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EXPERT VIEW

Genetic basis of plasticity in plants

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Abstract

The ability of an organism to change its phenotype in response to different environments, termed plasticity, is a particularly important characteristic to enable sessile plants to adapt to rapid changes in their surroundings. Plasticity is a quantitative trait that can provide a fitness advantage and mitigate negative effects due to environmental perturbations. Yet, its genetic basis is not fully understood. Alongside technological limitations, the main challenge in studying plasticity has been the selection of suitable approaches for quantification of phenotypic plasticity. Here, we propose a categorization of the existing quantitative measures of phenotypic plasticity into nominal and relative approaches. Moreover, we highlight the recent advances in the understanding of the genetic architecture underlying phenotypic plasticity in plants. We identify four pillars for future research to uncover the genetic basis of phenotypic plasticity, with emphasis on development of computational approaches and theories. These developments will allow us to perform specific experiments to validate the causal genes for plasticity and to discover their role in plant fitness and evolution.

Keywords: Genetic architecture, GWA, G×E interaction, hub genes, plant adaptation, plasticity, variance.

Introduction

Rapidly changing climate together with the emergence of genomic tools and high-throughput phenotyping technologies have awoken an interest in investigating the genetic and molecular mechanisms of phenotypic plasticity (Box 1). Phenotypic plasticity denotes the ability of a genotype to exhibit changes in a specific trait across different environments; difference in phenotypic plasticity between genotypes is commonly referred to as genotype–environment (G×E) interaction. Difference in plasticity, genetic variation among genotypes, and stochastic factors within and among genotypes can cause phenotypes also to vary within a single environment (Box 2).

Plasticity can be especially important for plants, as sessile organisms, to respond to changing conditions (Bradshaw, 1965; Pigliucci, 2005). Together with experimental developments in genotyping and phenotyping, a key to identifying causal genes underlying plasticity lies in the quantification of plasticity as a trait. Plasticity of a given trait is known to be a quantitative trait by itself (Kliebenstein *et al.* 2002; Ordas *et al.*, 2008; Rönnegård and Valdar, 2012; Shen *et al.*, 2012). However, the extent to which its genetic basis is independent of the mean value of the trait is as yet unknown (Kliebenstein *et al.* 2002; Joseph *et al.*, 2015; Kusmec *et al.*, 2017) but would offer new possibilities to control and modulate agronomically important traits. In addition, understanding G×E interactions provides a novel strategy in breeding crop varieties that may better cope with changing environments while securing stable yields.

However, plasticity is not beneficial in all traits (Abley *et al.*, 2016). Plasticity of a given trait is a matter of degree, such that the threshold at which a given trait is considered canalized (i.e. robust) or plastic may be difficult to pinpoint. Moreover, due to the interconnection of molecular networks and (sub) cellular organization, traits depend on one other. As a result, plasticity of a trait often both depends on and is determined by other traits. A recurrent scenario is that plasticity in one trait is required for robustness of another. For instance, it has been shown that plasticity in different traits defining root architecture in several crop species, including maize (Zhu *et al.*, 2005; Niones *et al.*, 2015; Sandhu *et al.*, 2016) and wheat (Ehdaie *et al.*, 2011), correlates with high robustness in yield (Sandhu *et al.*, 2016).

Phenotypic plasticity and robustness are both associated costs that may be difficult to disentangle (DeWitt *et al.*, 1998). For instance, maximized yield directly reflects the fitness of the plant, and therefore robustness may be desired. However, it has been further shown that robustness in yield has its costs, and it does not correlate with the maximum yield (Zhai *et al.*, 2014; Sandhu *et al.*, 2016).

Here we provide a succinct review of the most widely used approaches to quantifying phenotypic plasticity and the recent developments in approaches used to dissect its genetic architecture. In addition, we highlight recent experimental studies that have been undertaken to determine genetic and molecular mechanisms underlying phenotypic plasticity in plants (Box 1). Finally, we discuss the main challenges in identification and validation of the causal genes of plasticity (i) by development of a comprehensive theory of plasticity based on the established molecular networks, and (ii) by dissecting the role of plasticity in growth- and fitness-related traits.

Quantitative approaches to studying plasticity and its genetic basis

A first necessary step for dissecting the causal genes of trait plasticity is to quantify the trait plasticity in a given genotype. Principally, we can partition the existing approaches to quantifying plasticity of a trait for a single genotype into two groups: nominal and relative.

Nominal approaches

In the approaches for nominal quantification of plasticity, only trait data in a single genotype across multiple environments are used. The classical nominal 'measure' of phenotypic plasticity is based on the concept of the reaction norm that describes the pattern of trait values of a genotype across multiple environments (Sommer *et al.*, 2017). Therefore, reaction norms for traits can be regarded as a visualization tool. Nominal quantification of plasticity based on reaction norms uses two concepts—the slope of the regression line that provides the best fit to the environmental variable and the residual mean square error from this regression line. These concepts are applicable when the trait changes linearly with the change in an environmental variable (Box 3A). In those traits that do not change linearly with the change in the environmental variable (e.g. enzyme catalytic activity with respect to pH, Box 3A), other measures of plasticity have to be used.

To this end, various measures of statistical dispersion, systematically reviewed in Valladares *et al.* (2006), can be used as nominal measures of plasticity. These include the range, standard deviation, coefficient of variation (i.e. ratio of standard deviation to the mean), stability index (Fridman, 2015), as well as other functions of means and standard deviations over different environments (Box 3B). For instance, Fisher *et al.* (2017) have used the coefficient of variation for 18 traits, including yield and seed production, across different crop species (e.g. tomato, eggplant, pepper, melon, watermelon, sunflower, and maize) grown in a common crop garden experiment, and found that the phenotypic plasticity per used measure follows a bimodal distribution. In addition, measures from information theory, such as Shannon entropy, can also be used to quantify the uncertainty about the values that a trait can assume (Teschendorff and Enver, 2017; Box 3B).

Relative approaches

In contrast to nominal measures of plasticity, approaches for relative quantification of plasticity, data for a single trait from multiple genotypes over the same set of environments are employed. To arrive at the relative measures of plasticity, we recall that the variance of a given trait can generally be decomposed into three components due to: (i) genotype, (ii) environment, and (iii) genotype-by-environment interactions. Since the variance components can be determined by ANOVA, the relative measures of phenotypic plasticity are based on modifications of ANOVA. If plasticity is analysed as a trait, then the variance due to genotype-by-environment interaction quantifies the variance in the plasticity of the trait over the analysed genotypes (Marais *et al.*, 2013), but not for the individual genotypes themselves. To address this issue, Finlay and Wilkinson considered that the interaction between the genotype and environment is proportional to the environmental index (Finlay and Wilkinson, 1963). To quantify plasticity, the trait values in a given genotype are then regressed against the average trait values of all genotypes over the studied environments. As in the case of the reaction norm, both the slope and the mean square error from the genotype-specific regressions can be used as relative measures of plasticity for a single genotype (Box 3C). Computational approaches that consider genetic relationships between the analysed genotypes as well as simultaneous estimation of genotype and environmental parameters have also been proposed (Lian and de los Campos, 2016).

Quantitative genetics approaches

It has been suggested that there are three groups of loci based on their role in controlling (i) the mean only, (ii) the variance only, or (iii) both the mean and the variance. The genetic basis of a trait's plasticity, quantified by the variance, is then provided by those loci that control the variance only without confounding effects on the mean (see Box 3D). Comparative analysis of

Box 1. Key developments in dissecting the genetic basis of phenotypic plasticity in plants

- **Loci associated with GxE interaction are often located in the regulatory regions of the genome**

  Gage *et al.* (2017) used data from a large maize GxE project including 11 environments and 21 phenotypes to study the relationship between allelic variation and GxE as an indicator of phenotypic plasticity. Slopes and mean residual deviations from Finley–Wilkinson regression lines were used as responses modelled by GWA. The authors found that the loci associated to GxE interactions were often in regulatory regions of the genome.

- **Plasticity and means of traits show independent genetic basis**

  Kusmec *et al.*, (2017) used phenotyping data for 23 traits in a maize nested association mapping (US-NAM) population grown in multiple environments to perform genome-wide association (GWA) analysis with measures of phenotypic plasticity obtained from Bayesian formulation of Finley–Wilkinson regression. Measures for means and plasticity of the traits were moderately to highly correlated, but were shown to have an independent genetic basis, suggesting a different way for breeding to improve productivity and stability.

- **Several loci control oil-yield plasticity in response to cold in sunflower**

  Mangin *et al.*, (2017) used the slopes of the reaction norm as a measure of plasticity for oil yield in response to cold in sunflower, and further analysed the genetic basis of the plasticity by GWA analysis. Nine loci showed significant association of yield plasticity to cold, and the most strongly associated genes were known to be involved in cold stress responses.

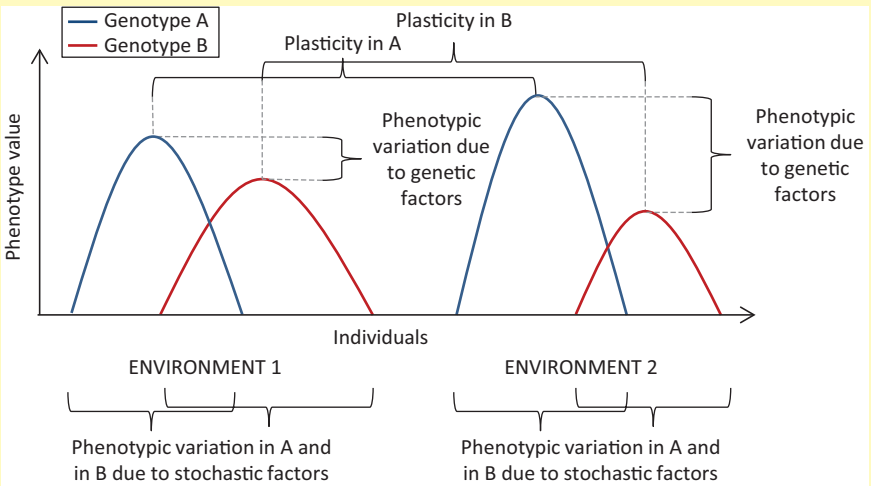
Box 2. Different sources of phenotypic variation

Phenotypes can vary within individuals of the same genotype or between individuals of different genotypes in one environment or different environments. Plasticity is phenotypic variation in response to different environments and can be due to genetic or stochastic factors or both.

Stochastic factors: factors that result in phenotypic variation in genetically identical individuals in a single environment; for instance, intrinsic noise of the system.

Genetic factors: factors that result in differences between the mean phenotypic values of different genotypes in a single environment.

Plastic phenotype: a phenotype with different mean values that are produced by a single genotype in response to different environments.



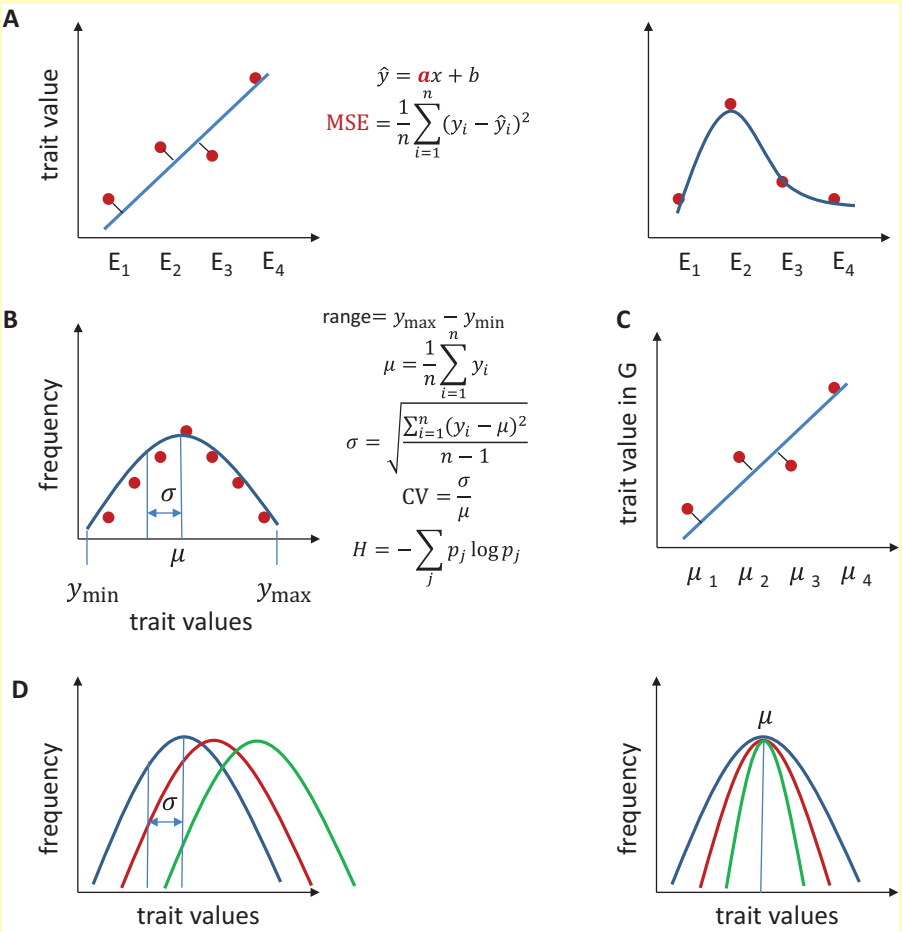
genotypes from different populations (e.g. introgression lines, natural variability of a species) can in turn be used to identify loci associated with variance.

For instance, with genetic populations suitable for QTL analysis, Levene's test and the Brown–Forsythe test for equality of group variances, both based on ANOVA on a transformation of response variable (using the mean and the median, respectively), have been used to dissect the genetic basis of variance as a measure of phenotypic plasticity (Alseekh *et al.*, 2017). In addition, analysis of means for variances (Nelson *et al.* 2005) has been used to compare the genotype means of the mean absolute deviation (MAD) to the mean of MADs over all genotypes (Ayroles *et al.*, 2015). Ability to reject the null hypotheses for these tests is employed to infer differences in phenotypic

Box 3. Quantitative approaches to studying plasticity

Nominal approaches: data from a single genotype over different environments are used.
Relative approaches: data from multiple genotypes over different environments are used.

(A) Nominal approaches based on reaction norm, suitable for trait that changes linearly with environment (left), in contrast to a trait that changes non-linearly with environment (right). (B) Nominal approaches based on frequency of trait values over different environments. (C) Relative approaches based on relationship between trait values in genotype G and mean trait values over a studied population of genotypes over different (here four) environments. Quantification based on nominal and relative quantitative approaches can be used in genome-wide association studies. (D) Three genotypes with respective trait distributions depicted in blue, red, and green exhibiting effects only on mean (left) and only on variance (right). a denotes the slope and b the intercept of the regression line modeling the association between y and x, where y denotes the predicted values. MSE is the mean square error of the model. Expressions for y and MSE are given in (A). μ represents the mean, σ, the standard deviation of a distribution of trait values, CV is the coefficient of variation, and *H* is the Shannon entropy; pj denotes the frequency of the trait value yj. Expressions for μ, σ, *H*, and CV are given in (B).



plasticity between compared genotypes. However, these tests do not consider covariates and random effects (e.g. population structure or relatedness of compared genotypes). A two-step procedure of first regressing on the covariates and then application of the aforementioned tests on the residuals offers a way around this drawback at the cost of inclusion of bias and reduction in power.

Double generalized linear models (Rönnegård and Valdar, 2012; Conley *et al.*, 2017, Preprint) are another option for determining the genetic basis of trait variance across different environments, as a measure of plasticity, while allowing for consideration of covariates and population structure. These models are employed in genome-wide association (GWA) studies in which parameters modelling the association of a locus to the mean and the variance across environments are simultaneously estimated (with the latter allowed to vary between genotypes) (Rönnegård and Valdar, 2012).

In principle, GWA mixed-effect linear models can be employed with any of the quantitative measures of plasticity discussed above. However, when the coefficient of variation is used as a measure, it is necessary to be careful about the confounding effects of the mean. Similar shortcomings can be observed when using measures derived based on the reaction norm, including those

obtained from Finlay–Wilkinson regression, referred to above. The reason is that these measures usually capture deviations from or dependence between means. One possibility to overcome these drawbacks is to conduct GWA for both the means and the selected measure of phenotypic plasticity, separately, and to then focus future confirmatory experiments on loci associate exclusively to phenotypic plasticity.

Genetic architecture of plasticity

While there are some universal factors whose presence is known to increase robustness or decrease plasticity, such as the molecular chaperone *HEAT SHOCK PROTEIN90* (*HSP90*) or circadian regulators *EARLY FLOWERING3* (*ELF3*) and *EARLY FLOWERING4* (*ELF4*), little is yet known about other genetic factors controlling trait plasticity in plants. Although, we are only at the beginning of understanding the specific genes that are causal for plasticity, GWA studies have taken us a step forward in unraveling the genetic architecture and discovering the candidate genes underlying trait plasticity.

Genetic loci associated with trait plasticity

Recently, GWA studies have been successfully used to identify candidate loci of trait plasticity in several plant species. For example, Kadam *et al.* (2017) used a diverse panel of 274 *indica* genotypes grown under control and water-deficit conditions to identify genes that underlie plasticity in traits related to root morphology and anatomy. The phenotypic plasticity was quantified as the relative change in trait value under water-deficit compared with control conditions, which was then used for GWA studies. The authors identified 76 loci associated with trait plasticity and predicted 233 *a priori* candidate genes within linkage disequilibrium blocks at these loci (Kadam *et al.*, 2017). Further, in another study Mangin *et al.* (2017) used the slopes of the reaction norm to cold stress as a measure of plasticity in oil yield in sunflower. They identified nine loci associated with oil yield plasticity in response to cold. The authors found that the associated single nucleotide polymorphisms were localized in genes previously shown to be involved in cold stress responses, including oligopeptide transporters, lipid transfer protein, cystatin, and alternative oxidase (Mangin *et al.*, 2017), suggesting that in some cases trait plasticity could be controlled by the same genes as the mean value of the trait. On the contrary, there is also recent evidence that the genetic basis of plasticity can also be independent from the mean value of the traits (Kusmec *et al.*, 2017). A large study of 23 different phenotypes in four environments showed that the candidate genes of mean phenotype values and plasticity measures (based on a Bayesian Finley–Wilkinson regression) comprise structurally and functionally distinct groups, suggesting independent control of the two traits (Kusmec *et al.*, 2017). Nevertheless, additional experiments are needed to confirm the role of these genes in controlling plasticity.

Metabolic changes are known to underlie many of the plant responses to different stresses to ensure optimized growth of the plants. There are a few studies that have addressed the stochastic variation in metabolism in plants. For instance, Joseph *et al.* (2015) showed that stochastic variation in recombinant inbred lines of Arabidopsis is controlled by genetic variation, and Li *et al.* (2016) identified several QTLs linked to stochastic variation in metabolism. In tomato, using introgression line populations, several QTLs were recently identified that decrease variability of primary or secondary metabolism and contribute to canalization of metabolism (Alseekh *et al.*, 2017). If and how canalization in metabolism influences fitness in yield remains to be investigated.

The role of hub genes in plasticity

It has been proposed that perturbations in genes representing hubs that stitch together genetic networks are likely to influence the overall plasticity or robustness of organisms. The role of 'hub genes' in buffering phenotypic variation is well known in yeast (Levy and Siegal, 2008) and they are suggested to play a more global role also in controlling variability in plants (Lachowiec *et al.*, 2016). In plants, evidence for the role of hub genes in controlling plasticity or robustness is still scarce. However, a recent genome-wide study of 11 maize phenotypes in 21 different environments supported that regulatory regions, likely harboring hub genes, contribute to phenotypic plasticity (Gage *et al.*, 2017). For example, the known plasticity gene *ELF3* was recently proposed to be a key hub gene integrating developmental and environmental signals in response to temperature (Anwer *et al.*, 2014; Boden *et al.*, 2014; Box *et al.*, 2015). In another example, plasticity in flowering time across six conditions in *Boechera stricta* identified the *near FLOWERING LOCUS T* (*nFT*) gene as the main QTL influencing variability in flowering time across different environments (Lee *et al.*, 2014). The *nFT* gene was previously found to exhibit a trade-off between flowering probability and fitness across varying environments (Anderson *et al.*, 2014). Moreover, the *FT* gene is known to act as a local hub (Pin and Nilsson, 2012) for several upstream signals of the Arabidopsis flowering time network, further supporting the role of hub genes in phenotypic variability. While flowering is known to be a plastic trait allowing plants to adjust their reproduction to the environmental conditions, flower size is an example of a robust trait in which mechanisms buffer the trait to show low variance (Hay *et al.*, 2014).

In a recent study, stochastic variance in petal number within a species of *Cardamine hirsuta* was shown to be a quantitative trait and further linked to two main loci (Monniaux *et al.*, 2016). Interestingly, the same QTLs also affected average petal size, indicating that petal size and variance in petal size have a shared genetic basis (Monniaux *et al.*, 2016). Whether petal size variance at different scales or across environments has a common genetic mechanism with stochastic variation remains to be investigated.

Future directions

While we are starting to understand the causal genes controlling plasticity of traits, this field of research is still in its nascent stages. Directing research to understand the genetic basis

of plasticity of specific traits will provide a different means to tackle the issue of controlling the yield of agronomically important crops in ever more fluctuating environments. We believe that future directions can focus on four points: (i) use of legacy data and accumulated knowledge on cellular networks, (ii) development of a network-based theory of plasticity leading to targeted experiments to investigate causal genes underlying plasticity, (iii) large-scale studies of specific traits across different environments, and (iv) precise studies to verify the genes controlling plasticity.

First, systems biology studies in model plants and crops have already produced various physiological, developmental, and molecular traits across different genotypes and environments. Future directions in understanding the genetic architecture controlling plasticity of traits can therefore benefit from taking a fresh look at the legacy data. These efforts will benefit from further development of computational approaches aimed at GWA studies that integrate knowledge on cellular networks to pinpoint genetic determinants of plasticity of different traits.

Second, there is increasing evidence of the existence of regulatory hub genes that underlie plasticity in phenotypic traits in response to different environments. Targeted perturbation of well-understood genetic networks harboring such hub genes can help us further understand the mechanisms by which a genetic change alters the network modulating the plasticity of a trait. Such an approach can also be taken with specific metabolic or signaling networks. These efforts may result in developing a sound network-based theory of trait plasticity that can be used to plan targeted experiments to modify plasticity of specific traits and conditions. Such a theory is currently elusive, since it requires modeling approaches to characterize the physiologically plausible ranges of values for modeled traits.

Third, to help test the theory and to understand the different genetic factors underlying plasticity, further experiments from large populations tested across multiple environmental conditions should be conducted. One bottleneck for this is the difficulty of experimentally dissecting the contribution of stochastic and genetic factors in fitness traits. Creation of genetic populations to specifically study plasticity of particular traits provides a tractable means to address this challenge.

Fourth, specific genetic and molecular studies are needed to validate the causality of the candidate genes in controlling the plasticity of different traits. These studies will enhance our understanding of the underlying genetic architecture and the variety of genes controlling plasticity.

Finally, in addition to genetic factors, DNA methylation has been recently associated with trait variation across environments in Arabidopsis and in response to stress, providing evidence for the contribution of epigenetic factors in trait plasticity (Zhang *et al.*, 2013; Kooke *et al.*, 2015 Aller *et al.*, 2018). Therefore, an additional future direction will be to investigate the role of epigenetic factors in plasticity. A comprehensive understanding of the genetic and epigenetic factors underlying plasticity will then allow us to further investigate their role in plant fitness and adaptation. Since plasticity, as an adaptive trait, could also be subject to natural selection and contribute to population dynamics, future studies on plasticity will also enhance our understanding of plant evolution.

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