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Research article

When the classical reaction norm is corrected by body size

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ABSTRACT

For nearly half a century, plasticity has been a controversial issue in ecology and evolution. Observed plasticity was classically quantified as the slope of a reaction norm or quantified in time series, which concealed its relationship with body size. With a theoretical framework and an experiment, here we demonstrated that: (1) body size significantly contributes to nearly all traits' variability, which produces apparent, but not true plasticity; (2) the classical reaction norm seriously misestimates the origins and levels of plasticity, and a size-correction to the reaction norm can eliminate its size-dependency and leave only the environmental-induced plasticity; and (3) the absence of true plasticity in beneficial traits can be compensated for by true plasticity in compensating traits. This study emphasizes that the role of true plasticity varies throughout ontogeny. It also suggests that the classical reaction norm or function-valued traits needs to be integrated with body size (i.e., size serial analysis) when we evaluate environmental effects on phenotypes.

1. Introduction

Phenotypic plasticity, the direct impact of the environment on the development of individual phenotypes (Laland et al., 2014), is defined as one genotype producing more than one phenotype in different environments (Bradshaw, 1965). Phenotypic plasticity is discussed almost everywhere in ecology or evolutionary biology (Pfennig et al., 2010; Forsman, 2014; Laland et al., 2014; Wray et al., 2014), yet its origin/role remains controversial (Pfennig et al., 2010; Laland et al., 2014). Historically, research has concentrated on genetic and ecological causes (genotype by environment or G × E interaction) of plasticity. Recently, development's contribution to plasticity has received much more attention (Pfennig et al., 2010; Laland et al., 2014). There has been renewed interest in clarifying phenotypic plasticity's origin/role (Forsman, 2014) and this has triggered a debate between 'genes act as leaders in plasticity (hereafter referred to as standard evolutionary theory or SET; Wray et al., 2014)' and 'genes act as followers in plasticity (hereafter referred to as extended evolutionary synthesis or EES; Laland et al., 2014)' in evolution. Although there have been significant advances in the understanding of phenotypic plasticity (Bradshaw, 1965; Kirkpatrick and Heckman, 1989; Kirkpatrick et al., 1994; Meyer and Hill, 1997; McConnaughay and Coleman, 1999; Pigliucci, 2003; Weiner, 2004; Pfennig et al., 2010; Adler et al., 2013; Munday et al., 2013; Robinson and Beckerman, 2013; Bartlett et al., 2014; Forsman, 2014; Laland et al., 2014; Valladares et al., 2014; Wray et al., 2014), three major problems have hampered our progress toward understanding the consequences of phenotypic plasticity for plant populations and communities.

First, most researches focused on 'single trait' plasticity in response to environment variance, but not on 'whole organism' plasticity. Recently, in a review, Forsman (2014) emphasized that a 'whole organism' (rather than 'single trait') approach is important to the understanding of the roles of plasticity in ecology and evolution. Within an individual, the absence of plasticity in focal traits must be compensated for by higher plasticity in some other traits (Forsman, 2014). The question is how to link the plasticity of so many focal traits at a 'whole organism' level?

Second, phenotypic plasticity is classically quantified as the slope of a reaction norm (Dejong, 1990) or based on it (Chevin et al., 2010). There is a long tradition in ecology of investigating how different environments lead to plasticity in traits on the basis of reaction norm. These trait-based frameworks have been used to explain many ecological processes (Pigliucci et al., 1996; McConnaughay and Coleman, 1999; Adler et al., 2013). However, studies have shown that classical representation of the reaction norm is too general (Weiner, 2004) to represent the complicated responses of traits (Valladares et al., 2006). Recent studies propose that many phenotypic expressions are better understood as

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Table 1
List of plasticity-related terms or concepts and the relationships among them.

Terms or concepts	Description
Apparent plasticity	Plasticity in traits turns out to be simply the result of body size. This phenomenon has been called 'passive plasticity' or 'apparent plasticity' or 'size-dependent plasticity', because this is not really plasticity (Weiner, 2004).
Developmental reaction norms	The set of multivariate ontogenies that can be produced by a single genotype when it is exposed to environmental variation (Pigliucci et al., 1996; Valladares et al., 2007).
Environmental canalization	A set of processes historically selected to keep the phenotype constant despite environmental variation (Debat and David, 2001). It is inferred from the slope of the phenotypic norm of reaction: a weak response or a flat reaction norm represents environmental canalization (Van Buskirk and Steiner, 2009).
Genotype	When we refer to a genotype we do so in a population genetic sense, not in reference to a molecular sequence of a single gene, but to the complete genome (Nicotra et al., 2010).
Ontogenetic drift	Changes of a biological trait in such a predictable way that it can be presented as a function of plant growth or development (size-dependent phenotypic plasticity; McConnaughay and Coleman, 1999).
Phenotypic canalization	The phenomenon in which a set of genotypes show nonparallel norms of reaction in environment 0 and 1; however, the phenotypes are constant around environment 0 (Lande, 2009).
Phenotypic plasticity	One genotype producing more than one phenotype in different environments (Bradshaw, 1965). For a linear norm of reaction, plasticity is measured by the slope b (Chevin et al., 2010).
Reaction norm	The expected phenotype of a given genotype as a function of the environment (Chevin et al., 2010).
True plasticity	Traits exhibits plasticity as an 'active' response to environmental variation (Weiner, 2004; Forsman, 2014).
Whole organism plasticity	Within an individual, the absence of plasticity in focal traits must be compensated for by higher plasticity in some other traits from a whole organism perspective (Forsman, 2014). In essence, individuals are functionally and phenotypically integrated complex units (not a single trait) in response to environment differences. In contrast, some studies are focused on 'single trait' in response to environment differences (single trait plasticity).

size-dependent processes (Müller et al., 2000; Enquist and Niklas, 2002; Weiner, 2004; Kingsolver and Huey, 2008; Bonser and Aarssen, 2009; Poorter et al., 2012; Xie et al., 2012). For instance, there is no doubt that allocation patterns are significantly affected by plant size (Poorter et al., 2012). Thus, size-dependent plasticity (Table 1) must be taken into account (differentiating apparent from phenotypic plasticity) when estimating true plasticity (Brakefield, 2006; Valladares et al., 2006; Forsman, 2014). Unfortunately, the observed plasticity was quantified by classical reaction norm, which totally ignored its relationship with body size.

Third, recent conceptual and computational advances have proposed that phenotypes or reaction norm changes as a function of independent continuous variables (Xiong et al., 2011; Buckley and Kingsolver, 2012; Kingsolver et al., 2012; Hadjipantelis et al., 2013; Wang et al., 2013; Granier and Vile, 2014). Detailed descriptions of this extension of classical quantitative genetics to the analysis of function-valued traits have been addressed by Kirkpatrick and Heckman (1989), Kirkpatrick et al. (1994), Meyer and Hill (1997) and Meyer (1998). Although any continuous variable is acceptable (e.g., the level of certain environmental factor), the most commonly used is the time (Jaffrezic and Pletcher, 2000; Stinchcombe et al., 2012). In short, the method assumes the observed phenotype is described by a function of time, $z(t)$ (Beder and Gomulkiewicz, 2007) and/or environmental factor (e.g., thermal performance curve). Therefore, its first order derivative, $z'(t)$, represents the growth rate. This approach can detect the differences in growth rate, but cannot distinguish whether the growth rate affects phenotypic plasticity (apparent plasticity in Fig. 2 of Weiner, 2004). Poorter et al. (2012) have also suggested in judging whether an observed variance in allocation pattern is a result of active response (true plasticity) to environmental variation or not, comparison at a common size is the best available option. Thus, this approach also needs to be integrated with body size in order to further distinguish apparent from true plasticity.

In essence, apparent plasticity is some of underlying mechanisms that allow plant parts to vary in response to the environment. However, whether the plant response to environmental variation is 'passive' or 'active' is also the division between apparent and true plasticity (Table 1; Weiner, 2004; Forsman, 2014). Active plasticity needs a signal cascade that transcribes an external stimulus (DeWitt et al., 1998; Nicotra et al., 2010; Forsman, 2014). These accessory processes could increase the cost or limits of true plasticity (DeWitt et al., 1998; Forsman, 2014). Therefore, apparent plasticity is a simpler strategy and in some sense cheaper than true plasticity (Weiner, 2004; Xie et al., 2012). The costs of plasticity are thought to have important ecological and evolutionary consequences (DeWitt et al., 1998; Van Buskirk and Steiner, 2009). Such costs have recently been included in theoretical models (Chevin et al., 2010) and, perhaps more importantly, relevant experimental studies have now also appeared (Van Buskirk and Steiner, 2009; Chevin et al., 2010). For example, the cost of plasticity decreases the critical rate of environmental change that a plant can endure (Chevin et al., 2010). Consequently, misestimating the true plasticity may lead to misestimating the cost of plasticity when predicting the plant response to environmental changes.

To solve these problems, we first propose that body size (plant size in plant science) is the key to understanding to 'whole organism' plasticity. Then we propose that the classical reaction norm need to be corrected by body size, and function-valued traits approach also needs to be integrated with body size. Third, we propose a theoretical framework that: (1) illustrates the quantitative relationship between classical and size-correction reaction norm (including four scenarios to judge the levels of plasticity); (2) illustrates how to quantify the apparent and true plasticity. Finally, we try to test these hypotheses in a single framework with an experiment.

1.1. The key to linking trait plasticity at 'whole organism' level

From the allometric perspective, the allometry of plant quantitative traits is often characterized by power laws of the form $Y = \alpha X^{\beta}$ or its linear form $\mathrm{Ln}^{Y} = \beta \mathrm{Ln}^{X} + \mathrm{Ln}^{\alpha}$, where Y is one trait, X is another trait, β is the scaling exponent and α is a normalization constant. Allometric relationships among traits are universal in plants (Weiner, 2004). For example, plasticity in one trait (e.g., shoots) may be constrained by plasticity in another trait (e.g., roots; de Kroon et al., 2005). The relationship between trait and body size is only one aspect in allometric relationships. However, it has been widely studied in literatures (Weiner, 2004; Poorter et al., 2012). Here, we focused on the allometric relationships between trait (Y) and body size (M, plant size in plant science). Weiner and Thomas (1992) have suggested that "the term 'size' be restricted to measures of biomass, and that other metrics be referred to only by name (e.g., diameter, height, leaf area, etc)" in plants (here, we also follow this suggestion).