

Chapter 2

IS PHENOTYPIC PLASTICITY ADAPTIVE?

Kimberly A. Hughes, Mary H. Burleson, and F. Helen Rodd

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Overview: Phenotypic plasticity is the tendency for organisms with the same genotype to produce different, but repeatable, phenotypes under different environmental conditions. Human behavior genetics is concerned with partitioning human variation into genetic versus non-genetic variation, and the non-genetic variation is due in part to phenotypic plasticity. Here we examine human phenotypic plasticity from the perspective of evolutionary biology. The question—to what degree is phenotypic plasticity adaptive—has been studied intensively during the past 15 years, but it has been studied almost exclusively in plants and non-human animals. Although the question has usually been asked of examples of plasticity that seem particularly likely to be adaptive, the results of empirical tests have been mixed. These mixed results suggest that many cases of apparently adaptive plasticity might fail to support the adaptive hypothesis when currently accepted methodology is applied. Here we describe the techniques that have been developed to test the adaptive hypothesis, and consider whether any of them can be successfully applied to the study of human phenotypic plasticity. We conclude that while many of the techniques of experimental evolutionary biology cannot be applied to humans, there are methods by which students of human behavior can successfully test the adaptive plasticity hypothesis..

1. Introduction

Many of the chapters in this volume describe genetic variation within human populations, and illustrate how that variation might translate into phenotypic variation in fertility. In this chapter, we consider another source of phenotypic variation: *phenotypic plasticity*. Plasticity is the tendency for organisms to produce different, but repeatable, phenotypes under different environmental conditions. For example, some plants produce leaves of different size or shape when grown in the shade and when grown in full sun (Bjorkman, 1981). Animals are also capable of phenotypic plasticity. Some aquatic invertebrates produce elaborate armored carapaces, but only when they are in the presence of predators; when predators are absent, the armor does not develop (Parejko & Dodson, 1991; Spitze, 1992). Even some vertebrates are capable of extreme phenotypic plasticity. In many reptiles, the temperature at which the eggs are incubated determines the gender of developing offspring (Shine, 1999). Several species of fish are sexually plastic—even as adults they can change from female to male (or vice versa), depending on the social environment in which they find themselves (Warner & Swearer, 1991).

In contrast, some traits and some species are highly *canalized*—they tend to produce invariant phenotypes under a wide range of environmental conditions. For example, all homeothermic (warm-blooded) vertebrates maintain nearly constant body temperature over a wide range of environmental temperatures. Other features of homeothermic vertebrates are less canalized. For example, development time (age at sexual maturity) depends upon nutritional status in many long-lived mammals (c.f., Robinson, 1996).

To a large degree, the field of human behavior genetics is concerned with partitioning human variation into genetic versus non-genetic variation. Assuming the partitioning is approximately correct, the non-genetic variation can be ascribed to sampling error, to pure environmentally induced variation (when individuals of similar genotypes produce different phenotypes in different environments, but respond in a similar way to the same environments), or to genotype-environment (GxE) interaction (when different genotypes respond to environmental variation in different ways). Both environmental and GxE variance can be composed of both random and non-random variation. Non-random variation is that due to a predictable, repeatable response of organisms to their environment—phenotypic plasticity. Non-random environmentally-induced variation can be thought of as that portion of the variation due to the average phenotypic plasticity of the average genotype, while the non-random GxE interaction is due to phenotypic plasticity that differs between different genotypes present in the population.

While many of the chapters that comprise the two volumes in this series deal directly with partitioning human variation into genetic and non-genetic components, some of them discuss variation that can be described as phenotypic plasticity (Belsky, 2000; Doughty & Rodgers, 2000; Mealey, 2002; Rowe, 2000). Phenotypic plasticity can be an important component of many behavioral, physiological, and morphological traits with effects on fertility. Although empirical data are scant, it seems reasonable to suppose that traits such as age at sexual maturity, fecundability, and child-rearing motivation might display phenotypic plasticity in addition to genetic variability.

Many of the chapters in this volume that deal with phenotypic plasticity are also concerned with whether this plasticity is adaptive (see Figure 1). That is, did the plasticity evolve by natural selection because genotypes displaying plasticity had higher Darwinian fitness than did genotypes displaying canalization? Here, we attempt to place this question in the broader framework of evolutionary biology. The question—to what degree is phenotypic plasticity adaptive—has been studied intensively during the past 15 years. There are several alternative hypotheses to explain plasticity: 1) passive, fitness-neutral response; 2) deleterious consequences of poor environmental conditions; 3) constraints imposed by biochemical and biophysical reactions. Although no consensus has been reached on the degree to which phenotypic plasticity is generally adaptive, agreement has arisen on methodologies for answering the question in specific cases. However, this progress has been achieved only in studies on non-human organisms. Our purpose here is to explore whether any of the methods developed for studies of plants and non-human animals can be successfully applied to the study of human phenotypic plasticity.

2. History and Background

The study of phenotypic plasticity has a long history within biology (see Schlichting and Pigliucci (1998) for a thorough review). Until recently, most discussion of plasticity has assumed that plastic responses to the environment are adaptive—that is, plasticity would be seen to increase individual fitness if plastic individuals could be compared to similar, but non-plastic individuals. As early as 1896, a comparative psychologist named James Mark Baldwin realized that plastic responses to the environment could arise by natural selection (Baldwin, 1896; Schlichting & Pigliucci, 1998). Later, Ivan Ivanovich Schmalhausen (1949) described many cases of plasticity that were apparently adaptive, along with some examples for which no adaptive explanation was obvious. In 1952, Conrad H. Waddington demonstrated that a plastic response to environmental cues could be changed by artificial selection in his famous “genetic assimilation”

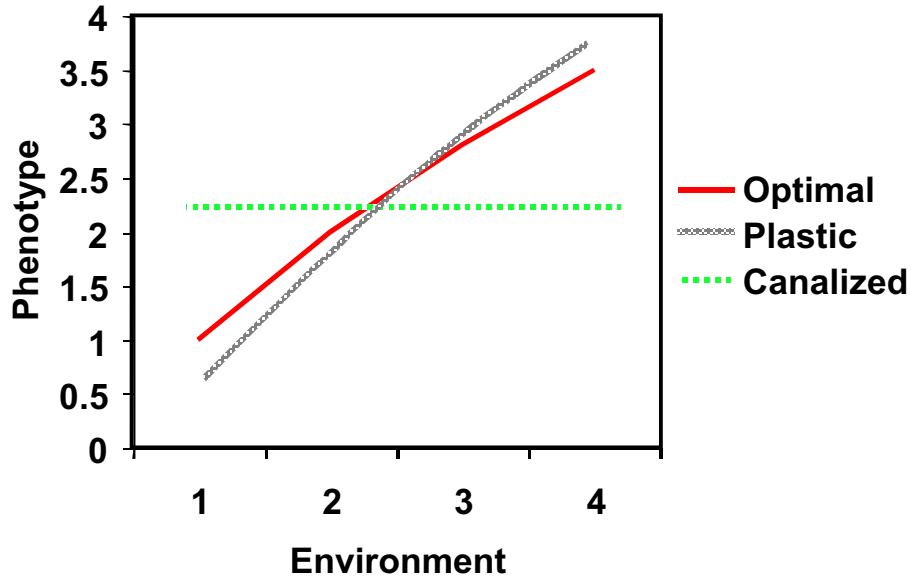


Figure 1: Illustration of adaptive phenotypic plasticity. Environmental variation is shown on the horizontal axis. This axis can represent four discrete environments (e.g., presence/absence of different predators) or a continuous environmental gradient measured on some scale (e.g., temperature). For a continuous-environment case, the solid line illustrates how the optimum phenotype (that with the highest fitness) differs in different environments. For example, the optimal body mass might increase as the temperature increases from 1 to 4. The shaded line shows the phenotype produced in each environment by a phenotypically plastic genotype, and the dotted line shows a canalized genotype that produces the same phenotype in all environments. In the case illustrated here, the plastic genotype produces a near-optimal phenotype in all four environments, while the canalized genotype produces phenotypes far from the optimum in environments 1 and 4. Because the plastic genotype is generally nearer the optimum phenotype, it can be assumed to have higher fitness than the canalized genotype.

experiments on fruit flies (Waddington, 1952). In those experiments he showed that the level of phenotypic plasticity could respond to selection, and was therefore a heritable characteristic. Waddington's experiments convincingly demonstrated that plasticity could potentially evolve by natural selection, and therefore could be an adaptive feature of organisms (also see Scheiner & Lyman, 1991).

Recently, studies of phenotypic plasticity have become a prominent component of the evolutionary literature. In an influential paper, Bradshaw (1965) emphasized that phenotypic plasticity is not necessarily adaptive. At about the same time, evolutionary biologists began to question and challenge the "adaptationist paradigm" that was common in some disciplines (Gould & Lewontin, 1979; Rose & Lauder, 1994).

One result of this renewed emphasis on adaptation as a hypothesis, rather than an unquestioned assumption, was that, in the early 1980s, both theoretical and experimental biologists began to ask how adaptive plasticity might be distinguished from non-adaptive environmentally-induced variation (Via & Lande, 1985). Phenotypic plasticity can, in theory, result from a number of different nonadaptive processes. For example, plasticity could be due to a passive reaction of the organism to different environmental conditions, with little or no effect on fitness (Winn, 1999). The bleaching of animal hair when exposed to sunlight is likely an example of this kind of passive response. Alternately, it could be caused by biophysical or biochemical constraints. For example, the reduced rate of development that ectotherms, such as amphibians and insects, experience at low temperatures is well documented (Newman, 1992). Slowed development certainly has fitness effects, but cannot necessarily be considered an adaptation to cold. Finally, plasticity could arise because some extreme environments can have deleterious effects and induce an aberrant phenotype that has generally low fitness (see Figure 2). Fruit flies (and many other organisms) develop aberrant, low-fitness phenotypes when exposed to high temperatures during development (Eshel & Matessi, 1998, see Figure 2). A human example of this phenomenon is found in Northeast Ethiopia. Diet shifts during droughts can lead to a debilitating neuromuscular disorder known as lathyrism. The disorder appears to be due to increased consumption of a drought-resistant legume, the grass pea (Getahun, Mekonnen, TekleHaimanot, & Lambein, 1999).

Several biologists have pointed out that functional arguments or arguments based on plausibility are not sufficient to demonstrate that plasticity is adaptive (Dudley and Schmitt 1996; Kingsolver & Huey, 1998; Winn 1998). It is now an accepted principle in evolutionary biology that adaptive hypotheses for plasticity should be tested explicitly. Given that adaptive hypotheses should be tested, is it possible to do so in humans? This

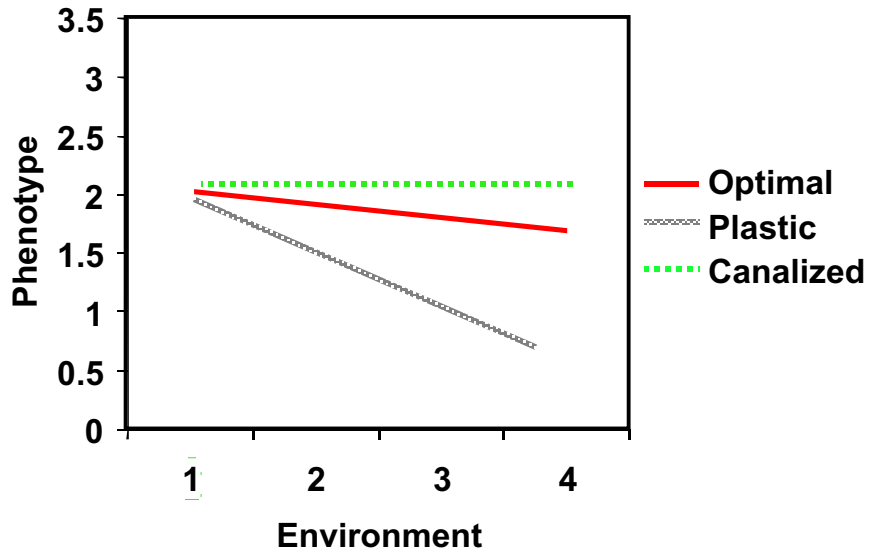


Figure 2. Illustration of nonadaptive plasticity. The plastic genotype produces a phenotype that is generally farther from the optimum than that produced by the canalized genotype. Over all environments, the canalized phenotype has highest average fitness.

is the question we have set out to answer in this chapter. We first describe how studies of plants and animals have tested whether phenotypic plasticity is adaptive (Dudley & Schmitt, 1996; Kingsolver & Huey, 1998; Thompson, Jeung, & Thoday, 1998). We next outline how current trends in the study of phenotypic plasticity in plants and animals might be used as models for designing studies of phenotypic plasticity in humans.

3. Theory

Models of Phenotypic Plasticity

Mathematical models of the evolutionary process indicate that the capacity for adaptive plasticity will evolve only under certain circumstances. Many different types of models have been used, but they can be broadly characterized as optimality models, quantitative-genetic models, and gametic models (after Scheiner, 1993). All these models share certain features: 1) at least two different environments are modeled, and the optimal phenotype (the phenotype with the highest fitness) differs in the different environments; 2) plastic phenotypes are compared to non-plastic phenotypes to determine under which circumstances the plastic types will have an overall fitness advantage; 3) often, plastic phenotypes incur a “cost of plasticity” that is not incurred by non-plastic phenotypes. A cost of plasticity is a reduction in fitness resulting from having the ability to produce different phenotypes in different environments. These costs can be due to the maintenance of sensory or developmental mechanisms, to risks associated with gathering information on the current environment, or to a cost of “mistakes” such as producing the wrong phenotype in a given environment (see detailed discussion below).

Optimality models are phenotypic models that ignore the genetic basis of phenotypes and the genetics underlying variability. These models are useful because they allow the investigation of complex types of costs and benefits of plasticity that would be cumbersome or impossible to model using a genetic approach. Several researchers have used optimality models to investigate which cost-benefit functions are expected to lead to the evolution of phenotypic plasticity (e.g., Houston & McNamara, 1992; Lively, 1986; Stearns & Koella, 1986).

Quantitative genetic models are based on the effects of selection on the genetic variances and covariances among a suite of traits that are assumed to be polygenic and to be affected by the environment (Falconer & Mackay, 1996). In one of the earliest models of the evolution of plasticity, Via and Lande (1985) used a quantitative genetic approach in which a trait expressed differently in two environments was treated as two genetically correlated traits. Each genotype was characterized by the mean phenotype it produced

in each environment. The genetic correlation expressed the degree of correspondence between the environment-specific phenotypes produced by the range of genotypes in the population. Gomulkiewicz and Kirkpatrick (1992) extended this approach by replacing discrete phenotypes produced in discrete environments with a continuous function that described the mean character state in a continuously varying environment. Each genotype within a population could then be characterized by a particular functional relationship between phenotype and environment (Figure 3).

The third class of models—gametic models—specifies the effects of different alleles at one or two loci. These models are particularly powerful because they can explicitly incorporate genetic phenomena that the other types of models cannot: linkage, pleiotropy, and epistasis. They can therefore tell us if different types of “genetic architecture” have important consequences for evolution. This approach is less mathematically tractable than the other approaches, however. Usually, only one or two loci can be modeled simultaneously, which limits the potential for costs and benefits and other kinds of interactions to be included. Examples of these types of models can be found in Levins (1968), Orzack (1985), De Jong (1989; 1990) and Scheiner (1998).

Factors Limiting the Development of Phenotypic Plasticity

Although details of the models vary enormously, many predictions have been consistent across models. First, evolution of plasticity depends upon availability of appropriate genetic variation. If the correct kind of genetic variation is not present, then a plastic genotype with high fitness in all environments cannot evolve (Gomulkiewicz & Kirkpatrick, 1992; Scheiner, 1993; Via et al., 1995). Gomulkiewicz and Kirkpatrick (1992) argue that appropriate variation may often be lacking when many different environments are experienced, or when environmental variation is continuous, because of the complexity of the phenotype-environment matching (Figure 3). To our knowledge this assumption has not been tested explicitly. Although a few experiments have shown that selection on plasticity in two or three different environments can be successful (e.g., Brakefield et al., 1996; Hillesheim & Stearns, 1991), we are not aware of any attempt to select on plastic response to many different environments. In principle, a model organism with short generation times could be used to determine whether a canalized phenotype is capable of evolving into a plastic phenotype that is well adapted to a complex range of environmental conditions.

The models also predict that plasticity will only evolve when the “cost” of plasticity is low (DeWitt, 1998; Gomulkiewicz & Kirkpatrick, 1992; Scheiner, 1993; Van Tienderen, 1991; Via et al., 1995; Via & Lande, 1985).

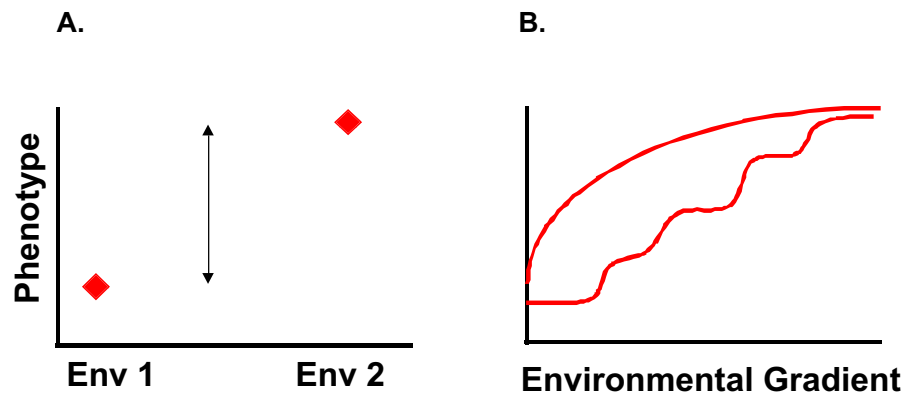


Figure 3. Different types of plasticity that have been modeled. Panel A: in the two-environment case, a genotype is characterized by the change in the mean phenotype it produces in the two different environments, indicated by the double-headed arrow. Panel B: in the continuous-environment case, a genotype is characterized by a functional relationship between phenotype and environment. The smooth curve shows a relatively simple functional relationship, while the wavy line shows a more complex relationship.

As mentioned above, the cost of plasticity is any fitness disadvantage suffered by a plastic genotype in comparison with a genotype that produces a fixed phenotype in all environments. Such costs can include the energy used by inducible regulatory mechanisms, energy use or risk involved in information acquisition (so that the correct phenotype is produced), developmental instability (possible mistakes), and genetic costs (e.g., deleterious effects of pleiotropy and epistasis). Costs of plasticity should lead to limits in the degree of plasticity that can evolve, sometimes to the extent that a single generalist phenotype is favored by selection (DeWitt, 1998; Newman, 1988; Scheiner & Berrigan, 1998).

Only a few studies have tested for the existence of a cost of plasticity. In a large experiment, DeWitt (1998) looked for a cost of plasticity in the freshwater snail *Physa heterostropha*, which exhibits plasticity in shell morphology in response to the presence of two different predators. Using 29 different families, he found a significant negative correlation between the average family plasticity in shell morphology and family mean growth rate. He found no evidence of a reduction in adult fitness with increasing plasticity in shell morphology. Newman (1992) reported a negative correlation between plasticity in size at metamorphosis and the time required to reach metamorphosis in five full-sib families of spadefoot toad tadpoles, demonstrating a cost of plasticity. These results suggests that costs of plasticity may tend to occur during development, rather than during adulthood. In contrast, Scheiner and Berrigan (1998) found no evidence of a cost of plasticity in defensive morphological traits among 47 clones of *Daphnia pulex*, and Donohue et al. (2001) found none for leaf shape in *Impatiens*.

Depending upon the type of trait and the type of environmental variation, other factors can also limit the evolution of plasticity. Two types of traits can display plasticity: traits that are fixed during the lifetime of an organism, and traits that can change during the lifetime of an organism (labile traits). Most behaviors are labile traits, as are some morphological traits (body weight and muscle mass, for example). The conditions for plasticity to evolve in labile traits are less stringent than those for fixed traits. Unless costs are prohibitive or the appropriate genetic variation does not exist, adaptive plasticity in labile traits is likely to evolve (Gomulkiewicz and Kirkpatrick 1992).

For fixed traits, plasticity is most likely to evolve when the following conditions are met: 1) differences between environments are large; 2) different environments are equally frequent; 3) strength of selection is equal in different environments; 4) individuals reproduce in the environment in which they have the highest fitness; 5) the environmental "cue" that triggers alternative phenotypic development is a reliable indicator of environmental conditions at the time of reproduction; 6) and the cost of plasticity is low

(reviewed in Scheiner 1993). To the extent that any of these conditions are violated, plasticity becomes less likely. If environmental cues during development are not good predictors of the environment experienced during reproduction, or if the cost of plasticity is high, then evolution of a single generalist phenotype is much more likely. If different environments are not equally frequent or the strength of selection is unequal in different environments, then selection favors specialist genotypes that are each adapted to a different environment.

Whitlock (1996) demonstrated yet another limitation on the evolution of adaptive plasticity. Using a gametic model that incorporated both mutation and genetic drift, he showed that phenotypically plastic populations evolve more slowly and accumulate more deleterious mutations than do more specialist populations. Further, in a spatially complex environment, where the fitnesses of different phenotypes across different environments are imperfectly correlated, natural selection will favor the evolution of habitat fidelity and mating within environments. Increased assortative mating and habitat fidelity leads to the evolution of specialization, rather than to plasticity.

4. Tests of the Adaptive Plasticity Hypothesis

During the last decade, many experimental biologists set out to test the hypothesis that phenotypic plasticity is adaptive. Several different types of plasticity were tested in many different groups of organisms. In general, these biologists concentrated on plasticity that seemed intuitively likely to be adaptive.

Two main criteria have been used for demonstrating that plasticity is adaptive. One is that adaptive plasticity should produce environment-specific phenotypes that have higher fitness in that environment than do alternative phenotypes (Dudley and Schmitt 1996, Kingsolver and Huey 1998). Applying this criterion requires that one can induce different phenotypes that are hypothesized to be adaptive in different environments. In addition, phenotypes should be statistically independent of genotype. The next step is to place replicates of these alternate phenotypes into the different environments, preferably under natural conditions. Finally, one needs measures of the relative fitness of the different phenotypes in the different environments. These measures are generated by counting offspring or seeds, or by measuring survival or mating success (Dudley & Schmitt, 1996; Kingsolver & Huey, 1998; Schmitt, McCormac, & Smith, 1995). The adaptive hypothesis is supported if the phenotype with the highest fitness in each environment is that which is normally produced in that environment.

The second method used to test the adaptive hypothesis is even more laborious than the first. This method assesses whether genotypes differ in

plasticity, and whether that difference has adaptive significance. Replicates of different genotypes, or sets of individuals of known relatedness (such as full or half-sibs) are needed. Individuals of each genotype (or family) are raised in a range of environments. [Note: if a labile trait is being investigated, then exposing the same individual to different environments provides the replication needed, so sets of relatives are not needed.] One then measures the relationship between the plasticity of a genotype (family), and the average fitness of that genotype (family) across environments (Scheiner, 1993). For example, level of plasticity could be estimated as the slope of a regression of phenotype on environment if there are many different environments, or it could be the amount (and direction) of change in the phenotype across two or three discrete environments (Figure 3). A positive relationship between the plasticity and average fitness of genotypes then supports the hypothesis that plasticity is adaptive. Of course, the relationship might not be linear. An intermediate optimum for plasticity might exist, in which case one expects a curvilinear relationship with the highest mean fitness achieved by families with intermediate levels of plasticity.

Using these methods, mixed results have been obtained. Even with the inherent bias of studying plasticity that seems likely to be adaptive, both positive and negative results have been reported (c.f., Dudley & Schmitt, 1996; Kingsolver & Huey, 1998; Scheiner, 1993; Winn, 1999). Dudley and Schmidt (1996) performed an experiment supporting adaptive plasticity using the first method described above. They tested the hypothesis that having elongated stems under crowded conditions is an adaptive form of plasticity in the jewelweed, *Impatiens capensis*. They used different light treatments to produce plants with either elongated or nonelongated stems, and then transplanted both types into both low and high-density populations. Their results supported the adaptive hypothesis: elongated plants were more fit at high density, while nonelongated plants were more fit at low density.

In contrast, Winn (1999) used the second method to examine the hypothesis that seasonal plasticity for leaf size and shape was an adaptive form of plasticity in the wild mint, *Dicerandra linearifolia*. Leaf shape is a labile trait, so that individuals express both a “winter” and a “summer” phenotype (large and thick in summer and small and thin in winter). The adaptive hypothesis predicts there should be a relationship between plasticity and fitness. However, Winn found no such relationship. She concluded, “Seasonal variation in leaf traits may persist simply because there is no selection against individuals in which these traits vary. My results underscore the importance of definitive tests of the hypothesis of adaptation to distinguish adaptive plasticity from neutral or nonadaptive phenotypic plasticity.”

5. Can the Adaptive Plasticity Hypothesis Be Tested in Humans?

Given the nature of the experiments used in non-human organisms, can we hope to test the adaptive plasticity hypothesis in humans? One is tempted to answer with a resounding 'No!' Clearly, the above experiments employ both genetic and environmental manipulation, neither of which can be used in studies of humans. However, that answer leaves students of human variation with two options: 1) to conclude that the question is unanswerable, and therefore not amenable to scientific inquiry, or 2) to simply assume that observed plasticity is either adaptive or not, based on guesswork or intuition. Since we view both these options as untenable, we hope to describe a few other options that might allow some objective testing of the hypothesis.

Although direct tests as outlined above will be very difficult for fixed traits, it may be possible to apply them to labile traits. For labile traits, a single individual expresses different phenotypes in response to different environments. For example, women sometimes cease menstrual cycling in response to intense exercise, insufficient food intake, or stress (c.f., Bronson, 1995; Bronson & Manning, 1991; Chen & Brzyski, 1999; Rivier & Rivest, 1991). For this type of trait, individual plasticity could be measured in sets of twins, perhaps as variation in cycling or hormone levels in response to different exercise regimes. This method tells us if genotypes differ in their level of plasticity. In order to show whether plasticity (range in phenotypic response) has adaptive significance, some measure of reproductive success would be needed. Clearly, this type of study would be logistically difficult and could take many years, but it is theoretically possible.

A more amenable type of trait would be one that responds to short-term stimuli in a laboratory or clinical setting. For example, plasticity for reproductive decision-making or sexual arousability could be studied using a social scenario approach. Reproduction-related physiological responses to short-duration psychological stressors could also be investigated. In fact, heritability of plasticity of this kind has been measured (c.f., Kirschbaum, Wüst, Faig, & Hellhammer, 1992, who conducted a small twin study on hormonal response to the stress induced by speaking in public and by mathematical tests). However, no measure of fitness was made, so adaptive significance of the plasticity could not be determined. Small sample sizes will be a limitation in this type of study, but this is a logistical, and not a conceptual limitation.

For fixed traits, the direct tests of the previous section will probably be impossible because of the kinds of families needed: sets of relatives matched for age and for any environmental condition not under investigation, where each set of relatives has been exposed to the same range of the particular environmental variable being studied, and where average

fitness data are available for each family. Such a study would not be feasible in humans. However, a few authors have outlined other predictions of the adaptive plasticity hypothesis that are potentially testable in humans. Kingsolver and Huey (1998) describe a comprehensive program for analyzing and testing phenotypic plasticity. Applying all aspects of their program will only be possible in organisms that are amenable to laboratory and field manipulations. Nonetheless, some of the methods they recommend should be applicable to data from humans. For example, two of the approaches they describe (description of the environmental conditions that induce plastic responses, and investigating the physiological mechanisms underlying plastic responses) are both feasible approaches for studies of human traits. While these methods are not useful for testing the adaptive hypothesis *per se*, they will provide information on the conditions that co-occur with plasticity and on the underlying biological basis of human plasticity.

Other approaches described by Kingsolver and Huey (1998) are more directly related to tests of the adaptive hypothesis. They recommended the direct tests of the previous section, but they also proposed two indirect tests that could be used on data from humans. One of these is to assess the predictive ability of environmental cues in order to test the assumption that environmental conditions during development are reliable indicators of environments that will be experienced during reproduction. Two recent theoretical studies have emphasized that cue reliability is critical for the evolution of plastic phenotypes. If cues are unreliable, local genetic differentiation evolves instead of phenotypic plasticity (de Jong, 1999; Tufto, 2000).

As an illustration of this method, Kingsolver and Huey (1998) tested whether temperature during larval development was a good predictor of temperatures experienced by adult butterflies. These butterflies are plastic for melanic coloration on their wings, and the adaptive hypothesis was that dark colors develop during cold periods and light colors develop during warm periods to aid the insects in thermoregulation. The authors measured the correlation between the average high temperature (T_{\max}) in April, when the insects are in the larval stage (when adult coloration is determined), and T_{\max} in July, when the insects are adults and are reproducing. The thermal environment during development was a poor indicator of the thermal environment during adulthood. Thus, in this case, the prediction based on the adaptive hypothesis was not supported.

A similar approach could be taken to test hypotheses of adaptive plasticity in humans. In the previous volume in this series, several papers dealt with a specific kind of plasticity in humans: the effects of family environment on age at menarche and pubertal development in girls (Belsky,

2000; Doughty & Rodgers, 2000; Rowe, 2000). These papers were motivated by an earlier prediction (Belsky, Steinberg, & Draper, 1991) that family instability serves as a cue that the environment is unstable, and should therefore lead to early menarche and early reproduction in females. Doughty and Rodgers (2000) and Rowe (2000) used comparisons between genetic and environmental variance components to test some predictions of Belsky's model. We suggest that a further test could be conducted by measuring the predictive value of pre-pubertal family stability for the social and economic conditions of the same women during their childbearing years. Such a study would test one of the assumptions underlying models of the evolution of plasticity, but not all of them. Even if environmental cues during development are reliable, plasticity will not evolve if there is a substantial cost of plasticity. To our knowledge, there have been no attempts to measure the cost of the plasticity described in Belsky's model.

Another of Kingsolver and Huey's indirect tests that could potentially be applied to human studies is to determine whether current selection favors plasticity. Measurement of current selection is a commonly used technique in evolutionary biology. For a simple trait such as body size, one measures the trait in a number of individuals within a population, and also measures some aspect of Darwinian fitness, such as lifetime reproductive success or survival. Regression analysis then gives an estimate of the direction and form of natural selection that is currently acting on the trait. For example, there could be a positive linear relationship indicating that current selection favors large body size; or there could be a nonlinear relationship indicating an intermediate optimum. These techniques have been extended to studies of selection on many traits at once, and there is currently a large literature on multivariate selection analysis (see Janzen & Stern, 1998, for a recent review of these techniques).

Measuring selection on plasticity is more complex because each trait must be measured in multiple environments, so that the degree of plasticity can be determined and then related to fitness. We believe the technique can be applied to humans, but only to labile traits. To measure selection on a labile trait, one needs to expose each individual to multiple environments, measure the individual's response to each environment, and then determine the association between the range of environmental responses (plasticity) and some measure of fitness. For example, one might measure plasticity in body weight by asking individuals to undertake at least two different regimes of diet and exercise. The change in body weight across the two different regimes provides a measure of plasticity. One then relates these measures of plasticity to an index of fitness to determine whether and how selection is currently operating on plasticity itself. Of more relevance to readers of this volume, we can imagine using this approach to ask whether plasticity in fertility-related behaviors, physiological processes, or psychological states is

currently under selection. One caveat is in order here. If plasticity in the trait of interest is correlated with some other trait (say height in the current example), then selection operating on that trait (height) will lead to indirect selection on plasticity. Such indirect selection will give the appearance that plasticity is adaptive, even if it is not under direct selection. The methods of multivariate selection analysis could be used to decompose selection into direct and indirect components (see Jansen and Stern 1998).

6. Conclusions

Phenotypic plasticity is one of several sources of human behavioral and reproductive variation. It can arise as an adaptation to variable environments, but plasticity is not always adaptive. Evolutionary biologists have recently defined criteria that can be used to distinguish adaptive and non-adaptive plasticity. Some of these criteria are potentially applicable to empirical data available on humans. Tests of this kind could help alleviate a criticism that has been directed toward many studies of human variation—that researchers in the field sometimes engage in untestable adaptationist story telling (Grantham & Nichols, 1999).

Unfortunately, the most direct techniques used to test for adaptive plasticity are not generally applicable to human studies. Several indirect techniques could be applied, but these tests may not be completely satisfying. There is, however, one class of traits for which some direct tests may be applicable. These are traits that are labile in individuals, and so can assume different states during the lifetime of a single person. Methods of estimating genetic variances, heritability, and phenotypic plasticity could potentially be used for these labile traits, since different character states would be expressed in identical genotypes. Even measures of the relative fitness of genotypes characterized by different degrees of plasticity might be possible, which would allow a direct assessment of the adaptiveness of plasticity.

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