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COMMENTARY

Aligning the Spaces: A Comment on Polly—Developmental Dynamics and G-Matrices

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In this issue, Polly (2008) discusses the appropriateness of using metaphors and visualization techniques like the adaptive landscape and morphospace and their mathematical descriptors for understanding phenotypic evolution. I was stimulated to explore further, how these abstract visualizations relate to each other and how variation at each level of biological organization (e.g. genotype, developmental program, phenotype) is manifest at other levels. In particular, what is the nature of the adaptive and phenotypic landscapes and what is the distribution of mutational effects in those landscapes? Does the structure inherent at one level promote clustering of organisms at a higher (e.g. phenotypic) level?

I prefer to view the relationships among genetic changes, phenotypes, and the adaptive landscape a little differently than in the accompanying paper. The environment interacts with an organism's *performance* (e.g. running speed, food crushing efficiency) to select those phenotypes that are more successful (Emerson and Arnold 1989). Different combinations of phenotypic (especially morphological) traits can yield the same performance values. This many-to-one mapping makes for a more complex and rugged adaptive landscape when fitness is graphed against individual phenotypic traits. The impact of that selection is filtered and translated through the various levels of organization to result in changes in allele frequencies, evolution. In that context, Rice's (2004) "phenotypic landscape" is placed within this hierarchy differently. Rather than inserting it between the adaptive landscape (fitness against genotype) and the phenotype, I view it as underlying the adaptive landscape (fitness against phenotype) and that Rice's (2004) "phenotypic landscape" is the mapping function that (1) translates the genotype, via development and environment, up the hierarchy into the phenotype, and (2) conducts the effects of selection down the hierarchy onto the genotype. There are several types of adaptive landscapes, depending on which level of organization is being compared to fitness, and as Polly (2008) points out, they can have different topographies or textures.

Adaptive landscapes are often presented where fitness is a function of one or more genetic traits ("factors" in Polly 2008). But what is represented by the scale called, for example, "genetic factor 1"? (Polly 2008). It is clear it must be a continuous variable, whether graphed against fitness in an adaptive landscape or against phenotype in a phenotypic landscape, because of the smooth surface where fitness values of adjacent genetic values are very similar (i.e., Moran's I, a measure of spatial autocorrelation, monotonically approaches 1.0 with decreasing distance). I would suggest however, that the only genetic factor that could be continuous is allele frequency within a population; this is Wright's original formulation of the adaptive landscape (Phillips and Arnold 1989; Wright 1932) and the one common in population genetics. But whenever the graph refers to individuals rather than populations, all genetic changes are discrete. There is no genetic scale that is both objective and continuous. How does a mutation at site 451 in locus X from an A to a T move an organism on such a scale? One could, for example, refer to magnitude of gene expression, but that is really a phenotypic trait. At the genetic level, all mutations are discrete with a wide range of possible phenotypic effects, from none to lethal. And no matter how you order those factors on a scale (e.g. adjacent nucleotide positions), the fitness surface of genetic factors

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will be exceedingly rough, with little autocorrelation evident in the fitness surface. One can decompose phenotypic variation into additive genetic and environmental variances, but can one assign a genetic value to a trait? In quantitative genetics, therefore, individual fitness surfaces and their associated adaptive landscapes are best conceived of as the relationship between phenotypes and fitness, not genotypes and fitness (Phillips and Arnold 1989). Genotypes obviously have fitnesses, but they cannot easily be ordinated to create a biologically interpretable space.

Even if one does develop a continuous scale below the phenotype and closer to the genotype, say, magnitude of gene expression, then as Polly and others have pointed out, there can be a discontinuous translation to the phenotype, as in the phenotypic landscapes Polly illustrates. In fact, that space (perhaps termed epigenetic or developmental space) may be even more complex due to threshold effects and phase changes, reminiscent of catastrophe theory (Thom 1988). For example, does a set of cells in a limb bud differentiate into one digit or two? Either the intervening cells undergo apoptosis or they do not. Thom's model provides one framework illustrating how small changes in gene expression can result in sudden, discontinuous modifications of the phenotype, potentially resulting in novel features.

In a similar vein, Polly (2008) suggests that evolutionary transitions in phenotype space might not be continuous even if changes in the underlying genetic parameters might be. I would turn that around because in my conceptualization there are no continuous changes in genetic traits, but there can be in phenotype space. Perhaps this discordance in metaphors could be resolved by substituting an intermediate level between the genotype and phenotype (e.g. gene expression, epigenetic space) for "genetic parameters" or "factors."

Whether quantitative genetic tools like the G-matrix are applicable to explaining phenotypic evolution (and modeling such in continuous morphospace) depends in part on the distribution of mutational effects. In particular, what is the proportion of effects that are less than the precision of measurement (or perhaps more practically, less than twice the precision)? If the vast majority of effects are less than that threshold, then using quantitative genetic methods that assume an infinite number of sites, each with very small effect, is a reasonable approximation that distills an exceedingly complex problem (if accounting for each allele separately) into elegant and tractable equations. However, it is not just the relative frequency of small (immeasureable) effects versus large effects ("macromutations", defined broadly). If rare macromutations are of sufficiently large magnitude, they can still account for a significant proportion of phenotypic variation. As an illustration, if the cumulative frequency of mutations is plotted against the magnitude of their effect on the phenotype, then what may matter is the relative areas under the curve for those mutational effects less than or greater than the level of precision. Furthermore, macromutations may be disproportionately responsible for major phenotypic transitions, such as shifts to alternate adaptive peaks. The proportion of evolutionary changes that behave like the highly simplified statistical models that we use remains an empirical question.

More broadly, nearly every phenotypic trait is the product of multiple genetic traits. Can we ever really construct an accurate genotype-phenotype map based on mathematical equations? Should we ever achieve that kind of detailed information, the statistical approaches like G-matrices may be superfluous.

On a side note, Polly's discussion suggests that it is not uncommon for evolutionary biologists to tend to reify the G-matrix—treating it as an inherent property of an organism rather than a statistical description of population variation, a warning made more directly elsewhere (Pigliucci 2006).

Finally, Polly ends by asking if any of these issues really matter? In the case of reconstructing ancestral states on a phylogeny, I agree, probably not much. The errors involved in applying incomplete continuous-variation models to reconstruct ancestral states, for example, are likely to be much smaller than the uncertainty of the reconstruction methods themselves. The error bars on ancestral node reconstructions can exceed the range of variation among descendant lineages (Schluter et al. 1997). G-matrices and related concepts might have real utility though if used to inform prior probabilities in Bayesian reconstructions, especially where the parameters of interest are trends or transformation rates rather than point estimates of specific ancestors (for a maximum likelihood implementation, see Hohenlohe and Arnold 2008). But in other aspects, such as accurately reconstructing historical selection and defining the most likely directions of phenotypic evolution, maybe—the jury is still out. But in the meantime, there is a benefit to refining our models of multivariate evolution and identifying the appropriate space to explore.

References

Emerson, S. B., & Arnold, S. J. (1989). Intra- and interspecific relationships between morphology, performance, and fitness. In D. B. Wake & G. Roth (Eds.), *Complex organismal functions: integration and evolution in vertebrates* (pp. 295–314). New York: John Wiley and Sons.

Hohenlohe, P. A., & Arnold, S. J. (2008). MIPoD: A hypothesis-testing framework for microevolutionary inference from patterns of divergence. *American Naturalist, 171*, 366–385.

Phillips, P. C., & Arnold, S. J. (1989). Visualizing multivariate selection. *Evolution; International Journal of Organic Evolution, 43*, 1209–1222.

Pigliucci, M. (2006). Genetic variance–covariance matrices: A critique of the evolutionary quantitative genetics research program. *Biology and Philosophy, 21*, 1–23.

Polly, D. (2008). Developmental dynamics and G-matrices: Can morphometric spaces be used to model phenotypic evolution? *Evolutionary Biology*. doi:10.1007/s11692-008-9020-0.

Rice, S. H. (2004). Developmental associations between traits: Covariance and beyond. *Genetics, 166*, 513–526.

Schluter, D., Price, T., Mooers, A. O., & Ludwig, D. (1997). Likelihood of ancestor states in adaptive radiation. *Evolution; International Journal of Organic Evolution, 51*, 1699–1711.

Thom, R. (1988). *Structural stability and morphogenesis*. Reading, MA: Addison-Wesley Publishing Co.

Wright, S. (1932). The roles of mutation, inbreeding, crossbreeding and selection in evolution. *Proceedings of the 6th International Congress on Genetics, 1*, 356–366.