Ann. N.Y. Acad. Sci. ISSN 0077-8923

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES
Issue: *The Year in Evolutionary Biology*

Evolution of morphological allometry

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Morphological allometry refers to patterns of covariance between body parts resulting from variation in body size. Whether measured during growth (ontogenetic allometry), among individuals at similar developmental stage (static allometry), or among populations or species (evolutionary allometry), allometric relationships are often tight and relatively invariant. Consequently, it has been suggested that allometries have low evolvability and could constrain phenotypic evolution by forcing evolving species along fixed trajectories. Alternatively, allometric relationships may result from natural selection for functional optimization. Despite nearly a century of active research, distinguishing between these alternatives remains difficult, partly due to wide differences in the meaning assigned to the term *allometry*. In particular, a broad use of the term, encompassing any monotonic relationship between body parts, has become common. This usage breaks the connection to the proportional growth regulation that motivated Huxley's original narrow-sense use of allometry to refer to power–law relationships between traits. Focusing on the narrow-sense definition of allometry, we review here evidence for and against the allometry-as-a-constraint hypothesis. Although the low evolvability and the evolutionary invariance of the static allometric slope observed in some studies suggest a possible constraining effect of this parameter on phenotypic evolution, the lack of knowledge about selection on allometry prevents firm conclusions.

Keywords: adaptation; allometry; evolutionary constraint; growth; macroevolution; microevolution; scaling; shape; size

Introduction

Allometry, in a broad sense, refers to the positive relationships generally observed between body size and other organismal traits. Allometry is important because variation in a wide variety of morphological, physiological, and life-history traits is highly correlated with variation in organism size.[1–3] These relationships generate intuitive hypotheses for understanding trait variation; for example, the fact that elephants are larger than mice can be used to explain why the brain mass of elephants is larger than the brain mass of mice. In many cases, however, traits do not enlarge proportionally to overall size, but instead follow a power–law function of the form $Z = aX^b$ (Box 1), where the trait value is Z, the body size is X, and a and b are parameters describing the relationship. If $b = 1$, the trait changes in proportion to body size, a condition referred to as isometry. When $b \neq 1$, trait size and body size will vary in different proportions and the shape of the organism will change with a change in size. For example, it has been argued that brain mass in mammals scales with body mass with a coefficient $b \approx 0.75$;[4] as a result, for the same unit increase in body mass, a larger organism will have a smaller increase in brain mass than a smaller organism. Consequently, relative to body mass, the brain mass of an elephant is about 14 times smaller than that of a mouse, while the brain mass of a human is similar to that of a mouse. Although allometry ($b \neq 1$) is often contrasted with isometry ($b = 1$), the study of allometry includes both

doi: 10.1111/nyas.12470

isometric and allometric relationships. Analyzed on a log scale, allometric relationships become linear: $\log(Z) = \log(a) + b \times \log(X)$, where $\log(a)$ is the allometric intercept and the allometric exponent b is often called the allometric slope.

Because body size can vary with age or developmental stage, across individuals, and across populations and species, three types of allometry have been defined: ontogenetic allometry refers to the relationship between the trait and size during growth; static allometry refers to the relationship between the trait and size observed across individuals measured at a similar developmental stage; and evolutionary allometry refers to the relationship observed among population or species means.[5–7]

Allometric relationships often fit very precisely over large size ranges, which may be found across ontogeny or across species. Furthermore, ontogenetic and static allometric slopes usually vary little among closely related species.[5] These observations support the idea that allometric relationships may reflect strong physical, physiological, or other biological mechanisms that constrain the rate and direction of evolution.[5,8–12] Accordingly, allometric relationships have been taken as prime evidence against the dominance of natural selection as an evolutionary force.[13] A role for allometric constraints has been widely accepted for physiological and life-history traits,[14,15] but has been more controversial for morphological traits, where allometries are usually thought of as a result of different body parts being under common growth regulation (see Box 1).

The alternative is that natural selection persistently favors the particular scaling relationships that are observed between traits and size. This hypothesis is at least implicitly accepted in traditional studies of functional allometry.[2] For example, the relationship between the cross-sectional area of the skeleton with body mass in terrestrial vertebrates may be explained with respect to optimal allocation, balancing the chances of breakage—favoring a robust skeleton—and the costs of locomotion favoring a lighter skeleton.[16] Accordingly, several authors have thought that allometric slopes evolve adaptively.[17–20] In this context, the evolution of the static allometry of primary and secondary sexual characters has been a popular topic. Secondary sexual displays have been predicted to evolve steep slopes (i.e., positive allometry: $b > 1$) under handicap models,[17,18,21–23] while male genitalia have been predicted to show negative allometry ($b < 1$) due to stabilizing selection on trait size.[19,24,25]

In principle, it should be possible to distinguish between constraint and selective explanations for allometric relationships by quantifying the evolvability of allometry and the selection acting upon it. The predictions from this can then be compared with observed patterns of evolution in allometry. The general prediction is that a constrained allometry will either be incapable of evolving (an absolute constraint[26]) or that fitness will decrease so rapidly when moved away from the optimal value that the possible advantages of the altered allometry would not outweigh the fitness costs. Unfortunately, despite renewed interest in the last two decades, progress in understanding the evolution of morphological allometry has been slow. A key reason for this is the rise of a very general notion of allometry that encompasses any monotonic relationship between trait size and body size[27,28] (Box 1). The consequence of this is that many studies of allometry become studies of shape evolution that are not directly informative about the existence or the evolution of the scaling relations that are the essence of the original meaning of allometry (Box 2). Importantly, the hypothesis that traits are constrained to follow a power law is not directly tested by studies of variation in or evolution of shape in general. Our aim in the current review is to develop predictions from the constraint and selection hypotheses on morphological narrow-sense allometries and review the recent literature in light of these predictions.

Evolutionary constraint and allometry

Evolutionary constraints are processes that preclude a trait from reaching a phenotypic optimum or slow down its evolution toward this optimum.[29,30] Depending on the perspective and processes involved, many types of constraint have been defined.[30–32] Allometry has been classified as a developmental constraint, that is, a constraint imposed by the developmental architecture in the production of variant phenotypes.[11] Although developmental constraints may themselves result from selection,[33,34] they may still limit the direction of phenotypic evolution. Such a perspective of allometry as a developmental constraint corresponds to the view defended by early students of allometry and heterochrony who explained patterns of species divergence as changes

Box 1. Narrow-sense and broad-sense allometry: conceptual and methodological issues

In recent years, the term *allometry* has been used for any type of monotonic relationship between two morphological variables, independent of the scale on which these variables are expressed. This broad definition of allometry is inconsistent with the biological interpretation of morphological allometry originally suggested.[10,112] Huxley[10] showed that if an arbitrary trait Z and body size X grow at different rates but under the control of a common growth parameter G, such that $dX/dt = \alpha XG$, and $dZ/dt = \beta ZG$, where α and β are specific constants for X and Z, respectively, and t the time during growth, the relationship between Z and X follows a power law $Z = AX^b$, where A is a constant that depends on the initial values of Z and X, and $b = \beta/\alpha$. On a log scale, this relationship is linear $z = a + bx$, where $z = \log(Z)$, $x = \log(X)$, $a = \log(A)$. A later generalization of this model showed that whenever two or more variables are connected in a dynamic synergistic system controlled by one variable, their relationship follows an allometric relationship.[113]

Therefore, allometry in its narrow sense is defined by two parameters of a power function that can be expressed as the intercept *a,* and the slope *b,* of a linear regression on log scale. When the explanatory variable x is mean centered, the intercept a becomes the elevation of the static allometry, that is, the trait size at the population mean body size.[114–116] By using linear regression on log-transformed data, the size of the trait on the y axis (response variable) can be predicted from the size of the trait on the x axis (predictor variable). Consequently, the choice of the predictor variable is not arbitrary but reflects our knowledge or intuition that variation in this trait will more accurately reflect differences in the growth parameter G than will variation in the trait used as a response variable. Body size or some proxy measurements are generally used as a predictor variable under the assumption that variation in these traits best reflects variation in overall growth (see Box 2 for a brief discussion on size).

The specific statistical model that should be selected to estimate allometric slope and intercept has been the subject of long debates. Packard suggested that the estimation of these parameters should be done from a power function fitted on arithmetic scale.[117,118] This approach would be justified if the processes producing the error (the variation not explained by the model) act in an additive manner. Although this may be the case for measurement error, it is most likely that biological error will be generated by multiplicative processes similar to those responsible for the growth of the trait.[119] Because most of the error in the response variable is likely to be of biological origin, estimates produced by linear regression on log scale should better estimate the true relationship than those produced by a power model fitted on arithmetic scale. Both approaches are valid, however, and the choice of model should ideally be conducted with proper modeling of both biological and measurement error, when those can be distinguished.

Major-axis and reduced major-axis regression are often used in place of ordinary least-squares regression to estimate allometric parameters. This practice seems based on the belief that these procedures correct for observational errors in the predictor variables or that they are more appropriate when there is no causal direction to the relation between the traits in question. It is important to realize that neither of these methods provides sensible estimates of allometric regression slopes when there is biological error (i.e., biological deviations from the allometric line) in the model.[105,120] These models compute slopes, but these slopes are not proper estimates of the exponent b of the narrow-sense allometry. A dramatic consequence of the reduced major-axis regression is that strong allometries are typically found even in the total absence of covariance between the two traits.

Finally, we notice that ignoring the consequences of trait dimension on the allometric slope has led to some erroneous statements in the literature on allometry. For example, a positive allometry between testes size and body length has been reported in the Hottentot golden mole (*Amblysomus hottentotus*) as an exception to the one-size-fits-all hypothesis.[121] However, this positive allometry was most likely generated by the difference in dimension between the traits (body length – length vs. testes mass – volume).

in timing or rate of development along constant ontogenetic trajectories.[5,8,10,35,36]

The introduction of the quantitative genetics framework in the study of allometry[6,37–39] shifted the hypothesis of allometry as a constraint from a developmental to a genetic perspective. Genetic constraints occur when the amount or pattern of genetic variation limits or channels the response to

Box 2. From bivariate to multivariate allometry: drifting away from Huxley's model

Jolicoeur[122] suggested analyzing multivariate relative growth using the first principal component (or eigenvector) of the variance–covariance matrix of log-scaled trait values (herein referred to as PC1). He showed the relation between this approach and the Huxley model,[10] arguing that the trait's loadings on PC1 are equivalent to their allometric exponents. This approach formalized multivariate isometric variation as a PC1 vector with all elements or loadings equal to $1/\sqrt{k}$, where k is the number of traits measured. Variation along such a $\{1/\sqrt{k}, \ldots, 1/\sqrt{k}\}$ vector is associated with variation in organism size, assuming an isometric relationship of all parts to size. Group difference in multivariate allometry is commonly estimated as the angle between two within-group PC1 in the multivariate space.[6,79] This approach has been used to describe the plasticity of static multivariate allometry of *Drosophila* exposed to different environmental conditions[94] or to investigate the diversity of allometric variation among species.[123,124] However, the biological meaning of the PC1 loadings in this approach is not equivalent to that of the standard regression estimates (i.e., Huxley's allometric exponent). Instead, these estimates are the slopes of the regressions of standardized trait variables on size, as defined by PC1. Therefore, the ratios of the loadings between pairs of variables do not correspond to their bivariate allometric coefficient as estimated by standard regression procedure (Box 1) but to the ratio of their respective covariances with PC1. This can be interpreted as the relative change in the two traits for a given change along PC1.[6] However, the orientation of the PC1 vector in the morphospace will vary with an increase in trait(s) variance even if covariances are kept constant.

This raises the problem of the use of an adequate definition of size in the study of its relation with shape (see Bookstein[125] for review). The two most commonly used approaches for estimating size are the PC1-as-size approach described above and Mosimann's[126] definition. Mosimann defined size as any function G of the measurement vector x that satisfies the property: $G(a\mathrm{x}) = aG(\mathrm{x})$, $G(\mathrm{x})$ having the same dimension as any element of x. Indeed, shape being on a ratio scale, multiplication of each element of the ratios by a constant a does not change shape, and G is uncorrelated with any ratios under fully isometric multivariate variation. It is worth noticing that Huxley[10] already defined traits' growth rates as affected by a common growth factor (G) that vanishes when the growth rates of the traits are put in relation to each other in the exponential relationship (Box 1).

Contrary to the PC1 approach, this geometric definition of size is not directly dependent on the variance–covariance properties of the studied samples. As a measure of size, geometric morphometrics conventionally use centroid size (CS), the square root of the sum of squared distances of a set of landmarks from the centroid of the entire landmark configuration. Although this choice does not generally rely on biological arguments, CS corresponds to a size vector, according to Mosimann's definition. The Procrustes superimposition algorithm scales landmark coordinates with CS, providing Mosimann's shape vectors. The effect of CS on remaining shape variation therefore corresponds to multivariate allometry.

Under specific conditions, a multivariate regression of shape on size, both on log scale or mean scaled, represents the multivariate approach that is most closely related to the original bivariate model from Huxley. On the other hand, the broadly used geometric morphometric approach abandons the notion of trait for a notion of shape analyzed as a whole and hampers interpretations of allometry in a context of relative growth.

selection.[30] Although these two perspectives are, in fact, linked because genetic constraints must result from developmental constraints, this change in perspective may have weakened the constraint hypothesis, given the ubiquity of genetic variation generally found.[40] More recently, the hypothesis of constraint due to low genetic variation has been refueled by the realization that genetic correlations among traits due to pleiotropy could seriously influence the direction of phenotypic evolution,[26,41–49] allometry being a perfect example of such a constraint.

Testing whether allometry represents an evolutionary constraint using quantitative genetics approaches requires quantifying the evolvability of allometry and comparing the observed evolutionary patterns with those patterns expected under specific selection pressures. If we are to test the allometry-as-a-constraint hypothesis, we should therefore

answer the following questions: Is allometry evolvable? Does allometry constrain patterns of phenotypic evolution? Data on static allometry play a central role for these questions, because it is the level at which developmental constraints can be easily measured and allows predictions about phenotypic evolution.

Is static allometry evolvable?

Testing whether a trait is evolvable can be achieved by quantifying either its propensity to vary (i.e., its evolvability[50]) or its actual evolution, that is, quantifying the changes in the trait when selected. In the following section, after considering the possible sources of constraint and selection acting on static allometry, we review evidence for both genetic variation (evolvability) and evolutionary changes in static allometry.

Sources of constraint

Evolution of static allometry depends on the evolution of the static allometric slope and intercept. One key challenge is that an individual organism expresses neither a slope nor an intercept. Doing standard quantitative genetics on these traits therefore requires groups of genetically related organisms of different sizes or at least repeated parts of modular organisms. Several growth models have been developed to explain how static allometry is generated from simple growth patterns.[10,22,36,51,52] Using these models, one can identify possible constraints on the variation of static allometry.

From Huxley's[10] model of relative growth (Box 1), it can be shown that when two traits expressed on log scale present an ontogenetic allometry, such as $z = a + bx$, with x and z varying during growth, the static allometric slope at any specific time t can be expressed as:

$$b_s = \bar{b} + \frac{\sigma(x_t, a) + \bar{x}_t \sigma(x_t, b)}{\sigma^2(x_t)}, \qquad (1)$$

where $\bar{b}$ is the ontogenetic allometric slope averaged across all individuals (i.e., the mean ontogenetic allometry of the population), and $\bar{x}_t$, the mean body size at time t.[53] This shows that the static allometric slope is affected by both the average slope of the ontogenetic allometry and the covariance between the parameters of the ontogenetic allometry (slope and intercept) and body size. Similarly, parameters of the ontogenetic allometry will affect the average value of the trait. This effect can be expressed with respect to the mean trait at the population mean body size $\bar{x}_t$ in the following way:

$$\bar{z}_t = \bar{a} + E(bx_t) = \bar{a} + \bar{b}\bar{x}_t + \sigma(x_t, b), \qquad (2)$$

where $\bar{a}$ is the average ontogenetic intercept across all individuals. Therefore, a positive covariance between the ontogenetic slope and body size at stage t will increase the trait mean value, while a negative covariance will decrease it. This model (Eqs. (1) and (2)) illustrates how variation in the static allometric slope and intercept can be generated by variation in the ontogenetic parameters. Importantly, it also shows that when ontogenetic and static allometries are different ($\sigma(x_t, b) \neq 0$ or $\sigma(x_t, a) \neq 0$ in Eq. (1)), invariance of static allometry across populations with different mean body sizes implies changes in the ontogenetic parameters.[53] Comparing ontogenetic and static allometries and estimating the variational properties of the ontogenetic parameters should therefore provide valuable insights into the possibility for static allometry to evolve.

Different body parts do not always grow in concert; some organs grow most rapidly early in development (e.g., the mammalian brain[54]) while other organs continue to grow even after overall body mass has leveled off (e.g., appendages in holometabolous insects,[55] secondary sexual traits in vertebrates,[56] and the special case of deer antlers[57]). Ontogenetic allometries resulting from this type of growth pattern are necessarily nonlinear[51,52] and sometimes the link between ontogenetic and static allometry can be difficult to establish. Nevertheless, even in such cases, the growth of the trait may be coordinated with the expected or achieved body size, despite the difference in the timing of growth (see Bondurinasky and Day[22] for such a model), and variation in the trait growth sensitivity to the growth of the whole organism could generate variation in the allometric slope. The difference in sensitivity to insulin observed among imaginal discs in holometabolous insects is an example of such a process[58] (and see below). In such cases, the lack of genetic variance in trait growth sensitivity to the size of the body, or the sharing of a large proportion of the developmental pathway between the two traits considered, may seriously constrain changes in static allometry. Importantly, such constraints would affect the allometric slope but not necessarily the intercept (i.e., mean trait size).

The final size of an organ may also be molded by a secondary loss of part of the structure via programmed cell death, apoptosis. For example, in the horned beetle genus *Onthophagus*, programmed cell death is responsible for sex- and species-specific horn development, with thoracic horns being partly or completely reabsorbed during the pupal stage.[59] Similarly, the complex shape of the hindwing anal lobe of the butterfly *Battus philenor* is generated by apoptosis along the wing margin during pupal wing development.[60] In these cases, tight allometric relationships between traits molded by apoptosis would reflect common regulation of cell death as well as growth. It remains unknown, however, whether traits regulated by apoptosis display tight allometric relationships with body size. Head horns in male *Onthophagus taurus*, for which a tight nonlinear allometric relationship with body size is observed,[61] show limited, if any, apoptosis.[59] It is, therefore, possible that programmed cell death is partly responsible (in species where it occurs) for the weak allometric relationships observed between some traits.

Sources of selection

Selection on trait and body size. Changes in the allometric slope may occur as side effects of directional selection on trait or body size. According to the above model (Eqs. (1) and (2)), ontogenetic and static allometry should be similar, and changes in body size should not affect the static allometric slope in the absence of covariance between body size and the ontogenetic parameters (Fig. 1, scenario A). If the covariance between body size and the slope or the intercept of the ontogenetic allometry is nonzero, changes in body size should generate changes in static allometric slope (Fig. 1, scenario B). Selection on the mean trait $\bar{z}_t$ is expected to affect either the mean ontogenetic intercept $\bar{a}$ (Fig. 1, scenario C), the mean body size $\bar{x}_t$ (Fig. 1, scenario A), the mean ontogenetic slope $\bar{b}$, the covariance between these last two terms, $\sigma(b, x_t)$, or any combination of these parameters. Changes in static allometric slope with increasing body size may also occur if there is a nonlinear ontogenetic relationship on a log scale between size and trait.[53]

Selection on the slope. The static allometric slope can evolve because of selection on the slope itself, with no change in $\bar{x}_t$ or $\bar{z}_t$ (Fig. 1, scenario D). This can happen if the bivariate distribution of the traits evolves to fit an adaptive ridge (Fig. 2A). We can completely separate selection on the allometric slope from selection on trait means by imagining an adaptive landscape that selects on trait (co)variances but not on trait mean (i.e., no directional selection). Because trait means evolve more rapidly toward fitness optima than trait variation, situations where only the (co)variances continue to evolve should be frequent. In theory, the presence of a selective ridge should be sufficient to favor a particular slope. However, under such a scenario, the strength of selection on the slope will be weak, because many individuals at the center of the distribution (i.e., with noninformative genotype) have high fitness, and stabilizing selection on one trait will lead to an indirect stabilizing selection on the other trait (Fig. 2A). Alternatively, selection for a decrease in trait variance combined with selection to maintain the variance in body size should lead to a decrease in the allometric slope, while selection for increasing variance in trait size (e.g., disruptive selection) combined with a decreasing variance in body size should increase the allometric slope (Fig. 2B). However, strong disruptive selection balanced precisely to generate no directional selection is probably an extremely rare event in nature.

Verbal models of selection on allometric slope have been proposed to explain the positive allometry often observed in sexually selected traits.[17,18,21,23,62] In these models, positive allometry evolves either because large males, presumably in better condition, are able to invest more in costly traits, or because the benefits of large traits increase with body size. The combination of various selection regimes with an allocation model of growth has suggested that the conditions under which positive allometry evolved were more restrictive than previously thought, but that an increase in the relative fitness gain for larger traits with an increase in body size should select for positive allometry.[22] Canalizing selection on trait size, on the other hand, has been suggested to generate negative allometries (e.g., in male copulatory organs in insects).[19,25]

The variational properties that generate variance and covariance among traits are therefore central to the evolution of static allometry. Some selection experiments have suggested that variation may respond to stabilizing and disruptive selection.[63] It remains unclear, however, how efficiently selection can mold genetic and environmental variation,[64–67]

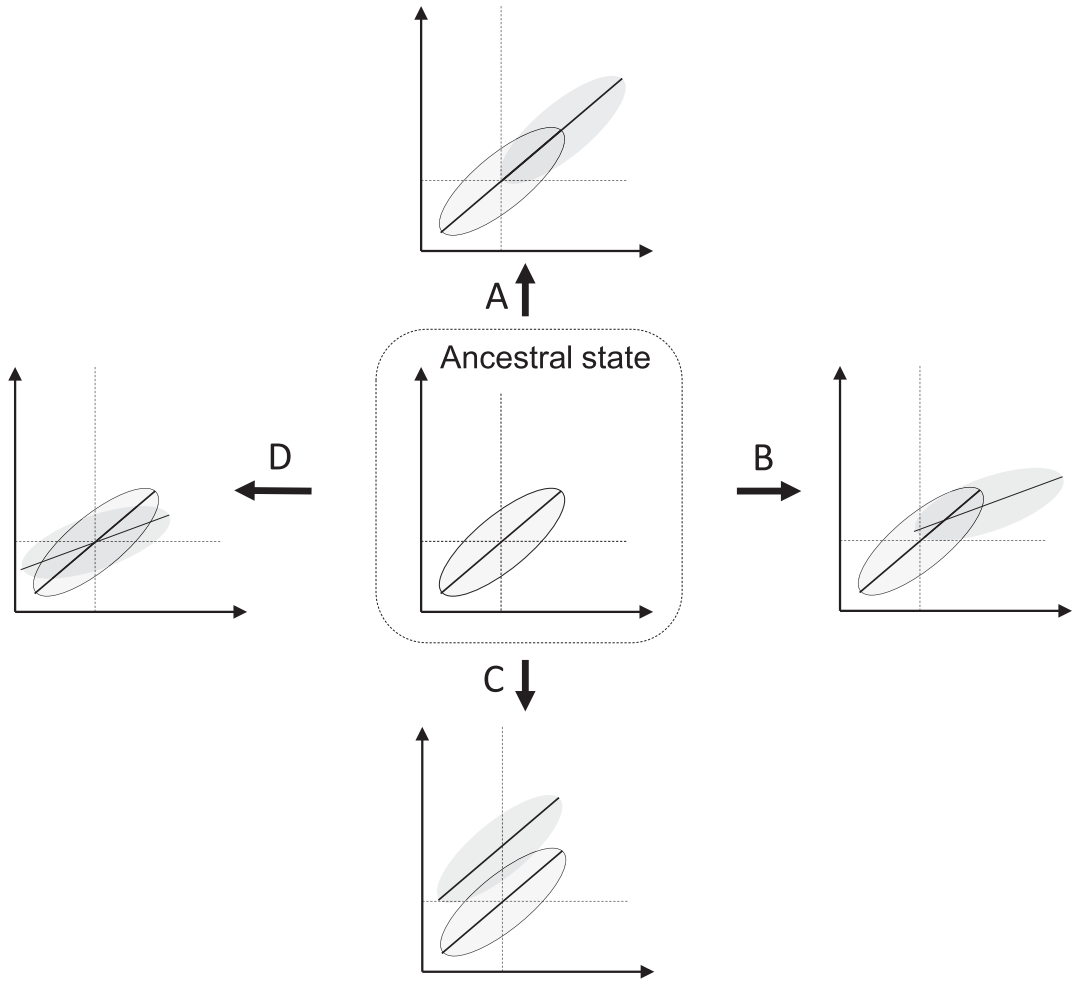

Figure 1. Evolution of static allometry. The scenarios considered here correspond to the expected evolution of static allometry from an ancestral state, depending on which parameters are constrained. Vertical and horizontal dashed lines represent the mean trait value and body size. Body size is mean centered, so the allometric intercept represents the trait value at the population mean (i.e., elevation of the allometric regression). In scenario A, both the slope and the intercept are constrained. Any change in body size will generate a change in trait size, and vice versa. In scenario B, the slope and intercept evolve. This pattern may be generated by the effect of a negative covariance between body size and the ontogenetic slope when selection on body size occurs. In scenario C, the slope is the constraining parameter, while the intercept can change. Finally, in scenario D, only the allometric slope can evolve, while the intercept (elevation) cannot.

and how covariation can respond to selection given that covariation may change without changes in the variational properties of individual traits. The evolution of covariance between trait size and body size directly links the evolution of the static allometric slope with the evolution of the phenotypic and genetic covariance matrices (**P** and **G** on log scale). Theoretical and empirical work shows that genetic covariances do evolve under selection,[65,68–72] but the responses to selection are likely to depend on genetic details, and it is unclear how rapidly they can take place. The various hypotheses listed here suggest that, in many cases, selection on the allometric slope is indirect and inefficient. If combined with a low evolvability, this may result in slow and erratic evolutionary changes in allometry.

Evolvability and evolution in static allometry

Because changes in ontogenetic allometry are expected to induce changes in static allometry (Eq. (1)), we first consider here evidence for the evolution of ontogenetic allometry. Such evidence

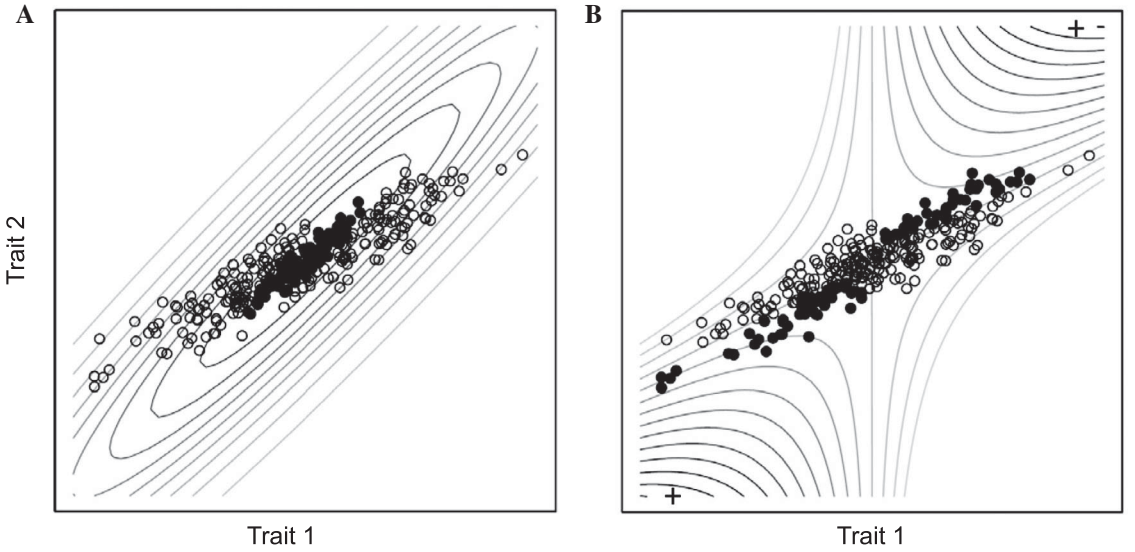

Figure 2. **Fitness landscape to change allometric slope via correlated selection on the trait and body size. (A) Selection is generated by the difference in direction between the phenotypic regression and the adaptive ridge. Black dots represent selected individuals (with the highest fitness). (B) Saddle fitness landscape that generates disruptive selection on one of the two traits (plus signs indicate regions of high fitness).**

is provided by several studies comparing ontogenetic allometries among sexes within[73] or among species.[74–77] However, few studies have tested the relationship between ontogenetic and static allometry,[6,7,78–80] and fewer have analyzed how variation in the parameters of ontogenetic allometry affects static allometry. The only study, to our knowledge, that compared the relationship between ontogenetic and static allometry among populations[53] showed that static allometry between caudal fin length and body length in female guppies (*Poecilia reticulata*) was similar among three populations despite differences in their mean ontogenetic allometry. This resulted from a negative covariance between adult body size and the steepness of the ontogenetic allometry, both within and among populations. The relevance of these results regarding the rate at which ontogenetic allometry evolves is unclear, however, because guppy populations from different drainages may have been separated for hundreds of thousands of generations.[81] Furthermore, although individual variation in ontogenetic allometry has been observed,[53] the genetic basis of this variation remains unknown. Overall, we found only one study that reported heritability of ontogenetic slopes. Atchley and Rutledge[82] reported heritabilities for chest circumference ($h^2 = 0.25 \pm 0.07$) and tail length ($h^2 = 0.39 \pm 0.08$) on body weight within six laboratory strains of rats selected for larger or smaller weight. Interpreting these results with respect to evolvability is difficult, however, because variance estimates were not provided.

Microevolutionary changes in phenotypic and genetic covariances among traits are suggested by studies reporting rapid changes in P- or G-matrices within populations,[83–85] but these observations are difficult to interpret with respect to evolution of allometry because traits are not always strongly correlated in the first place and G-matrices are rarely analyzed on log scale. Furthermore, studies analyzing changes in the P-matrix cannot distinguish between environmental and genetic changes in the patterns of covariation among traits.

Quantitative genetic estimates of genetic variance in static allometric slopes are scarce. We are only aware of a single study reporting heritabilities of allometric slopes measured on log scale.[86] In this study, heritabilities of the static allometric slope between the length of several bones and the cubic root of body weight, and between the weight of internal organs and body weight, were all statistically significant but relatively small (all h^2 less than 0.20), despite being measured under lab conditions.

Alternatively, artificial selection can be used to uncover genetic variation and covariation in quantitative traits.[87] During the last two decades, several authors have claimed to alter patterns of static allometry by artificial selection. In addition, a few artificial selection studies on size and experimental evolution have shown changes in the covariance patterns between the size of some traits and body size. Most of these studies have adopted the broad-sense definition of allometry and are not directly informative about the evolution of narrow-sense allometry.[27,28] For example, several experiments have exerted selection on the ratio between two traits.[88–90] Although changes in the intercept of the relationships between traits on the arithmetic scale have been observed in these studies, the authors did not assess whether the allometric slope evolved as well.

The only artificial-selection experiment on narrow-sense allometry conducted so far combined stabilizing and disruptive selection on body area and caudal fin area in the guppy (*poecilia reticulata*) to select for a change in static allometric slope[91] (a procedure similar to the one described in Fig. 2B). Up and down selection on the allometric intercept was also applied in two separate lines. After three generations, the results suggested that the allometric slopes had very little capacity to evolve compared to the allometric intercepts, but this conclusion was weakened by the small number of generations of selection.

In a selection experiment to increase or decrease body mass in the moth *Manduca sexta*, static allometry between wing mass and body mass became steeper in the line selected for smaller body mass and shallower in the line selected for larger body mass after 10 generations of selection[92] (and see Ref. 28 for reanalysis using standard regression). In a recent experiment on the seed beetle *Callosobruchus maculatus*, it was found that 21 generations of relaxed sexual selection had modified the allometric relationship between two traits of the male genital apparatus and elytron length.[93] It has been argued, however, that this finding is not very informative about the evolution of allometry, because there was a very poor fit to the allometric model in the first place.[28]

One factor that has not been considered in these last two experiments is the possibility of plasticity in the allometric slope. Plasticity in allometric relationships has been little studied, but two studies clearly show that static allometry varies in response to different environmental treatments.[94,95] Similarly, a selection experiment on *Drosophila* wings in which selection was performed on the relative position of some veins[66,96,97] showed erratic, but sometimes statistically significant, variation in static allometry (Fig. 3). The differences in slope could be, if generations are observed in isolation, misinterpreted as change in allometry due to selection. To avoid such problems, we strongly recommend, during selection experiments, to investigate the possibility of plasticity in the allometric slope and to follow the changes in allometry from generation to generation. Experiments that do not do this should be interpreted with caution.

In recent years, developmental biology provided particularly relevant observations regarding the developmental mechanisms responsible for scaling between appendage size and body size in holometabolous insects (see Emlen and Allen[55] and Shingleton *et al.*[98] for review). The first mechanism involves the genetic regulation of growth rate through regulatory genes, such as Decapentaplegic (*dpp*) or Wingless (*Wg*), that affect cell proliferation and differentiation. Genetic variation in these genes may affect the pathway specifying the shape and relative sizes of domains within imaginal discs and affect the size of the corresponding structure in adults. Trait-specific variation in the duration of growth may also affect scaling relationships between traits. In holometabolous insects, growth via cell proliferation is promoted during the prepupal period by a pulse of juvenile hormone and ecdysone. When juvenile hormone drops below a certain threshold, cell proliferation ceases. Differences in threshold sensitivity between imaginal discs may generate a different duration of growth and consequently a different size of the final trait. Trait-specific sensitivity to insulin may also affect allometric relationships. Insulin activates a signaling cascade by binding to specific receptors that stimulate cell proliferation.[99] Trait-specific modification of the signaling cascade (e.g., by increased expression of insulin receptors) can affect the size of specific imaginal structures and therefore the allometric relationship with body size. It remains largely unknown whether genetic changes in any of these mechanisms could affect the allometric slope and intercept, but recent work on the insulin pathway has uncovered mutations

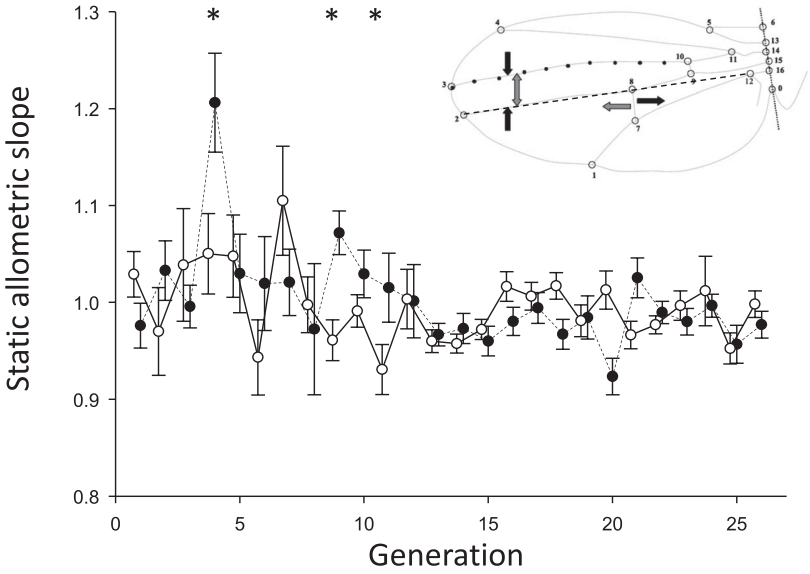

Figure 3. **Phenotypic plasticity in static allometry. The figure presents the variation in the slope of the static allometry between wing size (estimated by the centroid size) and the interlandmark distance 2–12 (dashed line) in two populations of *D. melanogaster* selected to increase (gray arrows, open dots) or decrease (black arrows, black dots) a selection index. Selection was performed for 26 generations. Idiosyncratic variation of the slope (estimated on 100 males at each generation) generates statistically significant differences between selection lines at generations 4, 9, and 11 (indicated by asterisks). The data presented here correspond to the LHM 1 replicate, but similar results were observed for the other three replicates: LHM 2, IV 1, and IV 2 (see Ref. 22 for more details regarding the selection experiment).**

at specific loci that could affect the organ sensitivity to insulin concentration and therefore affect the slope of the static allometry[58,100] (and see Shingleton and Frankino[101] for review). Although these studies provide evidence for genetic variation in allometric slope, it has also been suggested that this variation results from complex genetic architecture that may not easily respond to selection.[86] Overall, quantitative genetics studies provide conclusive evidence for genetic variation in the allometric intercept, but not for the allometric slope.

Does static allometry constrain phenotypic evolution?

Theoretical considerations

Microevolutionary studies reviewed in the last section suggest that the evolvability of the allometric slope is low relative to the evolvability of the intercept. At the macroevolutionary level, studies of the allometry of secondary sexual characters show that allometries of homologous traits can vary among sexes from the same species or among species (see Ref. 102 for review). Such information is, by itself, not very informative about the constraint hypothesis because we do not know anything about the strength of selection on the allometric relationships, and the time scale for divergence can be very long. Therefore, it remains unclear whether there are meaningful evolutionary constraints due to allometry.

In the absence of empirical knowledge regarding selection on allometry, we are left with the option of generating predictions on the basis of scenarios where static allometry constrains phenotypic evolution under various hypothetical selection regimes. In this context, a general prediction is that, if static allometric parameters represent evolutionary constraints, they should shape patterns of population and species divergence on some time scales.[5,8] This general idea was placed in a quantitative genetic framework by Lande[37,38] who showed how to predict the correlated response of trait size z to selection acting on body size x. In this special case, the evolutionary allometric slope b_e along which populations and species evolve corresponds to the ratio of the correlated response in z divided by the direct response in x, that is, the slope of the genetic regression between the two traits: $b_s = \sigma_A(x, z)/\sigma_A{}^2(x)$,

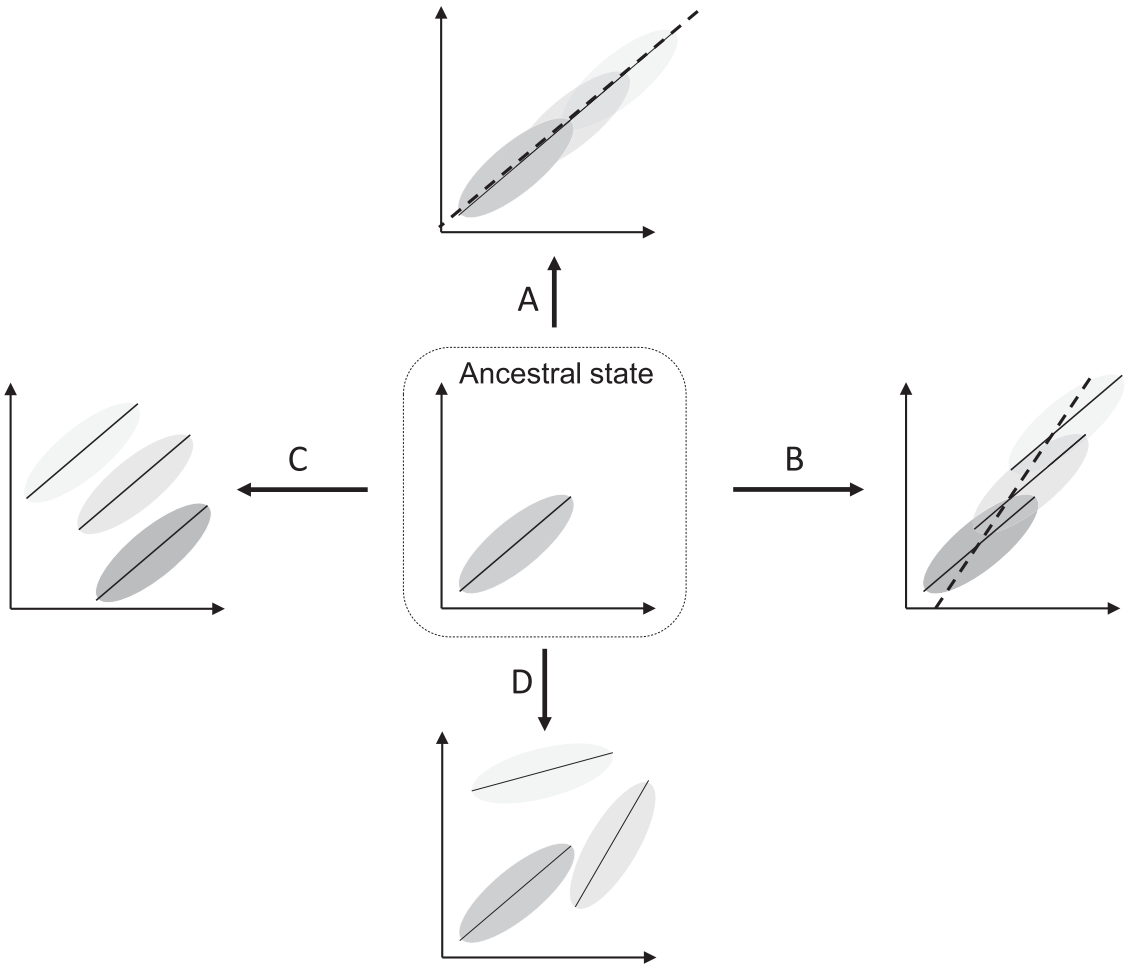

Figure 4. Static allometry as an evolutionary constraint. The various scenarios represent the effects of constraining parameters of the static allometry on the evolutionary allometry, assuming that selective optima are spread more or less randomly in the bivariate morphospace. In scenario A, neither the slope nor the intercept can vary. The divergence of population means follows the genetic allometric regression. If the slopes of the genetic and phenotypic allometry are similar, evolutionary allometry (dashed line) will follow the patterns of static allometry. In scenario B, the intercept is allowed to change but not the slope. Although such a pattern may result from the evolution of the intercept itself, it may also result from the difference between the phenotypic and genetic allometry. Scenario C presents a similar pattern but where selection does not favor any particular direction in the morphospace. In scenario D, both intercept and slope can evolve (no constraint).

where $\sigma_A{}^2(x)$ and $\sigma_A(x, z)$ are the additive genetic variance in x and the covariance between x and z, respectively (Fig. 4, scenario A).

Because static allometry is defined at the phenotypic level, the static allometric slope b_s combines both additive genetic and residual variances and covariances: $b_s = h^2\left(\sigma_A(x, z)/\sigma_A^2(x)\right) + \left(1 - h^2\right)\left(\sigma_R(x, z)/\sigma_R^2(x)\right)$, where h^2 is the heritability of body size x, that is, the ratio between the additive genetic variance and the phenotypic variance, and R denotes all other residual components of the variance, including environmental and nonadditive genetic variance (for justification, see the Appendix). Under this scenario, static and evolutionary regression coefficients, b_s and b_e, will be similar when $b_s = \sigma_A(x, z)/\sigma_A^2(x) = \sigma_R(x, z)/\sigma_R^2(x)$. In this case, the genetic and phenotypic variance–covariance matrices, **G** and **P**, will be proportional for these elements. If this condition is not fulfilled, evolutionary allometry will not follow the trajectory defined by the static allometry (Fig. 4, scenarios B and C[38]). Furthermore,

if the additive genetic and residual contributions to static allometry are different but remain constant, the static allometric slope should remain constant across populations and species while the intercept will change (Fig. 4, scenarios B and C).

It is important to realize that when Lande's model[38] is generalized to allow selection on trait and size, and there is at least some additive genetic variation in both, the means obtained in the long term depend only on the selective optima for the size and trait, and not on the patterns of covariance between them.[39] If this is the case, and if selective optima are not systematically placed in the bivariate morphospace, we expect no relationship between evolutionary and static allometry (Fig. 4, scenarios C and D). Scenario C in Figure 4 represents a situation where there is no genetic variation in the slope but where the trait and body size means can evolve more or less freely in the morphospace. This calls for an alternative explanation for evolutionary allometry in which population divergence is not dictated by patterns of genetic variation, but by patterns of selection.

These considerations underscore the impossibility of testing the allometric constraint hypothesis without knowing the patterns of selection acting on the allometric relationships. As a result, our interpretations of the macroevolutionary patterns remain speculative. Although specific predictions have been suggested regarding the effect of selection on allometric slope for genitals and secondary sexual characters, these predictions only concerned the type of allometry expected (positive or negative allometry), leaving unclear the expected rate at which such patterns evolve. One possible exception is a study on various species of stalk-eyed fly (Diopsidae) that estimated the rate of evolution of the static allometry between eye span and body length toward a predicted optimum, modeled as a function of the strength of sexual selection within each lineage.[103] This analysis showed that the static allometric slopes were tracking the optimum, but that the rate of evolution was slow, with estimated times of 2–3 Myr for adaptation in the static slopes to exceed ancestral influence on the trait.

Empirical patterns

Although many studies have compared relationships between trait size and body size among populations and species, a recent review by Voje *et al.* identified only 10 studies with sufficient information to compute interspecific variation in narrow-sense allometric slopes and intercepts.[28] Excluding genital traits, this review revealed considerable interspecific variation in static allometric slopes across species within genera (the median standard deviation corrected for sampling error was $SD = 0.27$) but very little across populations within species (median $SD = 0.07$). The patterns were quite similar for the allometric intercept, with substantial variation across species (median $SD = 0.15$) and much less among populations (median $SD = 0.02$).

Because the slope and intercept are on different scales, their levels of variation are not directly comparable. To assess their relative importance, the concept of conditional variance[43,45] was used to estimate their influence on the evolution of trait size. This allowed comparing the variation in slope and intercept on a common scale (i.e., variance in trait size).[28] Using this method, it was shown that 74% of the interspecific variation in trait size was associated with changes in body size, while the contribution of the static allometric slope and intercept were more limited, with 13% and 29% of the log trait variance explained, respectively (e.g., Fig. 5A). The contributions of these three parameters to trait diversification at the among-population level were similar, with size variation explaining 71%, slope explaining 36%, and intercept explaining 40% of the variation in trait size. However, as previously mentioned, much less variation in static allometry was observed among populations, and the evolutionary allometry was often very similar to the pattern of static allometry[28] (Fig. 5B). Finally, within species, the average static allometry across populations was a good predictor of the evolutionary allometry, while across species within genera the average static allometry was poorly correlated with the evolutionary allometry.[28]

Morphological evolution that is more constrained along static allometric trajectories at the within-species level than at the among-species level could be interpreted as a signature of evolutionary constraint. Therefore, these results are compatible with the allometry-as-a-constraint hypothesis if selective optima were spread more or less randomly in the bivariate morphospace, and not along adaptive ridges or "cordillera." Occurrence of such an adaptive ridge could explain the evolutionary allometry observed between the width and length of the M_1

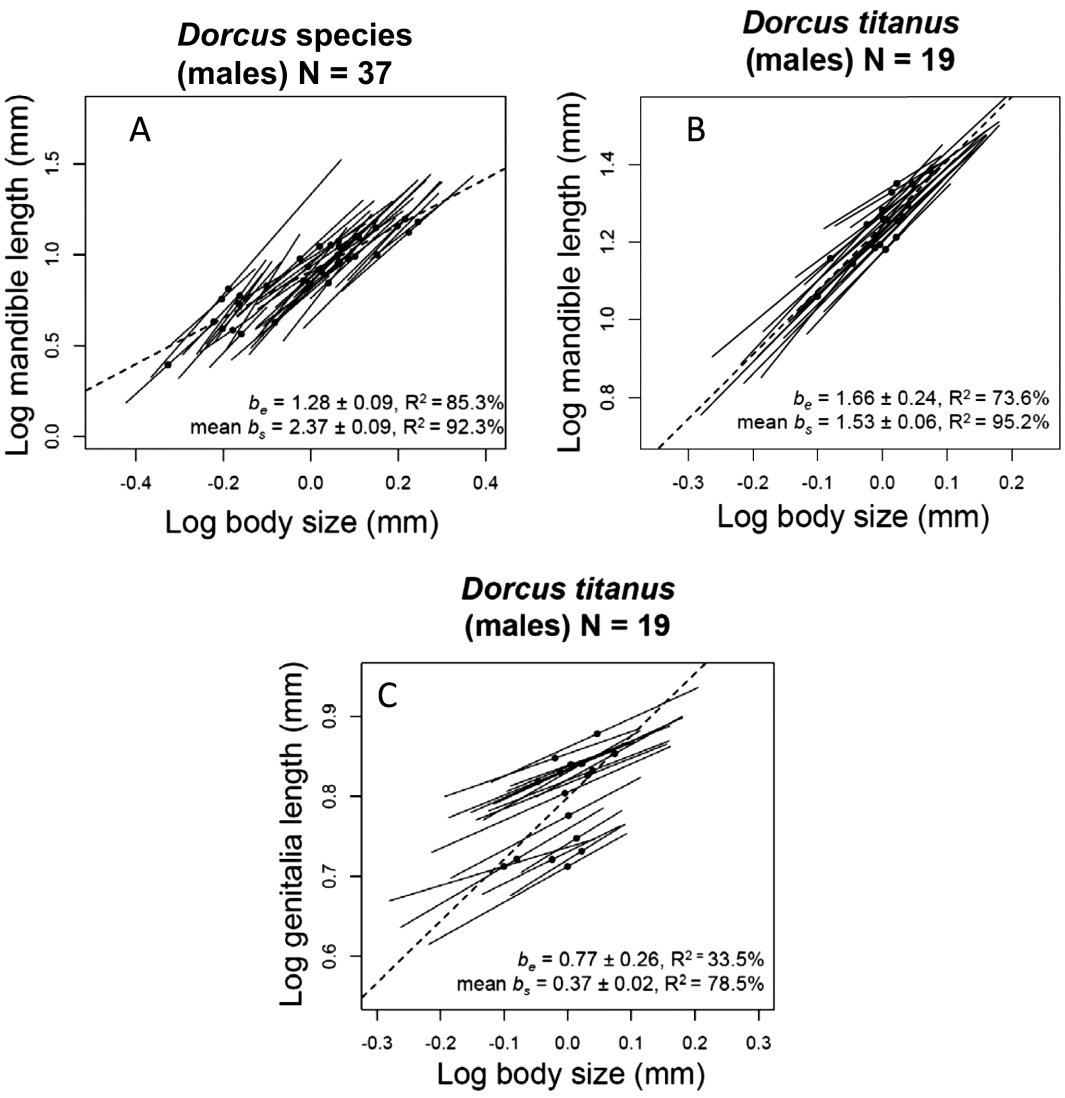

Figure 5. Example of variation in static allometry and its effect on evolutionary allometry among (A) species within genus, (B) populations within species, and (C) populations within species for genitalia. In each graph, the evolutionary allometric slope (b_e) and the average static allometric slope (b_s) are reported. Figure is from Ref. 28.

molar of the rodent species *Mimomys savini* during the Pleistocene in the Iberian Peninsula.[104] Using a well-calibrated paleontological sequence of dental measurements, it is shown that the evolutionary allometry among populations spread across 600,000 years follows the pattern of average static allometry. However, the relatively low r^2 of the static allometry observed within each population also provides some support for the existence of an adaptive ridge along which populations evolved.

The one-size-fits-all hypothesis

The various studies analyzing static allometry of genital traits in arthropods and vertebrates showed consistently shallow static allometric slopes ($b < 1$), as expected from the one-size-fits-all hypothesis.[19,25] A few exceptions to this pattern were reported,[25] but all came from studies using reduced major axis regression, a method that will seriously misestimate the slope unless the r^2 is very high.[105] For all these exceptions, the r^2 was low (between

0.00 and 0.58). When these cases were reanalyzed using standard regression methods, negative ($b < 1$) static allometries were found.[28,106]

The one-size-fits-all hypothesis has also been extended to reproductive organs in flowering plants with insect pollination, where the fit between pollen donor and pollen receiver organs and pollinators of relatively constant size and behavior is expected to produce stabilizing selection.[107] Several studies have provided evidence for the relative invariance of floral compared to vegetative traits[108] (and see Ref. 109 for review), and the two studies that have tested the effect of pollination accuracy on the allometry of the pollen transport organs both reported shallow allometry for these traits.[110,111]

If results from studies on the one-size-fits-all hypothesis provide clear evidence of a possible effect of selection on static allometry, they do not provide evidence for high evolvability of the allometric slope. Indeed, intraspecific comparisons of static allometry in insect genitalia show that variation in the allometric slope explains only a small proportion of the variation in genitalia size: 92% of the variance of log-size genitalia is independent of the variation in allometric slope.[28] This is particularly clear when observing the allometric slope between genitalia size and body size in various populations of the beetle species *Dorcus titanus* (Fig. 5C).

Conclusions

Despite great interest in morphological allometry over nearly a century, we still have a very limited understanding of its evolution and biological basis. Key observations, such as genetic variation in ontogenetic allometry or the static allometric slope, are surprisingly scarce, and if developmental biology offers several mechanisms capable of altering scaling relationships between traits, the evolvability of these mechanisms remains unknown. Although micro- and macroevolutionary patterns seem to point to a constraining effect of morphological allometry on phenotypic evolution, the complete lack of data on the nature of direct or indirect selection on allometry, and the near absence of data on genetic variation in allometric parameters, precludes the interpretation of these patterns as evidence for or against the constraint and adaptation hypotheses. Until such data are obtained, the evolution of allometry will remain a mystery.

Acknowledgments

The authors want to thank Jarle Tufto for great discussions on allometry and two anonymous reviewers for their constructive comments. This work was supported by Grant 196434/V40 from the Norwegian Research Council to C.P. at the Norwegian University of Science and Technology.

Appendix

The observed allometric slope b of a trait y on size (x), where x and y are on log scale, can be written as

$$b = \frac{\sigma(x,\, y)}{\sigma^2(x)},$$

where $\sigma(x,\, y)$ is the covariance between x and y and $\sigma^2(x)$ is the variance of x. By partitioning the variance and the covariance into an additive genetic and a residual component, assumed to be independent and denoted by subscript G and R respectively, we get

$$\begin{aligned} b &= \frac{\sigma_A(x,\, y)}{\sigma^2(x)} + \frac{\sigma_R(x,\, y)}{\sigma^2(x)} \\ &= h^2 \frac{\sigma_A(x,\, y)}{\sigma_A^2(x)} + (1 - h^2) \frac{\sigma_R(x,\, y)}{\sigma_R^2(x)}, \end{aligned} \quad \text{(A1)}$$

where $h^2 = \sigma_A^2(x)/\sigma^2(x)$ is the heritability of x. We define the genetic and residual allometric slope as $b_A \equiv \sigma_A(x,\, y)/\sigma_A^2(x)$ and $b_R \equiv \sigma_R(x,\, y)/\sigma_R^2(x)$.

Conflicts of interest

The authors declare no conflicts of interest.

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