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Phenotypic Plasticity in Response to Ecological Forces : A Review

***Imam, T. S.[1], Abubakar, K. I.[1], Rabiu, S.[1] , Rabiu, A. T.[2]**
[1]Biological Sciences Department, Bayero University, Kano, PMB 3011, Kano, Nigeria
[2]Department of Life Sciences, School of Basic Science and Research, Sharda University, Greater Noida, Uttar Pradesh 201306, India.
*Corresponding Author's E-mail Address: tsimam.bio@buk.edu.ng

Abstract : Phenotypic plasticity, the capacity of a single genotype to exhibit variable phenotypes in different environments, is common in insects and is often highly adaptive. Here we review terminology, conceptual issues, including variance partitioning, reaction norms, physiological mechanisms, adaptive value, and evolution. Most plasticity is physiological, but can manifest as changes in biochemistry, physiology, morphology, behavior, or life history. Virtually any abiotic or biotic factor can serve to induce plasticity, and resulting changes vary from harmful susceptibilities to highly integrated and adaptive alternative phenotypes. Understanding plasticity requires knowing the environment, physiological mechanisms, and fitness outcomes. Plasticity is thought to be evolutionarily favored under specific conditions, yet many theoretical predictions about benefits, costs, and selection on plasticity remain untested. The ecological consequences of plasticity range from simple environmental susceptibilities to mediating interspecific interactions, and extend to structuring of ecological communities, often through indirect effects. Phenotypic plasticity, through its ecological effects, can facilitate evolutionary change and speciation. Plasticity is important because it is an encompassing model to understand life on earth, it can increase fitness, generate novelity, and facilitate evolution, it structures ecological communities, and it has numerous practical applications.

Keywords: adaptive value, environment, evolution, genotype, phenotypic plasticity

Introduction: Evolution of Phenotypic Plasticity

Phenotypic plasticity also refers to Genetic plasticity is the ability of an organism to change its phenotype in response to changes in the environment (Prince,2003). Also, it can be broadly defined as the ability of one genotype to produce more than one phenotype when exposed to different environments, as the modification of developmental events by the environment, or as the ability of an individual organism to alter its phenotype in response to changes in environmental conditions (Kelly *et al*., 2012; Cridge *et al*., 2017).

Plasticity is usually thought to be an evolutionary adaptation to environmental variation that is reasonably predictable and occurs within the lifespan of an individual organism, as it allows individuals to 'fit' their phenotype to different environments (West-Eberhard,2005). If the optimal phenotype in a given environment changes with environmental conditions, then the ability of individuals to express different traits should be advantageous and thus selected for(Hurd and Lane *et al*., 1998). Hence, phenotypic plasticity can evolve if Darwinian fitness is increased by changing phenotype (Price, 2003).

Given the profound ecological importance of temperature and its predictable variability over large spatial and temporal scales, adaptation to thermal variation has been hypothesized to be a key mechanism dictating the capacity of organisms for phenotypic plasticity (Langerhans *et al.*, 2002). The magnitude of thermal variation is thought to be directly proportional to plastic capacity, such that species that have evolved in the warm, constant climate of the tropics have a lower capacity for plasticity compared to those living

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in variable temperate habitats. This idea is termed the "climatic variability hypothesis", which has been supported by several studies of plastic capacity across latitude in both plants and animals (Janzen *et al.*, 1967).

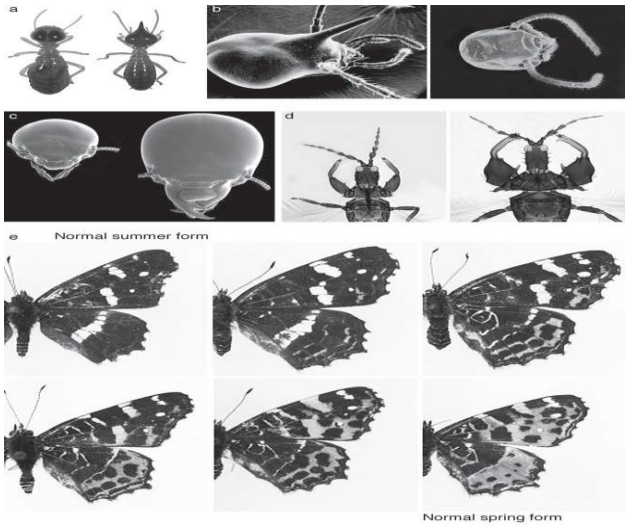

Fig. 1 (a) Worker and nasute-soldier of *Nasutitermes takasagoensis* termites from Japan (Hojo *et al.*, 2004). Photo by Masaru Hojo. (b) Head of soldier (left) and minor worker (right) of *Hospitalitermes medioflavus* termites (Miura & Matsumoto 1995; Miura *et al.*, 1998) (c) Head of worker (left) and soldier (right) of *Hodotermopsis sjostedti* termite (Miura, 2005). (d) Large and small males of *Ecacanthothrips tibialis* thrips (Mound, 2005). (e) Only two discrete forms of the nymphalid butterfly *Araschnia levana* are found in nature: the summer form (top left) and the spring form (bottom right). However, in the laboratory, intermediate phenotypes can be produced by subjecting individuals to intermediate environments or timed ecdysone injections, documenting that a continuous reaction norm lies at the base of this seasonal, diphenic polyphenism (Nijhout, 2003).

Most traits are both genetic and environmentally influenced (Roundtree and Nijhout, 1994). Example is the pigment melanin, which is the end product of well-known enzyme chains. The sequence instructions for these enzymes are coded by DNA and are heritable. But in many animals, melanin production and deposition are also environmentally influenced (True, 2003).

Characteristics of Phenotypic Plasticity

Scientists agree that phenotypic plasticity concerns environmentally induced changes to phenotypes; also most consider discrete morphological polytheisms as good examples of this concept.

Below, we discuss some of the complexities and controversies surrounding phenotypic plasticity (Kristan, 2003):

❖ Virtually any trait can show phenotypic plasticity. The concept was first applied to morphological traits (Woltereck and Schlichting, 1909). However, it is clear that organisms can also alter biochemistry, physiology, behavior, and life history in response to the environment (Agrawal, 2001).

❖ Phenotypic plasticities range from graded, continuous responses (phenotypic modulation), to discrete switches in phenotype with no intermediate forms (developmental conversion or threshold traits) (Woltereck and Schlichting, 1909).

❖ Phenotypic plasticity can be initiated by either environmental stimuli or cues (Nijhout, 2003).

❖ Some plastic responses are highly specific in either requisite stimuli or response. For example, some plants possess receptor proteins that detect only their most common natural enemy (Zhao *et al*., 2005)

❖ Some plastic responses are anticipatory, in that individuals initiate phenotypic change before the appearance of a harmful (or beneficial) environmental factor. Examples include diapausesinduction before the onset of winter, and detoxification induction in caterpillars. Some plants exhibit.

Adaptive Phenotypic Plasticity

Many examples of phenotypic plasticity are clearly adaptive, such as some immune responses, antipredator defenses, acclimatizations, diapause, life-history shifts, dispersals, etc. (WestEberhard, 2003).

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A specific altered trait may be highly beneficial in one context, but overwhelmingly detrimental in another. For example, plastic production of large spines or heavy armor in a prey in response to the presence of predators may aid antipredator defense, but reduce feeding, migration, mating, fecundity, etc. (Roff, 1996).
Phenotype is relative to a specific time and place and presence or absence of interacting individuals (Nykänen and Koricheva, 2004). Forsman (2015) opines that an increased understanding of the roles of plasticity in these contexts requires a 'whole organism' (rather than 'single trait') approach, taking into consideration that organisms are integrated complex phenotypes.To understand adaptive plasticity, one must consider benefits and costs of plastic phenotypes in several environments. Genetic and environmental correlations are themselves plastic to the environment (Roff, 1996).
Finally, "adaptive" implies past selection, but a population's history is often opaque (Doughty and Reznick, 2004). Hence, it is difficult to know the adaptive value of phenotypic plasticity, and, for that reason, "adaptiveness" cannot Horn polyphenism in *Onthophagus nigriventris*. Courtesy of exposed to fish-predator chemicals, right individual was not. The long spines reduce predation (Agrawal, 2001; Van Klunen and Fischer, 2005).

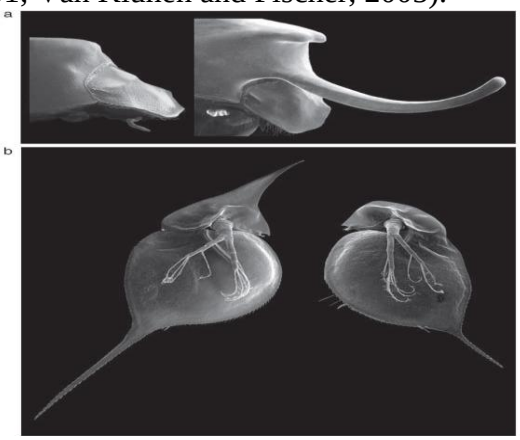

Fig. 2 (a) Horn polyphenism in *Onthophagus nigriventris*. Courtesy of Emlen,D. (Emlen *et al.,* 2006) (b) Predator-induced plasticity in Daphnia lumholtzi. Left individual was exposed to fish-predator chemicals, right individual was not. The long spines reduce predation (Agrawal, 2001).

Physiological Homeostasis in Phenotypic Plasticity

Rapid, short-term physiological homeostasis such as regulation of blood pH and osmolarity represents phenotypic plasticity. Somewhat counter intuitively, homeostasis is derived from monitoring internal and external conditions, and manipulating physiology, i.e., keeping some aspect of the phenotype constant by altering enzyme activity or other physiological or behavioral parameters, in response to a varying environment. Some traditional homeostatic mechanisms and phenotypic plasticity share similar physiological mechanisms. (Emlen *et al.,* 2006). Physiological changes, be they rapid and short-term, or delayed and long-term, represent altered phenotypes to altered environments, and, as such, have the potent to produce the same evolutionary effect – increased fitness for those genotypes that can show the beneficial plasticity.

Phenotypic Plasticity and Canalization

Phenotypic plasticity is often considered the opposite of canalization. However, reaction norms can be canalized (Scheiner, 1993). In addition, to hold one trait constant in the face of a changing environment often requires change (plasticity) in another trait. For example, some insect's exhibit canalized egg size, and when confronted with poor nutrition or end of season, such insects maintain egg size, but express plasticity in clutch size or oocyte development rates. In other species, clutch size or oocyte development may be canalized (Stearns *et al.,* 1992). Furthermore, what at first may appear to be a non-adaptive passive response? (For example, lowered clutch size under poor nutrition), may in fact be an evolved plastic response to maintain egg size, oocyte development rate, or female survival. As such, canalizations in physiology, life history, and development are often accomplished via phenotypic plasticity (Stearns *et al.,* 1992; Padilla and Savedo, 2013).

Contribution of Phenotypic Plasticity to Speciation

There are different hypothetical pathways for this to occur but one possible pathway would

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be the following (Jablonka *et al*., 1998; West-Eberhard, 2003):

1) *Trait Origin via Phenotypic Plasticity* - the production of an environment induced alteration of the phenotype. This could be passive, and be detrimental, neutral, or beneficial with regard to fitness.

2) *Phenotypic Accommodation*, whereby the individual accommodates the changed phenotype by adaptively altering additional phenotypic traits, such as physiology, behavior, or morphology (Jablonka *et al.*, 1998; West-Eberhard, 2003).

3) Genetic Accommodation — assuming population genetic variation in most traits, the recurrence of this particular environmental induction (Dark color) in numerous individuals and generations would allow this novel phenotype to be tested repeatedly in the new environment and among a vast assortment of genetic variants (West-Eberhard, 2003). Examples of genetic accommodation in this hypothetical case might be better nocturnal eyesight or longer antennae for non-visual sensing (Piggliucci, 2001).

4) *Adjustment of the capacity and shape of the reaction norm via the Baldwin effect* Here, natural selection alters the frequency of genes and gene combinations that influence the expression of the plasticity – genes that do not produce the optimal plastic response are eliminated (Agrawal, 2001;Mondor *et al.*, 2005).

5) *Genetic Assimilation* — an evolutionary mechanism by which environmentally induced (facultative) traits become genetically fixed (Obligatory) (Agrawal, 2001; Mondor *et al.*, 2005).

6)

Plasticity Change in the World

Unprecedented rates of climate change are predicted to occur over the next 100 years as a result of human activity. Phenotypic plasticity is a key mechanism with which organisms can cope with a changing climate, as it allows individuals to respond to change within their lifetime (Whiteman, 1994; Mondor *et al.*, 2005; Padilla and Savedo, 2013).

The North American Red Squirrel *Tamiasciurushudsonicus* has experienced an increase in average temperature over this last decade of almost 2°C. This increase in temperature has caused an increase in abundance of white spruce cones, the main food source for winter and spring reproduction. In response, the mean lifetime parturition date of this species has advanced by 18 days (Poinar and Yanoviak, 2008).

Phenotypic Plasticity and the Epigenetics of Human Disease and Ageing

The revolutionof phenotypic plasticity started when scientists realised that a human physical performance could be improved through physical training. Exercised muscles responded to the stimulation, and remodelled to improve performance. Scientists have characterised numerous physiological aspects of this phenomenon across a range of tissues, and with the advent of modern molecular tools, it has proved possible to uncover some of the mechanisms that underpin the phenomenon (Phillips, 2006).

Epigenetic lesions in human disease affect a cell's ability to change its phenotype as described by Feinberg, (2007):

a) There is impediment of normal development in the case monogenic disorders such as Rett syndrome, a defect in the normal epigenetic apparatus.

There is failure to completely silence genes appropriately during development because DNA methylation (brown circles on the DNA) proceeds normally but is not recognized owing to the absence of the MeCP2-methylationinteracting protein (large red oval).

b) there is involvement of many epigenetic lesions in cancer that could affect a pluripotent programme in tissue-specific stem cells, which may lead to an incorrect distribution of differentiated cell lineages (indicated by the bivalent euchromatin and heterochromatin proteins shown in the upper left panel) as well as normal tissue-specific silencing of gene A and activation of gene B after differentiation.

Examples of epigenetic lesions changes in cancer involve the following: changes in chromatin proteins in stem cells which is as a result of increased expression of MLL1 in leukaemia leading to aberrant HOX expression in differentiated cancer lineages and increased expression of EZH2 which leads to aberrant

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silencing of these genes in differentiated cancer lineages.

c) Ageing consists of a loss of the normal plasticity of response to internal and external environmental signals. Thus the epigenome could have an important role in ageing if the aged epigenome is less responsive to such signals.

For example, if a gene shows an increased H3K9-methylation or DNA methylation, it might be relatively refractory to environmentally induced activation more than if the gene had not undergone age dependent epigenetic change.

Variance Partitioning

Phenotypic plasticity represents measureable variation, and as such can often be expressed and analyzed by Analysis of Variance, ANOVA (Pigliucci, 2001). A statistical measure of variation is variance, which quantifies the deviation of values around a mean (Pigliucci, 2001). The variance of a phenotypic trait can be partitioned as follows:

$$VP = VG + VE + VGE + V \text{ error}$$

Where:
V*P* = Total phenotypic variance for a trait
V*G* = Genetic variance (proportion of phenotypic variation attributable to genes)
V*E* = Environmental variance (proportion of variation caused by the environment)
V*G* x *E* = Genotype environment interaction (Genetic variation for phenotypic plasticity)
error = Unexplained variance, including developmental noise, measurement error, etc.
ANOVA can partition phenotypic variation into the above components.
However, these terms, especially the expression of genetic variance, are often further divided into component parts (Debat and David *et al.,* 2001). Thus, experimental designs with some form of genetic structure (i.e., using clones, half-sibling families, multiple populations, etc.) and environmental treatments are extremely powerful for studying phenotypic plasticity. Nonetheless, genetic structure is not required for the study of plasticity (Debat and David *et al.,* 2001). Here, *VG* and X *VE* are unknown, but *VP* can still be partitioned into what is explained by *VE* (i.e., phenotypic plasticity) and all other sources of phenotypic variation. *VG X E* is an important term because it shows that different genotypes express different plastic responses. Such genetic variance in plasticity allows plasticity to evolve (Schlichting and Pigliucci 1998).

However, it is clear that organisms can also alter biochemistry, physiology, behavior, and life history in response to the environment (Agrawal, 2007) (Agrawal, 2007).

Conclusion

Phenotypic plasticity is widespread in nature, and often involves ecologically relevant behavioral, physiological, morphological and life-historical traits. As a result, plasticity alters numerous interactions between organisms and their abiotic and biotic environments. Here, we highlight an expanding body of work that examines how plasticity can affect all levels of ecological organization through effects on demographic parameters, direct and indirect species interactions, such as competition, predation, and coexistence, and ultimately carbon and nutrient cycles.

Recommendation

1. Phenotypic plasticity should be tested using techniques of molecular biology.

2. Industries should dispose their waste properly to make sure that the trash does not harm living organism.

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