

Mapping phenotypes: canalization, plasticity and developmental stability

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The relationship between genotype and phenotype is not one to one. This statement is central to our understanding of how natural selection shapes phenotypic evolution. Here, we clarify the links between canalization, plasticity and developmental stability, the three major processes involved in the control of phenotypic variability. We present a short historical review, including the original definitions of these concepts, and then summarize their current meaning and use, highlighting possible sources of confusion. Some of the perspectives allowed by a more synthetic conceptual framework are presented, in the light of the recent advances in molecular and developmental genetics.

One of the most important advances achieved through the evolutionary synthesis is the consideration of phenotypic variation as a quantity of interest, whereas, according to the classic typological view, it is considered a nuisance. Thus, the factors affecting phenotypic variation have been given considerable attention by evolutionists. It is widely accepted that variability (defined as the ability to vary^{1–3}) results from two antagonistic trends⁴: on the one hand, sources of variation, including genetic mutations, environmental effects and developmental errors; on the other hand, a set of regulatory processes, including buffering and enhancing mechanisms. Three such processes are commonly considered: canalization, phenotypic plasticity and developmental stability. Much effort has been made recently to understand the genetic bases, evolvability and evolutionary implications of these processes^{5–8}. However, the literature can be confusing, because the historical definitions usually referred to do not take into account later conceptual and empirical advances. Most modern authors consider each of the three processes separately, although their distinction might appear arbitrary⁵.

Historical context: the origin of disorder

The study of developmental regulatory processes is rooted in the old concept of physiological homeostasis, defined by Bernard⁹ and Cannon¹⁰.

Developmentalists, such as Waddington and Schmalhausen, suggested that developmental pathways, as well as physiology, must be strongly controlled. The concepts of canalization, plasticity and stability were developed to describe such control systems.

Canalization

Canalization was first defined by Waddington^{11,12} as the ability to produce a consistent phenotype in spite of variable genetic and/or environmental features (Box 1). Later^{13,14}, Waddington broadened this definition, focusing on phenotypes that, if not strictly invariable, are 'to some extent resistant to modification'. Simultaneously, he developed the idea of canalizing selection, implying a genetic control of canalization. Waddington thought that the same process counteracted both genetic and environmental disturbances. This duality is the basis of 'genetic assimilation', the process by which a response to unusual environments can be converted by selection into a permanent, genetically determined, phenotypic change. In parallel, Schmalhausen¹⁵ developed the related concept of the 'auto-regulatory mechanism' (Box 1), a process that stabilizes the morphology against environmental influences and mutations. He also recognized that some labile organisms showed environmentally induced changes in development, although he never used the term 'plasticity'. Thoday¹⁶, in defining 'developmental flexibility', explicitly introduced an adaptive value to the processes involved in developmental control (Box 1).

Phenotypic plasticity

Woltereck¹⁷ introduced the concept of the 'reaction norm' as early as 1909, but Johannsen was the first to point out the general importance of environmental influences for genotype–phenotype relationships¹⁸. Schmalhausen (Box 1) used two different terms to describe such influences, depending on their adaptive or nonadaptive nature¹⁵. The definition of plasticity given by Bradshaw¹⁹ encompasses both Schmalhausen's concepts (Box 1). Smith-Gill²⁰ recently distinguished two different components in plasticity, focusing on the discrete versus continuous variation of the traits in relation to their adaptive value (Box 1).

Developmental stability

Physiological homeostasis ensures a constant end product in spite of disturbances¹⁰. Lerner's²¹ extension of this concept ('genetic homeostasis', Box 1) is the basis of many later studies dealing with correlations between heterozygosity and morphological variance^{22,23}. In this context, individual deviations from the population mean were often interpreted as developmental errors, revealing a lack of developmental stability or developmental homeostasis, taken as synonyms of Waddington's canalization. However, the use of the word 'homeostasis' at both the individual and population levels is confusing and was rejected by Waddington¹³, and many researchers argued that these concepts were unnecessary to explain the observed correlations²⁴. In addition to morphological variance, the deviation from bilateral symmetry, or 'fluctuating asymmetry' (FA),

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Box 1. Canalization, plasticity and developmental stability: some historical landmarks in the evolution of three concepts

We provide original definitions whenever they are short and self-explanatory. Otherwise we provide a concise summary of the author's ideas.

Canalization

- Waddington, 1942^a: canalization: 'Adjustment of developmental reactions so as to bring about one definite end result regardless of minor variations in conditions during the course of the reaction'.
- Waddington, 1961^b: canalization: 'The property of a developmental process, of being to some extent modifiable, but to some extent resistant to modification'.
- Schmalhausen, 1949^c: autoregulatory mechanism: 'a set of processes historically selected, monitoring developmental path'.
- Thoday, 1953^d: developmental flexibility: 'an individual organism may be said to possess flexibility either if its genotype is such that it can develop different phenotypes in different environments, each phenotype better adapted than the others to the environment that evokes it, or if its genotype is so balanced that development is buffered against environmental variables and hence apparently the same adaptive phenotype results in a range of environmental conditions'.

Plasticity

- Schmalhausen, 1949^c: morphosis and dependent autoregulatory morphogenesis. Schmalhausen distinguishes two types of 'environment-dependent development': 'morphosis', a nonadaptive, continuous change in development correlated to environmental variations; and 'dependent autoregulatory morphogenesis', an environmentally induced variation of adaptive value, historically selected.

- Bradshaw, 1965^e: phenotypic plasticity: 'Plasticity is shown by a genotype when its expression is able to be altered by environmental influences...it does not have any implication concerning the adaptive value of the change occurring, although many types of plasticity may have important adaptive effects'.
- Smith-Gill, 1983^f: plasticity, developmental conversion and phenotypic modulation. Plasticity is separated by Smith-Gill into two different components: 'developmental conversion' is an adaptive discrete change; whereas 'phenotypic modulation' is a continuous nonadaptive variation. Only the former is supposed to be genetically based and therefore moulded by selection. Additionally, she proposed modulation to result from 'a failure of the organism to completely buffer development against environmental perturbations', or explicitly formulated, as a lack of canalization.

Developmental stability

- Lerner, 1954^g: genetic homeostasis, developmental homeostasis, developmental stability. Lerner defined genetic homeostasis as 'the property of the population to equilibrate its genetic composition and to resist to sudden changes'. According to him, this property depends on population heterozygosity, as heterozygous individuals buffer developmental variation more efficiently than do homozygous ones. Later researchers adopted this view to interpret the existence of negative correlations between individual heterozygosity and morphological variance: homozygous individuals would exhibit variable phenotypes and/or high levels of fluctuating asymmetry because of a lack of developmental stability or developmental homeostasis.

- Bradshaw, 1965^e: developmental instability is plasticity. According to Bradshaw, stochastic errors during development (referred to as 'developmental instability') must be viewed as a manifestation of plasticity, considering that 'such changes ultimately have definite environmental causes'.
- Zakharov, 1992^h: developmental homeostasis, homeorhesis, developmental stability. Zakharov redefined developmental homeostasis (or 'homeorhesis') as the set of mechanisms ensuring phenotypic consistency, including two different and well-separated processes: canalization, corresponding to Schmalhausen's 'autonomous-regulatory development'; and developmental stability, a set of mechanisms buffering developmental noise *sensu* Waddington, that is, developmental variation among replicated or symmetrical organs within a single organism.

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was used as a canalization index^{25,26}. However, Waddington^{12,13} again argued that FA had nothing to do with his canalization concept, because the FA of a character does not diminish following canalizing selection. Waddington proposed the terms 'developmental noise', to refer to differences among homologous replicated parts within a single individual, and 'developmental stability', to refer to the lack of noisiness. Waddington's position has been recently re-advocated by Zakharov¹ (Box 1).

Current uses of the three concepts

Several definitions of canalization, plasticity and developmental stability are currently in use (Box 2). The latest review on canalization is by Gibson and Wagner⁶, who define it as 'the reduction in variability of a trait'. However, unlike Waddington, they highlight the difference between genetic and environmental canalization, possibly based on different (although potentially overlapping) genetic and developmental systems. Environmental canalization, defined as the

Box 2. Current definitions of the three concepts

Canalization

- Zakharov, 1992^a: 'Similarity of the expression of the phenotypic character under different conditions of development'.
- Stearns *et al.*, 1995^b: 'The process by which phenotypic variation is reduced by developmental mechanisms.'
- Wilkins, 1996^c: 'Genetic capacity to buffer developmental pathways against mutational or environmental perturbations'.
- Wagner *et al.*, 1997^d: 'The suppression of phenotypic variation.'

Plasticity

- Via *et al.*, 1995^e: 'A change in the phenotype that depends on the environment...Historically, two main types of phenotypic plasticity have been recognized: graded responses and discrete or switched responses'.
- Callahan *et al.*, 1997^f: 'The ability of an organism to alter its physiology, morphology or development in response to changes in its environment'.
- Wagner *et al.*, 1997^d: 'Macroenvironmental sensitivity'.
- Stearns *et al.*, 1995^b: 'The sensitivity of a trait to change in an environmental factor measures its phenotypic plasticity'.

- Eshel and Matessi, 1998^g: 'Plasticity occurs in the many cases in which organisms are able to respond to changing conditions of the environment with specific modifications of certain traits, in a way that is appropriate to preserve the quality of its vital activities'.
- Behera and Nanjundiah, 1995^h: 'Plasticity is roughly analogous to a mixed strategy in the Evolutionary Stable Strategy (ESS) language'.

Developmental stability

- Zakharov, 1992^a: 'The capability for stable development under given conditions'.
- Palmer, 1994ⁱ: 'The developmental stability of an organism is reflected in its ability to produce an "ideal" form under a particular set of conditions'.
- Clarke, 1998^j: 'Developmental stability refers to buffering processes that reduce the variation resulting from developmental accidents'.
- Auffray *et al.*, 1999^k: 'The ability of organisms to withstand genetic or environmental disturbances during development, so as to produce a predetermined phenotype'.
- Van Dongen and Lens, 2000^l: 'An individual's ability to buffer its development against random perturbations'.

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Table 1. The interpretation of canalization, phenotypic plasticity and developmental stability in a quantitative genetic context

	Partition of phenotypic variance ^a	Genetic mechanisms	Effect on variance
Genetic canalization	$V_P = V_A + V_{NA} + V_E$	Epistatic modifiers (genetic-canalizing genes)	Negative on V_A
Environmental canalization	$V_P = V_A + V_{NA} + V_E$	Environment-canalizing genes	Negative on V_E
Phenotypic plasticity	$V_P = V_A + V_{NA} + V_{\mu E} + V_{ME}$	Gene expression dependent on macroenvironment	Positive on V_{ME}
Developmental stability	$V_P = V_{ind} + V_{within}$	Genes controlling homogeneous development of homologous body parts	Negative on V_{within}

^a V_P , phenotypic variance; V_A , additive genetic variance; V_{NA} , nonadditive genetic variance; V_E , environmental variance; $V_{\mu E}$, microenvironmental variance; V_{ME} , macroenvironmental variance; V_{ind} , among-individual variance; V_{within} , within-individual variance.

'insensitivity of a character to environmental factors'³ seems to be the opposite of phenotypic plasticity^{27,28}. By contrast, genetic canalization and phenotypic plasticity are not mutually exclusive: they can 'combine to form canalized reaction norms'²⁷. Gibson and Wagner⁶ do not distinguish developmental stability from canalization, because they list FA studies^{29,30} as examples of canalization. Therefore, they assume implicitly that the regulation of symmetry is just a special case of the canalizing mechanisms controlling phenotypic variance, still a controversial position^{31–33}.

Canalization, plasticity and developmental stability as patterns: a quantitative genetic interpretation
Each of the concepts of canalization, plasticity and developmental stability is associated with a component of phenotypic variance that can be measured using the appropriate quantitative genetic design (Table 1). Genetic canalization has traditionally been associated with directional selection experiments: if selection proves relatively inefficient in moving the trait value outside a certain interval, this trait is said to be canalized within this interval. The quantitative genetic interpretation of

this resistance to selection is a decrease in additive genetic variance (V_A) owing to a network of epistatic interactions. Two populations of genes are usually considered³: additive genes that increase or decrease the deviation from the mean by a certain number of phenotypic units; and genetic-canalizing genes that multiply these deviations by a constant, pushing the phenotype away from, or close to, the mean. Similarly, environmental canalization can reflect the action of environment-canalizing genes that decrease V_E , the environmental variance.

Phenotypic plasticity is usually measured using an experimental design involving several replicates of a genotype (such as clones or full siblings) distributed among a few treatments (e.g. temperatures). The environmental variance is decomposed into a microenvironmental component (within treatment, $V_{\mu E}$), and a macroenvironmental component (among treatment, V_{ME}). Only V_{ME} is used to quantify plasticity. This suggests that environmental canalization and plasticity involve different kinds of phenotypic variation ($V_{\mu E}$ and V_{ME} , respectively). However, the distinction between macro- and microenvironment is artificial: macroenvironment (e.g. temperature) merely represents the environmental parameters one chooses to manipulate, whereas microenvironmental variation represents the uncontrolled parameters (e.g. small uncontrolled variation in developmental temperature). In natural populations, the environments experienced by different individuals will differ in many ways, and there is no distinction between macro- and microenvironment. Therefore, plasticity and environmental canalization must act on the same kind of phenotypic variation.

Developmental stability is not measured using the classic genetic/environmental partition of phenotypic variance. Instead, within-individual variance (represented by FA) is separated from among-individual variance. The underlying assumption is that symmetrical parts of the organism represent the replicated expression of a single genotype in a single environment: the differences between replicates therefore reflect the intrinsic variance of the developmental process, when environmental variation is reduced to a minimum. However, this minimum variance must originate in minute, undetected, environmental events (including statistical fluctuations in cellular processes) that differentially affect the development of replicated parts within an individual. It is implicitly assumed that, even in an ideal world, the environment would vary among individuals more than it would among replicated parts of the same individual. According to this view, developmental noise is considered as the lowest attainable value of V_E , and developmental stability could be interpreted as a special component of environmental canalization, dealing with these small environmental differences among different parts of the body. However, this view ignores potential developmental correlations among these parts: for example, symmetry might depend on

mutual adjustment of growing left and right parts during development. Therefore, developmental stability, as measured by symmetry, might involve mechanisms other than environmental canalization.

Canalization, plasticity and developmental stability as processes: the mark of adaptationism

Since they were first established, the concepts of canalization, plasticity and developmental stability have been associated with adaptive interpretations, whether in the form of open statements or hidden assumptions.

Canalization is considered to be adaptive in a context of stabilizing selection. Both Waddington's 'canalization' and Schmalhausen's 'auto-regulatory mechanism' are assumed to evolve as adaptive fine-tunings following a major environmental and/or genetic change that initially increased phenotypic variance. For example, after a major mutation has been fixed, many minor genes will be selected to canalize the new developmental pathway. Genetic models incorporate stabilizing selection explicitly as the motor of the evolution of canalization³. They assume that the canalized pathway is optimal in all genetic and environmental conditions prevailing at that stage in the population. Recently, fluctuating selection was shown to promote canalization, in apparent contradiction with previous studies³⁴. However, fluctuating selection only works when the best long-term strategy is to remain phenotypically invariant: this kind of fluctuating selection can, therefore, be considered as a special case of stabilizing selection.

Developmental stability is considered adaptive in much the same way as is canalization. First, developmental homeostasis^{26,35} was taken by Lerner²¹ to be a property directly related to fitness. Second, in all FA studies the optimum phenotype is always considered to be perfect symmetry (FA = 0). When there are reasons to reject this assumption (e.g. directional asymmetry or antisymmetry), the data are excluded or statistically corrected to obtain a mean FA of zero⁸. Several studies have questioned seriously the validity of the relationship between FA and fitness³⁶. However, direct or indirect correlations between FA and fitness have been documented. For example, wing FA might decrease flight precision directly³⁷, and FA in male sexual ornaments might decrease mating success directly³⁷. FA might also reflect indirectly a fitness-related quality, such as individual condition³⁷. Finally, the existence of developmental stability, a process reducing random departure from an ideal phenotype, cannot be justified without assuming that this ideal (e.g. perfect symmetry) is an adaptive optimum. The occasional lack of empirical evidence for a relationship between phenotypic deviance (e.g. asymmetry) and fitness might be a consequence of the efficiency of developmental stability processes, keeping the departures from the optimum at an extremely low level in natural populations. Unfortunately, this hypothesis seems hardly falsifiable.

Phenotypic plasticity is used in a different context, in which the selective optimum differs among environments. Although most quantitative geneticists do not refer to adaptation in the definition of plasticity, experimental studies almost always present adaptive arguments, by defining the macroenvironment in such a way that different phenotypic optima are expected under different macroenvironmental conditions. Indeed, plasticity plays a prominent role in adaptation to environmental conditions in many organisms¹⁹. Some authors even include adaptation explicitly in the definition of plasticity^{38,39}.

The need for synthetic definitions

Although definitions are always conventional, those of canalization, plasticity and developmental stability remain unclear because evolutionists disagree on which convention to use. First, should adaptive arguments be included in definitions? On the one hand, purely phenomenological definitions are simpler and acknowledge the fact that phenotypic variation is often a nonadaptive matter of fact. On the other hand, most people referring to canalization, plasticity or developmental stability are, in fact, interested in the adaptive aspects of these processes. Second, the adaptive view itself is subdivided into two options, strongly dependent on the trait being studied. People studying plasticity consider the ability to vary as the adaptive strategy. For those studying developmental stability or canalization, the adaptive strategy is phenotypic consistency. As a consequence, a given environment-dependent phenotypic change will be called plasticity if it is believed to be adaptive, and lack of canalization if it is not.

Synthetic definitions of canalization, plasticity and developmental stability must emphasize the tight relationship between living organisms and their variable external environment. Organisms must keep their internal milieu constant enough to preserve physiological reactions and adjust themselves to changing external conditions, simultaneously. Any organism will stand within the theoretical limits of total environmental dependence and total autonomy, all intermediary positions being influenced by developmental constraints and historically inherited regulating mechanisms. Because of the history of the concepts of canalization, plasticity and developmental stability, it seems impossible to remove adaptive considerations from the definitions. Moreover, purely phenomenological definitions are not free of complications: for example, according to Bradshaw¹⁶, the microenvironmental variance is plasticity, because it must represent developmental changes related to unidentified and/or small variations in the environment. However the existence of environmental variance is not usually considered as a proof of plasticity. Rather, some sort of reaction norm is needed. In Box 3, we propose definitions of the three

concepts, considered as manifestations of adaptation, the assumptions necessary to refer to them, and some of the patterns one might use as evidence for their action in empirical contexts. We are aware of the difficulties involved in defining adaptation itself precisely⁴⁰. However, this problem is beyond the scope of this paper.

Questions that remain

Both canalization and plasticity are often heritable and can therefore evolve; but are there genes that are specialized in plasticity or canalization? Are the mechanisms of canalization and plasticity organism-wide or character-specific? Is there any relationship between environmental and genetic canalization, or are they independent? What is the link, if any, between developmental stability and canalization?

Studies of developmental stability or canalization have often highlighted heterogeneous responses among traits^{37,41,42}. A long-standing assumption was that traits highly constrained by natural selection were more buffered against both developmental noise and environmental/genetic influences (i.e. both stable and canalized). For example, it has been argued that characters for which asymmetry largely affects fitness might exhibit less FA than other traits^{31,33,34}. However it is hard to recognize such characters *a priori*, and no consensus has been achieved, inconsistency being the most consistent result³⁷.

New methodologies for new perspectives

In the past two decades, new approaches based on mathematical models of shape have been developed. Geometric morphometrics is a powerful way to quantify complex shape variation and has been used increasingly in ecology, palaeontology and systematics^{43,44}. Landmark-based studies⁴⁵ and outline analyses⁴⁶ are now common. However, the determinants of phenotypic variability have been addressed only recently. The relationship between developmental stability and canalization has been investigated^{32,33} using landmark data. In these studies, shape variation and asymmetry of complex structures (fly wing and mouse skull) were quantified to evaluate the congruence between canalizing and stabilizing effects. Although these studies led to different results, this approach is powerful and could be extended fruitfully to a wide range of models and characters. The same methodology could also be used to investigate the relationship between genetic and environmental canalization.

Recently, molecular developmental genetics has provided new perspectives on monitoring mechanisms. Indeed, Rutherford and Lindquist⁴⁷ reported an increase in morphological variation in Hsp90 mutants in *Drosophila*. Following artificial selection, these variants increased in frequency and their expression became independent of Hsp90, suggesting that they relied on cryptic mutations initially buffered by Hsp90. This escape from

Box 3. The concepts: definitions, assumptions and evidence

Genetic canalization

A set of processes historically selected to keep the phenotype constant in spite of genetic variation. Assumption: the canalized trait must have the same selective optimum in the range of genetic contexts considered. Possible evidence: resistance to selection when the mean phenotype is close to an optimum; resistance to mutation pressure; and existence of uncanalized variants with increased response to selection and/or to mutation.

Environmental canalization

A set of processes historically selected to keep the phenotype constant in spite of environmental variation. Assumption: the canalized trait must have the same selective optimum in the range of environments considered. Possible evidence: resistance to nonspecific environmental variation, such as among-individual environmental variation in a population. Sudden increase in phenotypic variance (uncanalization) when extreme environments are used, to which the species has not been able to adapt during its history. Existence of different variants with different values of $V_{\mu E}$ in the same environment.

Adaptive phenotypic plasticity

A set of processes historically selected to produce different phenotypes in relation to some environmental parameter. Assumption: the plastic trait has different optima for different values of the environmental parameter. Possible evidence: each form has higher fitness in the environment in which it developed, than that seen in other forms developed in different environments. The case for adaptive phenotypic plasticity is strengthened when: the sensitivity to environmental variation is restricted to one particular environmental parameter or set of parameters; and/or the proximal signal eliciting the developmental switch between two forms does not influence fitness by itself, but is a correlated indicator of

changes in selection regime (e.g. photoperiod allows plants to prepare themselves to low temperatures). Although many authors associate discontinuous changes with adaptive plasticity and continuous responses with nonadaptive physiological constraints, we do not think that discontinuity is a strong argument for adaptiveness. Many traits (binary or meristic) have intrinsically discontinuous responses, and these responses are not necessarily optimized by natural selection. Conversely, continuous responses might often be adaptive.

Some reaction norms (i.e. the set of phenotypes produced by one genotype in a range of environmental parameters) cannot be said *a priori* to be either adaptive or counteradaptive. In this case, one should stick to a phenomenological description such as 'developmental sensitivity to parameter x ' (corresponding to Bradshaw's original definition of phenotypic plasticity). The use of the word 'plasticity' alone is ambiguous, given its controversial link with adaptation.

Developmental stability

A set of mechanisms historically selected to keep the phenotype constant in spite of small, random developmental irregularities potentially inducing slight differences among homologous parts within individuals. Assumption: symmetry (or identical development of replicated body parts) represents the fitness optimum. Possible evidence: within-individual variation is smaller than is microenvironmental variation among individuals ($V_{\mu E}$), even when all individuals are genetically identical (clones) and placed in the best-controlled environment. Different genetic variants have different levels of fluctuating asymmetry.

N.B. Some of the developmental and genetic mechanisms ensuring environmental canalization might also ensure developmental stability, but this is not necessarily true.

canalization is a 'plausible mechanism for promoting evolutionary change in otherwise entrenched developmental processes'³⁶. This is, to our knowledge, the first direct evidence of a molecule involved in canalization. Although it is not formally proven that Hsp90 has been historically selected for its buffering properties, it remains the best candidate so far for such a scenario⁴⁸.

To investigate the fundamental nature of the monitoring processes, the advances achieved through shape variation quantification, developmental modelling⁴⁹ and developmental molecular genetics⁵⁰ must be integrated. The example of Hsp90 will boost such complementary approaches that might root canalization, plasticity and developmental stability firmly in a developmental molecular framework^{49–52}.

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