduced, with permission from M. Burcham (a–b), A. Badyaev (c), and A.J. Rapone (d–f).

grown adults has left several unresolved questions. How do we reconcile the striking diversity of ontogenetic processes that produce SSD with highly conserved genetic variation in adult SSD? What accounts for the lack of sex-biased genetic variation in most size traits in adults (i.e. in fully implemented developmental programs): an adaptation to maintain developmental integration in the face of a fluctuating and unpredictable environment or conservatism in gene action? Finally, how does sex bias and sex specificity in developmental processes arise and evolve?

The recent integration of knowledge of regulatory aspects of development that are sex specific with quantitative and population genetics theory provides a promising approach to answering these questions. The central thesis of this approach, illustrated here with a focus on avian and mammalian studies, is that the combination of conserved developmental processes shared between the sexes and the rapid evolution of sex-specific modifiers of these developmental processes provides a compromise between what is favored by selection for SSD and what is possible to achieve without destroying the integration of essential components of an organism's development.

Why study the ontogeny of sexual dimorphism?

SSD is produced proximately by differences in patterns of growth between the sexes; thus, selection acting on the growth of males and females will result in changes in SSD of adults. Consequently, although the ontogenetic stage might represent an arena for a developmental conflict between the sexes, it also represents an arena for the resolution of this conflict (Fig. 2). Whereas the evolution of genetically based SSD in adults is extremely slow, there is a rapid evolution of differences between males and females in growth patterns, and these differences evolve not just among related species (Box 2), but also among populations and even among different traits within an organism [4,5]. In poultry breeding, the greatest change in body size of adult males or females is accomplished by programs that capitalize on sex-specific differences expressed during development [6]. Thus, although the ultimate targets of sex-specific selection might be the traits of adult males and females, the proximate targets are sex-specific aspects of development that can produce SSD whilst minimizing the ontogenetic conflict between the sexes (Fig. 2).

Knowledge of the details of growth is essential for our understanding of the ultimate mechanisms underlying SSD evolution. Similar patterns of adult SSD can be produced by distinct growth processes (Box 2). Thus, without knowing the details of ontogeny and of selection during growth, we cannot understand the evolutionary change in SSD. This is illustrated clearly by long-growing species in which selection on males and females during growth, and not during the adult stage, is the most important determinant of adult SSD [7] (Table 2). Moreover, the need for within-organism integration during prolonged and complex development might determine the aspects of variation available to selection [8]; thus, the internal dynamics of the developmental program that is shared between the sexes might have a profound influence on the evolution of SSD (Fig. 2a).

More generally, understanding the proximate mechanisms of change in SSD provides insight into all of organogenesis and its developmental controls. Sexual differentiation of initially similar morphological structures occurs commonly at relatively late ontogenetic stages and involves an organism-wide coordination of sexually dimorphic and sexually monomorphic growth of different tissues [9]. Uncovering the mechanisms by which such organism-wide realignment is accomplished is invaluable for understanding morphological evolution.

Establishment of ontogenetic intersexual conflict

The extent of the intersexual conflict at each developmental stage can be quantified by the strength of between-sex genetic correlations, the measure of covariation in the genetic determination of a trait between the sexes (Box 1). The adaptive evolution of SSD in response to changing environments will be

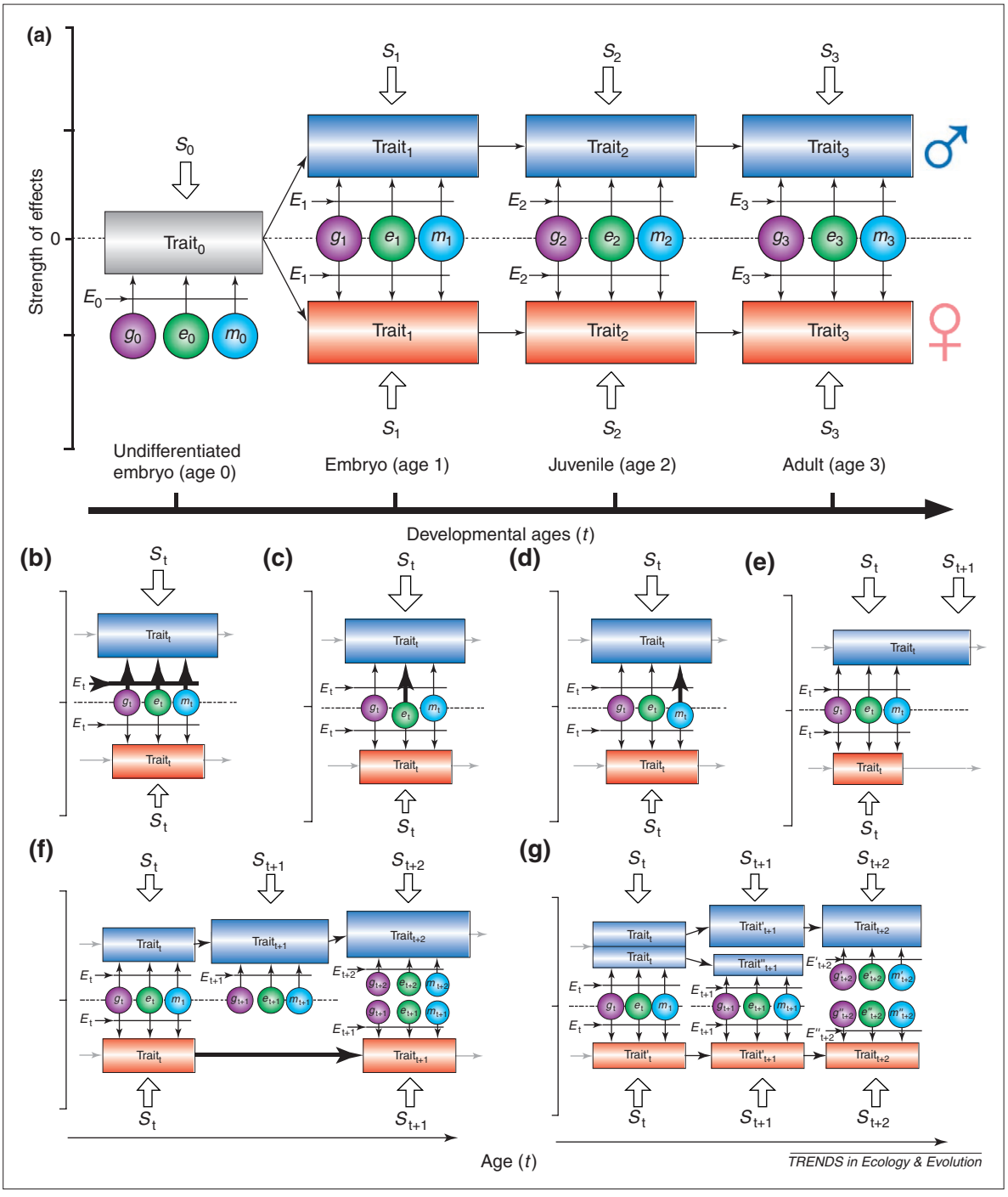

Fig. 2. (a) Conceptual model of ontogeny of sexual-size dimorphism (SSD) for a trait with no sex-biased genetic determination. At any given age (t) and in both sexes, the trait value depends on age-specific genetic effects (g_t), environmental effects (e_t), maternal effects (m_t) and age-specific epigenetic interactions (E_t; e.g. indirect genetic effects, transcription factors or secreted morphogens). Contributions of these effects vary between ages and sexes. SSD at age t depends on the sex-specific differences in the external (environment-dependent) and internal (organism's internal developmental and functional coordination) selection pressures (S_t) at this age. Ontogenetic intersexual conflict arises when S_t is different between sexes, whereas the contribution of shared genetic effects (g_t) is not. (b–g) Resolution of the conflict at each age might include (b) sex-specific epigenetic context (e.g. hormone interactions), (c) sex-biased effects of environment, (d) sex-biased maternal effects, and (e–g) stage-specific trait expression accomplished by (e) sex-specific differences in duration of developmental stages, leading to (f) dissociation of ages between the sexes that still share a common genetic developmental program, and (g) dissociation of traits within each sex (e.g. duplications that generate new sex-biased developmental program). Age-specific changes in contribution of E, e, and m (b-d) lead to subsequent changes in sex-specific ontogenetic trajectories (f-g) and thus to the resolution of intersexual ontogenetic conflict.

slowed by high between-sex genetic correlations, and sex-specific selection should favor weakening these correlations. That is, sex-specific selection should favor the evolution of sex bias in some aspect of the developmental programs (Fig. 2b–g).

However, high between-sex genetic correlations might be adaptive because they themselves can be produced by long-term selection for the developmental stability of physiological and developmental processes that are shared between the sexes. Consistent selection for such stability might reduce phenotypic and genetic variation in growth processes and thus limit the opportunity for the evolution of sex-specific growth. A conflict can arise, however, if selection for SSD, which often acts outside of the developmental period, favors sex-specific growth (Fig. 2a). When such selection is fluctuating, it might be advantageous for the developmental program not to respond rapidly to environmental change by producing sex-specific growth patterns. Instead, a well-buffered developmental program shared between the sexes might integrate environmental demands for sex-specific expression gradually and without disruption of shared developmental processes (Fig. 2b–g).

Thus, whereas high between-sex genetic correlations might limit the speed of adaptive

Box 1. Predicting the evolution of sexual-size dimorphism

The morphological change in each sex under selection is due to the direct response of that sex and the correlated response of the other sex [a]. Thus, the response (R) of male (m) and female (f) for each trait can be presented as Eqn I and II.

$$R_m = 1/2\,(h^2_m S_m I_m + h_m h_f r_g S_m I_f) \quad \text{[Eqn I]}$$

$$R_f = 1/2\,(h^2_f S_f I_f + h_m h_f r_g S_f I_m) \quad \text{[Eqn II]}$$

where h^2 is the narrow sense heritability, S is the standard phenotypic deviation, I is the selection intensity and r_g is the between-sex genetic correlation [b]. Therefore, the response of sexual size dimorphism (SSD) to selection (R_{SSD}) can be defined as the difference between male and female responses. When R_m and R_f are not equal, sexual dimorphism will evolve such that (Eqn III):

$$R_{SSD} = 1/2\,[h^2_m S_m I_m - h^2_f S_f I_f + h_m h_f r_g (S_m I_f - S_f I_m)] \quad \text{[Eqn III]}$$

Sex-specific differences in either selection parameters (I) or in phenotypic and/or genetic variance parameters (S, h^2) can lead to changes in SSD [b]. Under the most common scenario, when heritabilities and phenotypic and genetic variances are not different between sexes, and when males and females are subject to distinct selection pressures, the evolutionary response of SSD will be proportional to the difference in selection pressures between the sexes ($I_m - I_f$) adjusted by r_g (Eqn IV).

$$R_{SSD} = 1/2\; h^2 S (I_m - I_f)(1 - r_g) \quad \text{[Eqn IV]}$$

When selection on each sex is similar, but the sexes differ in the amount of phenotypic variation for a trait, the response of SSD will be proportional to $S_m - S_f$, adjusted by r_g (Eqn V) [b].

$$R_{SSD} = 1/2\; h^2 I\,(S_m - S_f)(1 + r_g) \quad \text{[Eqn V]}$$

Under both scenarios, the strength of r_g plays a decisive role in determining the evolution of SSD (Table 1, main text). However, recent tests of these models suggest that the strength of r_g is more informative about patterns of past selection than about the long-term future evolution of SSD.

Reeve and Fairbairn's [c,d] evaluation of the quantitative genetics framework of SSD evolution produced two important results. First, they showed that fluctuations in genetic variance of both sexes (as a result of variation in the strength and targets of selection as well as in the distribution of allelic effects) can produce extensive and rapid changes in SSD in the presence of high r_g. These results question important assumptions of the standard quantitative genetics models, pointing to the possibility of asymmetrical and sex-specific distribution of genetic variance. Second, they showed that selection acting on ontogeny of body size (e.g. on growth rate and duration) can produce rapid changes in adult SSD. They emphasized that SSD evolution cannot be understood without detailed knowledge of phenotypic and genetic variation during growth; lack of this knowledge could account for the poor explanatory power of existing models [c].

These ideas are formalized further by models that emphasize the evolution of sex-specific regulatory mechanisms that enable the sex-specific expression of genes shared between sexes. Because most genes that underlie SSD are not sex-linked, the regulatory mechanisms specific to males and females account for most SSD. Rhen [e] developed a population genetics model to examine SSD evolution via sex-limited effects at autosomal loci. This approach is promising because the models that explicitly focus on sex-biased ontogenetic expression (i.e. sex-specific timing and extent of gene action) provide a better fit with existing data on the neuroendocrinological regulation of SSD.

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morphological change in each sex in response to local selection, these constraints could be beneficial if selection pressures fluctuate (Table 1). For example, in stoats *Mustela erminea*, populations with an intermediate level of SSD have the highest fitness because an increase in SSD in years with an abundant food supply during growth is opposed by higher mortality in populations with greater SSD during years with poor food supplies [10]. Similarly, populations and species with greater SSD are at a higher extinction risk when exposed to novel environments compared to monomorphic species [11] partly because of the inability to change body size (species size) rapidly in response to novel conditions when male and female morphologies are highly distinct.

Resolution of ontogenetic intersexual conflict

Given the adaptive significance of maintaining the coordination and integration of the developmental

Table 1. Examples of constraints on the evolution of sexual dimorphism in size or mass in birds and mammals[a]

Species	Evidence for constraint	Suggested mechanism	Refs
Human *Homo sapiens*	Predicted evolution of SSD under selection is 65 times slower than change in mean size	Lack of sex-biased genetic variance	[47]
	Slow change in SSD in spite of selection favoring adult SSD	Selection against SSD during growth	[48]
Collared flycatcher *Ficedula albicolis*	Predicted reduction in optimum response to selection by 50% in males and by 200% in females	Lack of sex-biased genetic variance	[49]
Lemurid primates *Eulemur, Hapalemur, Varecia* spp.	Absence of SSD in spite of selection favoring adult SSD	Selection against SSD during growth	[50]
White-tail deer *Odocoileus virginianus*	Decrease in SSD in spite of selection favoring adult SSD	The same as above	[51]

[a]Abbreviation: SSD, sexual-size dimorphism.

Table 2. Examples of rapid and adaptive change in sexual dimorphism in size or mass in birds and mammals[a]

General mechanism	Species	Evidence for rapid change (proposed mechanism)	Refs
Sex-specific sensitivity to condition during growth	Human *Homo sapiens*	30% increase in SSD over one generation (greater male capitalization on improving growth conditions)	[52]
	White-tailed deer *Odocoileus virginianus*	Decrease in SSD over 16 years (changes in male growth because of increasing population density)	[26]
	Snow goose *Anser c. caerulescens*	Seasonal variation in SSD (changes in male growth due to food supply fluctuations)	[53]
	Red deer *Cervus elaphus*	Increase in SSD over 32 years of warmer climate (16% increase in male weight, 10% decrease in female weight)	[24]
	Pied flycatcher *Ficedula hypoleuca*	SSD changes with nest mite exposure (greater male sensitivity during growth)	[18]
	Feral sheep *Ovis* spp.	Seasonal variation in SSD depending on time of birth (greater male sensitivity during growth)	[54]
	Alpine ibex *Capra i. ibex*	Decrease in SSD in cohorts born after winters with higher snow cover (greater male sensitivity during growth)	[25]
	Bighorn sheep *Ovis canadensis*	Decrease in SSD over 19 years of increasing population density (greater male sensitivity during growth)	[27]
	Domestic pig *Sus scrofa*	Change in SSD under experimental change in competition during growth (greater male sensitivity during growth)	[55]
Sex-biased maternal investment during growth	Human *Homo sapiens*	Extensive SSD variation among 76 populations (change in value and maternal care of daughters)	[28]
	Spotless starling *Sturnus unicolor*	Seasonal variation in SSD (sex-biased maternal transference of nutrients to eggs)	[29]
	House finch *Carpodacus mexicanus*	Population divergence in SSD (egg-based maternal effects)	[56]
Sex-specific selection during growth period	Carnivorous marsupials Dasyuridae	Greater SSD during lactation period in captive- versus free-living animals (less sex-biased lactation and greater growth of male offspring)	[57]
	Sifakas *Propithecus* spp.	Population difference in SSD (diet during growth and sex-specific dominance patterns because of food dispersion)	[58]
	Black bear *Ursus americanus*	Population difference in SSD (differences in diet during growth and growth patterns among environments)	[59]
	Mandrill *Mandrillus sphinx*	Decrease in SSD in captive-born versus free-living populations (less sex-biased selection during growth)	[42]
Sex-biased genetic variance in growth and adult traits	Domestic chicken *Gallus domesticus*	Increase in SSD (78%) over 13 years of selection on growth rate (additive genetic variance for SSD)	[60]
		Change in SSD over 36 generation of selection on mass (sex differences in heritability of growth and of body size)	[23]

[a]Abbreviation: SSD, sexual-size dimorphism.

program shared between the sexes, the resolution of ontogenetic intersexual conflict should involve the evolution of regulatory processes that enable the sex-specific expression of shared genes, whilst maintaining the integration of shared development. At different levels of organization, the processes involved in the evolution of sex specificity during development include genetic redundancy and substitution, the co-option of genes for new sex-specific regulatory functions, changes in the timing and duration of growth between sexes and the evolution of a sex-specific epigenetic context of development, resulting ultimately in a developmental trajectory that minimizes intersexual ontogenetic conflict [8] (Fig. 2b–g).

Sex- and age-specific developmental processes

Sex- and age-specific expression of morphological variation can be accomplished by hormonal modifiers of a developmental program (Fig. 2b). On the proximate level, the remarkable diversity of sexually dimorphic growth patterns (Box 2) is established by two contrasting paradigms. First, across all studied vertebrates, the growth of both sexes depends on the concentration of circulating growth hormone (GH) [12]. For example, growth rates are correlated closely with the concentration of GH independently of sex in domestic fowl [13,14], which include some of the most sexually-dimorphic bird species [e.g. in Muscovy ducks *Cairina moschata*, males (5 kg) are twice as heavy as females (2.5 kg); in wild turkey *Meleagris gallopavo* males (14 kg) are more than four times heavier than the females (3.5 kg)]. Second, in spite of the consistent dependency of growth on GH in both sexes, growth is modified because of the high sex specificity in the patterns of GH synthesis and secretion, as well as in the sensitivity of tissues to GH (Box 3). For example, continuous administration of GH to rats *Rattus norvegicus* does not achieve changes in growth patterns, whereas administration of GH in sex-specific pulse patterns produces a strong and sex-specific response [15]. Furthermore, the pathways by which the sex-specific regulation of GH is accomplished vary widely among taxa [12]. Importantly, all known mechanisms that underlie age- and sex-specific expression of GH-induced growth do so without

Box 2. Different means to achieve the same end

The two general developmental processes that produce sexual size dimorphism (SSD) are sex-specific differences in growth rate and growth duration [a]. These processes themselves are the subjects of selection and their relative contribution to the SSD of adults is informative about the direction and patterns of SSD evolution. Figure I shows schematically the relative contribution of dimorphism in growth rate and growth duration to the final SSD (Figure I modified, with permission from [b]).

In many vertebrates, especially in large terrestrial herbivorous mammals, SSD of adults is produced primarily by sex-specific differences in growth duration [c]. However, in most species, for example many primates, sexes differ in both growth rate and growth duration, and sexual dimorphism in these growth patterns leads to adult SSD in several ways. For example, the SSD in gorillas *Gorilla gorilla* and pygmy chimpanzees *Pan paniscus* is produced largely by sex-specific differences in growth duration, whereas in the common chimpanzee *Pan troglodytes,* it is a result largely of sex-specific differences in growth rate [d]. Sex-specific differences in growth patterns can rapidly produce high SSD. For example, in mandrills (animals that show some of the highest SSD of terrestrial mammals) there are no sex-specific differences in either size or growth rate until a year of age. Over the next eight years, however, body mass of males increases by 21.6 kg, whereas females gain only 1.5 kg over the same period of time [e]. In 45 species of primates, adult SSD is produced by a wide range of ontogenetic processes, from sex-specific growth rates in the absence of sex-specific differences in the duration of growth, to SSD produced only by sex-specific duration of growth, with most cases representing a mixture of the two mechanisms [b,f]. Figure II shows a remarkable diversity of ontogenetic pathways that produce SSD in primates. Note the differences in growth patterns of males (blue line) and females (red line) between closely related species. (Figure II modified, with permission from [g]).

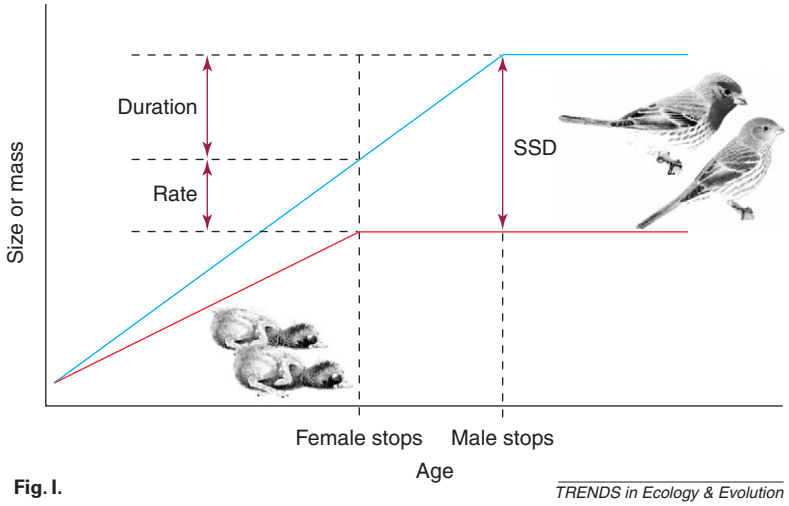

Fig. I. TRENDS in Ecology & Evolution

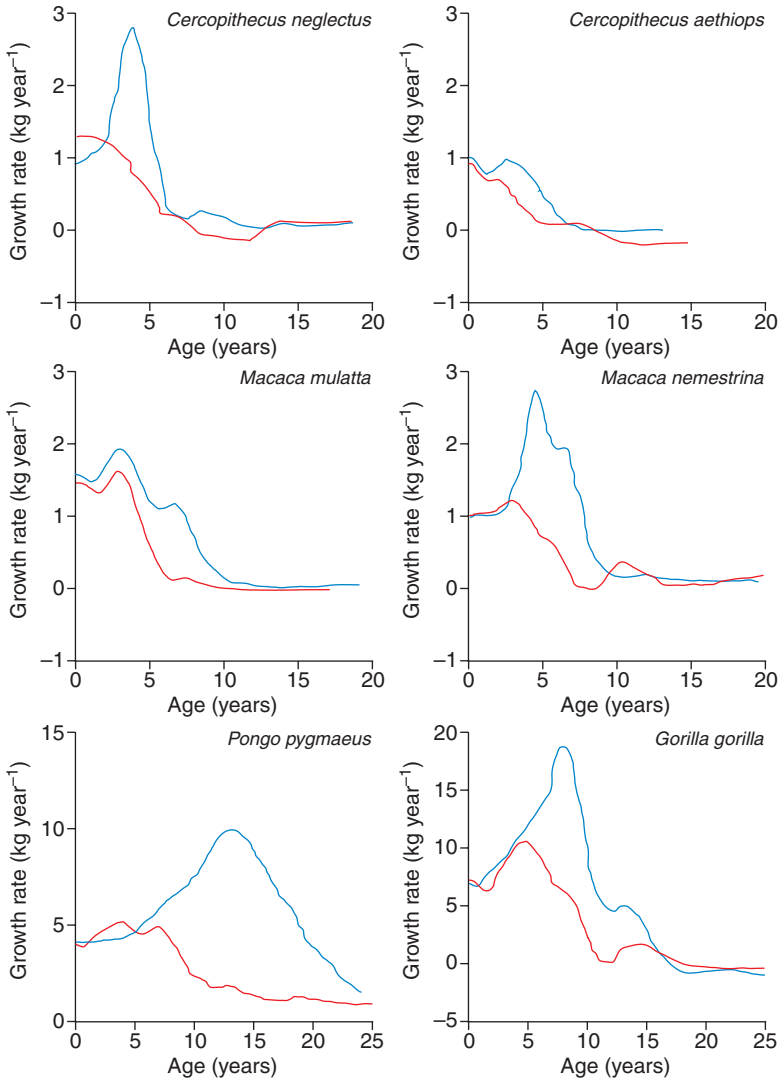

Fig. II. TRENDS in Ecology & Evolution

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Box 3. Conservative way to accomplish diversity: hormonal effects

The secretion of growth hormone (GH) is the fundamental determinant of body size in both sexes of vertebrates. GH is released by the anterior pituitary gland in a highly sex- and species-specific manner [a] under the control of two hypothalamic hormones, the stimulatory GH-releasing factor and the GH-inhibiting factor. In turn, the synthesis and secretion of these two factors are under control of gonadal steroids; both androgens and estrogens stimulate secretion of GH, but their relative importance, the onset of their action and the biochemical pathways by which their effect is accomplished differ between species [b,c]. Age- and sex-specific expression of GH-induced growth is accomplished by four general processes. First, prenatal exposure to steroids (including those of maternal origin) determines the sex-specific sensitivity of the pituitary gland to GH- controlling hormones [c]. Correspondingly, a subsequent exposure to steroids (e.g. during puberty) produces strongly sex-specific GH release and thus sexually dimorphic growth [d]. Second, gonadal steroids can influence hypothalamic secretion of GH-controlling hormones directly [e], and thus produce sex-specific concentrations of GH. Under this scenario, sex-specific growth patterns are due to temporal differences between males and females in the secretion of gonadal steroids [f]. Third, in early development, sex steroids can produce a sex-specific density and distribution of hormone receptors [g] and hormone secreting cells [h], which results ultimately in a long-term differential sensitivity to hormones across tissues that can persist regardless of the subsequent exposure to steroids. Finally, age-specific growth is accomplished by both the duration of exposure to steroids (i.e. age) and age-related changes in GH production [i]. All these mechanisms result in highly age- and sex-specific patterns of growth whilst preserving basic features of development that are shared between the sexes.

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interfering with major regulatory aspects of development (Box 3). Such aspects of development are shared between males and females and are vital for the functional integration of sexually monomorphic and sexually dimorphic traits within an organism. Whereas most known sex-specific genetic controls of shared development are autosomal, the sex-specific regulation of GH in some taxa is attributed to sex linkage of GH-controlling genes. For example, study of hybrids of Pekin duck *Anas platyrhynchos domesticus* (<3.5% SSD) and Muscovy duck (>50% SSD) found heritable suppression of female growth that was consistent with the genetic sex linkage of an as yet unknown regulatory mechanism [16].

Sex-biased variation in some aspects of development might account for cases in which the sexes respond differently to selection during growth. For example, in poultry, equally strong long-term selection for an increase in adult body size of both sexes results in different changes in growth patterns. Across generations, females increase their growth rate progressively, whereas males grow for a longer period [17]. In the pied flycatcher *Ficedula hypoleuca*, the sexes have different growth timing and patterns depending on the exposure to nest mites. Males raised under stressful conditions allocate preferentially to increase in mass at the expense of skeletal maturation, whereas the pattern is opposite for females [18,19].

Sex-specific effects of environmental variation

Sexes differ in their sensitivity to environmental conditions during growth (Fig. 2c), and this is the main cause of population divergence in SSD in many species (Table 2). In birds and mammals, male growth is affected more by nutritional and environmental stress than is female growth [20]. Males commonly have higher mortality during growth, especially in mammals, and improvement in maternal condition and food availability commonly has greater effect on male growth and development than on that of females [21–23].

Predictable and consistent patterns of environmental change during growth, such as seasonal changes in food supply or parasite infestation, lead to seasonal and temporal variation in SSD (Table 2). For example, recent climatic trends [24,25] and population management practices [26,27] resulted in changes in SSD of large mammalian herbivores because of the differences in sensitivity to the environment between the sexes during growth (Table 2).

Selection on ontogeny of sexual size dimorphism

Maternal selection and sex-specific expression

Parents, especially mothers, can influence SSD of their offspring both by modifying the environment during growth and by restricting SSD at birth because of maternal size (Fig. 2d). Population divergence in SSD is often due to population differences in sex-biased maternal effects (Table 2). For example, preferential treatment of children according to gender varies among cultures and might account for the population variation in human SSD [28]. In the spotless starling *Sturnus unicolor*, breeding females transfer different amounts of nutrients to male and female embryos throughout the year, producing temporal patterns of SSD in their offspring [29]. An example of sex-biased maternal selection in relation to offspring SSD is where mothers lay larger eggs for the smaller sex, apparently to mitigate the effects of within-brood competition and lessen parental expenditure. In the American kestrel *Falco sparverius* [30] and spotless starlings [29], mothers produced larger eggs for the structurally smaller sex (males in kestrels and females in starlings). In addition, exposure to steroids

of maternal origin can permanently affect the sensitivity of an embryo's tissues to GH (Box 3).

Maternal size can have strong effects on the evolution of SSD by physically restricting the size of the neonate of the larger sex and thus SSD at prenatal and natal stages. In an analysis of 109 species of primates, Smith and Leigh [31] found that even the species with the highest adult SSD had very small, although detectable, SSD at birth. Interactions between the benefits of greater neonate SSD and costs of greater SSD on pelvic structures is thought to play a role in population differences in adult SSD in humans [32]. Similarly, an analysis of 446 bird species [33] showed that although those with greater adult SSD laid larger eggs, this was not related to SSD of offspring.

In addition, the SSD at birth and during early growth is limited strongly by the mother's body size and by the costs of lactation and provisioning. For example, mandrills *Mandrillus sphinx* (males weigh 31 kg, females, 9 kg), California sea lions *Zalophus californianus* (males weigh 270 kg, females, 90 kg) and the southern elephant seals *Mirounga leonina* (males weigh 3600 kg, females, 900 kg) are some of the most sexually dimorphic mammals with adult males being up to four times heavier than females. Yet, even in these species, the SSD at birth does not exceed a 1:1.2 ratio. Similarly, offspring SSD during the lactation is low, because mothers do not provision males and females preferentially even in the most size-dimorphic species [34]. The lack of the sex-biased provisioning is a powerful selection pressure on the ontogeny of the larger sex, leading to the evolution of an increased rate and duration of growth as well as adaptations that allow greater sensitivity to (and capitalization on) environmental variation during growth. For example, in the California sea lion and Antarctic fur seal *Arctocephalus gazella*, the maternal expenditure during lactation is equal between the sexes, but sons are able to grow larger because of their lower metabolic rate compared with smaller, but more active daughters [35,36].

Selection for stage-specific expression

One of the best examples of the ontogenetic conflict between the sexes is the change in the fitness consequences of a particular size between developmental stages [37]. Because of the cascading effects of growth, the between-sex integration during early development can carry over to later life stages, preventing the sexes from reaching their adaptive optimum in size (Fig. 2a). For example, it is often difficult to separate the activational and organizational roles of steroids that affect development; sex steroids that induce sex-specific growth patterns often regulate the time of growth termination and thus final size and SSD [38]. One solution to conflicting intersexual effects is the evolution of regulatory mechanisms that, in both sexes, would limit trait expression to stages in which such expression is favored by selection; that is, to produce some independence between adult and juvenile stages (Fig. 2e–g). This solution generates two general predictions that are well supported by empirical studies.

First, sexually dimorphic size traits that are expressed only in late ontogenetic stages (and therefore do not require extensive developmental integration with other traits) should develop with the lowest intersexual conflict (Fig. 2f–g). A good example is the sex-specific pattern of fat deposition in humans, which is expressed only late in development (age 3 in Fig. 2a) and shows some of the most sex-specific expression and sex-biased genetic determination among size-related and nonsex-biased traits [39]. Similarly, most sexually dimorphic traits of the human skeleton grow during late ontogeny and have low developmental integration with sexually monomorphic traits [4] that grow early in development. In many species, the most sexually dimorphic growth occurs during short periods late in development (Box 2).

Second, the intensity of ontogenetic intersexual conflict depends on the duration of growth. More generally, it depends on the relative duration of stages when selection favors or opposes the similar appearance of sexes and on the relative intensity of selection during these periods (Fig. 2e). Species with longer growth periods experience more age-specific selection pressures and thus are more likely to evolve greater dissociation in growth patterns between the sexes (Fig. 2f). Typically, SSD is favored by selection acting during adult stages when differences in size contribute to the reproductive success of both sexes. In the analysis of sex-specific growth in 31 bird species, Teather and Weatherhead [19] concluded that the differences between sexes in growth rate and duration are explained best by the allometric requirements of adult SSD, and are not an adaptive consequence of selection during growth as had been often suggested.

However, many species exist in which sex-specific selection during growth favors the evolution of SSD during the juvenile period. An excellent example is the sex-specific difference in the growth of primates (Box 2). Primates have the most prolonged juvenile (pre-breeding) period of any mammal, with a juvenile period of >50% of the entire life span in many species. In many species, sexes show significant differences in the timing and duration of growth spurts (Box 2). Female primates commonly have earlier and more prolonged growth spurts, apparently to minimize the interference between growth and a potentially early pregnancy [40]. In males, a delayed and more condensed growth spurt is favored because of the benefits of retaining a small size during the prolonged juvenile period to reduce competition and agonistic interactions with older males [41]. Thus, sexual dimorphism in growth patterns evolves as a result of selection pressures during the juvenile

Box 4. Unresolved questions and future studies

- In spite of a striking diversity and versatility of both initial molecular triggers of sex-specific development in an undifferentiated embryo and subsequent regulatory pathways [a], sex-specific ontogenies are remarkably coordinated (i.e. no intermediate phenotypes between the sexes are produced). What maintains this developmental coordination given such diverse starting points? How do different developmental pathways arise and how are they maintained? Why is there such a remarkable diversity of initial triggers of sex-specific development and early ontogeny of sexual-size dimorphism (SSD)?
- Early ontogenetic stages are commonly highly canalized and resistant to modifications, whereas later stages show greater potential for evolutionary change. Yet, the pattern seems to be reversed for the early ontogeny of SSD, where upstream regulators of development are less conserved among evolutionary lineages than are the effects of downstream gene effects [b,c]. How are these regulatory hierarchies formed? What constrains the diversity in downstream regulators of SSD ontogeny?
- The lack of sex-biased genetic variation in the size of fully grown males and females is often documented. Might this represent an adaptation to fluctuating sex-specific selection on size during ontogeny? Is this an adaptation to the needs of maintaining internal integration during development or a limitation imposed by shared gene effects during development? Could direct selection on aspects of development (e.g. on time of maturity or any other developmental event) be more effective in altering the developmental trajectory (and thus the adult phenotype) than direct selection on the adult phenotype?
- Epigenetic aspects of development in general, and the sex-specific epigenetic context of SSD ontogeny in particular (i.e. genetic and non-genetic developmental agents that modify the expression of genes shared between the sexes), are very poorly known [d]. For example, why are the GH effects on growth remarkably constant across evolutionary lineages, whereas other aspects of SSD ontogeny, including those of GH regulation, evolve rapidly?
- Sex-biased maternal effects are ubiquitous in the evolution of SSD and are especially strong in species with long generation times. Interestingly, these effects are often a direct consequence of maternal condition and social rank [e]. What are the effects of population variation in condition and age of breeding females on the evolutionary dynamics of offspring SSD?

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period (Box 2; age 2 in Fig. 2a). Interestingly, in these primates, the removal of sex-specific selection during growth (e.g. restricting access of adult males to juvenile males and females) reduces sexual dimorphism in growth patterns significantly and, therefore, reduces SSD in final size. For example, in mandrills, zoo-living juvenile females have a longer growth period and juvenile males grow faster compared with juveniles in free-living populations, thus accounting for the reduced SSD in zoo populations [42]. In primates, a close correlation between the social and ecological conditions experienced during growth and the patterns of SSD acquired during growth illustrates further that sex-specific selection during ontogeny is one of the main determinants of SSD in these species [7] (Table 2). But perhaps the best illustration of an association between the timing of selection favoring SSD and the development of SSD (and thus the resolution of ontogenetic intersexual conflict) is the ontogeny of SSD in the orang-utan *Pongo pygmaeus*. In this species, development of SSD is inhibited hormonally by the presence of adult males and thus is linked proximally to the timing of selection for SSD by male–male competition [41] (Box 2).

Strong selection for SSD can occur as a result of developmental processes *per se*, as shown by the sex-specific selection for different rates of growth in as yet morphologically undifferentiated male and female embryos (age 0 in Fig. 2a). In large mammals, including humans, the differences between male and female growth rates are evident as early as 6 h post-fertilization at the 32-cell stage [43], when male embryos grow up to five times faster than do female embryos. These sex-specific differences in growth are driven by the need for early gonadal differentiation of males and the corresponding production of gonadal testosterone necessary to sustain their normal sex-specific development in spite of the increase in maternal estrogens as pregnancy progresses [44]. In turn, the faster initial growth of male embryos in mammals sets the stage for the greater sensitivity of male embryos and juveniles to environmental conditions during growth (Table 2).

Ontogenetic conflict between the expression of SSD at different life stages might be exaggerated further because selection during the growth period is experienced by more members of the population than at other life stages and mortality is often the highest during the juvenile period. Intersexual ontogenetic conflict is enhanced when selection favors the opposite pattern of SSD during juvenile and adult stages [45,46]. Thus, of special interest is the estimation of the relative strength and direction of selection acting on juvenile versus adult males and females.

Future directions

In spite of a large volume of research and a wealth of information, there is a remarkable lack of integration between the molecular, genetic, physiological and developmental approaches to the study of SSD. A growing understanding of the evolution of developmental processes holds great promise for answering the questions left unresolved (Box 4). Current studies of the molecular and genetic mechanisms of SSD ontogeny are uncovering striking variation in the origin and maintenance of SSD among evolutionary lineages and have the potential to advance our understanding of SSD evolution.

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