New perspectives on the evolution of exaggerated traits

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The scaling of body parts is central to the evolution of morphology and shape. Most traits scale proportionally with each other and body size such that larger adults are essentially magnified versions of smaller ones. This pattern is so ubiquitous that departures from it – disproportionate scaling between trait and body size – pique interest because it can generate dramatically exaggerated traits. These extreme morphologies are frequently hypothesized to result from sexual selection and their study has a long history, with several hypotheses seeking to explain their evolution. Despite this effort, surprisingly little progress has been made in demonstrating the forms of selection that produce different scaling patterns or in identifying the mechanisms that underlie the expression and evolution of scaling relationships. Here we review recent insights regarding the proximate mechanisms that regulate and integrate trait growth and that offer a new framework for studying the evolution of morphological scaling.

Keywords:
allometry; diversification; morphological scaling; morphology; sexual selection

DOI 10.1002/bies.201200139

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Abbreviations:
CNS, central nervous system; **IIS,** insulin/IGF-signaling; **ILP,** insulin-like-peptide.

Variation in relative trait size

Organismal shape is determined largely by the size of morphological traits relative to body size [1, 2]. Within species, the size of traits typically covaries strongly with body size such that overall shape is maintained across all sizes. In *Drosophila*, for example, wing size relative to body size is approximately the same among all members of a population, making large individuals essentially proportionally scaled-up versions of smaller ones [3] (Fig. 1A). Such low variation in relative trait size is generally believed to result from selection for ecological function [4]; improperly scaled organisms are less fit as they cannot effectively move, feed, etc. (although data explicitly addressing this point are elusive). In contrast to low levels of variation in relative trait size within biological groups, however, differences in relative trait size among groups are common. In fact, changes in the relative size of traits is perhaps *the* major pathway by which morphology diversifies [5–7].

Not all traits scale proportionally with body size. For example, the size of the male genitalia in most arthropods [8], and the size of the central nervous system (CNS) in mammals [9] is relatively invariant across body sizes within species. Conversely, some traits increase dramatically with body size, as observed in many signaling traits or weapons of sexual selection [2, 10–14] such as the elongated eye-stalks of stalk-eyed flies [15] or the enormous antlers of the extinct Irish elk [12]. In cases where trait size changes disproportionately with body size, small and large individuals do not resemble each other as shape changes with body size (Fig. 1C and D).

Because disproportional scaling is atypical and often conspicuous, considerable effort has been applied over at least 100 years to determining the selective patterns that produce such morphologies. What has been missing from this effort, until very recently, is an understanding of the developmental mechanisms that regulate and integrate trait growth and that are the target of selection for changes in morphological scaling. Now, a new paper by Emlen et al. [16] synthesizes the developmental mechanisms that regulate trait growth and the selective pressures that lead to changes in morphological scaling to propose an exciting and novel model for the evolution of exaggerated traits. Here, we place this new work in the context of recent studies on the developmental regulation of body proportion and examine the implications for

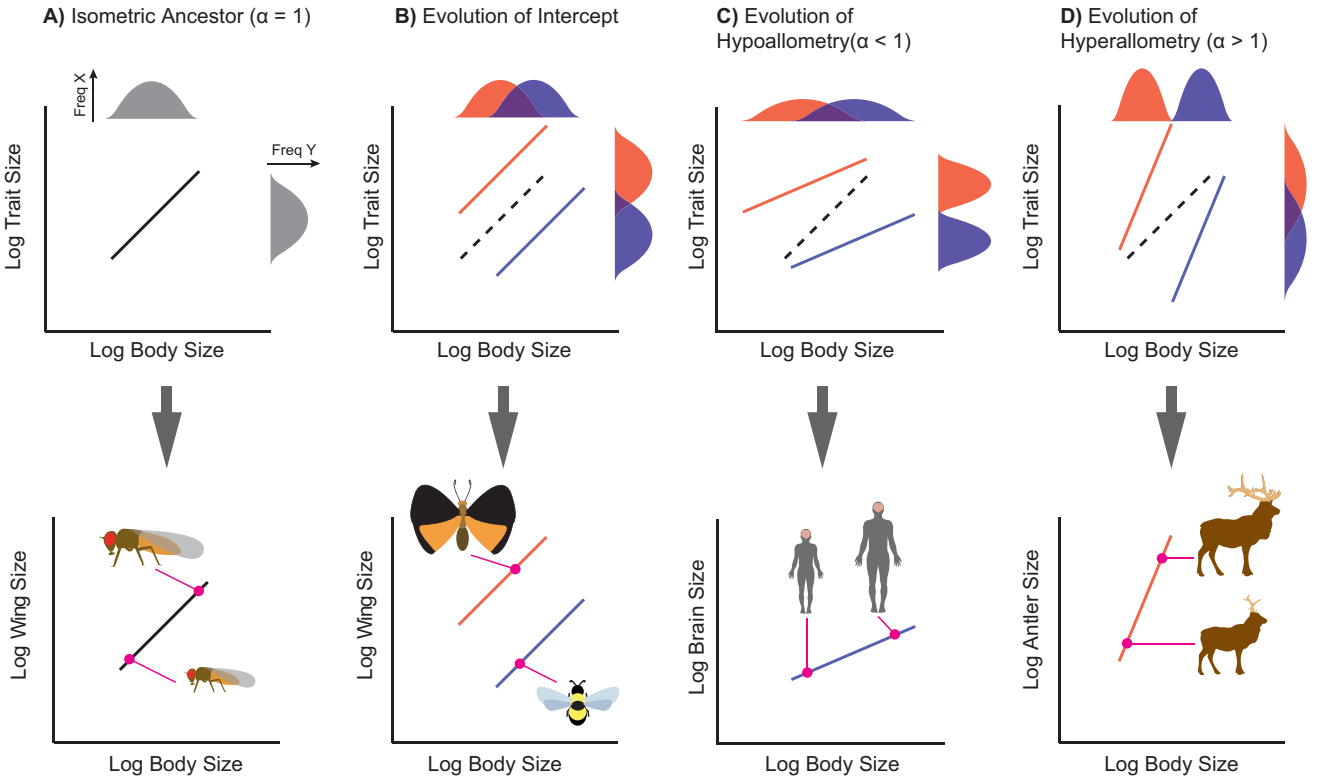

Figure 1. Evolution of morphological scaling and trait plasticity. **A:** Isometric (1:1) scaling relationships occur when both trait and body size show the same size response to variation in a genetic or environmental regulator of size (gray frequency distributions). Examples include the relationship between wing size and body size in *Drosophila* [3]. **B:** Evolution of the intercept occurs when both trait and body size maintain the same sensitivity to variation in a size regulator, but the mean size of either the trait or body size increases or decreases (red vs. blue frequency distributions) relative to the ancestor (dashed line). Examples include the difference in the wing/body scaling relationship between bees and butterflies. **C:** Evolution of hypoallometry occurs when there is a decrease in the sensitivity to variation in a size regulator in the trait relative to the body, producing a trait with low relative variation (red and blue frequency distributions). Two evolutionary pathways from the ancestral isometric relationship (dashed line) to hypoallometry are shown. The red trait (top line) achieves hypoallometry by becoming disproportionately large in small individuals, whereas the blue trait (bottom line) achieves hyperallometry by becoming disproportionately small in large individuals. Examples include the relationship between brain and body size in humans [67]. **D:** Evolution of hyperallometry occurs when there is an increase in the sensitivity to a size regulator in a trait relative to the body producing a trait with high relative variation (red and blue frequency distributions). Again, two evolutionary pathways from the ancestral isometric relationship (dashed line) are shown. The red trait (top line) achieves hyperallometry by becoming disproportionately large in large individuals (i.e. the trait is exaggerated), whereas the blue trait (bottom line) achieves hyperallometry by becoming disproportionately small in small individuals. Examples of hyperallometry include the relationship between antler size and body size in the Irish Elk [12].

understanding the expression and evolution of morphological scaling relationships.

Morphological scaling relationships evolve through changes in slope and intercept

Morphological scaling relationships are often (but not always [17]) linear on a log–log scale (Box 1) and so their evolution can be partitioned into changes in the intercept and the slope of the relationship (Fig. 1, Box 2). The ability of the intercept to respond to selection is likely affected by the genetic variances of the traits comprising the scaling relationship, their covariance, and the form and strength of selection acting on their individual and joint values, as is the case for any set of correlated traits [18–21]. Comparative work on many traits across a variety of taxa [6] and artificial selection experiments (e.g. [22–24]) demonstrate clearly that the intercept of morphological scaling relationships can evolve.

Sexual selection is frequently invoked when trait size changes disproportionally with body size (Box 2; [11–13, 25, 26], although see [27]). For example, stabilizing selection on male genitalia size imposed by mating ability and/or female choice is believed to favor a constant genital size regardless of body size in arthropods, resulting in hypoallometry [8, 28]. In contrast, sexual selection for ever-increasing size in weapons or morphological traits used in sexual signaling is thought to explain instances where traits scale hyperallometrically with body size [14, 29, 30]. Numerous models explain how sexual selection can lead to the evolution of hyperallometry [29–35]; collectively, they posit fitness gains for individuals possessing traits scaled disproportionally for their body size but no such gains (or perhaps natural-selection imposed fitness losses) for smaller individuals expressing similarly proportioned traits. Particularly relevant to much

Box 1

Describing morphological scaling relationships mathematically

Traditionally, scaling relationships have been modeled using Huxley and Tessier's allometric equation:

$$y = bx^{\alpha}$$

where, x and y are the size of two traits, b is the intercept and α is the allometric coefficient [70]. Typically, y is the size of the focal trait, such as an appendage or organ, while x is body size. Log-transformation of the allometric equation yields a linear relationship $\log(y) = \log(b) + \alpha[\log(x)]$ which offers a convenient method of summarizing scaling relationships within biological groups. Here, b (the intercept) describes the size of the trait relative to body whereas α (the slope) describes how this relative size changes with body size [6, 71]. Importantly, log transformation makes the traits scale-independent and thus, permits meaningful comparison of scaling relationship parameters among traits or biological groups. This latter point underscores the value of log transformation, even when scaling relationships are already linear; by removing the scale-dependence, trends in the expression or evolution of scaling relationship parameters will be revealed, free from nuanced caveats regarding variation in measurement units, trait types or plotting approaches (Box 2) [72, 73].

Box 2

Three types of log-linear morphological scaling relationships

Log transformation produces three categories of scaling relationship. Isometry occurs when $\alpha \sim 1$. Here, large individuals are proportionally magnified versions of smaller individuals and shape does not change with body size (Fig. 1A). For isometric scaling relationships, differences in intercept reflect a constant difference in trait size across all body sizes and thus large individuals are simply proportionally magnified versions of small ones (Fig. 1B). Departures from isometry produce changes in shape across body sizes. Hypoallometry describes the condition where $\alpha<1$. Here, trait size is relatively invariant across body sizes, and the trait is disproportionally large in small individuals or the converse (Fig. 1C). Hyperallometry occurs when $\alpha>1$, a condition where trait size is more variable than body size and the trait is disproportionally large in large individuals or disproportionally small in small individuals (Fig. 1D). Thus, departures from isometry result in overall shape change across body sizes; large individuals are not proportionally magnified versions of small ones (Fig. 1C and D).

Absent log transformation, however, scaling relationships remain scale-dependent and thus categorizing and comparing them in this manner is problematic [74]. For example, consider the scaling relationship between wing size and body size in a population of the honeybee *Apis melifera* and butterfly *Bicyclus anynana*. Body size in both insects is more or less the same ($\sim$30 mg), but wing size is approximately twice as large in the butterfly than the bee ($\sim$60 mm^2 vs. 160 mm^2) [22, 75]. Since we expect that larger morphological traits will vary more than smaller morphological traits on a linear scale (compare body size variation in a mouse versus an elephant) [72], across the same range of body sizes, we would a priori expect more variation in wing size in the butterfly than the bee. Because the slope of a scaling relationship is controlled by the extent to which trait size varies with body size (see Fig. 1 and text for more details), on a linear scale the slope of the wing-body scaling relationship will therefore be greater for the butterfly than the bee, even though in both species wing size scales more-or-less proportionally with body size. Log-transformation resolves this issue and allows the comparison of slopes independent of scale [72] (although see [17] for further issues relating to log-transforming morphometric data).

of the work discussed below, sexual selection for honest (unfakeable) signals of the quality of rivals or potential mates is hypothesized to favor enlargement of morphological signals relative to the body, frequently producing hyperallometric scaling of traits [29, 36]. Absent from many of these models, however, is explicit inclusion of a developmental perspective on the mechanisms that regulate and integrate trait growth (except see [1]). Because selection on these mechanisms ultimately underlies evolved changes in scaling, their elucidation is essential if we are to understand if, or how, a particular mode of selection might produce changes in the slope or intercept of a morphological scaling relationship.

Organ-specific insulin-sensitivity regulates the slope of morphological scaling relationships

Morphological scaling relationships reflect the covariation between trait and body size, and arise because both trait and body size respond to common factors that regulate the rate and duration of growth. These factors may be genetic – for example, the effects of allelic variation on growth rate – or environmental – for example, the effects of temperature or nutrition on growth rate. It follows that, for log-linear scaling relationships, the slope reflects the relative effect of these growth-regulatory factors, be they genetic or environmental, on trait size versus body size (Fig. 1). An isometric scaling relationship results when a factor has the same effect on trait and body size [37], whereas hypo- or hyperallometric relationships occur when the effects on trait and body size differ (Fig. 1) [37].

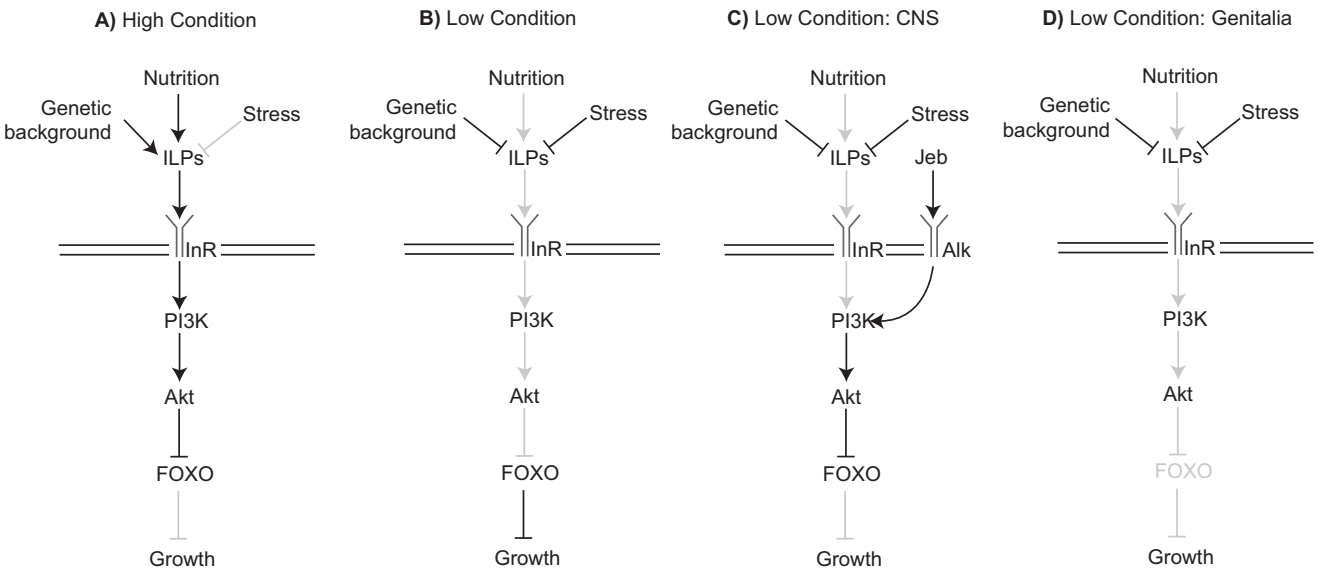

Figure 2. The molecular-genetic regulation of insulin sensitivity in *Drosophila*. **A:** When circulating levels of ILPs are high due to high nutrition, low stress or genetic background, the insulin-signaling pathway promotes growth primarily by suppressing the activity of negative growth regulators, for example FOXO. **B:** When circulating levels of ILPs are low, this de-represses FOXO activity and inhibits growth [68, 69]. **C:** In the CNS, insulin-sensitivity is reduced by the activation of Pi3K by Jeb/Alk in an ILP-independent manner [55]. **D:** In the male genitalia, insulin-sensitivity is reduced by down-regulating expression of *FOXO*, ensuring that growth is not suppressed even when ILP-levels are low [56]. Black lines indicate active components of the pathway; gray lines indicate inactive components of the pathway.

The insulin/IGF-signaling (IIS) pathway is a primary regulator of variation in trait size, specifically organ size, and body size in animals (Fig. 2) [38–42]. The IIS pathway regulates growth in response to circulating levels of insulin-like-peptides (ILPs), which bind to the insulin receptor (Inr) of dividing cells [43, 44]. This initiates a signal transduction cascade that ultimately regulates cell growth and proliferation in growing organs, primarily by deactivating negative growth regulators (e.g. TSC1/2 and forkhead transcription factors, FOXOs; Fig. 2; reviewed in [45]). The result is that systemic changes in the level of circulating ILPs regulate organ-autonomous cell growth and proliferation, coordinating growth across the body (Fig. 2A and B). Circulating ILP levels are affected by environmental factors such as nutrition and infection, and it is primarily through the IIS pathway that environmental variation is transduced to affect trait and body size [46–49]. However, evidence suggests that genetic variation in circulating ILP levels and the activity of components of the IIS pathway also generates variation in trait and body size [50]. Thus variation in insulin/IGF, be it environmental or genetic in origin, may account for a substantial amount of trait and body size variation.

If (i) the slope of a morphological scaling relationship reflects the relative organ and body sensitivities to systemic factors that affect size, and (ii) variation in IIS-pathway activity is the proximate factor generating intraspecific variation in organ and body size, it follows that the relative response of an organ to variation in IIS-activity, i.e. the trait's *insulin-sensitivity*, determines the slope of the organ/body size scaling relationship [37, 51–53]. This hypothesis is well supported: male genitalia and the central nervous system are strongly hypoallometric in *Drosophila*, and likely all arthropods [3, 8, 54–56], and both traits exhibit less size response to reductions in developmental nutrition and insulin-signaling than does the body [55, 56].

Insulin-sensitivity is regulated by at least two mechanisms in *Drosophila*

The insulin-insensitivity of the CNS and male genitalia in *Drosophila* is achieved through different mechanisms. In the case of the CNS, a second receptor, anaplastic lymphoma kinase (Alk), can also activate the insulin/IGF-signaling pathway even when nutrition and circulating ILPs are low (Fig. 2C) [55]. As a consequence, growth of the CNS is less sensitive than the rest of the body to changes in nutrition, resulting in a near-constant CNS size across body size [55]. In contrast, the male genitalia reduce their response to changes in nutrition and insulin/IGF-signaling by down-regulating the expression of *FOXO* (Fig. 2D), a transcription factor that is negatively regulated by the IIS pathway and which targets negative growth-regulator expression [56]. When nutrition and IIS is low, *FOXO* is activated and growth is suppressed. However, by reducing *FOXO* expression in the genitalia, larvae maintain genital growth even under nutritional stress, reducing the effects of nutrition on genital size and rendering the genitalia hypoallometric to body size (Fig. 2D).

Organ-specific changes in IIS gene-expression generates exaggerated traits

Since low levels of *FOXO* expression make an organ hypoallometric to body size, it follows that increasing *FOXO* expression in an organ should increase the slope of its scaling relation-

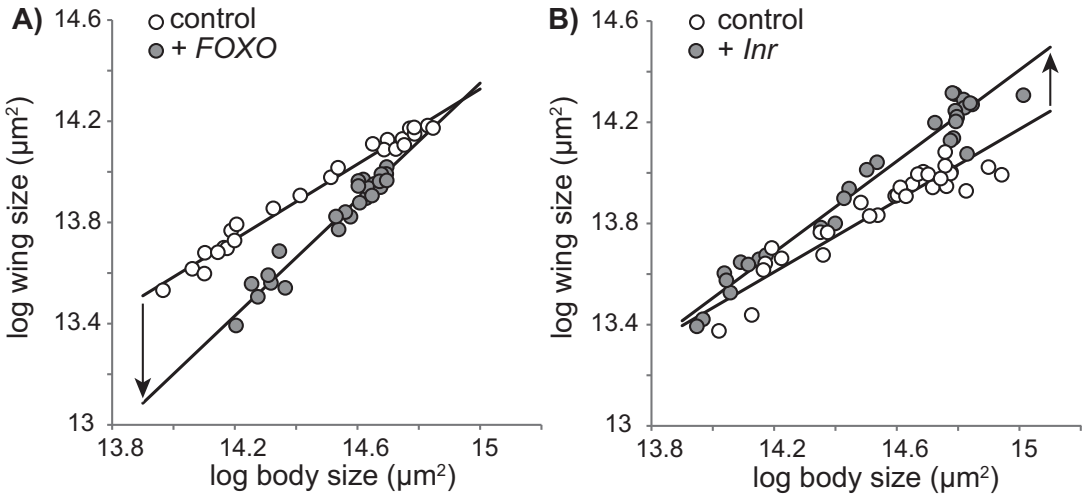

Figure 3. Alternative developmental mechanisms for achieving trait hyperallometry in *Drosophila* wings. **A:** An increase in wing-specific expression of *FOXO* generates hyperallometry by suppressing wing growth in small individuals where nutrition and insulin-signaling is low. **B:** An increase in wing-specific expression of *Inr* generates hyperallometry by enhancing wing growth in large individuals where nutrition and insulin-signaling is high. Arrows indicate direction of treatment effect. Figure modified from [59].

ship with the body. This is indeed the case for the *Drosophila* wing and male genitalia (Fig. 3A) [56]. This slope change occurs via disproportionate reduction in organ size in individuals that are small because of malnourishment. For many hyperallometric traits, however, steep scaling relationships are achieved through a different pattern: traits are disproportionally increased in large individuals (compare red and blue lines in Fig. 1D). Such hyperallometric traits are considered *exaggerated*, and this exaggeration is evident when comparing scaling relationship slopes in one sex (usually the male) relative to the other, or by comparing slopes between the evolutionarily derived and ancestral states (e.g. [53, 57, 58]; Fig. 1D). Note that here we restrict the term *exaggeration* to traits that scale disproportionally with body size and are thus disproportionally larger in large individuals, as opposed to traits that are proportionally enlarged across the full range of body sizes relative to the ancestral condition, as with the trunks of elephants. Work in *Drosophila* suggests that hyperallometry resulting from trait exaggeration is also achieved through organ-specific changes in the insulin-signaling pathway [59]: Increasing *Inr* expression in the developing wing increases relative wing size in large (i.e. well nourished) flies but has almost no effect on relative wing size in small, (i.e. malnourished) flies [59] (Fig. 3B). Thus, changes in organ-specific insulin-sensitivity can generate the type of trait exaggeration observed in sexually selected traits.

Integrating the developmental regulation and evolution of morphological scaling

A recent paper by Emlen et al. [16], illustrates how such developmental information can deepen our understanding of morphological scaling evolution [16]. Rather than focusing on the details of which patterns of sexual selection might generate hyperallometry, they examined the mechanisms that regulate and coordinate growth. Their central hypothesis is that the extreme growth in showy ornaments and weapons of sexual selection in large individuals occurs through an increase in a trait's insulin-sensitivity.

To test their hypothesis, first Emlen et al. document the scaling relationship between the size of three traits and the body in male rhinoceros beetles (*Trypoxylus dichotomus*). Male *T. dichotomus* posses exaggerated horns, and intrasexual selection appears to favor large males with disproportionately large horns [60]. As Emlen et al. demonstrate, the strong hyperallometric scaling of the horn contrasts with the near isometry of the wings and the hypoallometry of the genitalia (Fig. 4). Their hypothesis predicts that the steepness of these scaling relationships should reflect the relative sensitivity of each trait to changes in insulin/IGF signaling: hypoallometric traits should be insensitive, hyperallometric traits should be highly sensitive, and isometric traits should exhibit intermediate sensitivity. RNAi against the insulin receptor to suppress insulin/IGF-signaling during trait growth revealed support for their prediction: the growth response to RNAi was greater in the horns (15% reduction in size) than in the wings (2% reduction) or the genitals (0% reduction). Thus it appears that sexually selected exaggerated traits are more insulin-sensitive than other traits.

As discussed above, however, there are two ways in which traits can become hyperallometric, both of which result from increased insulin-sensitivity (Fig. 1D, 3). Implicit in Emlen et al.'s hypothesis is the assumption that exaggerated traits are hyperallometric because they are more growth-responsive to circulating ILPs when ILP levels are high, as opposed to being less growth-responsive to circulating ILPs when ILP levels are low. That is, they assume that hyperallometry is achieved in the same manner as *Drosophila* wings in which *Inr* expression has been increased rather than as *Drosophila* wings in which *FOXO* expression has been increased (Fig. 3) [59]. Unfortunately, distinguishing between these alternatives is challenging in a non-model organism. However, it is clear that hyperallometry of the horns is not achieved by trait-specific increases in *Inr* expression as *Inr* levels are the same

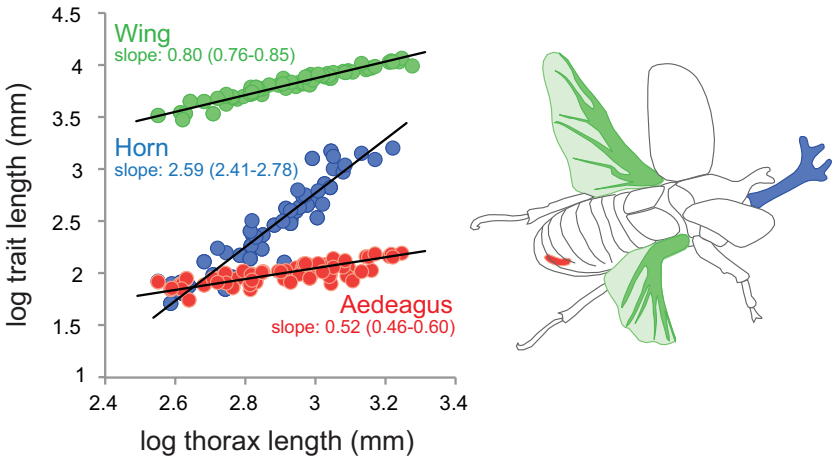

Figure 4. Morphological scaling for three traits in male rhinoceros beetles. Slopes (with 95% CI in parentheses) are calculated using standardized major axis regression, as described in [16]. Data and beetle image from [16].

in the developing horns, wings and genitals, despite their different insulin-sensitivities. Moreover, a reduction in *Inr* expression (through *Inr* RNAi) does not reduce the horn's hyperallometry, but changes only the intercept of its scaling relationship with body size [16].

Mechanisms other than insulin-signaling regulate the growth of exaggerated traits

Whilst Emlen et al.'s study implicates the IIS pathway as a mechanism targeted by selection for trait exaggeration, recent work has indicated that other hormonal systems could also be involved. In particular, the application of juvenile hormone (JH) increases the intercept but does not change the slope of hyperallometric mandibles in male stag (*Cyclommatus metallifer*) and flour (*Gnatocerus cornutus*) beetles [61, 62]. Thus, while JH does not appear to be a regulator of trait exaggeration *sensu stricto*, it seems to be involved in controlling the growth of exaggerated traits. Intriguingly, changes in diet quality also affect the intercept but not the slope of the mandible/body allometry in the flour beetle [63] and JH seems to be required to couple nutritional variation with organ growth in the tobacco hornworm (*Manduca sexta*) [64]. Together, these findings suggest that JH may increase a trait's response to nutrition across the full range of nutritional conditions without affecting its sensitivity to changes in nutrition, a subtle but important difference. Whether JH plays a similar role in rhinoceros beetle is not known, but as JH apparently affects only the intercept and not the slope, it seems unlikely that changes in JH underlies the evolution of horn hyperallometry.

Which mode of selection leads to hyperallometry and trait exaggeration?

Emlen et al.'s results are rightly and interestingly cast in the context of sexual selection for non-isometric scaling relationships. However, the particular pattern(s) of selection likely to produce exaggerated traits from an ancestral isometric condition remain unclear. The stag and flour beetle data suggest that it is possible for relative trait size to increase without hyperallometry, even for secondary sexual characteristics. Indeed, artificial selection for changes in absolute mandible size in the flour beetle affected the intercept but not the slope of the mandible's scaling-relationship with body size [65]. Similarly, artificial selection for increased relative caudal-fin size in male guppies (*Poecilia reticulata*) does not increase fin hyperallometry [24]. Both traits are used by females to assess male quality [24, 65], demonstrating that sexual selection for enlarged relative trait size need not produce trait exaggeration, i.e. need not target the developmental mechanisms leading to trait hyperallometry. Why then in the rhinoceros beetle should sexual selection appear to have focused on a developmental mechanism that increases relative trait size disproportionately in larger individuals, generating hyperallometry?

The answer may lie in a key observation made by Emlen et al.: trait enlargement through enhanced insulin-sensitivity necessarily increases relative trait size disproportionally to body size and other measures of condition. Consequently, this mechanism can generate hyperallometry as an indirect response to selection for increased relative trait size. The developmental link binding condition, body size and relative trait size has important implications for the evolution of hyperallometric traits by sexual selection. Emlen et al. argue that if insulin/IGF-signaling tracks condition, and if condition is positively correlated with body size, then inter- or intrasexual selection will favor individuals that evaluate the quality of potential mates or rivals via structures that are particularly sensitive to changes in insulin/IGF-signaling. Thus, because individual quality can be reliably assessed by the (relative) size of traits that are highly sensitive to environmentally induced variation in insulin/IGF-signaling, traits that achieve hyperallometry via insulin/IGF make good targets for sexual selection.

Conclusions

Emlen et al.'s hypothesis provides a mechanistic context for models of sexual selection that propose exaggerated traits evolve as reliable indicators of quality or condition. These models posit that signals are reliable either because they

cannot be faked (traits are ''indices'') or because they are too costly to produce in low quality signalers (traits are ''handicaps'') [29]. Since traits exaggerated through increased insulin-sensitivity do not require that the trait be physiologically costly to produce, such traits likely serve as indices of quality rather than handicaps. Of course, not all exaggerated traits may have arisen through the same evolutionary processes. For example, Fisher's runaway-process [66], does not require traits to indicate any aspect of male quality. The extent to which exaggerated sexually selected traits show heightened insulin-sensitivity in other taxa, or among exaggerated traits within taxa, is therefore an open question that demands more investigation.

Emlen et al.'s experiment, combined with the kinds of data from other systems we describe above, indicate that much can be inferred regarding the evolution of scaling relationship parameters by taking a developmental perspective. As the proximate bases of the transduction of environmental variation into size variation and covariation are further elucidated, we may be able to finally explain patterns in the evolution of morphological scaling – and thus of the evolution of diversity itself – that have vexed researchers for over a century.

Acknowledgments

We thank the editor for the invitation to write this review and Doug Emlen for kindly providing the data for Fig. 4. The writing of this review was supported by funding from the National Science Foundation to A.W.S. (IOS-0845847 and IOS-0919855) and W.A.F. (IOS-0920720).

References

1. **Bonduriansky R**, **Day T.** 2003. The evolution of static allometry in sexually selected traits. *Evolution* **57**: 2450–8.
2. **Huxley JS.** 1932. *Problems of Relative Growth*. London: Methuen & Co. Ltd.
3. **Shingleton AW**, **Estep CM**, **Driscoll MV**, **Dworkin I.** 2009. Many ways to be small: different environmental regulators of size generate distinct scaling relationships in *Drosophila melanogaster*. *Proc Biol Sci* **276**: 2625–33.
4. **Newell ND.** 1949. Phyletic size increase, an important trend illustrated by fossil invertebrates. *Evolution* **3**: 103–24.
5. **Thompson DW.** 1942. *On growth and form*. New York, Cambridge, UK: Cambridge University Press.
6. **Gould SJ.** 1966. Allometry and size in ontogeny and phylogeny. *Biol Rev* **41**: 587–638.
7. **Emlen DJ**, **Allen CE.** 2003. Genotype to phenotype: physiological control of trait size and scaling in insects. *Integr Comp Biol* **43**: 617–34.
8. **Eberhard WG.** 2009. Static allometry and animal genitalia. *Evolution* **63**: 48–66.
9. **Guthrie HA**, **Brown ML.** 1968. Effect of severe undernutrition in early life on growth, brain size and composition in adult rats. *J Nutr* **94**: 419–26.
10. **Petrie M.** 1988. Intraspecific variation in structures that display competitive ability: large animals invest relatively more. *Anim Behav* **36**: 1174–9.
11. **Petrie M.** 1992. Are all secondary sexual display structures positively allometric and, if so, why? *Anim Behav* **43**: 173–5.
12. **Gould SJ.** 1973. Positive allometry of antlers in the ''Irish Elk'', *Megaloceros giganteus*. *Nature* **244**: 375–6.
13. **Green AJ.** 2009. Multiplicative by nature: why logarithmic transformation is necessary in allometry. *J Theor Biol* **257**: 519–21.
14. **Kodric-Brown A**, **Sibly RM**, **Brown JH.** 2006. The allometry of ornaments and weapons. *Proc Natl Acad Sci USA* **103**: 8733–8.
15. **Burkhardt D**, **Motte I.** 1985. Selective pressures, variability, and sexual dimorphism in stalk-eyed flies (diopsidae). *Naturwissenschaften* **72**: 204–6.
16. **Emlen DJ**, **Warren IA**, **Johns A**, **Dworkin I**, et al. 2012. A mechanism of extreme growth and reliable signaling in sexually selected ornaments and weapons. *Science* **337**: 860–4.
17. **Nijhout HF.** 2011. Dependence of morphometric allometries on the growth kinetics of body parts. *J Theor Biol* **288**: 35–43.
18. **Lande R**, **Arnold SJ.** 1983. The measurement of selection on correlated characters. *Evolution* **37**: 1210–26.
19. **Brodie ED III.** 1992. Correlational selection for color pattern and antipredator behavior in the garter snake *Thamnophis ordinoides*. *Evolution* **46**: 1284–98.
20. **Phillips PC**, **Arnold SJ.** 1989. Visualizing multivariate selection. *Evolution* **43**: 1209–22.
21. **Schluter D.** 1996. Adaptive radiation along genetic lines of least resistance. *Evolution* **50**: 1766–74.
22. **Frankino WA**, **Zwaan BJ**, **Stern DL**, **Brakefield PM.** 2005. Natural selection and developmental constraints in the evolution of allometries. *Science* **307**: 718–20.
23. **Frankino WA**, **Zwaan BJ**, **Stern DL**, **Brakefield PM.** 2007. Internal and external constraints in the evolution of morphological allometries in a butterfly. *Evolution* **61**: 2958–70.
24. **Egset C**, **Hansen T**, **Le Rouzic A**, **Bolstad GH**, et al. 2012. Artificial selection on allometry: change in elevation but not slope. *J Evol Biol* **25**: 938–48.
25. **Lupold S**, **Mcelligott A**, **Hosken D.** 2004. Bat genitalia: allometry, variation and good genes. *Biol J Linn Soc* **83**: 497–507.
26. **Tatsuta H**, **Mizota K**, **Akimoto S.** 2001. Allometric patterns of heads and genitalia in the stag beetle *Lucanus maculifemoratus* (Coleoptera: Lucanidae). *Ann Entomol Soc Am* **94**: 462–6.
27. **Bonduriansky R.** 2007. Sexual selection and allometry: a critical reappraisal of the evidence and ideas. *Evolution* **61**: 838–49.
28. **Eberhard WG**, **Huber BA**, **Rodriguez RL**, **Briceño RD**, et al. 1998. One size fits all? Relationships between the size and degree of variation in genitalia and other body parts in twenty species of insects and spiders. *Evolution* **52**: 415–31.
29. **Smith JM**, **Harper DD.** 2003. *Animal Signals*. USA: Oxford University Press.
30. **Andersson M.** 1986. Evolution of condition-dependent sex ornaments and mating preferences: sexual selection based on viability differences. *Evolution* **40**: 804–16.
31. **Tomkins JL**, **Radwan J**, **Kotiaho JS**, **Tregenza T.** 2004. Genic capture and resolving the lek paradox. *Trends Ecol Evol* **19**: 323–8.
32. **Lorch PD**, **Proulx S**, **Rowe L**, **Day T.** 2003. Condition-dependent sexual selection can accelerate adaptation. *Evol Ecol Res* **5**: 867–81.
33. **Iwasa Y**, **Pomiankowski A.** 1999. Good parent and good genes models of handicap evolution. *J Theor Biol* **200**: 97–109.
34. **Parker GA.** 1974. Assessment strategy and the evolution of fighting behaviour. *J Theor Biol* **47**: 223–43.
35. **Zahavi A.** 1975. Mate selection—A selection for a handicap. *J Theor Biol* **53**: 205–14.
36. **Searcy WA**, **Nowicki S.** 2010. *The Evolution of Animal Communication: Reliability and Deception in Signaling Systems*. Princeton, NJ: Princeton University Press.
37. **Shingleton AW**, **Frankino WA**, **Flatt T**, **Nijhout HF**, et al. 2007. Size and shape: the developmental regulation of static allometry in insects. *BioEssays* **29**: 536–48.
38. **Oldham S**, **Hafen E.** 2003. Insulin/IGF and target of rapamycin signaling: a TOR de force in growth control. *Trends Cell Biol* **13**: 79–85.
39. **Goberdhan DCI**, **Wilson C.** 2002. Insulin receptor-mediated organ overgrowth in *Drosophila* is not restricted by body size. *Dev Genes Evol* **212**: 196–202.
40. **Claeys I**, **Simonet G**, **Poels J**, **Van Loy T**, et al. 2002. Insulin-related peptides and their conserved signal transduction pathway. *Peptides* **23**: 807–16.
41. **White MF.** 1998. The IRS-signalling system: a network of docking proteins that mediate insulin action. *Mol Cell Biochem* **182**: 3–11.
42. **Dupont J**, **Holzenberger M.** 2003. Biology of insulin-like growth factors in development. *Birth Defect Res C* **69**: 257–71.
43. **Grewal SS.** 2009. Insulin/TOR signaling in growth and homeostasis: a view from the fly world. *Int J Biochem Cell Biol* **41**: 1006–10.
44. **Brogiolo W**, **Stocker H**, **Ikeya T**, **Rintelen F**, et al. 2001. An evolutionarily conserved function of the *Drosophila* insulin receptor and insulin-like peptides in growth control. *Curr Biol* **11**: 213–21.
45. **Taniguchi CM**, **Emanuelli B**, **Kahn CR.** 2006. Critical nodes in signalling pathways: insights into insulin action. *Nat Rev Mol Cell Biol* **7**: 85–96.
46. **Ikeya T**, **Galic M**, **Belawat P**, **Nairz K**, et al. 2002. Nutrient-dependent expression of insulin-like peptides from neuroendocrine cells in the CNS contributes to growth regulation in *Drosophila*. *Curr Biol* **12**: 1293–300.

Recently in press

47. **Slaidina M**, **Delanoue R**, **Gronke S**, **Partridge L**, et al. 2009. A *Drosophila* insulin-like peptide promotes growth during nonfeeding states. *Dev Cell* **17**: 874–84.
48. **Arquier N**, **Geminard C**, **Bourouis M**, **Jarretou G**, et al. 2008. *Drosophila* ALS regulates growth and metabolism through functional interaction with insulin-like peptides. *Cell Metab* **7**: 333–8.
49. **Geminard C**, **Arquier N**, **Layalle S**, **Bourouis M**, et al. 2006. Control of metabolism and growth through insulin-like peptides in *Drosophila*. *Diabetes* **55**: S5–8.
50. **Sutter NB**, **Bustamante CD**, **Chase K**, **Gray MM**, et al. 2007. A single IGF1 allele is a major determinant of small size in dogs. *Science* **316**: 112–5.
51. **Shingleton AW**, **Mirth CK**, **Bates PW.** 2008. Developmental model of static allometry in holometabolous insects. *Proc Biol Sci* **275**: 1875–85.
52. **Nijhout HF.** 2003. The control of body size in insects. *Dev Biol* **261**: 1–9.
53. **Emlen DJ**, **Nijhout HF.** 2000. The development and evolution of exaggerated morphologies in insects. *Annu Rev Entomol* **45**: 661–708.
54. **Cheng CM**, **Reinhardt RR**, **Lee WH**, **Joncas G**, et al. 2000. Insulin-like growth factor 1 regulates developing brain glucose metabolism. *Proc Natl Acad Sci USA* **97**: 10236–41.
55. **Cheng LY**, **Bailey AP**, **Leevers SJ**, **Ragan TJ**, et al. 2011. Anaplastic lymphoma kinase spares organ growth during nutrient restriction in *Drosophila*. *Cell* **146**: 435–47.
56. **Tang HY**, **Smith-Caldas MSB**, **Driscoll MV**, **Salhadar S**, et al. 2011. FOXO regulates organ-specific phenotypic plasticity in *Drosophila*. *PLoS Genet* **7**: e1002373.
57. **Cotton S**, **Fowler K**, **Pomiankowski A.** 2004. Condition dependence of sexual ornament size and variation in the stalk-eyed fly *Cyrtodiopsis dalmanni* (Diptera: Diopsidae). *Evolution* **58**: 1038–46.
58. **Emlen D**, **Hunt J**, **Simmons L.** 2005. Evolution of sexual dimorphism and male dimorphism in the expression of beetle horns: phylogenetic evidence for modularity, evolutionary lability, and constraint. *Am Nat* **166**: S42–68.
59. **Shingleton AW**, **Tang HY.** 2012. Plastic flies: the regulation and evolution of trait variability in *Drosophila*. *Fly (Austin)* **6**: 1–3.
60. **Karino K**, **Niiyama H**, **Chiba M.** 2005. Horn length is the determining factor in the outcomes of escalated fights among male Japanese horned beetles, *Allomyrina dichotoma* L. (Coleoptera: Scarabaeidae). *J Insect Behav* **18**: 805–15.
61. **Gotoh H**, **Cornette R**, **Koshikawa S**, **Okada Y**, et al. 2011. Juvenile hormone regulates extreme mandible growth in male stag beetles. *PLoS ONE* **6**: e21139.
62. **Okada Y**, **Gotoh H**, **Miura T**, **Miyatake T**, et al. 2012. Juvenile hormone mediates developmental integration between exaggerated traits and supportive traits in the horned flour beetle *Gnatocerus cornutus*. *Evol Dev* **14**: 363–71.
63. **Okada K**, **Miyatake T.** 2010. Plasticity of size and allometry in multiple sexually selected traits in an armed beetle *Gnatocerus cornutus*. *Evol Ecol* **24**: 1339–51.
64. **Truman JW.** 2006. Juvenile hormone is required to couple imaginal disc formation with nutrition in insects. *Science* **312**: 1385–8.
65. **Okada K**, **Miyatake T.** 2009. Genetic correlations between weapons, body shape and fighting behaviour in the horned beetle *Gnatocerus cornutus*. *Anim Behav* **77**: 1057–65.
66. **Fisher RA.** 1930. *The genetical theory of natural selection*. USA: Oxford University Press.
67. **Koh I**, **Lee M**, **Lee N**, **Park K**, et al. 2005. Body size effect on brain volume in Korean youth. *Neuroreport* **16**: 2029–32.
68. **Kramer JM**, **Davidge JT**, **Lockyer JM**, **Staveley BE.** 2003. Expression of *Drosophila* FOXO regulates growth and can phenocopy starvation. *BMC Dev Biol* **3**: 5.
69. **Puig O**, **Marr MT**, **Ruhf ML**, **Tjian R.** 2003. Control of cell number by *Drosophila* FOXO: downstream and feedback regulation of the insulin receptor pathway. *Genes Dev* **17**: 2006–20.
70. **Huxley J**, **Tessier G.** 1936. Terminology of relative growth. *Nature* **137**: 780–1.
71. **Huxley J.** 1924. Constant differential growth-ratios and their significance. *Nature* **114**: 895–6.
72. **Kerkhoff AJ**, **Kerkhoff AJ**, **Enquist BJ.** 2009. Multiplicative by nature: why logarithmic transformation is necessary in allometry. *J Theor Biol* **257**: 519–21.
73. **Knell RJ.** 2009. On the analysis of non-linear allometries. *Ecol Entomol* **34**: 1–11.
74. **Warton DI**, **Wright IJ**, **Falster DS**, **Westoby M.** 2006. Bivariate line-fitting methods for allometry. *Biol Rev* **81**: 259–91.
75. **Hepburn HR**, **Radloff SE**, **Fuchs S.** 1999. Flight machinery dimensions of honeybees, *Apis mellifera*. *J Comp Physiol B* **169**: 107–12.