



EPIGENETIC VARIATIONS BROUGHT ABOUT BY CLIMATIC CHANGE IN PLANTS – AN ASSESSMENT

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ABSTRACT

The earth is experiencing major changes in global and regional climates and changes are believed to escalate in the future. The organism will be facing the threat of extinction. Detoriated environment conditions can bring about epigenetics changes in plant and also in gene expression. This may help plants to adapt to environmental change. Also they are reversible. The major challenge for the investigation of epigenetic adaptation theories is therefore to identify genomic loci that undergo epigenetic changes in response to environmental conditions, which alter their expression in a heritable way and which improve the plant's ability to adapt to the inducing conditions. This review focuses on the role of DNA methylation as a prominent epigenetic mark that controls chromatin conformation, and on its potential in mediating expression changes in response to environmental signals.

KEYWORDS: *Adaptation, DNA methylation, epigenetic variation, stress response, climate acclimation.*



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INTRODUCTION

Rapid and extreme climate changes are predicted, raising questions as to the capacity of plants to adjust to and survive the new environments. In clonal plants, limited dispersal and lack of recombination as a source of new gene combinations might compromise their capacity to migrate or evolve fast enough. Recent work in epigenetics has revealed an alternative path to adaptation involving variation in gene regulation, whereby genotypes can respond to environmental change without genetic recombination¹⁻³, that has consequences for clonal plants⁴⁻⁵. The most recent Intergovernmental Panel for Climatic Change (IPCC) report (IPCC, 2014) predicts increasing temperatures among a range of carbon emission scenarios, with greatest changes occurring at higher latitudes and elevations. A second prediction from the IPCC report is increased frequency of extreme climatic events, such as heat, droughts, floods and storm damage. Indirect effects of these extreme events will include reduced plant defenses, increased attacks from pests and diseases and subsequent episodes of fire and soil erosion. Rates of climate change predicted by the climate models for the next century are unprecedented and may exceed by an order of magnitude the rates of climate warming during the Holocene deglaciation⁶. The last major period of climatic change, during the Pleistocene, saw major species' distributional shifts⁷ and changes in community structure as a result of unequal species' responses⁸. Ecologists have already monitored plant responses to recent climate change that include colonization, as altitudinal and latitudinal displacements occur⁹ and adaptation, such as spring-time advances in phenological processes¹⁰. Currently, mortality through increased attacks by pests and diseases following drought stress is putting some ecosystems at risk¹¹⁻¹². Because of the anticipated rates of climate change, population relocations through dispersal and colonization are expected to be more successful responses than survival through adaptation *in situ*¹³. Community structure will likely change as species respond unequally; modeling suggests that community diversity decreases and non-analog communities are most likely to form when dispersal differences among species are high¹⁴. Those species with limited ranges, or low dispersal potential are the most likely to face extinction. The sedentary nature of plants imposes constraints on the velocity of response to rapidly changing environmental conditions. The potential for plants to track climate change depends on long distance dispersal events that allow colonization of new habitat and added genetic variation on the colonizing front¹⁵. For many clonal plants that spread vegetatively from attached organs (rhizomes, roots, stem bases), the opportunity for populations to track environmental change through long distance dispersal will be limited¹⁶⁻¹⁷. If clonal plants are limited in the ability to disperse, will species that depend on this mode of reproduction be able to take advantage of *In situ* adaptation as are response to climate change? In the absence of meiotic recombination, adaptation requires fitness beneficial mutations (mitotic recombination is unlikely to provide a sufficient source of genotypic variation that could be selectively

advantageous.). Accumulation of beneficial mutations is slow and, for phenotypic traits, antagonistic interactions among traits are likely to impede adaptation¹⁸. Nevertheless, clonal plants have persisted for thousands, or even millions of years during past environmental changes¹⁹, and clonal plants have been successful in colonizing new habitats²⁰⁻²¹, occupying broad geographic ranges²²⁻²³ and have come to dominate some ecosystems²⁴. Epigenetic mechanisms alter the probability or competence of genetic information to be expressed in a heritable but still reversible way. This is mediated by changes in chromatin structure that alter the accessibility of a genetic region for the transcription machinery, or by changes in turnover rates of selected transcripts. In many, but not all, cases these changes are implemented by small RNAs or longer non-coding RNAs that serve as sequence- or locus-specific guides for DNA methylation, chromatin modification, or transcript degradation/amplification mechanisms. While epigenetic changes can influence mutation and recombination rates, epigenetic target loci do not change their DNA sequence. A local epigenetic modification, as long as it is maintained, therefore alters the conversion of genetic information into a phenotype, while reversal to the original epigenetic state restores the previous status quo. This provides plants with an efficient tool to alter gene function in specific cell types, developmental stages, or under specific environmental conditions, and to pass on the altered epigenetic state during somatic cell division or even via the germ line to subsequent generations. Depending on the epigenetic modification, this can lead to the silencing of a previously active gene or to the activation of a functional but so far silent genetic region. Reversible epigenetic modifications include histone marks, in particular methylation, acetylation, or phosphorylation marks at histone tails, and methylation of cytosines. So, how do the classic paradigms for clonal plants fail to capture their ecological potential in a dynamic world? First, obligate clonality is rare in nature²⁵. Even in extreme environments where clonal reproduction is expected to predominate⁽²⁶⁾, high genetic diversity can be maintained by episodes of sexual recruitment²⁷. Second, clonal growth permits a range of advantageous ecological strategies including resource sharing⁽²⁸⁾, niche specialization²⁹⁻³⁰ and rapid vegetative growth, particularly in pioneer habitats. Thirdly, plastic phenotypic responses may include an adaptive component that to some extent substitutes for, and can be far more rapid than adaptation through genetic selection⁴⁻⁵. Accommodation through plasticity has commonly been invoked as important in permitting clonal plants to respond to heterogeneous environments^{31, 22}. Furthermore, phenotypic accommodation to changing environments ultimately can lead to evolutionary change through selection on elevations and slopes of norms of reaction³²⁻³³. Recently, an increasing number of studies have shown that plastic responses can be mediated through epigenetic modifications³⁴⁻³⁵, the most commonly studied being DNA methylation that results in changes in gene expression. These epigenetic marks may be stable across somatic generations³⁶⁻³⁷ and across germlines^{2, 38, 4, 5}; the latter known as transgenerational⁽³⁹⁾. Stable epigenetic variations that result in phenotypic variation are thought to offer both

short and long-term possibilities for plants to be buffered against their environment^{21, 35, 40-41} and provide an alternative, or are complementary to genomic adaptations. Because epigenetic variations can be heritable and reversible^{2,4-5,36,38,42} they offer a potentially flexible mechanism for plant adaptation. Therefore, epigenetic diversity could provide a crucial source of adaptive potential in asexual plants^{4-5,37}.

EXTREME CLIMATIC EVENTS

Extreme climatic events can be the most devastating for plant survival by pushing systems beyond thresholds of tolerance⁴³. The predicted increase in extreme events will place many organisms under stress (i.e., drought) and impose episodes of extreme environmental events (i.e., fire) and potentially devastating biotic interactions (i.e., pests and diseases). It has been suggested that extreme events may have impacts on ecosystems even before the progressive changes in temperature or rainfall⁴⁴. Evolutionary responses to select for stress-tolerant genotypes are unlikely to be rapid enough to protect populations against extinction in the face of extreme stresses. However, the potential for epigenetic responses to stress may provide the phenotypic variation necessary to sustain populations during events that could push plants past threshold tolerance levels. We now know that environmental stresses can elicit changes in DNA methylation. Examples mostly from *in vitro* tests on well-studied systems such as crop plants or *Arabidopsis* have been reviewed⁴⁵. In many cases, these involve histone mediated epigenetic changes that are reversed when the environmental cue is removed. The reversibility of epigenetic changes can provide an important additional source of variation. It would be interesting to test whether reversals can provide a pre-adaptation to future change. In other words, once an epigenetic change has occurred, even if reversed, could it be easier for the same change to occur again in the future (a form of hormesis,⁵). Further studies of stress-induced DNA methylation in genetically identical apomictic dandelions revealed more than 75% of epigenetic modifications to be transmitted to offspring not exposed to the environmental stress⁴¹. These epigenetic changes may be stress-targeted or random (subject to natural selection), but in either case they contribute to the overall response to environmental stimuli and indicate an added potential for phenotypic diversity⁴. Although the potential for stress-related epigenetic changes may occur in both sexual and asexual plants, the combination of these responses and the ecology of clonal species can explain in part the success of clonality in environments subject to severe stresses. Many species are particularly vulnerable during the seed development, germination and seedling establishment phase, particularly in drought-prone habitats. Indeed, the switch from sexual to asexual reproduction is commonly associated with the risks associated with sexual reproduction²⁶. Clonal reproduction provides an escape from the seedling phase, coupled with rapid vegetative growth because of the existing root system. The moderating effect epigenetic variation has on reduced genetic recombination, to some extent tempers the genetic disadvantage of this sex-avoidance strategy. Increased fire frequencies and severity are expected to be the new norm in many parts of the world as a result

of higher temperature and increased drought⁴⁶⁻⁴⁷. Although some sexual reproductive systems are fire-adapted, in a great many resprouting systems, clonal reproduction provides the most rapid recovery after fire⁴⁸. Another effect of drought-stress is the increased incidence of disease and pest outbreaks¹¹⁻¹². The search for heritable variation in disease resistance traits usually assumes variations in DNA sequence. However, recently⁴⁹ reported the epigenetic inheritance of response to the defense hormones, jasmonic acid and salicylic acid, in *Arabidopsis thaliana*. The role of epigenetics in plant defense to pests and diseases is an area that deserves much more attention as it seems likely to hold considerable promise in understanding disease dynamics in natural populations. The assumption that resistance is genetically based would suggest that clonal genotypes may be at a disadvantage as disease spreads through a population. However, if stress-related epimutations arise in populations, it is possible that disease outbreaks could induce defense responses regardless of the host genotype.

BIOLOGICAL EFFECTS OF DNA METHYLATION

Changes in DNA methylation are the easiest to detect and most precisely positioned indicators and modifiers of epigenetic change, which influence gene expression directly or in combination with histone marks. Changes in DNA and histone methylation influence gene expression, in particular transcription⁵⁰, splicing⁵¹, and polyadenylation⁵², but they also affect DNA repair⁵³, recombination⁵⁴, and meiotic crossover in euchromatic regions⁵⁵. The multiple mechanistic effects make it difficult to differentiate between direct changes mediated by DNA methylation and their secondary effects. While the literature is full of reports that correlate DNA methylation and specific phenotypes, there are many fewer reports that demonstrate a direct role for DNA methylation in the transcriptional regulation of one or several distinct target loci, which are responsible for a defined effect or phenotype. Examples of mechanisms and phenotypes under direct control of DNA methylation include parental imprinting⁵⁶, floral symmetry⁵⁷, flowering time⁵⁸, pigmentation⁵⁹, fruit ripening⁶⁰, sex determination⁶¹, and stomatal development⁶²⁻⁶³. Seed yield, determined by energy use efficiency, was the first quantitative trait associated with distinct, heritable DNA methylation patterns⁶⁴. Flowering time and primary root length are two other complex quantitative traits linked to DNA methylation patterns at differentially methylated regions (DRMs). Methylation patterns of some DRMs are heritably altered in epigenetic mutants, which suggest that they are specific targets of an epigenetic system that enhances expression variability. Accordingly, many DRMs display a considerable level of variability in natural *Arabidopsis* populations⁶⁵.

STRESS-INDUCED EPIGENETIC CHANGES

While epigenetic *Arabidopsis* mutants have proven useful to test the significance of epigenetic functions in stress responses^{52, 66}, we have to be careful when drawing conclusions about a direct role for epigenetic functions, especially when using epigenetic mutants that display a range of phenotypes due to secondary effects. Mutation of the *MET1* gene, for example, inhibits expression of DNA demethylases and leads to the establishment of histone H3K9 methylation and RNA-

directed methylation marks in new genomic regions⁶⁷. This generates a variety of stochastic epi-mutations and phenotypes, many of which probably do not represent direct MET1 targets but reflect randomly established novel epigenetic marks. Another factor that complicates the comparison of epigenetic mutants and wild-type lines is background differences in gene expression profiles frequently observed among different plant lines due to epigenetic diversity⁶⁸. The use of epigenetic mutants to link phenotypic effects to distinct epigenetic changes is further complicated by the mutagenic consequence of certain epigenetic alterations, which induce genetic changes that could be mistaken for stable epi-mutations. This is exemplified by the *bal* variant that was isolated from an inbred *ddm1* mutant background and that contains a 55 kb duplication within the *RPP5* (*recognition of Peronospora parasitica* 5) locus, which includes a cluster of disease Resistance (*R*) genes. Duplication is accompanied by hypermutation and up-regulation of *SNC1* (*SUPPRESSOR OF NPR1-1, CONSTITUTIVE 1*), which co-ordinately activates *RPP5* locus *R* genes and induces a distinct dwarfism and curled leaf phenotype⁶⁹. It is unclear if these changes represent a random, independent event, or if recombination and mutation rates at the *RPP5* locus are increased by *DDM1* deletion. If hypomethylation induced by mutation of *DDM1* or other methylation functions stimulates recombination and mutation events at distinct loci, this could lead to genetic changes of identical regions in different DNA methylation mutants that could be mistaken for epi-mutations. To identify direct epigenetic targets for stress effects among a background of epi-alleles and genetic mutations, it will therefore be important to link expression changes at potential epigenetic target loci in epigenetic mutants with corresponding epigenetic changes in response to the stress effect. An example for this strategy is the discovery of epigenetic target loci that are activated in response to bacterial pathogens⁷⁰. Indications for a role for DNA methylation in biotic stress responses came from infection studies of methylation mutants *met1-3* and *ddc* (*drm1-2 drm2-2 cmt3-11*), which showed enhanced resistance to pathogenic and avirulent strains of *Pseudomonas syringae*. A screen for DMRs in wild-type plants, in response to bacterial infection, identified methylation changes at DMRs that correlated with activation of pathogen response genes. While methylation differences were relatively modest due to the high background of unaffected tissue that was not involved in the local response to bacterial infection, they were significant to identify distinct target regions for pathogen-induced DMRs. These mainly comprised changes in CG and CHH marks in intergenic regions and at 5' and 3' boundaries of protein-coding genes. Infections with virulent and avirulent strains induced similar changes at CG and CHG sites but different changes at CHH sites, which suggests that certain non-symmetrical methylation marks are modified in a stress-specific way. Hypomethylation at non-genic regions correlated with a moderate increase in transcript abundance of proximal genes, while transcript levels were more strongly increased for genes with hypomethylated coding regions. Genes affected by hypomethylation in the wild type after infection were also misregulated in *met1-3* and *ddc* mutants, which implies

that all three methyltransferases were involved in their transcriptional control⁷⁰. Various biotic⁷¹ and abiotic stress conditions⁷² have now been shown to correlate with changes in DNA methylation profiles. We still, however, lack clear evidence for a model case demonstrating that a stress-specific epigenetic modification is transmitted to subsequent generations, improving the progeny's capability to cope with the relevant stress⁷³. Some reports demonstrate heritable changes in DNA methylation at distinct loci in response to stress but do not show the relevance of these loci to stress tolerance⁷⁴⁻⁷⁵. Others detect a correlation between stress conditions and overall or tissue-specific methylation changes in putative stress response genes but do not report on the heritability of these changes⁷⁶⁻⁷⁷. Factors that make it difficult to assess the relevance of defined epigenetic changes in stress adaptation are the lack of control over the combined effects of multiple stress conditions a population has been exposed to and the high level of epigenetic variability in populations⁷⁸⁻⁸⁰. It is also unclear if epigenetic changes at distinct loci are the direct consequence of changing environmental conditions or if they are the secondary consequences of other stress-induced changes. In this context, it is worth noting that certain environmental stress conditions alter the expression levels of epigenetic regulators. The Geminivirus Rep protein, for example, reduces transcript levels of the *NbMET1* and *NbCMT3* methyltransferase genes in *Nicotiana benthamiana*⁸¹, and in *Arabidopsis*, *MET1* and *DDM1* transcript levels are down-regulated in response to biotic stress or salicylic acid⁷⁰, and various stress conditions increase transcript levels of histone deacetylases HDA6⁸² and HDA19⁸³. At least for certain loci that are sensitive to heritable epigenetic variation in response to environmental conditions, the local concentration of regulatory factors may therefore mediate environmental influences on epigenetic patterns. Environmental effects that alter the concentration of DNA methyltransferases, their interacting histone modifiers, or potentially their regulatory siRNA or transcripts⁸⁴⁻⁸⁵, may induce epigenetic changes at loci that are sensitive to quantitative changes of key regulators of methylation. Even transient exposure to stress conditions may add to epigenetic diversity if it influences efficiency and fidelity of epigenetic maintenance.

TRANSPOSABLE ELEMENTS: MEDIATORS OF EPIGENETIC RESPONSE

Transposable elements (TEs) and their derivatives, which make up more than half of the DNA in many species, play a prominent role in the epigenetic regulation of adjacent genes, and in the transmission of epigenetic memory effects due to the conversion of epigenetic states in response to environmental change⁸⁶⁻⁸⁸. TEs are controlled by different, frequently interacting epigenetic pathways that determine the stability and fidelity of their transcriptional repression, activation, and re-setting⁸⁹⁻⁹⁰. TEs can be activated by stress conditions leading to transient⁹¹, cell-specific⁹², or widespread⁷⁰ expression. Activation of TEs can alter expression of adjacent genes and of genes adjacent to new integration sites, into which new TE copies have transposed. Environmental conditions influence the activity of TEs if these contain specific stress response elements, and they influence the activation of TEs if they

change their epigenetic state⁹³⁻⁹⁴. Examples of stress-responsive TEs that insert into genic regions are *mPing*, a miniature inverted-repeat rice TE, and the *Arabidopsis* *ONSEN* retroelement. Amplified copies of *mPing*, which are produced after cold and salt stress, preferentially insert into 5' regions of genes, avoiding potential mutagenic damage via insertion into exons⁹⁵. *ONSEN* has acquired a heat-responsive element that regulates its activation⁹⁶ and that induces heat responsiveness in genes adjacent to its new insertion sites⁹⁷.

HOW USEFUL IS AN EPIGENETIC STRESS MEMORY?

The responsiveness of DNA methylation patterns to environmental stress⁹⁸ has been suggested to act as a molecular switch for evolutionary adaptation of plants to environmental change⁷⁴. In many cases, however, the continuous activity of stress-responsive genes will be undesirable due to secondary effects or the associated energy burden. This may make it advantageous for stress response pathways with secondary effects to remain active only for the duration of the inducing stress. Under this concept, epigenetic changes should be more useful if they did not cause permanent expression of target genes but rather they enabled the gene to respond more quickly and efficiently to frequently re-occurring stress conditions. To detect these kinds of epigenetic changes, we would face the much harder task of searching for changes in transcriptional competence and/ or response time to secondary challenges, not for changes in expression levels. Under continuous stress conditions, it may be advantageous if epigenetic changes lead to continuous activity of stress response genes that were previously only temporarily active. A potential example where durable changes in environmental conditions could have caused continuous activation of stress response genes may be mangrove populations that grow in close vicinity in riverside or salt marsh locations, respectively. The two populations differ more significantly in their methylation patterns than in DNA sequence. Plants in the salt marsh population, which display shrub-like phenotypes, have a lower level of methylation diversity than the tree-like plants in the riverside population⁹⁹. This may reflect a loss of epigenetic flexibility in response to permanent adaptation to salt stress. If this assumption was correct, one would expect to identify active genes in salt marsh populations that are associated with variable methylation patterns in riverside populations, and that are responsible both for improved salt tolerance and changes in plant architecture. While heritable epigenetic changes may be advantageous to adapt to continuous changes in environmental conditions, a transmission of any stress-induced epigenetic state would probably compromise plant growth and development. Plants have therefore developed several layers of control mechanisms that revert activated epi-alleles to their silent states. Heritability and transmission efficiency of epigenetic patterns are target specific and dependent on different epigenetic functions. The siRNA pathway plays an important role in restricting retrotransposition triggered by environmental stress. The heat-stress-activated

copia-type *ONSEN* retrotransposon is silenced in the next generation⁹⁷ but remains active in plants with compromised siRNA biogenesis.

Hypomethylation patterns of RdDM-dependent TEs and their derivatives are faithfully restored within a few generations¹⁰⁰, while other hypomethylation patterns are stably retained over at least eight generations⁹³. DDM1 and Morpheus'Molecule1 (MOM1) have recently been shown to act redundantly to restore silencing of some loci that are activated by heat stress¹⁰¹. This does, however, only affect ~10% of all stress-activated genes, which suggests the presence of one or several other resetting mechanisms that prevent trans-generational transmission of epigenetic changes. Current models and discussions for plants are dominated by the RdDM pathway, and many publications exclusively refer to DNA methylation being established by the guiding function of small RNAs that are generated and transported by RdDM pathway components. While, at least for *A. thaliana*, it is certainly correct that DNA methylation of most genomic regions is controlled by the RdDM pathway, we should not ignore the presence of RdDM-independent DNA methylation targets^{68, 90, 102-105}. Methylation at some RdDM independent target loci requires specific epigenetic functions, including HDA6, DDM1, or MET1. These may act as mediators of environmental change if certain stress conditions influence their steady-state levels and if this affects maintenance and stability of their methylation targets.

CONCLUSION

The velocity of future climate change is commonly viewed as necessitating rapid plant movements as natural selection cannot operate fast enough to generate novel beneficial gene combinations. However, the range of origins of epigenetic variation could provide phenotypic variation that would buffer against all but the most extreme climatic events. Clonal plants will continue to be an important component of ecosystems because of the attributes that they offer under heterogeneous environments, including rapid vegetative growth and multiplication, resource sharing and niche specialization among connected individuals. With our improved understanding of epigenetic systems and their mode of transmission among clonal copies and across sexual generations, we are uncovering only the superficial skin of a layer of complexity that drives phenotypic responses to the environment. The epigenome is likely to be particularly important in biological systems that lack genetic recombination, and under environmental changes, when the velocity of change exceeds the adaptation possible through natural selection. The added phenotypic diversity offered through epigenomic change should provide the buffer against environmental change that will permit more stable genetic systems to evolve.

CONFLICT OF INTEREST

Conflict of interest declared none.

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