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The evolution of phenotypic plasticity: Genealogy of a debate in genetics

Antonine Nicoglou

IHPST Paris, CNRS, University of Paris 1, ENS, 13 rue du Four, 75006 Paris, France

antoninenico@gmail.com

+33 6 81 44 64 25

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Abstract: The paper describes the context and the origin of a particular debate that concerns the evolution of phenotypic plasticity. In 1965, British biologist A. D. Bradshaw proposed a widely cited model intended to explain the evolution of norms of reaction, based on his studies of plant populations. Bradshaw's model went beyond the notion of the "adaptive norm of reaction" discussed before him by Dobzhansky and Schmalhausen by suggesting that "plasticity" – the ability of a phenotype to be modified by the environment – should be genetically determined. To prove Bradshaw's hypothesis, it became necessary for some authors to identify the pressures exerted by natural selection on phenotypic plasticity in particular traits, and thus to model its evolution. In this paper, I contrast two different views, based on quantitative genetic models, proposed in the mid-1980s: Russell Lande and Sara Via's conception of phenotypic plasticity, which assumes that the evolution of plasticity is linked to the evolution of the plastic trait itself, and Samuel Scheiner and Richard Lyman's view, which assumes that the evolution of plasticity is independent from the evolution of the trait. I show how the origin of this specific debate, and different assumptions about the evolution of phenotypic plasticity, depended on Bradshaw's definition of plasticity and the context of quantitative genetics.

Keywords: phenotypic plasticity, evolution, Bradshaw, quantitative genetics, adaptive plasticity, extended synthesis

1. Introduction

Since the end of 1970s, some biologists (e.g., Gould, 1977; Hamburger, 1980; Coleman, 1980; Lauder, 1982; Wallace, 1986) have started to question the adequacy of the genocentric conception of the Modern Synthesis – which brought together Mendelian genetics, and evolution through population genetics (Huxley, 1942; Pigliucci & Müller, 1942) – in explaining the evolution of phenotypic traits, suggesting that developmental issues should also be included in the synthesis (Gilbert, Opitz & Raff, 1996). More recently, some authors have stressed that phenotypic plasticity should be seen as one important element in an extended synthesis of evolution including these developmental issues (see, Pigliucci & Müller, 2010). The present paper comes back on a precise controversy in the history of phenotypic plasticity in biology, which is a debate in the 1980s between two representative views (among others) – these of Via & Lande and Scheiner & Lyman – concerning the evolution of phenotypic plasticity. The aim of the paper is to lay the groundwork for a genealogy of phenotypic plasticity and to show that the notion was defined and discussed since a long time before the 2010s, and that it adopted a specific meaning in the emerging field of quantitative genetics. Through a clarification of the origins and the basis of a specific debate concerning the evolution of phenotypic plasticity that occurred in the 1980s, the purpose here is to enlighten some of the implicit ideas, which used to be associated with the notion of plasticity at that time, and to show the reasons why it remains difficult to associate the notion with something different from the Modern Synthesis.

In 1965, Anthony D. Bradshaw (1926–2008) published an article entitled *The Evolutionary Significance of Phenotypic Plasticity in Plants*. In the article, he proposed for the first time a model intended to explain the evolution of norms of reaction mainly based

on his studies of plant populations, realized during his sabbatical year in California[1] (e.g., Bradshaw, 1959; Allard & Bradshaw, 1964; Jain & Bradshaw 1966). Bradshaw's article has been widely cited and discussed thereafter. It is considered, by most of the commentators, as one of the founding articles defining the notion of phenotypic plasticity: the ability of a genetically determined phenotype to be modified by the environment (see, Sarkar, 1999; Pigliucci, 2001).

Almost twenty years later, in the 1980s, a heated debate erupted between biologists over the possible ways to understand the evolution of phenotypic plasticity, which was precisely the topic of Bradshaw's seminal article. While Bradshaw's purpose was to demonstrate that phenotypic plasticity evolved using a kind of optimality approach before the heyday of optimality studies, Via & Lande and Scheiner & Lyman tried to explicitly model how plasticity evolves using quantitative genetics theory. In the 1980's, quantitative genetics[2] – the branch of population genetics dealing with phenotypes which vary continuously, and that employs the frequencies of trait variation in breeding populations, combined with principles from Mendelian inheritance, in order to analyze inheritance patterns across generations and descendant lines (i.e., their evolution) – was a central discipline in biology, and most of the scientists were looking for models to explain the evolution of phenotypic traits. Consequently, since the protagonists of the debate were active research members in the field, they tried to offer precise models to explain the evolution of phenotypic plasticity. However, their approaches differed. On the one side, biologists Russell Lande and Sara Via wanted to analyze trait evolution in environments with discrete states given a specific genetic architecture. That is essentially expanding existing quantitative genetics models of multiple traits into single trait in multiple

[1] For further details on Bradshaw's works, see E. Peirson, this issue.

[2] The field was founded by some of the proponents of the Modern Synthesis, R.A. Fisher, S. Wright and J.B.S. Haldane, and aimed to predict the response to selection given data on the phenotype and relationships of individuals.

environments. And in such a perspective, they concluded that the evolution of plasticity should be seen as a by-product of evolution, assuming that selection would not act directly upon plasticity, which Via did not consider to be a distinct trait with its own genetic etiology. On the other side, Samuel Scheiner and Richard Lyman wanted a tool by which they could model adaptive evolution of reaction norms. And in their perspective, they considered that plasticity had its own genetic basis, independent from the one of the plastic trait, and, therefore, that it had its own model of evolution (Via & Lande, 1985, 1987; Scheiner & Lyman, 1991).

Since both authors refer to Bradshaw's seminal article of 1965, one purpose of the present paper is to analyze in what way Bradshaw's understanding of phenotypic plasticity might have influenced the works of the main protagonists of the debate and so how it might have perpetuated in the current literature. In other words, the purpose is both to understand whether those divergent approaches concerning the evolution of plasticity might have a common ground and whether the differences between them could help to enlighten the current understanding biologists have of phenotypic plasticity. There are two interrelated problems here. The first one concerns their common reference to Bradshaw and the relationship of their models with Bradshaw's view on plasticity. The second one concerns the disagreement, our precise understanding of it, and the potential consequences for the current understanding of phenotypic plasticity in biology. However, in order to answer the second problem – that is to say: what was the disagreement about? –, it seems necessary to provide some main clues concerning the first one – that is to say: what was Bradshaw's influence on the protagonists of the debate? With this being said, I shall then come back to my main goal here and conclude on the reasons why the disagreement seems to have disappeared in the current understanding biologists have of the evolution of phenotypic plasticity. But let us first start with Bradshaw's view on phenotypic plasticity.

2. Bradshaw and the notion of "phenotypic plasticity"

In the article of 1965, British ecologist and geneticist Anthony Bradshaw proposed a model to explain the evolution of what was previously considered as the "shape of reaction norms" and that he will call "plasticity." In the article, Bradshaw pointed out the importance of the environmental effects on organisms: "We are becoming increasingly aware that the individual cannot be considered out of the context of its environment" (Bradshaw, 1965, p. 115). Before him, the "instability" produced by the environment was mostly conceived as a source of perturbation for geneticists (Falconer, 1952). And as Bradshaw claimed: "Any modifications induced by the environment during the course of an experiment are usually considered only an embarrassment" (Bradshaw, 1965, p. 148). Therefore, they mostly tried to eliminate it in their studies.

Another example, which is somehow less expected, is that of Conrad Hall Waddington (1905–1975), who was interested in the issue of "stability" through the process of canalization. His most quoted book *The Strategy of the Genes* (Waddington, 1957) has been often, and justifiably, used by biologists and theorists to argue that he was one of the first evolutionary biologists to make an effort to bring genetics and development together, and, therefore, to pay a specific attention to environment. However, in most other books where he dealt with the question of "stability" (or "canalization"), he did not refer explicitly to the environment. In *Principles of Embryology* (Waddington, 1956), in which he developed and explained the process of "canalization," the term "environment" and its derivatives (e.g., "environmental") appeared only 16 times in a book of 528 pages. In the book *How Animals Develop*, (Waddington, 1962[3], [1935]) the term "environment" and its derivatives appeared only 6 times in a book of 148 pages. Waddington considered

[3] When the book was republished in 1962, a new section entitled "gene activity during development" is added.

the process of canalization as the expression of some robustness to genetic variability and not to *environmental variability*. It appears that Waddington, like many geneticists of his time, mostly depicted – with the notable exception of *The Strategy of the Genes* – the environment, even if influent, as a source of perturbation that should be removed from the analysis of phenogenesis for experimental purposes. He also explicitly considered that environmental effects were minimal during early development of organisms (Waddington, 1962, p. 122). Despite this major rejection of the "unstable" factor, the problem remained, nevertheless, to understand how the individual could maintain some stability in an unstable environment. After World War II, the question became one of the main issues among geneticists (e.g., Dobzhansky & Wallace, 1953; Mather, 1953; Jinks & Mather, 1955; Kimura, 1955; Lerner, 1954; Lewontin, 1957; Waddington, 1959; Levins, 1963).

Unlike most of his colleagues and even though he was a geneticist, Bradshaw did not see the environment as a disruptive force. Far from it, he explicitly incorporated the environment in his genetic analysis (see Fitter, 2010). Therefore, in the article of 1965, Bradshaw was striving to demonstrate: "first, that environmental effects on the phenotype were as important as genetic effects (rather than simply inconvenient error), and second, that these effects were themselves under genetic control and could therefore evolve" (Fitter, 2010, p. 31). Moreover, Bradshaw undermined, at least partly, some of his predecessor's contribution by downplaying the importance of development for understanding plasticity (i.e., Waddington, 1957). In Bradshaw's view, if plasticity could be related to the general pattern of development of a character, it should not be seen as properly *explained* by these developmental pathways:

> It has been argued […] that at the present, owing to the complexities of the developmental pathways concerned, the interactions between pathways and environment will be so complex that it is unlikely that much progress in understanding can be made until more

> detailed work on the pathways has been carried out. Considerations of basic causes, however, have led to the argument that the degree of plasticity shown by a character can be related to the basic pattern of its developmental pathway. (Bradshaw, 1965, p. 117)

> While basic developmental pathways are important, it does not seem possible for them [Stebbins, 1950 & Harper, 1961] to provide an explanation of all observable difference in plasticity. (Bradshaw, 1965, p. 118)

In other words, Bradshaw's purpose was not only to emphasize the importance of plasticity for the understanding of the evolution of phenotypic traits, it was also a way, for him, to establish a strong position in the overall theoretical debate, by downplaying some of his protagonists assumptions. Furthermore, Bradshaw depicted "plasticity" – as he chose, after Nilsson-Ehle (1873–1949) to call it – as having, in many circumstances, an adaptive value, which should be studied per se. For Bradshaw,

> *Plasticity* is therefore shown by a genotype when its expression is able to be altered by environmental influences. The change that occurs can be termed the *response*. Since all changes in the characters of an organism which are not genetic are environmental, plasticity is applicable to all intragenotypic variability. (Bradshaw, 1965, p. 116)

In Bradshaw's view, the "response" to environmental variation can be understood as an adaptation. Plasticity embodies phenotypic variability as a consequence of the genotypic response to environmental variation. Indeed, Bradshaw considered that plasticity is "genetically determined" (Bradshaw, 1965, p. 145). This conception lies squarely in the lines of the genotype/phenotype distinction's definition offered by Johannsen in 1911. Such a definition had already influenced many biologists interested in phenogenesis, such as Timoféeff-Ressovsky and Timoféeff-Ressovsky (1926) or Dobzhansky (1937, 1955). Dobzhansky had precisely suggested that in the study of phenogenesis one should focus on

"determined tendencies" of variation rather than on its "indetermined tendencies." Before him, in 1909, Richard Woltereck had introduced the notion of *Reaktionsnorm*. Where Woltereck interpreted the genotype through the reaction norm as a permissive agent of phenogenesis, Johannsen saw it as a determinant agent (see Sarkar, 1999; Fuller 2003). By considering that the reaction norm was nearly synonymous with the genotype, Johannsen tended to "smooth" Woltereck's definition of *Reaktionsnorm* being aware of, but not doing, the distinction between "the variability of the phenotypic responses to environment" (what Woltereck used to call *Reaktionsnorm*) and "the variability of the trait" (what Johannsen used to call *Reaktionsnorm*). Where Woltereck emphasized variability Johannsen preferred to emphasize stability. Dobzhansky and many geneticists interested in evolution (rather than in development/phenogenesis for instance) followed and reinforced Johannsen's path by linking the notion of "norm of reaction" to a genocentric framework (see Sarkar, 1999). From the moment, the norm of reaction was considered as an adaptation, such as any other traits genotypically determined, it also became a possible target for natural selection. When Dobzhansky disseminated the notion of "adaptive norm" to the West and particularly to the United States, he did it trying to demonstrate that a mutation does not alter a particular morphological character but that it introduces a change in the reaction norm (Dobzhansky, 1955). Gradually, the notion of "norm of reaction" was replaced, in the literature, by the notion of "reaction range" (Sinnott, Dunn & Dobzhansky, 1950, p. 22).[4] It was a way to indicate that phenotypic variability depended largely on interactions between inherited (or genetic) factors and the environment inside a precise range. However, the stated objective was to put a new emphasis on variability (as opposed to a strong genetic determinism, which was completely leaving aside the environment in the explanation of the evolution of traits). Yet, the use of the notion of "norm of reaction"

[4] For more on this question see Sarkar, 1999, p. 246.

by Dobzhansky led to its conceptual change: it became in 1955 essentially a problem of genetics since the norm of reaction itself was interpreted as a unit of Mendelian inheritance. With Bradshaw, the conceptual shift associated with the reaction norm initiated by Dobzhansky (from a developmental/phenogenesis perspective with Woltereck, to a population genetic/evolutionary perspective with Dobzhansky) received a new notion – phenotypic plasticity – to escape confusions with the controverted concept of "norm of reaction" and its potential derivatives (e.g. norm of reactivity, reaction range, *Reaktionsnorm,* etc.), and "plasticity" became mainly a tool of population genetics/evolution.

Therefore, even if Bradshaw followed Herman Nilsson-Ehle's (the Swedish geneticists who first referred explicitly to the notion of "plasticity" to describe the effect of the environment on the *phenotype*) conclusions[5] concerning the adaptive value of plasticity, his interpretation concerning the genetically determined character of plasticity depicts a major difference between the two authors. Nilsson-Ehle used to understand plasticity as something "purely adaptive" (as opposed to "evolutionary adaptive" in Darwinian terms): he considered it from being part of self-regulation mechanisms (mechanisms by which an organism responds to environmental changes by phenotypic changes without necessarily a genetic basis). Therefore, Nilsson-Ehle, contrary to all expectation (since he realized his works in the context of the emerging Mendelian genetics), did not define plasticity as "a property of a single genotype" such as Johannsen (1911) who understood the "norm of reaction" as the range of reaction of a single genotype to changing environments. The fact that Nilsson-Ehle did not refer to "plasticity" in accordance with theories developed by Johannsen – one of the most influential scientists of

[5] For Nilsson-Ehle's explicit reference to plasticity, see Nilsson-Ehle (1914).

the rising discipline of genetics at that time – explains maybe why geneticists, before Bradshaw's seminal study in the mid-1960s, would not use so much the term "plasticity."

Bradshaw, on his side, considered "adaptive plasticity" in an evolutionary perspective. As such, he thought that it might be submitted to a genetic determination of some sort. If the formulation of the notion of "norm of reaction," in the beginning of the 20th century could be understood as a key-notion in the story of Mendelian genetics, involving a mitigation of genetic determinism, with Bradshaw the notion of "plasticity" became part of the story of quantitative genetics. The review he offered of it opened the possibility for a precise analysis of its evolution.

However, if Bradshaw opened a new field of study – the study of the evolution of plasticity with optimality models – that will be further developed in the 1980s, in his review of 1965, he was mostly discussing his predecessor's positions. It seems that some of these discussions might have introduced confusions for quantitative geneticists who started being interested on plasticity in 1980's. For instance, Bradshaw distinguished what he called "physiological plasticity" (which corresponds to all different forms of plasticity)[6] from "morphological plasticity" (which corresponds to a particular manifestation of physiological plasticity, that refers to developmental changes). Such a distinction should be linked to the fact that in the 1940s, the Austrian embryologist and geneticist Richard Goldschmidt (1878–1958) had made a clear distinction between "norm of reaction" and "norm of reactivity" (Goldschmidt , 1940, p. 250). He used the distinction to stress the genetically determined nature of the "norm or reaction" compared to the "norm of reactivity" (that would be applied to any physiological reactivity of the organism), while Bradshaw equated "physiological plasticity" and "morphological plasticity" and

[6] That is not the current standard usage anymore, which mainly limits physiological plasticity to physiological traits (like photosynthetic rate). However there is a current tendency to say that phenotypic plasticity should be seen as developmental plasticity (e.g., West-Eberhard, 2003, Gilbert & Epel, 2009; for more on this topic, see Nicoglou, 2013a).

considered that both of them were genetically determined. According to Bradshaw, in both cases, one was dealing with "adaptive plasticity." Therefore, the only reason, for him, to keep drawing a distinction between the two notions – morphological plasticity versus physiological plasticity – was to make clear that the kind of plasticity biologists were mainly dealing with, in their work, was "morphological plasticity," since this type of plasticity was most easy identified in nature. However, since physiological plasticity would start to be broadly studied in quantitative genetics – but with a different meaning from that of Bradshaw[7] – such a distinction (between physiological and morphological plasticity) might appear nowadays as a source of confusion for biologists who are dealing with plasticity.

In order to refine his definition, Bradshaw also made a distinction between plasticity and another phenomena: "phenotypic flexibility." This was likewise used by Thoday (1953) to describe the ability of an organism to function in a range of environments. In Bradshaw's view, he difference between "plasticity" and "flexibility" is that "plasticity" depicts the *variability* of the norm of reaction while "flexibility" may also include stable responses to environmental variation. For Bradshaw, "flexibility" had nothing to do with "norm of reaction," since there was no actual phenotypic change. Therefore, he did not consider heat resistance as a plasticity phenomenon but only as phenotypic flexibility. However, it seems that his position concerning such a distinction remains somehow unclear to his reader, since he kept pointing out the fact that phenotypic flexibility itself could occasionally involve both stable and plastic responses (Bradshaw, 1965, p. 117). This position would also be a source of confusion in the interpretation Bradshaw's readers did of his conception of plasticity. They would either consider

[7] See previous footnote.

plasticity as something definite, either as something that should, itself, be related to an adaptive trait.

After the publication of Bradshaw's article, in 1965, this difficulty will precisely become the subject of many controversies regarding the definition of "phenotypic plasticity." In this respect, the debate between the two pair of opponents Lande and Via on the one side, and Sheiner and Lyman on the other side – and more specially the precise opposition between Sara Via and Samuel Scheiner[8] – can partly be seen as a symptom of a tacit confusion, introduced by Bradshaw, concerning his definitions of plasticity. In order to understand more precisely some of the reasons and expressions of the controversy, I will now turn to a sharper analysis of Bradshaw's evolutionary significance of phenotypic plasticity.

Since, for Bradshaw, plasticity was genetically determined, and since he saw it as an adaptation, he was, therefore, convinced that the general assumption, which associates the plasticity of a trait to its developmental pattern, was not exhaustive enough. And as a plant biologist, Bradshaw mainly argued against plant biologists on this topic. His argument was the following. First, Bradshaw related the fact that,

> Stebbins (1950) has argued that characters formed by long periods of meristematic [the meristem is the tissue in most plants that contains undifferentiated cells and that one founds in zones of the plant where growth can take place] activity (such as over-all size, leaf number, etc.) will be more subject to environmental influences and are likely to be more plastic than characters formed rapidly (such as reproductive structures) or than characters whose pattern is impressed on primordia at an early stage of development (such as bud scales, leaves, etc.). This argument can be supported by evidence of the differences in plasticity shown by different characters in *Achillea* and *Potentilla* in the experiments of Clausen and associates (1940, 1948). The plasticity shown by the characters of these

[8] It seems that the personality of the two biologists has been a major reason in the crystallization of the debate. This clearly appears in the interviews of Scheiner and Via conducted by Erick Peirson, that he has kindly brought to my attention.

> species can be related to their general pattern of development, i.e., its duration, complexity, the number of interacting processes involved, etc. (Bradshaw, 1965, p. 117-118)

However, Bradshaw referred to a deductive theoretical reasoning to reject this argument:

> If it was assumed that the degree of plasticity shown by a character was the outcome of the basic pattern of its developmental pathway, certain deductions would follow. First, since general pathways of characters cannot be changed readily, it should not be possible for plasticity to change readily. Second, since the same organ, e.g., leaf, usually has the same basic developmental pathway in different species, the organ should show the same plasticity in different species. (Bradshaw, 1965, p. 119)

Bradshaw argued that those assumptions were false by referring to a number of studies, which showed that plasticity differs among different species that exhibit the same type of development.[9] Therefore, he considered that the plasticity is not specifically linked to the development of the plastic trait. For similar reasons, he also rejected the idea that plasticity is a property of the entire genome. Finally, he based his conception of plasticity on three main points:

(1) Plasticity is a property specific to individual characters in relation to specific environmental influences.

(2) Since plasticity of a trait changes among the different species of one genus and among the different varieties of the same species, Bradshaw concluded to a genetic independence of the plastic trait with the plasticity of a trait. He stressed that "such differences are difficult to explain unless it is assumed that the plasticity of a trait is an

[9] Most of the studies refer to a conspicuous type of plasticity: the heterophylly shown by certain water plants. Heterophylly is rarely characteristic of a whole genus; more commonly, it is found only in particular species. For this reason, this type of plasticity is particularly handy to Bradshaw's argument.

independent property of that trait and is under its own specific genetic control" (Bradshaw, 1965, p. 119).

(3) Finally, he quoted Waddington's works on canalization and genetic assimilation (1953b) and argued that a large measure of control on the degree of modification possible is afforded by the developmental pathway, the epigenetic landscape, which has its own genetic determinism as much as plasticity which offer the possibility to switch from one developmental pathway to another.

Taken together, these three points allowed Bradshaw to conclude that plasticity – genetically determined – is necessarily submitted to natural selection as any other trait in nature. Consequently, Bradshaw's approach was to introduce 'more' genetics rather than more development or environment when trying to make sense of plasticity.

In 2006, two years before his death, Bradshaw was invited to speak at a symposium organized by the Journal *The New Phytologist* and to comment his article of 1965. In the resulting article (his last published article), Bradshaw described the conference and offered a fascinating perspective on the origin of his ideas in the field (Bradshaw, 2006). The article reveals, among other things, that his position has ultimately not much changed since the 1965 article, except, at least, that its has been further informed by recent works in the field. Such a determination also demonstrates a fixation of Bradshaw's views of the concept of "phenotypic plasticity," after he gave it its first literature review in 1965.

Bradshaw viewed his legacy correctly; the concept of "phenotypic plasticity" became widely used in biological literature after the publication of his review (see next section). "Phenotypic plasticity" became an *operative concept* in biology where, in the past, and especially in embryology, the adjective "plastic" was mainly used with a metaphorical and an architectonic connotation linked to its use in natural philosophy from the 17th century

onwards.[10] Because of this scientific meaning, Bradshaw also permanently bound plasticity to the field of genetics (and even quantitative genetics), since the term "plasticity" became widely used in biology after Bradshaw's definition in the field of quantitative genetics.

It can also be noted that various past uses of the term are overshadowed by Bradshaw's definition of plasticity (Nilsson-Ehle's use but also the embryologists and zoologists' uses before him, such as Gavin de Beer's use (see, de Beer, 1940)) mainly because of the rise of genetics at this time and its importance for evolutionary studies. This settles a disciplinary division in which the geneticists are taking control of the terms of the debate, the scope of the conceptions, and above all are defining the parameters of evolutionary biology, versus the embryologists, the zoologists and even the botanists whose conception of plasticity is left out. It is in this precise context that was settled the debate, which arose in the 1980s concerning the evolution of phenotypic plasticity. Therefore, before detailing both views concerning the evolution of phenotypic plasticity, I shall first detail the general context and conditions of the debate.

3. The debate between Lande & Via and Scheiner & Lyman

Following Bradshaw, and in the context of the Modern Synthesis in evolutionary biology, population geneticists interested in plasticity would mainly study its *evolution*. More precisely, they sought to understand *to what extent* natural selection could explain the evolution of plasticity. Therefore, their attention focused on what they called "adaptive plasticity."[11] Like Bradshaw, these authors also downplayed the importance of developmental patterns in their understanding of plasticity. However, at this time – in

[10] For more about the use of the concept of « plasticity » in the philosophy of nature in the 17th and 18th centuries, see Nicoglou (2012).

[11] It should be noted that the different authors of the debate did not have exactly the same view on what "adaptive" means. However, since they used the same term "adaptive," they were under the impression that they referred to the same thing.

1980s – long after Bradshaw, when the discipline of quantitative genetics was already settled, it was not a claim for these authors but rather a matter of fact linked to the disciplinary constraints confining them to certain questions, such as: how to understand with genetic tools the evolution of phenotypic plasticity?

When first quantitative genetics models were established in order to describe the evolution of "phenotypic plasticity," the notion of plasticity was broadly defined in the same way Bradshaw used to define it: to characterize the phenotypic response to the environmental variations. At the beginning of the synthetic theory of evolution (in the 1930s-1940s), quantitative genetics became a common tool used in biological studies of evolution (e.g., Fischer, 1930; Wright, 1949). Later, it never stopped drawing attention in the analysis of phenotypic evolution (e.g., Lande, 1980; Falconer, 1981; Cheverud *et al.*, 1983; Lande & Arnold, 1983; Slatkin, 1987; Barton & Turelli, 1989; Shaw *et al.*, 1995; Roff, 1997). It became therefore natural that these techniques were also used to assess the evolution of phenotypic plasticity in works that heralded a new line of research on plasticity (e.g., Falconer, 1952; Via, 1984a, 1984b; Via & Lande, 1985). At this point, I would like to suggest that even if people dealing with studies in evolution were mainly dealing with genetic factors, the problem of the evolution of plasticity was not only related to the question of knowing in which way genes were involved in the determination of plasticity. It was also related to the *definition* of plasticity.

In an article of 1986, Carl Schlichting, with an early interpretation, suggested that Via and Lande's type of study illustrated a renewed interest in the study of plasticity, after a period of twenty years, from Bradshaw, when such studies had been limited. At the same time, he admitted failing to understand the precise reasons for such a long eclipse and could only offer few suggestions:

> Until 1980, theoretical work on plasticity was limited; and empirical research, with the notable exception of Subodh Jain's efforts [Marshall and Jain 1968; Jain 1978, 1979], was largely unfocused. The reasons for such neglect are puzzling, especially considering the clarity of Bradshaw's review. Surely part of the problem was the growing fascination with the detection and measurement of "genetic" variation, of which plasticity must have seemed the antithesis. Another problem was that environmentally induced variability in an experiment is typically avoided at all costs. Experimental complexity and the problem of measuring plastic responses also retarded progress. (Schlichting, 1986, p. 669)

Schlichting is certainly true from a disciplinary point of view, in this interpretation. However, he does not highlight the fact that at this time, when biologists were facing the problem of the evolution of plasticity, they had to do it mainly through a *genetic approach*, since their tools of analysis for phenotypic evolution were quantitative genetics. Like Bradshaw, they did not specifically distinguish different possible *types* of plasticity with different definitions (such as "histogenetic plasticity," "phylogenetic plasticity" or even "genetic plasticity" itself).[12] Most of them naturally focused on what they knew the best: "genetic plasticity," which had handily been defined by Bradshaw. Schlichting suggested, then, that the observed neglect between 1965 and 1980, concerning plasticity, was probably the result of an effect of myopia due to an exclusive focus on the genetic basis of variation rather than on its "plasticity."

However, I argue that if one adopts a larger scope than those of the discipline of quantitative genetics, the causes of this perceived absence are not only the result of a focus on stability/genetics (rather than unstability/plasticity), but they are also linked to some remaining difficulties in *defining plasticity*. Indeed, as I have shown previously, Bradshaw's review was sometimes source of confusions concerning his understanding of the notion (e.g., concerning "adaptive plasticity"). Furthermore, when one looks at the uses

[12] It was the British zoologist Gavin de Beer who first offered, in 1940, such a distinction between the different kinds of plasticity in his book *Embryos and Ancestors.*

of the term "plasticity" throughout the different disciplines of biology, one realizes that its study has, ultimately, still remained active between 1965 and 1980 (even if it was not specifically in genetics). Indeed, between 1965 and 1980 many ecologists, evolutionary biologists and botanists were interested in issues related to plasticity, both from a morphological and a physiological point of view (e.g., Harper, 1967; Rehfeld, 1979; Baker, 1974). Their understanding of plasticity in biology relied on dynamic phenomena analysis such as homeostasis (e.g., Ashby, 1952; Thoday, 1953, 1955, 1958; Hyde, 1973) or canalization (e.g., Waddington 1953a, 1957). Although all these studies did not explicitly and technically referred to the term "plasticity," it remains clear that they were all dealing with "plastic phenomena." In other words, in the 1980s, the term is still not fixed in biology, but is a malleable term about to be variously deployed and defined. Thus, its meaning and construction was still under debate (exposing the assumption about the local processes of that construction).

However, in 1985, when the field of quantitative genetics was already settled, the *idea* of plasticity in biology became mostly associated with the notion of "phenotypic plasticity" – previously defined by Bradshaw – and was mostly seen as a notion of (quantitative) genetics (as opposed to a more intuitive or philosophical notion of plasticity).[13] With the article of 1965, Bradshaw had already contributed to the rejection of an "indirect" analysis of the evolution of plasticity, through the analysis, for instance, of the *evolution of stability* or through the analysis of its different forms of expression (i.e., homeostasis or canalization). He believed that the observation of a certain "lack of stability" should be considered as something different from "plasticity" (Bradshaw, 1965, p. 117). Therefore, the study of plasticity has been relatively neglected for a number of years after Bradshaw especially if one has in mind the study of evolution of plasticity. But

[13] For more on this question, see Nicoglou, forthcoming.

the key point is that, during all these years, some noticeable studies continued to address the issue of plasticity and even question its definition (e.g., Shapiro, 1976; Cavalli-Sforza, 1974; Caswell, 1983; Smith-Gill, 1983). This illustrates the fact that, even after Bradshaw, two related problems remained that cannot be totally separated: with what model should one describe the evolution of plasticity? And what general definition could be given to the notion? If the first problem has mostly been worked out (despite the fact, or because, it provided a debate in 1980s), the second problem was not acknowledged, from the beginning, and led to a disputed genealogy of phenotypic plasticity after the debate (that could, or could not, be linked to development/phenogenesis, depending on the conception each members of the debate had of plasticity). I will now look more precisely at those conceptions.

3.1 Sara Via and Russell Lande's conception of the evolution of plasticity

Sara Via met Russ Lande while she was in graduate school and became one of her disciples, before working with him as a post-graduate student. Before working with Lande, Via was interested in how populations adapt to heterogeneous environments, and more precisely in population divergence and differentiation. Her question was about the status of individuals among the species: are the species that looks like generalist only like that because the individuals are placed in a specific environment; if they were placed in different environments would they act differently? In other words, her question was: in the generalist species, are individuals generalists or specialists? From this question, she then got into genotype-environment interaction by figuring out that she could study this question, which concerned generalist species, by using genotype-environment interaction and by testing populations in two environments (in her case, two host plants for leaf miners insects, her animal study model) (Via, 1984a; 1984b). From this first study, her interests

turned into how specialization could evolve and she started working with Russ Lande on the measure of – not specialization – but variation in response to the environment.[14] The variation in response to the environment had been described by Bradshaw, who focused on plasticity shown by a genotype when its expression is able to be altered by environmental influences. Quantitative genetics focused on variation in multiple environments. For this reason, Via left the context of adaptation to heterogeneous environment, genotype-environment interaction, and went straight to the study of the evolution of plasticity.

Therefore, while Lande and Via's explanatory model for the evolution of plasticity required quantitative genetics tools, it did not strictly speaking rely on a "quantitative genetic" definition of plasticity. Via was interested in plasticity because of the question of *adaptation* to heterogeneous environment, rather than because of the question of *variation* in multiple environment (the focus of quantitative genetics). Furthermore, she did not see the genotype as synonymous with "the reaction norm," and had come to plasticity because of her interest in genotype-environment interaction. Indeed, Via and Lande defined phenotypic plasticity by referring to Bradshaw's definition, but also to Georgy Gause (1910–1986) and Ivan Schmalhausen's (1884–1963) conceptions of plasticity:

> Environmental modification of the phenotype is common in the quantitative (polygenic) characters of organisms that inhabit heterogeneous environments. The profile of phenotypes produced by a genotype across environments is the 'norm of reaction' (Schmalhausen, 1949); the extent to which the environment modifies the phenotype is termed 'phenotypic plasticity' (Gause, 1947; Bradshaw, 1965). Because phenotypic response to environmental change may facilitate the exploitation of some environments and provide protection from others, the level of plasticity in a given trait is thought to be molded by selection (Gause, 1947; Schmalhausen, 1949; Bradshaw, 1965). (Via & Lande, 1985, p.505)

[14] Via thought that the moment you analyze two environments, it implies variation whether it is about specialization or not and that it implies genotype-environment interaction. From Sara Via's interview by E. Peirson on Monday, May 13, 2013.

From the start, Via and Lande's definition of plasticity provided a more nuanced picture, compared to the definition proposed by Bradshaw, which was: "*Plasticity* is […] shown by a genotype when its [phenotypic] expression is able to be altered by environmental influences" (Bradshaw, 1965, p. 116). Obviously, while the definition introduced by Bradshaw had tended to focus on the genotype, Via and Lande had in mind the conceptual controversies based on Soviet authors' views embracing genotype-environment interactions more fully (i.e., Gause, 1947; Schmalhausen, 1949), and which were still relevant in their opinion in 1985.

Via's interview by Peirson (May 13, 2013) seems to confirm that her conception of plasticity was not only influenced by Bradshaw's definition but also by those Russian authors.[15] For Via, plasticity was "different phenotypes in different environments." She was mainly thinking "in terms of genetic correlations and what causes a genetic correlation." She thought of plasticity as "adaptation to different environments." It means, "in some cases, the same gene could be expressed in different ways" (more or less expressed), or "some genes have an impact only in one environment." In other word, she associated plasticity with behavioral ecology rather than with a direct explanation by quantitative genetics.

In line with the model for the evolution of plasticity proposed with Lande in 1985, Via[16] offered, in 1993, a theoretical definition of phenotypic plasticity in accordance with their common model. In the article of 1993, she tried to show that there was a kind of agreement within population geneticists' community interested on plasticity (e.g.,

[15] It should be noted that there were many earlier studies of genetic divergence vs phenotypic plasticity that should be intimately linked to Via's own work (e.g., Berven, 1982; Stearns, 1977).

[16] Because of a personality issue between Sara Via and Samuel Scheiner, the dispute concerning the understanding of the evolution of phenotypic plasticity would then mainly be seen as being between those two authors.

Schlichting, Stearns, etc.), based on the idea that adaptive plasticity evolves under natural selection. She argued that the disagreement only arose between them concerning the description of the precise means of its evolution. According to Via, "the assertion that phenotypic plasticity is a character that is independent of trait means and the attendant implication that plasticity itself is the target of selection" (Via, 1993, p. 352) was based on an allegedly erroneous analysis of certain phenomena by Bradshaw. She thought that this erroneous analysis was due to the lack of sufficient data in quantitative genetics in 1965, when Bradshaw wrote his article. But the reasons of Via's disagreement with her colleagues should rather be related to a certain confusion expressed by Bradshaw, himself, concerning the definition of "adaptive plasticity," since he was not properly doing quantitative genetics.

Furthermore, from her observations of genetic studies with Lande (Via and Lande 1985, 1987; Via 1987), Via concluded that "partial genetic independence between the character states expressed in two environments is a minimal requirement for the evolution of a new reaction norm." From the idea that two genetic mechanisms can produce some degree of genetic independence between character states, she assumed in 1993 that "(1) two character states expressed in different environments will have some genetic independence if allelic sensitivities to the environment differ. […] (2) Partial independence between character states in different environments can also result if some loci influence the character state only in one of the environments, that is, if there is some environment-specific gene expression […]" (Via, 1993, pp. 355-356). Via was then convinced that the evolution of the adaptive norm of reaction could only occur through the phenotypic traits themselves. Selection did not directly act on plasticity, and therefore plasticity could not be considered as a separate trait with its own genetic etiology. However, one problem she had

to deal with was the correlation between the evolution of plasticity and the evolution of the trait expressed in the environment.

Therefore, from 1986, Carl Schlichting opened the controversy (he would be followed by others) by defending a very different position. By comparing two species of purslane (*Portulaca grandiflora* and *Portulaca oleraca*), Schlichting attempted to show that the plasticity of a trait might evolve independently of the trait. And after Via's paper of 1993, Peter Van Tienderen, in 1994, and Gerdien de Jong, in 1995, would establish quantitative genetic models to highlight the variation of plasticity in accordance with Schlichting's model. In these models, the existence of "plasticity genes[17]" appears attested, since the independence between trait mean evolution and the evolution of plasticity was empirically established.

The controversy between Via and Schlichting illustrates the persistence of theoretical and conceptual disputes, rather than actual empirical obstacles to the identification of a "genetic" basis of plasticity. While some biologists thought that a genetic basis of plasticity exists, the problem remains precisely what they understood by "genetic basis." This issue ties in with a certain degree of uncertainty concerning the proper conceptual approaches of plasticity to adopt, sometimes assumed to refer to *another phenotypic trait* that would be subject to selection, sometimes assumed to refer to a *property of living beings* for which the evolution should be analyzed without knowing the causes of the property.

3.2 Samuel Scheiner and Richard Lyman's model for the evolution of plasticity: defining "plasticity genes"

[17] The expression "plasticity genes" highlights the fact that the direct linear model between a single gene and a specific phenotype is rarely true. Models that are developed around the 1990's are polynomial models, which assess the plurality of genes implied in the expression of a phenotypic trait.

In line with a conception of plasticity as a specific "phenotypic trait," and in the context of researches in quantitative genetics, Scheiner and Lyman established, in 1991, a classification of what they considered as the "genetic basis of plasticity.[18]" According to them, three distinct categories cover the genetic basis of the plastic response. First, "overdominance[19]" expresses the fact that there is an inverse relationship between heterozygosity[20] and plasticity: the more a genotype is homozygous, the more its norm of reaction (its phenotypic response in different environments) is plastic. This model assumes, without formal demonstration, that plasticity is somehow an "accident" that is the result of a loss, or a reduction, of genotypic homeostasis[21], which leads to an excess of genotypic homozygosity (see Lerner, 1954; Gillespie & Turelli, 1989). Second, "pleiotropy" shows that plasticity is a function of the differential expression of the same gene (the same set of alleles) in different environments (e.g., Falconer, 1981; Via & Lande, 1985, 1987; Via, 1987). It is analogous to the case, in classical quantitative genetics, where several traits share a common genetic control due to the pleiotropic effects of genes. Finally, "epistasis" indicates that two classes of genes control two fundamental features of a norm of reaction: its plasticity and its overall mean. Therefore, for Scheiner and Lyman, plasticity is the result of the interaction between the genes that determine the magnitude of the response to the environmental effects with the genes that determine the trait mean expression (see Lynch & Gabriel, 1987; Jinks & Pooni, 1988; Scheiner & Lyman, 1989).

[18] Scheiner and Lyman conception is less sharp cut than De Jong's (1995) and Van Tienderen's (1991) conceptions because it does not focus on "genes of plasticity" but on the basis of plasticity's genetic expression. See Scheiner & Lyman (1991).

[19] The concept of overdominance is based on Lerner (1954) and Waddington's (1961) works.

[20] A heterozygous individual is a diploid individual that has different alleles at one or several genetic loci. A homozygous individual is a diploid individual that has equal alleles at one or several genetic loci.

[21] Homeostasis, or the "return to a genetic equilibrium", is linked to a homogenous repartition between maternal alleles and paternal alleles leading as a result to heterozygosity. See Pigliucci (2001) p. 69; p. 88–91.

This model assumes, in contrast to Via's, that the trait mean and the environmental variance are two independent features. For Scheiner and Lyman (1991), these three categories are not mutually exclusive and their uses apply to the effects that occur not in a single environment, but in different environments over time. For this reason, their conception is mainly phenomenological, that is to say, based on the observation of "types" rather than on an investigation of the "real causes" of plasticity. However, the underlying idea is that all the different types could be analyzed with the same tools, those of genetics. In this context, statistical quantitative genetic studies should be adequate to study any kind of models without knowing what is the actual role of the genes (de Jong, 1995). The fact that the model functions properly, together with its predictive ability, was somehow sufficient to prove its validity and to question the specificity of Via's conception.

While Via's conception was facing challenges by the polynomial approach[22] (Van Tienderen, 1991; Scheiner, 1993; Van Tienderen & Koelewijn, 1994), she kept arguing that it was not entirely rejected by the latter approach (Via *et al.*, 1995). For Via, who deeply disagreed with Scheiner, the "presupposed" independence between the "trait mean" and the plasticity, supported by the polynomial approach, remained to be confirmed. Via showed that the "trait mean" could be both measured for a single environment (independent environmental variability) and from a potential range of expression of the trait, which reflected, this time, the environmental variation in which the trait would be expressed. In the latter case, Via called it the "grand mean." Finally, she suggested that with a distinction between these two measures – the trait mean and the grand mean – it becomes possible to address, in a new way, the issue of the correlation between the evolution of the trait and the evolution of plasticity. For instance, in the case of a trait such as plant growth, the trait mean might be the same for two different species in two different

[22] The polynomial approach of plasticity involves considering the measure of plasticity as a polynomial function, which represents its genetic determinism, see Scheiner (1993).

environments (i.e., comparatively, the two species will grow likewise), while, in a single environment, the trait mean might be different for each species (i.e., one of the species will grow more than the other in the environment E_1 and vice versa in the environment E_2). The consequence of such a difference, in Via's view, is that, in the same environment, different species of plants might grow differently, whereas, in changing environments, their average growth might be the same. From this observation, Via concluded that phenotypic plasticity should be considered as a "by-product" of the selection exerted on different phenotypic trait means in changing environments and not as a specific trait like Scheiner suggested (Via, 1993, p. 164). In her understanding of the evolution of plasticity, she tried to include what she considered to be essential when trying to characterize plasticity: the specificity of genes-environment interaction (an aspect that Scheiner had voluntarily left out). Unfortunately, it seems that her attempt was not considered as a success because it appeared useless in a quantitative genetic approach.

Indeed, after the paper of 1995 (Via *et al.*, 1995), the debate was considered solved in favor of Scheiner's conception, redefined by Schlichting and Pigliucci (1993). However, some of the issues opened by this debate remained unsolved. The most contentious issue was not about how plasticity evolves, or what were its genetic bases, but it was about how plasticity should be defined, including or not the recognition of its causes. If neither side ever discussed the genotype-phenotype map (which is the underlying process[23]), Via's interest to the genotype-environment interactions in the beginning of her work, certainly drove that issue forward, despite the fact that she distanced herself from the topic when she started paying attention to plasticity, in the way it had been outlined by Bradshaw. Therefore, a common aspect between the two views (Via and Scheiner's views), and despite their differences, is that, like Bradshaw, they never analyzed precisely the

[23] Few quantitative geneticists did, although there were exceptions, e.g., Atchley's work (e.g., Atchley, 1984).

genotype-phenotypic map. This might help to explain why it remains difficult nowadays to associate this question with plasticity.

4. Conclusion

In order to solve the controversy between Via's approach and the polynomial approach, defended by Scheiner, Schlichting and Pigliucci proposed, in 1993, a new definition for "genes of plasticity" as: "regulatory loci that exert environmentally dependent control over structural gene expression and thus produce a plastic response. Such loci represent a genetic mechanism for plastic response that is distinct from that assumed in quantitative genetic models of reaction norm evolution" (Schlichting & Pigliucci, 1993, p. 366). The two authors concluded that the debate between Via and Scheiner could be solved, since they saw it as a result of a "semantic ambiguity" rather than as the expression of a fundamental divergence between two distinct positions. Therefore, they saw the distinction between the two opposite conceptions – "existence of plasticity genes" versus "plasticity as a by-product of selection" – only as something apparent, since both options were not mutually exclusive. However, even if this precise debate between Via and Scheiner appeared to be solved for quantitative geneticists, the impression that it might have at least two different possible views concerning plasticity in an evolutionary perspective remained (e.g., depending if one look, or not, at the causes of plasticity). If this problem exists concerning any phenotypic traits, it seems even more problematic concerning plasticity. Recently, Mary Jane West-Eberhard has suggested a third way forward, by taking into account a developmental perspective[24] (West-Eberhard, 2003, p. 33).

[24] For more on this perspective see Nicoglou, 2013a, 2013b.

The debate between Via and Scheiner concerning the evolution of phenotypic plasticity illustrates the fact that, even if empirical proofs of a genetic basis of plasticity have become more and more numerous since the mid-1980's, the change linked to a better understanding of the underlying molecular mechanisms (not only to the genetic ones) at the origin of phenotypic traits has led biologists to look at the underlying causes of plasticity. But if understanding how plasticity evolves – that is to say understanding the role of natural selection on plasticity – does not necessarily require referring to its causes, it does require having an idea of what plasticity is. With Bradshaw, plasticity had acquired a clear definition, at least in a certain field of biology – that of quantitative genetics – and a renewed position within scientific concepts. With the emergence of genetics and Bradshaw's works, the term "plasticity" became mainly used as an operating concept of genetics, whereas in the past it was more a metaphorical concept. This new status relies on seminal Bradshaw's article of 1965 but also on a renewed enthusiasm for the area of research of plasticity, first in ecology in the 1970s, then with the development of new tools in quantitative genetics in the 1980s. Despite their disagreement Via and Scheiner were both referring to Bradshaw's definition of plasticity (which did not give any detail concerning the cause of the process, or the genotype-phenotypic map). After the resolution of the debate between Via and Scheiner, Bradshaw's definition remained quite unchanged in biological literature probably because neither of the two camps had questioned it.

That being said, definitions rely on biological tools and since biological tools are continuously evolving it should be the same for their attached notions. Finally, the debate that occurred between Via and Scheiner illustrates the fact that in the 1980s, and despite Bradshaw's precise definition of phenotypic plasticity, it had already become fairly difficult for biologists to overcome some of the difficulties Bradshaw already encountered when he defined the notion of plasticity, as well as to separate it from the field of

quantitative genetics. If the debate concerning the evolution of phenotypic plasticity could be solved in the 1990s, it left open to considerations that did not follow from that theoretical framework, since it urged a precise analysis of the phenotype-genotype map. For this reason, I suggest that biologists who have an interest in "phenotypic plasticity" within the context of an extended synthesis of evolution including development should be aware of this aspect, and should maybe learn in the light of this knowledge how to distance themselves from Bradshaw's legacy.

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