Chapter

Phenological Plasticity of Wild and Cultivated Plants

Amber L. Hauvermale and Marwa N.M.E. Sanad

Abstract

The future survival of wild and cultivated plant species will depend on their ability to adapt to environmental changes caused by climate change. Phenological plasticity describes physiological, developmental, cellular, and epigenetic mechanisms that contribute to genetic diversity and adaptability. Many studies evaluating plasticity using trees, cereals (barley, wheat, and rice), pulses, and weeds have discovered that plasticity mechanisms differ between wild and cultivated plant populations. Major findings indicated by these studies are: (1) invasiveness and adaptability in wild and/or “weedy” plant species may be controlled by specific plasticity genes, (2) adaptability is directly connected to adaptive responses and fitness, and (3) domestication and cultivation have altered plasticity mechanisms. Therefore, selective breeding requires a holistic understanding of plant plasticity. Breeding strategies should consider differences in plasticity mechanisms between wild and cultivated plant populations to reintroduce genetic diversity of plasticity from wild relatives.

Keywords: cellular plasticity, climate change, developmental plasticity, drought response, epigenetic plasticity, germination, hormone signaling, physiological plasticity, phenological plasticity, seed dormancy, selective breeding

1. Introduction

Global climate changes undoubtedly impact adaptability in plants by altering mechanisms of physiological plasticity [1]. Modifications in mechanism occurring at the morphological, anatomical, and physiological level are regulated by the capacity of a plant to adjust to abiotic and biotic stresses [1–4]. The resulting survival response and survival capacity may vary depending on plant life stages [1–4]. Plasticity mechanisms discovered in plants are like those described in animals and humans, illustrating the conserved connection between environmental selection and adaptive response [2, 3, 5–11]. Research into the connection between environmental stress, environmental selection, and plant plasticity has also identified both general and unique plasticity mechanisms that differ between wild, i.e., non-cultivated, and cultivated plant species [1, 12–15]. However, a review analyzing the contribution of key traits responsible for varied plasticity mechanisms in wild and cultivated plants has not occurred. Thus, the range of plasticity occurring in wild plants will be compared with plasticity mechanisms in cultivated plants. Similarities and differences in plasticity responses will be highlighted between the two groups, with a specific focus on climate imposed global abiotic stresses like drought [14].
All plants have evolved unique life cycle characteristics that enhance survival and adaptation to diverse short and long-term climatic events that limit resources. Phenotypic responses occur at every stage of plant development, and influence overall plasticity from one generation to the next. Understanding and tracking phenotypic plasticity of wild plants in cultivated plants first requires defining biological reaction norms and their alternatives to clearly illustrate the differences between biological plasticity and non-plastic responses. Examples of phenotypic responses include: (1) rapid seedling growth (2) a short vegetative phase, (3) deep root systems, (4) high seed output, (5) discontinuous or extensive seed dormancy, (6) efficient cellular defense machinery, and (7) environmental plasticity. Although all plants exhibit phenotypic responses, the level of response is largely influenced by the degree of cultivation. Several species of trees and weeds are exceptional models for defining and tracking the range of both short and long-term heritable characteristics of wild plasticity [1, 4, 12, 16–21]. Drought response studies in agronomically important, and highly cultivated crops like wheat, add perspective about the contributions of selective breeding programs; how increased cultivation results in gains or losses in adaptive responses and plasticity [7]. Transitional plant models, such as Chenopodium quinoa (quinoa) and Hordeum vulgare (barley) will be used to illustrate the evolutionary path from wild plasticity to cultivated plasticity.

2. Environmental changes impact phenotypic plasticity

Climatic events trigger heterogeneous responses in plants. Plant responses occurring from biotic or abiotic factors drive two distinct adaptation mechanisms, natural selection and phenotypic plasticity. Both mechanisms reveal the full genetic capacity of plants [22, 23]. The genetic makeup or genotype of each plant species determines how a plant will react in new environments [24]. Accumulated exposure to novel environmental stresses over many generations may increase selection toward the frequency of favorable alleles versus a reduction of unfavorable alleles, and results in less genetic diversity [22]. Otherwise, in natural selection, any change in plant phenotype is defined as phenotypic plasticity [25]. Changes in phenotypic plasticity impact individual fitness without changing genetic diversity [22, 26]. Sometimes a novel genotypic response does not deviate from a normal range of reactions, i.e., the reaction norm, and sometimes it does [27]. Thus, plants have a wide array of genotypic responses that impact phenotype. Non-cultivated plant species like trees acquired wild plasticity through the combination of both the long-term accumulation of genetic changes and the conservation of favorable survival strategies through time [24]. Adaptive responses result in phenotypic plasticity [22, 26]. Adaptive responses also maximize phenotypic fitness, or the ability to respond and survive in changing environments [27]. Breeding programs have accelerated the adaptive process to abiotic stresses, like drought, in domesticated plant species by selecting for tolerance to drought or increased resource-use efficiency [28]. This approach has allowed breeders to select for favorable plant responses based on flexibility to varied environmental changes. A broad understanding of wild plasticity in non-domesticated plant species will enhance and extend our current understanding of the range of plasticity mechanisms in cultivated plants [2, 3, 5–11].

3. The plasticity spectrum

All terrestrial plants are stationary and adjust phenotypic responses to survive in fluctuating environments [22, 26]. A wide spectrum of adaptive variation occurs
with a specific phenotypic response, and which is defined as phenotypic plasticity [14]. Three recognizable outcomes associated with a phenotypic response, as illustrated in Figure 1, are: (1) a neutral response, (2) an adaptive response, or (3) a maladaptive response [13, 22, 27]. Each panel illustrates the relationship between a phenotypic response and a change in environment. Red, green, or blue colored lines represent different genotypes or individuals [13, 22, 27].

A neutral response occurs when there is no observable change in plant fitness or plasticity after exposure to novel environmental stress (Figure 1a). Canalisation and developmental stability are components of neutral responses that create some confusion in understanding and mapping phenotypes [29]. Canalisation describes the occurrence of a constant phenotype in a given population that is not influenced by environmental or genetic regulation [29]. Developmental stability describes the degree to which organisms withstand environmental changes or genetic perturbations during development [29]. Canalisation measures gene rigidity or the resistance of genes to altered function during environmental changes [29, 30]. Canalisation is a useful measure of genetic robustness and is more frequently described than adaptive plasticity in plants [29, 30].

Adaptive responses occur in new environments and may or may not occur as a direct result of genetic variation [29]. Adaptive responses result in beneficial changes that maximize phenotypic fitness (Figure 1b–d) [27]. Not all phenotypic changes occur because of beneficial adaptive responses [27, 29, 30]. Individuals within a population may experience random passive phenotypic changes that are limited to specific phenotypic traits or that act more broadly impacting adaptive performance at all stages of plant development [29, 30]. Plasticity may be controlled by a single gene or many genes [31, 32]. The plasticity threshold of a plant is a function of individual, pleiotropic, and collective responses within a population. This mosaic of responses influences genotypic selection [33, 34].

![Figure 1. Recognition of the different reaction norms. The three major responses; neutral, adaptive, and maladaptive, which occur within the plasticity spectrum [13, 22, 27, 34, 35]. A neutral response (a). An adaptive response without genetic variation (b), or with genetic variation (c, d). A non-plastic or maladaptive response (e). All three phenotypic responses occurring simultaneously (f).](image-url)
Not all adaptive strategies are beneficial for plants and often result in decreased fitness or yield [35]. A maladaptive response describes a phenomenon which reflects the absence of plasticity (Figure 1e) [34]. Maladaptive responses are not easy to distinguish from neutral responses because the average response of the population may mask any decline in response by individuals within the population over a long period of time [35]. Maladaptive responses are often misinterpreted as adaptive responses and difficult to study genetically [34, 35].

All phenotypic responses, neutral, positive, and negative, may occur simultaneously within an individual or across a population (Figure 1f) [13]. Changes in plasticity may be measured by examining the relationship between a specific genotype (G) in a specific environment (E) [13]. A genotype-by-environment (GXE) study tracks genetic plasticity and is a powerful tool for targeted genotypic selection [13, 33, 34].

4. Characteristics of wild plasticity: examples in trees and weeds

Phenotypic plasticity, especially within wild plant populations, is a mechanism that enhances plant invasion and survival [12]. The invasiveness of a plant species is influenced by many phenotypic characteristics and responses [12]. The three major phenotypic characteristics that impact plasticity in wild plant populations are plant development, plant morphology, and plant physiology (Figure 2) [36]. Phenotypic responses associated with each characteristic occur at every stage of plant development, influencing the overall plasticity from generation to generation (Figure 2). Common phenotypic responses known to be associated with plant development, plant morphology, and plant physiology include: (1) rapid seedling growth allowing maximum capture of light, water, and nutrients [37–41], (2) a short vegetative phase allowing life cycle completion in various growing seasons and conditions [42–47], (3) deep root systems allowing plants to survive through drought conditions [47, 48], (4) high seed output ensuring spatial and temporal dispersal, (5) discontinuous or extensive seed dormancy ensuring germination only in favorable conditions [49–51], (6) efficient
cellular machinery for scavenging reactive oxygen species (ROS) [52, 53], and (7) environmental plasticity, or the ability to respond to changing biotic or abiotic environmental factors [1, 13].

Phenotypic plasticity was first described for non-cultivated plants species including trees and weeds [1, 4, 12, 17–20, 54]. Trees are excellent models for studying phenotypic plasticity due to their longevity [12]. Trees have developed a diverse set of plasticity mechanisms that are specific for both short and long development programs occurring in different developmental tissues at the same time [12]. Simultaneous root and leaf canopy development are an example of parallel programming [12, 54]. Phenotypic plasticity in trees occurs through a diverse collection of physiological, anatomical, and morphological responses [12, 54]. Many studies exploring global warming have investigated the possibility of using physiological or morphological indicators of beneficial adaptive responses as predictors of species survival [1]. Adaptive mechanisms in trees, as well as other plants, are important for mitigating the stress that is associated with fluctuations in native environments or, after new colonization, for rapid adaptation to novel environments [54–57].

Studies investigating drought stress in trees have shown that by reducing the leaf canopy and increasing root proliferation, trees become more drought tolerant because both phenotypic responses limit water loss [58]. The occurrence of phenotypic responses occurring in parallel suggests that there may be a coordinated regulation of these traits [59, 60]. Other traits indicative of drought responses and plasticity in trees include leaf area, leaf dry mass, leaf mass per area (LMA), leaf tissue density, net photosynthesis, stomatal conductance, leaf respiration, water use efficiency, leaf water potential at midday, total chlorophyll content, relative water content, gross photosynthesis, leaf transpiration, and the ratio between leaf respiration and net photosynthesis [58]. Drought avoidance may also be viewed as a strategy for drought tolerance by altering the timing of growth and reproduction [14]. By maximizing the adaptive response of traits related to drought response, the overall fitness of an existing population of trees has the potential to adapt to a new environment [58]. However, if a given climatic event exceeds the limit of adaptive capacity, the same population of trees may also be replaced by a new, more adapted species [14].

Plasticity in weeds, as with trees, is governed by adaptive responses that impact physiology, morphology, and anatomy [36]. However, unlike trees, many weed species have relatively short life spans and must make rapid and frequent adjustments to environmental changes to ensure survival [16]. In addition to the wild characteristics for plasticity listed above, other characteristics in weeds that demonstrate enhanced phenotypic plasticity include: discontinuous or extensive seed dormancy ensuring germination only in favorable conditions, indeterminant or simultaneous flowering and vegetative growth, self-compatibility allowing genetic divergence from previous generations without requiring special pollinators to ensure seed viability, long-distance seed dispersal by air or water; competition with crop plants resulting in reduced crop yield, sexual and asexual reproduction strategies; and allelopathy, or the ability to produce chemicals that retard or kill other plants (Figure 2) [16, 36, 61]. Adaptive responses in weeds occur throughout development [16, 36]. Sometimes adaptive responses are more apparent in plant architecture than in signaling responses, are more pronounced at certain developmental stages or in specific populations, or involve the same tissue types during different developmental phases [1, 16, 36, 61]. The invasiveness of weeds is thought to be associated with several phenotypic plasticity traits including plant height, flower development, flowering, and light quality, [62, 63]. A direct correlation between plant height and invasiveness remains unclear. However, there may be an association between tall plant phenotypes, increased phenotypic diversity,
and higher plant abundance in unfavorable environments [63]. Associations have not been observed with flowering phenology among native and non-native plant populations, but this may be because flowering time is dependent on the environment [62–64]. Flower development and invasiveness in Purple loosestrife (Lythrum salicaria) demonstrate that both anther and stigma respond to changes in soil moisture during either of vegetative and reproductive development [65]. Tufted knotweed (Polygonum cespitosum) has been enhanced through adaptive responses to drought and high temperature without any observable decrease in fitness when grown in the shade [66]. Narrow-leaf plantain (Plantago lanceolata) is very sensitive to changes in light quality and modulates seed germination and leaf size as a mechanism for shade avoidance [67]. It is also the case in this weed species that leaf size and germination patterns share common physiological mechanisms where the short leaf phenotype is more plastic than the long leaf phenotype in shady conditions [20]. This discovery illustrates that wild plasticity is a dynamic network of processes that work synergistically to enhance the likelihood of survival [20]. Both trees and weeds demonstrate how, through the process of natural selection, non-cultivated plants have adopted very different and dynamic strategies that ensure reproduction and survival [1, 4, 12, 16–21, 24].

5. Genetic regulation of plasticity

There is not enough data about the genes, promoters, and regulatory elements that control “invasive” or “weedy” phenotypes commonly observed in wild plant populations. However, the phenotypes provide key insights into potential gene families and signaling pathways. Adaptive phenotypes also provide evidence that plasticity responses are controlled genetically and by specific plasticity genes [6]. The accumulation of genetic modifications associated with adaptive responses can be tracked through time and are genetically controlled [6]. Two models have been proposed to explore how changes in adaptive response occur. The first model proposes that the expression of structural genes varies as the environment changes [68]. Genetic plasticity is not regulated by plasticity genes, rather by changes in gene expression of structural genes resulting in phenotypic changes or plasticity [68]. The second model proposes that specific regulatory genes, i.e., plasticity genes mediate responses for structural genes [69, 70]. The resulting change in expression of the regulatory genes in response to environmental changes is what ultimately controls the pattern of plasticity [69, 70].

6. Plasticity mechanisms

There are four primary mechanisms in wild plant populations that regulate plasticity through adaptive responses [6]. The four plasticity mechanisms are physiological, developmental, cellular, and epigenetic responses [6]. Physiological plasticity describes all physiological responses associated with phenotypic traits and signaling networks [40, 71]. Developmental plasticity is associated with human or animal neural developmental, and plant embryonic development, in response to stress [6, 40]. Cellular plasticity describes adaptive responses within cells that are often associated with reducing reactive oxygen species accumulation through redox mechanisms [72]. Epigenetic plasticity describes changes to molecular mechanisms in response to abiotic stresses resulting in altered gene expression and function without changes in the DNA [71].
6.1 Physiological plasticity

Physiological plasticity is the most dynamic of plasticity mechanisms and is often involved in all other mechanisms of plasticity [40, 71]. Novel and emerging environments trigger many physiological responses such as carbon dioxide (CO$_2$) assimilation, changes in chlorophyll content, water use efficiency, sugar sensing and photosynthesis [73]. Physiological changes correlate directly to plant fitness, and changes in plasticity determine how a plant responds to environmental stresses [73, 74]. Studying the association between a physiological phenotype and changes in gene expression within wild populations will make it possible to identify and target genes that are responsible for adaptive responses, i.e., plasticity genes [24, 73, 74]. In this way plasticity genes and gene variants become a selective tool for understanding plasticity heritability dynamics, as well as identifying positively adapted populations [24, 73–75].

Seed dormancy is an excellent example of physiological plasticity [73, 74]. Seed dormancy prevents germination out of season, even under favorable conditions, and ensures species survival of natural catastrophes [16, 76]. Environmental cues such as light, temperature, and moisture impact the depth of seed dormancy and the length of time required for dormancy release [76]. In weeds, discontinuous or extensive seed dormancy ensures germination only in favorable conditions and confers environmental plasticity, or the ability to respond to changing biotic or abiotic environmental factors [16].

Discontinuous or extensive seed dormancy impacts environmental plasticity through variable emergence timing throughout a growing season [76]. Discontinuous seed dormancy is likely a major “weedy” characteristic contributing to physiological plasticity in many wild plants and weed populations [76]. Downy brome (Bromus tectorum L.) is an invasive grass weed in both natural and agricultural environments which produces seeds with discontinuous seed dormancy [77–80]. New downy brome seedlings have the capacity to emerge in any season; early and late in the fall, before and after cool season crops or native grasses emerge, and even in the spring [80]. Differences in emergence timing in downy brome may be due to differences in dormancy status and may occur because of phenotypic and genotypic variation within a single population cluster, the presence of multiple population clusters within a single location, and the viability of seed in the seed bank [80]. The successful invasion of wild plant populations is measured by the number of individuals in a population, reproductive output, the range of habitats occupied, and the ability for survival and adapt in new environments through time [16]. Therefore, downy brome is an excellent example of a model colonizing species as it allocates most of the developmental time to seed production [16]. Downy brome increases the chances for survival of future generations, by maximizing contributions to seed banks [76]. Physiological plasticity mechanisms like prolific seed production, discontinuous seed dormancy, and variable germination in weeds increase the likelihood of outcompeting wild and cultivated plant species in native and non-native environments [76].

There is currently very little information about the specific genes or molecular mechanisms regulating dormancy or dormancy loss in many weeds or wild plant species [77]. Gaps in molecular information slow the progress for understanding the impact of wild plasticity on adaptability [1, 16]. However, detailed physiological observations and translational research are useful tools. These are powerful tools for studying the mechanisms that drive physiological plasticity in the seed and throughout all plant life stages, in natural and agricultural environments, and in both wild and cultivated plant populations [1, 16, 82–86].
Basic research has established that a seed’s transition from dormancy to germination is controlled by the plant hormones, abscisic acid (ABA) and gibberellin (GA) [81, 82]. ABA establishes seed dormancy during embryo maturation and maintains dormancy in mature seeds, whereas GA stimulates seed germination [34, 36]. Dormancy studies in model systems including Arabidopsis thaliana, Brachypodium distachyon, Hordeum vulgare, and Triticum aestivum draw a clear connection between ABA, GA signaling mechanisms, seed dormancy and dormancy loss [81–84]. These studies also provide a framework for comparing the similarities, differences of mechanisms regulating physiological plasticity, and the degree of conservation within wild plant populations [50, 76, 83–100]. Carefully documenting development from seedling to seed in wild plant populations including weeds, provides a wealth of information about phenotypic plasticity in varied environments, and demonstrates the value in using wild species as models for understanding the full capacity of phenotypic plasticity in nature [85].

6.2 Developmental plasticity

Developmental plasticity was first identified in, and is most often associated with, human and animal development [40]. Developmental plasticity refers to the impact of environmental stimuli on embryonic development [6]. Within the plant biology community, there remains some skepticism surrounding the existence of plant developmental plasticity mechanisms, and how to best identify and characterize them [5–7]. Despite these challenges, recent paradigm shifts in conventional thought have resulted in significant efforts toward studying the impact of developmental plasticity in cultivated plant species [5–7, 40, 72].

Developmental plasticity directly impacts phenotypic plasticity and is characterized using GXE experiments that investigate the interactions of genotype in a given environment [7, 101–104]. Developmental plasticity occurs commonly within plant populations when a given population inhabits moderate environments [2, 18]. Abiotic stresses, like drought, trigger physiological and developmental plasticity in plants [7]. The degree of developmental plasticity observed in plants resulting from abiotic stress is directly connected to a plant’s development phase [7]. Some phases of development are more responsive to environmental changes and display a more plastic response than others [7]. It was found in spring wheat that the early developmental stages tillering and heading (after spike formation) show more morphological and physiological plasticity than other developmental phases [7]. Cold tolerance in quinoa is also based on developmental plasticity, and associated with grain formation [105, 106]. Flowering time is another trait associated with developmental plasticity across plant species [6]. A shift in flowering time in response to drought allows for accelerated seed set, thus ensuring species survival, even in non-ideal growing conditions [6].

6.3 Cellular plasticity

Plant cellular plasticity allows cells to respond to the negative impacts of biotic and abiotic stresses. Cellular plasticity occurs through long-range signaling via hydraulic, electrical, and chemical signaling mechanisms [107]. One example of chemical signaling directly connected with plant cellular plasticity occurs when plants experience oxidative stress. Environmental stresses stimulate the production of toxic chemicals known as reactive oxygen species (ROS) [108]. The function of scavenging enzymes is to quench the flux of ROS [108–114]. When ROS levels are elevated due to environmental stress, the activity of scavenging enzymes, including ascorbate peroxidase, superoxide dismutase (SOD), and catalase (CAT) increases [108–114].
Cellular plasticity is a very dynamic process whereby ROS scavengers are acting simultaneously in different cellular compartments including the cell wall membrane, cytoplasm, chloroplast, mitochondria, peroxisomes, and the apoplast [115–117]. The peroxisomes are the most important indicators of environmental stress, ROS-scavenger activity, and cellular plasticity [115]. Peroxisomes proliferate in response to an array of environmental stresses including light, ozone, metal, and salt [119]. Peroxisome number may fluctuate depending on cultivar or genotype [118–122]. An emerging hypothesis about cellular plasticity is that relative peroxisome abundance may be a good predictor for cellular plasticity mechanisms [123, 124]. Peroxisomal proliferation occurs because of environmental stress, and, any change in a phenological trait occurring from a change in environment is defined as cellular plasticity [123, 124]. Investigations of peroxisome proliferation in response to drought tolerance demonstrate that peroxisome abundance is correlated with abiotic stress response and impacts GXE interactions [123, 124]. A negative correlation also exists between peroxisome abundance and several phenological traits including plant biomass, root dry weight, and grain yield [123, 124]. Therefore, peroxisome abundance is an emerging tool for measuring cellular plasticity mechanisms of adaptation, and ROS homeostasis [123, 124].

### 6.4 Epigenetic plasticity

Plasticity responses exist as both the inherent genetic machinery (past regulatory events), and as part of regulation occurring outside of the genetic code (epigenetically) [71]. Epigenetic mechanisms include DNA methylation, non-coding RNA, chromatin remodeling, and histone modifications [71]. Changes in the environment trigger heritable changes in gene expression which result in stable phenotypes [71]. DNA methylation is the most common, and perhaps best understood mechanisms controlling epigenetic plasticity in plants [71]. Studies using Arabidopsis epigenetic recombinant inbred lines (epiRILS), i.e., lines with nearly identical genomes but contrasting DNA methylation patterns, demonstrated that plasticity to water availability and nutrient loss is controlled through changes in DNA methylation [80]. Epigenetic changes rather than genetic changes contribute to changes impacting phenotypic plasticity [71]. Other research has demonstrated that epigenetic regulation impacts heritability in specific phenological traits like plant height, plant biomass, seed/fruit production, the root-to-shoot ratio, and flowering time [71, 125–127]. Heritable traits are very important in breeding programs, and the role of epigenetics in regulating these traits is now only being characterized and understood [128].

### 7. The path to domestication: learning from transitional models

Decreased genetic diversity in plants populations is often associated with increased cultivation [7, 129]. Less cultivated plant populations tend to have more genetic diversity or “wildness” than plants that have been domesticated [129]. Wild characteristics broaden genetic responses and are valuable for maintaining phenotypic plasticity [129–131]. Leveraging broad genetic responses to enhance plasticity is especially important for the survival of plant species in unpredictable and changing climates [74, 75].

Since the dawn of agriculture, farmers have used selective breeding techniques for cultivating and domesticating wild plants for food [132]. Seeds from wild plant populations are smaller, an adaptation thought to enhance dispersal [132]. From an agricultural perspective, increased domestication is useful for reliable germination, uniform emergence, uniform stand establishment, larger seed size, increased yield,
and improved nutrition [132]. Domestication of wild maize, soybean, and barley has resulted in significant increases in seed size [86]. However, there has also been a negative cost associated with domestication [86]. In maize, soybean, and rice, domestication and intensive cultivation have resulted in the elimination of genetic loci in modern crop cultivars [86, 133–136]. Breeding strategies that do not address adaptation and plasticity decrease trait diversity and may limit the development of new crop varieties with the ability to adapt to insects and extreme environmental fluctuations [133–136].

A reduction in heritability of favorable traits within breeding populations has been one of the main reasons plant breeders have explored the possibility of integrating genetic diversity from wild populations (landraces) back into selective breeding programs [133–137]. Two wild plant models that have been very instrumental in the effort to introduce diversity back into breeding population are: (1) barley (*Hordeum vulgare*); a standard model for monocots, and (2) quinoa (*Chenopodium quinoa* Wild); a model for dicots [137–145]. These two models are very powerful because they highlight a clear transition from wild populations to domesticated cultivars. They also provide tools for understanding plasticity by comparing characteristics that have remained constant, changed, or been lost through a history of domestication [137–145].

Barley was domesticated very early in history from the wild grass relative, *Hordeum spontaneum* [137]. Barley, along with einkorn (*T. monococcum*, genomes AmAm) and emmer (*T. turgidum ssp. dicoccoides*, genomes BBAA), marked the beginning of domestication in cereals [137]. Barley is often used as a model to improve crops like wheat (*Triticum aestivum*) [138]. Barley demonstrates a wide range of plasticity including superior growth in nutrient-limited environments, and adapted root architecture [139, 140]. Although there are evolutionary similarities between barley and other monocots like wheat, the orthologous genomic regions between the two species have a completely diverged [141–143]. However, genomic similarities between barley and wheat have enriched the comparative studies of plasticity and provide new information about horizontal gene transfer [141–143].

Quinoa, like barley, was recognized as a valuable food resource, and was domesticated very long ago [144]. Although quinoa has been highly domesticated, it retains vast genetic variability and plasticity with a wide range of resistance to many abiotic and biotic stresses [144, 145]. Quinoa thrives in extreme environmental conditions including in regions with high salinity soils, areas of extremely low precipitation, and environments with extremely cold temperatures [105, 146]. Moreover, quinoa grain is resistant to starch degradation in environments susceptible to extreme temperature and moisture fluctuations [147]. The differences in plasticity discovered between wild and domesticated quinoa species illustrate the importance of continued studies identifying physiological and genetic mechanisms regulating plasticity [147]. These discoveries also highlight the feasibility and importance of selectively breeding for gene targets that improve adaptability and fitness [133–136]. Additionally, because quinoa is a polyploid, it is a rich resource for studying how complex genomes contribute new dimensions of genetic regulation to phenological plasticity [147]. Recent studies investigating modern cultivated varieties of quinoa show that cellular plasticity mechanisms, and more specifically ROS homeostasis, are dependent on both genotype and type of stress [123]. The emerging discoveries in quinoa are important because they provide a model for how plasticity mechanisms present in other polyploid crop species may be regulated [123].

8. Discussion

The discovery and utilization of improved traits that enhance the adaptability of crops to increasingly variable environments will help to ensure long-term crop
stability in changing climates [74, 128, 129]. Knowledge of phenological plasticity in wild populations will continue to benefit breeding programs [28]. Although wild genomes increase genetic complexity and may impact plasticity and fitness in unpredictable ways through changes in development, morphology, or physiology, one of the discovered benefits of increased diversity is increased adaptability [71, 129]. Over the last decade, advancements in genetics, molecular biology, systems biology, and statistical modeling have removed many of the barriers for understanding the regulation of complex plasticity networks in plants [13]. Association mapping, next generation sequencing, and genotype-by-phenotype (GWAS) approaches have greatly improved our comprehensive understanding of plasticity and the impacts of genomic selection [141–143]. Additionally, translational approaches utilizing a wealth of genomic information from both model plant systems and non-domesticated relatives have provided a framework for parallel studies in a wide range of plant populations. These studies have helped to uncover the developmental, cellular, and epigenetic mechanisms that regulate plasticity in all plants [6, 13, 71, 74, 142, 143].

9. Conclusions

One of the benefits of increasing genetic diversity in domesticated populations, from a long-term agricultural perspective, is the increased likelihood of plant population survival in unpredictable environments. In the past, evaluating the contributions of specific traits on phenological plasticity in plants was challenging due to experimental limitations and gaps in knowledge. However, emerging research continues to be extended from model systems directly to wild and cultivated plant populations to uncover the full potential of plasticity. New areas of research will need to investigate plasticity using a systems biology approach. Work should continue to explore the degree of conservation of plasticity existing between monocots and dicot crops, as well as comparing the contributions of ploidy on diversity. Other areas of research should address how DNA methylation and epigenetic mechanisms contribute to plant plasticity and may be fully utilized in plant improvement programs. Additional work should focus on how the simultaneous deployment of multiple plasticity mechanisms during plant developmental shift in changing environments using newly identified plasticity markers like the peroxisomes. Continued plasticity research will be critical for understanding how to maximize the benefits of both domestication and wild genetic diversity to maximize adaptation and fitness in a new area of climate diversity.
Author details

Amber L. Hauvermale¹ and Marwa N.M.E. Sanad²*

1 Department of Crop and Soil Sciences, Washington State University, Pullman, WA, USA

2 Department of Genetics and Cytology, National Research Centre, Giza, Egypt

*Address all correspondence to: mn.sanad@nrc.sci.eg and marwa.sanad@wsu.edu

IntechOpen

© 2019 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

12
References

[1] Gratani L. Plant phenotypic plasticity in response to environmental factors. Advances in Botany. 2014;2014:1-17

[2] Sultan SE. Commentary: The promise of ecological developmental biology. Journal of Experimental Zoological Biology and Molecular and Developmental Evolution. 2003;296:1-7

[3] West-Eberhard MJ. Developmental Plasticity and Evolution. New York: Oxford University Press; 2003. pp. 1-794

[4] Pigliucci M, Murren CJ, Schlichting CD. Phenotypic plasticity and evolution by genetic assimilation. The Journal of Experimental Biology. 2006;209:2362-2367

[5] Palmer CM, Bush SM, Maloof JN, et al. Phenotypic and Developmental Plasticity in Plants. Chichester, UK: John Wiley & Sons, Ltd; 2011

[6] De Jong M, Leyser O. Developmental plasticity in plants. Cold Spring Harbor Symposia on Quantitative Biology. 2012;77:63-73

[7] Sanad MNME, Campbell KG, Gill KS. Developmental program impacts phenological plasticity of spring wheat under drought. Botanical Studies. 2016;57:1-35

[8] Horton TH. Fetal origins of developmental plasticity: Animal models of induced life history variation. American Journal of Human Biology. 2005;17:34-43

[9] Minelli A, Fusco G. Developmental plasticity and the evolution of animal complex life cycles. Philosophical Transactions of the Royal Society, B: Biological Sciences. 2010;365:631-640

[10] Bateson P, Barker D, Clutton-Brock T, Deb D, D’Udine B, Foley RA, et al. Developmental plasticity and human health. Nature. 2004;430:419-421

[11] Nettle D, Bateson M. Adaptive developmental plasticity: What is it, how can we recognize it and when can it evolve? Proceedings of the Royal Society B: Biological Sciences. 2015;282:1005

[12] Funk JL. Differences in plasticity between invasive and native plants from a low resource environment. Journal of Ecology. 2008;96:1162-1173

[13] Nicotra AB, Davidson A. Adaptive phenotypic plasticity and plant water use. Functional Plant Biology. 2010;37:117-127

[14] Richter S, Kipfer T, Wohlgemuth T, Moser B. Phenotypic plasticity facilitates resistance to climate change in a highly variable environment. Oecologia. 2012;1(69):269-279

[15] Brachi B, Aimé C, Glorieux C, Cuguen J, Roux F. Adaptive value of phenological traits in stressful environments: Predictions based on seed production and laboratory natural selection. PLoS One. 2012;7:32069

[16] Baker H. Characteristics and Modes of origin of weeds. In: Genetics of Colonizing Species. New York: Academic Press Inc.; 1965. pp. 147-172

[17] Marshall DR, Jain SK. Phenotypic plasticity of Avena fatua and A. barbata. The American Naturalist. 1968;102:457-467

[18] Sultan SE. Phenotypic plasticity for plant development, function and life history. Trends in Plant Science. 2000;5:537-542

[19] Callaway RM, Pennings SC, Richards CL. Phenotypic plasticity and interactions among plants. Ecology. 2003;84:1115-1128
[20] Daehler CC. Performance comparisons of co-occurring native and alien invasive plants: Implications for conservation and restoration. Annual Review of Ecology, Evolution, and Systematics. 2003;34:183-211

[21] Rejmánek M, Richardson DM, Pylek P. Plant invasions and invasibility of plant communities. In: Maarel E, Franklin J, editors. Vegetative Ecology. 2nd ed. Wiley; 2013. pp. P387-P424

[22] Grenier S, Barre P, Litrico I. Phenotypic plasticity and selection: Nonexclusive mechanisms of adaptation. Scientifica. 2016;7021701:1-9

[23] Fay JC, Wittkopp PJ. Evaluating the role of natural selection in the evolution of gene regulation. Heredity. 2008;100:191-199

[24] Anderson JT, Willis JH, Mitchell-Olds T. Evolutionary genetics of plant adaptation. Trends in Genetics. 2011;27:258-266

[25] Przybylo R, Sheldon BC, Merila J. Climatic effects on breeding and morphology: Evidence for phenotypic plasticity. The Journal of Animal Ecology. 2000;69:395-403

[26] Reed TE, Schindler DE, Waples RS. Interacting effects of phenotypic plasticity and evolution on population persistence in a changing climate. Conservation Biology. 2011;25:56-63

[27] Ghalambor CK, McKay JK, Carroll SP, Reznick DN. Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. Functional Ecology. 2007;21:394-407

[28] Fritsche-Neto R, DoVale C. Breeding for stress-tolerance or resource-use efficiency? In: Fritsche-Neto R, Borém A, editors. Plant Breeding for Abiotic Stress Tolerance. Springer; 2012. pp. 13-19

[29] Waddington CH. Canalization of development and the inheritance of acquired characters. Nature. 1942;150:563-565

[30] Debat V, David P. Mapping phenotypes: Canalization, plasticity and developmental stability. Trends in Ecology & Evolution. 2001;16:555-561

[31] Van Gestel J, Weissing FJ. Is plasticity caused by single genes? Nature. 2001;45():746-754

[32] Belsky J, Jonassaint C, Pluess M, Stanton M, Brummett B, Williams R. Vulnerability genes or plasticity genes? Molecular Psychiatry. 2009;14:746-754

[33] Pigliucci M, Schmitt J. Genes affecting phenotypic plasticity in Arabidopsis: Pleiotropic effects and reproductive fitness of photomorphogenic mutants. Journal of Evolutionary Biology. 1999;12:551-562

[34] Des Marais DL, Juenger TE. Pleiotropy, plasticity, and the evolution of plant abiotic stress tolerance. Annals of the New York Academy of Sciences. 2010;1206:56-79

[35] Dewitt TJ, Sih A, Wilson DS. Costs and limits of phenotypic plasticity. Trends of Ecology & Evolution. 1998;13:77-81

[36] Sutherland S. What makes a weed a weed: Life history traits of native and exotic plants in the USA. Oecologia. 2004;141:24-39

[37] West-Eberhard MJ. Phenotypic plasticity and the origins of diversity. Annual Review of Ecology and Systematics. 1989;20:249-278

[38] Sultan SE. What has survived of Darwin's theory? Phenotypic plasticity and the neo-Darwinian legacy. Evolution of Trend Plant. 1992;6:61-71
[39] Sánchez-Gómez D, Valladares F, Zavala MA. Functional traits and plasticity in response to light in seedlings of four Iberian forest tree species. Tree Physiology. 2006;26:1425-1433

[40] Borges RM. Plasticity comparisons between plants and animals: Concepts and mechanisms. Plant Signaling & Behavior. 2008;3:367-375

[41] Chapin FS III. The mineral nutrition of wild plants. Annual Review of Ecology and Systematics. 1980;11:233-260

[42] Lambers H, Poorter H. Inherent variation in growth rate between higher plants: A search for physiological causes and ecological consequences. Advances in Ecological Research. 1992;23:187-261

[43] Aerts R, Peijl M. A simple model to explain the dominance of low-productive perennials in nutrient-poor habitats. Oikos. 1993;66:144-147

[44] Chapin FS III, Autumn K, Pugnaire F. Evolution of suites of traits in response to environmental stress. The American Naturalist. 1993;142:78-92

[45] Valladares F, Martínez-Ferri E, Balaguer L, Perez-Corona E, Manrique E. Low leaf level response to light and nutrients in Mediterranean evergreen oaks: A conservative resource-use strategy? The New Phytologist. 2000;148:79-91

[46] Pearson T, Burslem D, Goeriz R, Dalling J. Regeneration niche partitioning in neotropical pioneers: Effects of gap size, seasonal drought and herbivory on growth and survival. Oecologia. 2003;137:456-465

[47] Sapkota TB, Askegaard M, Laegdsmann M, Olsen JE. Effects of catch crop type and root depth on nitrogen leaching and yield of spring barley. Field Crops Research. 2012;125:129-138

[48] Alvarez-Flores R, Nguyen-Thi-Truc A, Peredo-Parada S, Joffre R, Winkel T. Rooting plasticity in wild and cultivated Andean Chenopodium species. Plant and Soil. 2018;425:479-492

[49] Finch-Savage WE, Cadman CS, Troorop PE, Lynn JE, Hilhorst HW. Seed dormancy release in Arabidopsis Cvi by dry after-ripening, low temperature, nitrate and light shows common quantitative patterns of gene expression directed by environmentally specific sensing. The Plant Journal. 2017;51:60-78

[50] Finch-Savage WE, Leubner-Metzger G. Seed dormancy and the control of germination. The New Phytologist. 2006;171:501-523

[51] Simpson GM, editor. Seed Dormancy in Grasses. New York: Cambridge University Press; 2007

[52] Miller G, Suzuki N, Ciftci-Yilmaz S, Mittler R. Reactive oxygen species homeostasis and signaling during drought and salinity stresses. Plant, Cell & Environment. 2010;33:453-467

[53] Sharma P, Jha AB, Dubey RS, Pessarakli M. Reactive oxygen species, oxidative damage, and antioxidative defense mechanism in plants under stressful conditions. Journal of Botany. 2012;26:1-27

[54] Vitasse Y, Bresson CC, Kremer A, Michalet R, Delzon S. Quantifying phenological plasticity to temperature in two temperate tree species. Functional Ecology. 2010;24:1211-1218

[55] Williams DG, Mack RN, Black RA. Ecophysiology of introduced Pennisetum setaceum on Hawaii: The role of phenotypic plasticity. Ecology. 1995;76:1569-1580
[56] Yeh PJ, Price TD. Adaptive phenotypic plasticity and the successful colonization of a novel environment. The American Naturalist. 2004;164:531-542

[57] Atkin OK, Loveys BR, Atkinson LJ, Pons TL. Phenotypic plasticity and growth temperature: Understanding interspecific variability. Journal of Experimental Botany. 2006;57:267-281

[58] Aroca R, editor. Plant Responses to Drought Stress: From Morphological to Molecular Features. Verlag Berlin Heidelberg: Springer; 2012. 413 p

[59] DeLuccia E, Maherali H, Carey E. Climate-driven changes in biomass allocation in pines. Global Change Biology. 2000;6:587-593

[60] Markestijn L, Poorter L. Seedling root morphology and biomass allocation of 62 tropical tree species in relation to drought-and shade-tolerance. Journal of Ecology. 2009;97:311-325

[61] Baker HG. Weeds-native and introduced. Journal of California Horticulture Society. 1962;23:97-104

[62] Pyšek P, Richardson DM, editors. Traits associated with invasiveness in alien plants: Where do we stand? In: Biological Invasions. Berlin Heidelberg: Springer Verlag; 2008. pp. 97-125

[63] Goodwin BJ, Allister AJMC, Fahrig L. Predicting invasiveness of plant species based on biological information. Conservation Biology. 1999;13:422-426

[64] Pyšek P, Brock JH, Bímová K, Mandák B, Jarosík V, Koukolíková I, et al. Vegetative regeneration in invasive Reynoutria (Polygonacea) taxa: The determinant of invisibility at the genotype level. American Journal of Botany. 2003;90:1487-1495

[65] Mal TK, Lovett-Doust J. Phenotypic plasticity in vegetative and reproductive traits in an invasive weed, Lythrum salicaria (Lythraceae), in response to soil moisture. American Journal of Botany. 2005;92:819-825

[66] Sultan SE, Matesanz S. An ideal weed: Plasticity and invasiveness in Polygonum cespitosum. Annals of the New York Academy of Sciences. 2015;1360:101-119

[67] Van Hinsberg A. Morphological variation in Plantago lanceolata L.: Effects of light quality and growth regulators on sun and shade populations. Journal of Evolutionary Biology. 1997;10:687-701

[68] Via S. Adaptive phenotypic plasticity: Target or by-product of selection in a variable environment? The American Naturalist. 1993;1(42):352-365

[69] Schlichting CD. The evolution of phenotypic plasticity in plants. Annual Review of Ecology and Systematics. 1986;17:667-693

[70] Schlichting CD, Pigliucci M. Gene regulation, quantitative genetics and the evolution of reaction norms. Evolutionary Ecology. 1995;9:154-168

[71] Zhang L, Lu X, Lu J, Liang H, Dai Q, Xu G-L, et al. Thymine DNA glycosylase specifically recognizes 5-carboxylcytosine-modified DNA. Nature Chemical Biology. 2012;8:328-330

[72] Kuwabara A, Nagata T. Cellular basis of developmental plasticity observed in heterophyllous leaf formation of Ludwigia arcuata (Onagraceae). Planta. 2006;224:761-770

[73] Kimball S, Gremer JR, Angert AL, Huxman TE, Venable DL. Fitness and physiology in a variable environment. Oecologia. 2012;169:319-329

[74] Becklin KM, Anderson JT, Gerhart LM, Wadgymar SM,
Wessinger CA, Ward JK. Examining plant physiological responses to climate change through an evolutionary lens. Plant Physiology. 2016;172:635-649

[75] Mitchell-Olds T, Willis JH, Goldstein DB. Which evolutionary processes influence natural genetic variation for phenotypic traits? Nature Reviews Genetics. 2007;8:845-856

[76] Bewley JD, Bradford KJ, Hilhorst HWM. In: Nonogaki H, editor. Seeds: Physiology of Development, Germination and Dormancy. Springer; 2013. pp. 1-15

[77] Rydrych D, Muzik T. Downy brome competition and control in dryland wheat. Agronomy Journal. 1968;60:279-280

[78] Young JA, Evans RA, Eckert RE. Population dynamics of downy brome. Weed Science. 1969;17:20-26

[79] Stahlman P, Miller S. Downy brome (Bromus tectorum) interference and economic thresholds in winter wheat (Triticum aestivum). Weed Science. 1990;38:224-228

[80] Blackshaw R. Downy brome (Bromus tectorum) control in winter wheat and winter rye. Canadian Journal of Plant Science. 1994;74:185-191

[81] Koornneef M, Jorna ML, der Brinkhorst-van Swan DLC, Karssen CM. The isolation of abscisic acid (ABA) deficient mutants by selection of induced revertants in non-germinating gibberellin sensitive lines of Arabidopsis thaliana (L.) heynh. Theoretical and Applied Genetics. 1982;61:385-393

[82] Finkelstein RR, Reeves W, Ariizumi T, Steber CM. Molecular aspects of seed dormancy. Annual Review of Plant Biology. 2008;59:387-415

[83] Karssen CM, Lačka E. A revision of the hormone balance theory of seed dormancy: Studies on gibberellin and/or abscisic acid-deficient mutants of Arabidopsis thaliana. Plant Growth Substances. Verlag Berlin Heidelberg: Springer; 1986. pp. 315-323

[84] Walker-Simmons M. ABA levels and sensitivity in developing wheat embryos of sprouting resistant and susceptible cultivars. Plant Physiology. 1987;84:61-66

[85] Morris CF, Moffatt JM, Sears RG, Paulsen GM. Seed dormancy and responses of caryopses, embryos, and calli to abscisic acid in wheat. Plant Physiology. 1989;90:643-647

[86] Taylor IB, Burbidge A, Thompson AJ. Control of abscisic acid synthesis. Journal of Experimental Botany. 2000;51:1563-1574

[87] Kushiro T, Okamoto M, Nakabayashi K, Yamagishi K, Kitamura S, Asami T, et al. The Arabidopsis cytochrome P450 CYP707A encodes ABA 8′-hydroxylases: Key enzymes in ABA catabolism. The EMBO Journal. 2004;23:1647-1656

[88] Chono M, Honda I, Shinoda S, Kushiro T, Kamiya Y, Nambara E, et al. Field studies on the regulation of abscisic acid content and germinability during grain development of barley: Molecular and chemical analysis of pre-harvest sprouting. Journal of Experimental Botany. 2006;57:2421-2434

[89] Millar AA, Jacobsen JV, Ross JJ, Helliwell CA, Poole AT, Scofield G, et al. Seed dormancy and ABA metabolism in arabidopsis and barley: The role of ABA 8′-hydroxylase. The Plant Journal. 2006;45:942-954

[90] Okamoto M, Kuwahara A, Seo M, Kushiro T, Asami T, Hirai N, et al. CYP707A1 and CYP707A2, which
encode abscisic acid 8′-hydroxylases, are indispensable for proper control of seed dormancy and germination in Arabidopsis. Plant Physiology. 2006;141:97-107

[91] Barrero JM, Talbot MJ, White RG, Jacobsen JV, Gubler F. Anatomical and transcriptomic studies of the coleorhiza reveal the importance of this tissue in regulating dormancy in barley. Plant Physiology. 2009;150:1006-1021

[92] Schramm EC, Abellera JC, Strader LC, Campbell KG, Steber CM. Isolation of ABA-responsive mutants in allohexaploid bread wheat (Triticum aestivum L.): Drawing connections to grain dormancy, preharvest sprouting, and drought tolerance. Plant Science. 2010;179:620-629

[93] Barrero JM, Jacobsen JV, Talbot MJ, White RG, Swain SM, Garvin DF, et al. Grain dormancy and light quality effects on germination in the model grass Brachypodium distachyon. New Phytologist. 2012;193:376-386

[94] Ariizumi T, Hauvermale AL, Nelson SK, Hanada A, Yamaguchi S, Steber CM. Lifting DELLA repression of Arabidopsis seed germination by non-proteolytic gibberellin signaling. Plant Physiology. 2013;162:2125-2139

[95] Chono M, Matsunaka H, Seki M, Fujita M, Kiribuchi-Otobe C, Oda S, et al. Isolation of a wheat (Triticum aestivum L.) mutant in ABA 8′-hydroxylase gene: Effect of reduced ABA catabolism on germination inhibition under field condition. Breeding Science. 2013;63:104-115

[96] Schramm EC, Nelson SK, Kidwell KK, Steber CM. Increased ABA sensitivity results in higher seed dormancy in soft white spring wheat cultivar ‘Zak’. Theoretical and Applied Genetics. 2013;126:791-803

[97] Barrero JM, Downie AB, Xu Q, Gubler F. A role for barley CRYPTOCHROME1 in light regulation of grain dormancy and germination. The Plant Cell. 2014;26:1094-1104

[98] Hauvermale AL, Tuttle KM, Takebayashi Y, Seo M, Steber CM. Loss of Arabidopsis thaliana seed dormancy is associated with increased accumulation of the GID1 GA hormone receptors. Plant & Cell Physiology. 2015;56:1773-1785

[99] Tuttle KM, Martinez SA, Schramm EC, Takebayashi Y, Seo M, Steber CM. Grain dormancy loss is associated with changes in ABA and GA sensitivity and hormone accumulation in bread wheat, Triticum aestivum (L.). Seed Science Research. 2015;25:179-193

[100] Lawrence NC, Hauvermale AL, Dhingra A, Burke IC. Population structure and genetic diversity of Bromus tectorum within the small grain production region of the Pacific Northwest. Ecology and Evolution. 2017;7:8316-8328

[101] Chaves MM, Maroco JP, Pereira JS. Understanding plant responses to drought from genes to the whole plant. Functional Plant Biology. 2003;30:239-264

[102] Rizza F, Badeck FW, Cattivelli L, Lidestri O, Di Fonzo N, Stanca AM. Use of a water stress index to identify barley genotypes adapted to rainfed and irrigated conditions. Crop Science. 2004;44:2127-2137

[103] De Leonardis AM, Marone D, Mazzucotelli E, Neffar F, Rizza F, Di Fonzo N, et al. Durum wheat genes up-regulated in the early phases of cold stress are modulated by drought in a developmental and genotype dependent manner. Plant Science. 2007;172:1005-1016
[104] Milad SI, Wahba LE, Barakat MN. Identification of RAPD and ISSR markers associated with flag leaf senescence under water-stressed conditions in wheat (Triticum aestivum L.). Australian Journal of Crop Science. 2011;5:334-340

[105] Espindola G. Respuestas fisiológicas, morfológicas y agronómicas de la quinoa al déficit hídrico [thesis]. Chapingo, México: These de maitrise, Colegio de Postgraduados Institución de Enseñanza e Investigaciér': Ciencias Agrícolas; 1986

[106] Rea J, Tapia M, Mujica A. Practicas agronómicas. In: Tapia M, Gandarillas H, Alandia S, Cardozo A, Mujica A, editors. Quinua y Kaiiwi. Cultivos Andinos. Rome, Italy: FAO; 1997

[107] Huber AE, Bauerle TL. Long-distance plant signaling pathways in response to multiple stressors: The gap in knowledge. Journal of Experimental Botany. 2016;67:2063-2079

[108] Gamble PE, Burke JJ. Effect of water stress on the chloroplast antioxidant system I. Alterations in glutathione reductase activity. Plant Physiology. 1984;76:615-621

[109] Smirnoff N. The role of active oxygen in the response of plants to water deficit and desiccation. New Phytologist. 1993;125:27-58

[110] Noctor G, Foyer CH. Ascorbate and glutathione: Keeping active under control. Annual Review of Plant Physiology and Plant Molecular Biology. 1998;49:249-279

[111] Rubio MC, González EM, Minchin FR, Webb KJ, Arrese-Igor C, Ramos J, et al. Effects of water stress on antioxidant enzymes of leaves and nodules of transgenic alfalfa overexpressing superoxide dismutases. Physiologia Plantarum. 2002;115:531-540

[112] Jiang M, Zhang J. Water stress-induced abscisic acid accumulation triggers the increased generation of reactive oxygen species and up-regulates the activities of antioxidant enzymes in maize leaves. Journal of Experimental Botany. 2002;53:2401-2410

[113] Guo Z, Ou W, Lu S, Zhong Q. Differential responses of antioxidative system to chilling and drought in four rice cultivars differing in sensitivity. Plant Physiology and Biochemistry. 2006;44:828-836

[114] Møller IM, Jensen PE, Hansson A. Oxidative modifications to cellular components in plants. Annual Review of Plant Biology. 2007;58:459-481

[115] Foyer CH, Noctor G. Redox sensing and signaling associated with reactive oxygen in chloroplasts, peroxisomes and mitochondria. Physiologia Plantarum. 2003;119:355-364

[116] Apel K, Hirt H. Reactive oxygen species: Metabolism, oxidative stress, and signal transduction. Annual Review of Plant Biology. 2004;55:373-399

[117] Nyathi Y, Baker A. Plant peroxisomes as a source of signaling molecules. Biochimica et Biophysica Acta (BBA)—Molecular Cell Research. 2006;1763:1478-1495

[118] Ferreira RMB, Bird B, Davies DD. The effect of light on the structure and organization of lemna peroxisomes. Journal of Experimental Botany. 1989;40:1029-1035

[119] Morre DJ, Sellden G, Ojanpéra K, Sandelius AS, Egger A, Morre DM, et al. Peroxisome proliferation in Norway spruce induced by ozone. Protoplasma. 1990;155:58-65

[120] Romero-Puertas MC, McCarthy I, Sandalio LM, Palma JM, Corps FJ, Gómez M, et al. Cadmium toxicity and oxidative metabolism of pea leaf...
peroxisomes. Free Radical Research. 1999;31:25-31

[121] Oksanen E, Häikiö E, Sober J, Karnosky DF. Ozone-induced H$_2$O$_2$ accumulation in field-grown aspen and birch is linked to foliar ultrastructure and peroxisomal activity. New Phytologist. 2004;161:791-799

[122] Mitsuya S, El-Shami M, Sparkes IA, Charlton WL, Louisa CDM, Johnson B, et al. Salt stress causes peroxisome proliferation, but inducing peroxisome proliferation does not improve NaCl tolerance in Arabidopsis thaliana. PLoS One. 2010;5:9408

[123] Fahy D, Sanad MNME, Duscha K, et al. Impact of salt stress, cell death, and autophagy on peroxisomes: Quantitative and morphological analyses using small fluorescent probe N-BODIPY. Scientific Reports. 2017;7, 39069

[124] Marwa NM, Sanad E, Andrei S, Kimberley A. Garland-Campbell. Differential dynamic changes of reduced trait model for analyzing the plastic response to drought: A case study in spring wheat. Frontiers in Plant Science. 2019. DOI: 10.3389/fpls.2019.00504

[125] Lynch M, Walsh B. Genetics and analysis of quantitative traits. Oxford University Press; 1998. pp. 1-980

[126] Johannes F, Porcher E, Teixeira FK, et al. Assessing the impact of transgenerational epigenetic variation on complex traits. PLoS Genetics. 2009;5:e1000530

[127] Roux F, Colomé-Tatché M, Edelis C, Wardenaar R, Guerche P, Hospital F, et al. Genome-wide epigenetic perturbation jump-starts patterns of heritable variation found in nature. Genetics. 2011;188:1015-1017

[128] Galluscip D, Dai Z, Génard M, Guaffretau A, Leblanc-Fournier N, Richard-Molard C, et al. Epigenetics for plant improvement: Current knowledge and modeling avenues. Trends in Plant Science. 2017;22:610-623

[129] Zhang H, Mittal N, Leamy LJ, Barazani O, Song B-H. Back into the wild; applying untapped genetic diversity of wild relatives for crop improvement. Evolutionary Applications. 2017;10:5-24

[130] Pigliucci M, Kolodynska A. Phenotypic plasticity to light intensity in Arabidopsis thaliana: Invariance of reaction norms and phenotypic integration. Evolutionary Ecology. 2002;16:27-47

[131] Bossdorf O, Pigliucci M. Plasticity to wind is modular and genetically variable in Arabidopsis thaliana. Evolutionary Ecology. 2009;23:669-685

[132] Osborne C. The conversation, an academic rigour, journalistic flair [Internet]. 2017. Did the first farmers deliberately domesticate wild plants? Available from http://theconversation.com/did-the-first-farmers-deliberately-domesticate-wild-plants-77434

[133] Wright SI, Bi IV, Schroeder SG, Yamasaki M, Doebley JF, MD MM, et al. The effects of artificial selection on the maize genome. Science. 2005;308:1310-1314

[134] Hyten DL, Song Q, Zhu Y, Choi I-Y, Nelson RL, Costa JM, et al. Impacts of genetic bottlenecks on soybean genome diversity. Proceedings of the National Academy of Sciences. 2003;103:16666-16671

[135] Xu X, Liu X, Ge S, et al. Resequencing 50 accessions of cultivated and wild rice yields markers for identifying agronomically
Phenological Plasticity of Wild and Cultivated Plants
DOI: http://dx.doi.org/10.5772/intechopen.85070

important genes. Nature Biotechnology. 2012;30:105-111

[136] Zhou Z, Jiang Y, Wang Z, et al. Resequencing 302 wild and cultivated accessions identifies genes related to domestication and improvement in soybean. Nature Biotechnology. 2015;33:408-414

[137] Harlan JR, Zohary D. Distribution of wild wheats and barley. Science. 1996;153:1074-1080

[138] Kartha KK, Nehra NS, Chibbar RN. Genetic engineering of wheat and barley. In: Robert J. Henry, John A. Ronalds, editors. Improvement of Cereal Quality by Genetic Engineering. New York: Springer; 1994:21-30

[139] Elberse IAM, van Damme JMM, van Tienderen PH. Plasticity of growth characteristics in wild barley (*Hordeum spontaneum*) in response to nutrient limitation. Journal of Ecology. 2003;91:371-382

[140] Bingham IJ, Bengough AG. Morphological plasticity of wheat and barley roots in response to spatial variation in soil strength. Plant and Soil. 2003;250(2):73-282

[141] Ramakrishna W, Dubcovsky J, Park Y-J, Busso C, Emberton J, Sanmiguel P, et al. Different types and rates of genome evolution detected by comparative sequence analysis of orthologous segments from four cereal genomes. Genetics. 2002;162:1389-1400

[142] SanMiguel PJ, Ramakrishna W, Bennetzen JL, Busso CS, Dubcovsky J. Transposable elements, genes and recombination in a 215-kb contig from wheat chromosome 5Am. Functional and Integrative Genomics. 2002;2:70-80

[143] Dubcovsky J, Dvorak J. Genome plasticity a key factor in the success of polyploid wheat under domestication. Science. 2007;29:316-393

[144] Del Castillo C, Winkel T, Mahy G, Bizoux J-P. Genetic structure of quinoa (*Chenopodium quinoa* Willd.) from the Bolivian altiplano as revealed by RAPD markers. Genetic Resources and Crop Evolution. 2007;54:897-905

[145] Rojas W, Mamani E, Pinto M, Alanoca C, and Ortúño T. Identificación taxonómica de parientes silvestres de quinoa del Banco de Germoplasma de Granos Altoandinos. En Revista de Agricultura. Revista de Agricultura-Año 60, Nro. 44. Cochabamba, Bolivia. 2008. pp. 56-65

[146] Jacobsen SE. The worldwide potential for quinoa (*Chenopodium quinoa* Willd.). Food Reviews International. 2003;19:167-177

[147] Ahamed NT, Singhal RS, Kulkarni PR, Pal M. A lesser-known grain, *Chenopodium Quinoa*: Review of the chemical composition of its edible parts. Food and Nutrition Bulletin. 1998;19:61-70