Review Article **Molecular Biology**

International Journal of Pharma and Bio Sciences

ISSN
0975-6299

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

PARUL SAKSENA*[1], ASHA AGARWAL[2]

1-4Depatment of Zoology & Biotechnology, Sri Sathya Sai College for Women, Bhopal, M.P. India

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. Detoriating 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.***

PARUL SAKSENA*

1-4Depatment of Zoology & Biotechnology, Sri Sathya Sai College for Women, Bhopal, M.P. India

***Corresponding Author**

Received on: 20-03-2017
Revised and Accepted on : 20-05-2017
DOI: http://dx.doi.org/10.22376/ijpbs.2017.8.3.b230-238

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 recombination [1- 3,] that has consequences for clonal plants [4- 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 deglaciation [6]. The last major period of climatic change, during the Pleistocene, saw major species' distributional shifts [7] and changes in community structure as a result of unequal species' responses [8]. Ecologists have already monitored plant responses to recent climate change that include colonization, as altitudinal and latitudinal displacements occur [9] and adaptation, such as spring-time advances in phenological processes [10]. Currently, mortality through increased attacks by pests and diseases following drought stress is putting some ecosystems at risk [11- 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 *insitu* [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 high [14]. 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 front [15.] 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 limited [16-17].If clonal plants are limited in the irability 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 adaptation [18.] Nevertheless, clonal plants have persisted for thousands, or even millions of years during past environmental changes [19], and clonal plants have been successful in colonizing new habitats [20- 21], occupying broad geographic ranges [22-23] and have come to dominate some ecosystems [24]. 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 nature [25]. Even in extreme environments where clonal reproduction is expected to predominate [(26)], high genetic diversity can be maintained by episodes of sexual recruitment [27]. Second, clonal growth permits a range of advantageous ecological strategies including resource sharing [(28)], niche specialization [29-30] 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 selection [4,-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 reaction [32-33]. Recently, an increasing number of studies have shown that plastic responses can be mediated through epigenetic modifications [34- 35,] the most commonly studied being DNA methylation that results in changes in gene expression. These epigenetic marks may be stable across somatic generations [36-37] 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 tolerance [43]. 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 rainfall [44] 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 reviewed [45]. 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 stress [41]. 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 diversity [4]. 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 reproduction [26]. 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 drought [46-47]. Although some sexual reproductive systems are fire-adapted, in a great many resprouting systems, clonal reproduction provides the most rapid recovery after fire [48]. Another effect of drought-stress is the increased incidence of disease and pest outbreaks [11-12]. The search for heritable variation in disease resistance traits usually assumes variations in DNA sequence. However, recently [49] reported the epigenetic inheritance of response to the defense hormones, jasmonic acid and salicylicacid, 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 transcription [50], splicing [51], and polyadenylation [52], but they also affect DNA repair [53], recombination [54], and meiotic crossover in euchromatic regions [55]. 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 imprinting [56], floral symmetry [57], flowering time [58], pigmentation [59], fruit ripening [60], sex determination [61], and stomatal development [62-63]. Seed yield, determined by energy use efficiency, was the first quantitative trait associated with distinct, heritable DNA methylation patterns [64]. Flowering time and primary root length are two other complex quantitative traits linked to DNA methylation patterns at differentially methylated regions (DRMs). Methylation patters 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* populations [65].

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 regions [67]. 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 diversity [68]. 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 phenotype [69]. 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 pathogens [70]. 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 control [70]. Various biotic [71] and abiotic stress conditions [72] 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 stress [73]. 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 tolerance [74-75]. 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 changes [76-77]. 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 populations [78-80]. 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 acid [70], and various stress conditions increase transcript levels of histone deacetylases HDA6 [82] and HDA19 [83]. 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 transcripts [84-85], 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 change [86-88]. TEs are controlled by different, frequently interacting epigenetic pathways that determine the stability and fidelity of their transcriptional repression, activation, and re-setting [89-90]. TEs can be activated by stress conditions leading to transient [91], cell-specific [92], or widespread [70] 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 state [93-94]. Examples of stress-responsive TEs that insert into genic regions are *mPing*, a minature 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 exons [95]. *ONSEN* has acquired a heat-responsive element that regulates its activation [96] and that induces heat responsiveness in genes adjacent to its new insertion sites [97].

HOW USEFUL IS AN EPIGENETIC STRESS MEMORY?

The responsiveness of DNA methylation patterns to environmental stress [98] has been suggested to act as a molecular switch for evolutionary adaptation of plants to environmental change [74]. 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 population [99]. 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 generation [97] but remains active in plants with compromised siRNA biogenesis.

Hypomethylation patterns of RdDM-dependent TEs and their derivatives are faithfully restored within a few generations [100], while other hypomethylation patterns are stably retained over at least eight generations [93]. DDM1 and Morpheus'Molecule1 (MOM1) have recently been shown to act redundantly to restore silencing of some loci that are activated by heat stress [101]. 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.

REFERNCES

1. Richards EJ. Inherited epigenetic variation—revisiting soft inheritance. J Nat. Rev. Genet. 2006. 7: 395–401.
2. Jablonka E., and Raz G. Transgenerational epigenetic inheritance: prevalence, mechanisms, and implications for the study of heredity and evolution. Q. Rev. Biol. 2009. 84: 131–76.
3. Massicotte R and Angers B. General-purpose genotype or how epigenetics extend the flexibility of a genotype. J Genet. Res. Int . (2012). 2012: 317-175.
4. Verhoeven KJF and Preite V Epigenetic variation in asexually reproducing organisms. J Evolution. 2014. 68: 644–55.
5. Douhovnikoff V. and Dodd RS. Epigenetics: a potential mechanism for clonal plant success. J Plant Ecol. 2015. 216: 227–33.
6. Diffenbaugh NS. and Field CB. Changes in ecologically critical terrestrial climate conditions. J Science. 2013. 341: 486–92.
7. Hewitt GM. A climate for colonization. J Heredity. 2004. 92: 1–2.
8. Huntley B. Species distribution and environmental change: considerations from the site to the landscape scale, in Ecosystem Management: Questions for Science and Society, eds E. Maltby, M. Holdgate, M. Acreman, and A. Weir (Virginia Water: Royal Holloway Institute for Environmental Research) 1999. 115–30.
9. Parmesan C, and Yohe G. A globally coherent fingerprint of climate change impacts across natural systems. J Nature. 2003. 421: 37–42.
10. Hughes L. Biological consequences of global warming: is the signal already apparent? Trends Ecol. Evol. 2000. 15: 56–61.
11. Woods A, Coates KD and Hamann A. Is an unprecedented dothistroma needle blight epidemic related to climate change? J Bioscience. 2005. 55: 761–69.
12. Hicke JA, Logan JA, Powell J and Ojima DS. Changing temperatures influence suitability for modeled mountain pine beetle (Dendroctonus ponderosae) outbreaks in the western United States. J. Geophys. Res. Biogeosci. 2006. 111:G02019.
13. Aitken SN, Yeaman S, Holliday JA, Wang T and Curtis-McLane S. Adaptation, migration or extirpation: climate change outcomes for tree population. J Evol. Appl. 2008; 1: 95–111.
14. Urban MC, Tewksbury JJ and Sheldon KS. On a collision course: competition and dispersal differences create no-analogue communities and cause extinctions during climate change. J Proc. Biol. Sci. 2012. 279: 2072–80.
15. Kremer A, Ronce O, Robledo-Arnuncio J.J, Guillaume F, Bohrer, G and Nathan, R. Long-distance gene flow and adaptation of forest trees to rapid climate change. Ecol. Lett. 2012. 15: 378–92.
16. Winkler E, and Fischer M. The role of vegetative spread and seed dispersal for optimal life histories of clonal plants: a simulation study, J Evol. Ecol. 2002. 15: 281–301.
17. Winkler E, and Stöcklin J. Sexual and vegetative reproduction of Hieracium pilosella L. under competition and disturbance: a grid-based simulation model. J Ann. Bot. 2002. 89: 525–36.
18. Etterson JR and Shaw RG. Constraint to adaptive evolution in response to global warming. J. Science. 2001. 294: 151–54.
19. Neiman M, Meirmans, S, and Meirmans PG. What can asexual lineage age tell us about the maintenance of sex? Ann. N. Y. Acad. Sci. 2009. 1168: 185– 200.
20. Ahmad R, Liow PS, Spencer DF, and Jasieniuk M. Molecular evidence for a single genetic clone of invasive Arundo donax in the United States. J Aquat. Bot. 2008; 88: 113–20.
21. Zhang YY, Fischer M, Colot V and Bossdorf O. Epigenetic variation creates potential for evolution of plant phenotypic plasticity. J New Phytol. 2013. 197: 314–22.
22. Geng Y, Pan X, Xu C, Zhang WJ, Li B and Chen, J. K. Phenotypic plasticity rather than locally adapted ecotypes allows the invasive alligator weed to colonize a wide range of habitats. Biol. Invasions. 2007. 9: 245–56.
23. Ganie AH, Reshi ZA, Wafai BA and Puijalon S. Clonal growth architecture and spatial dynamics of 10 species of the genus potamogeton across different habitats in Kashmir Valley, India. J Hydrobiologia. 2016. 767: 289–99.
24. Hollingsworth ML and Bailey JP. Evidence for massive clonal growthin the invasive weed Fallopia japonica (Japanese Knotweed). Bot. J. Linn. Soc. 2000. 133: 463–72.
25. Savidan Y. Apomixis: genetics and breeding. J Plant Breed. Rev. 2010.18: 13–86.
26. Eckert CG. The loss of sex in clonal plants. Evol. Ecol. 2002. 15: 501–20.
27. de Witte, L. C., and Stöcklin, J. Longevity of clonal plants: why it matters and how to measure it. J Ann. Bot. 2010. 106: 859–70.
28. Alpert P. Water sharing among ramets in a desert population of Distichlis spicata (Poaceae). Am. J. Bot. 1990; 77: 1648–51.
29. Gómez S, and Stuefer JF. Members only: induced systemic resistanceto herbivory in a clonal plant network. J Oecologia. 2006. 147: 461–68.
30. Louâpre P, Bittebière AK, Clément B, Pierre JS and Mony C. How past and present influence the foraging of clonal plants? PLoS ONE. 2012. 7:e38288.
31. Parker IM, Rodriguez J and Loik ME. An evolutionary approach to understanding the biology of invasions: local adaptation and general-purpose genotypes in the weed Verbascum thapsus. J Conserv. Biol. 2003.17: 59–72.
32. West-Eberhard MJ. Phenotypic accommodation: adaptive innovation due to developmental plasticity. J. Exp. Zool. 2005. B 304, 610–18.
33. Lande R. Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation. J. Evol. Biol. 2009. 22: 1435–46.
34. Jaenisch R and Bird A. Epigenetic regulation of gene expression: how the genome integrates intrinsic and environmental signals. J Nat. Genet. 2003. 33: 245–54.

35. Richards CL, Bossdorf O, Muth NZ, Gurevitch J and Pigliucci M. Jack of all trades, master of some? On the role of phenotypic plasticity in plant invasions. Ecol. Lett. 2006. 9: 981–93.
36. Bossdorf O, Richards CL. and Pigliucci M. Epigenetics for ecologists. Ecol. Lett. 2008. 11: 106–115.
37. Castonguay E. and Angers B. The key role of epigenetics in the persistence of asexual lineages. Genet. Res. Int. 2012; 2012: 534289.
38. Richards CL, Schrey AW., and Pigliucci M. Invasion of diverse habitats by few Japanese knotweed genotypes is correlated with epigenetic differentiation. Ecol. Lett. 2012. 15: 1016–25.
39. Boyko A, Golubov A, Bilichak, A and Kovalchuk I. Chlorine ions but not sodium ions alter genome stability of Arabidopsis thaliana. J. Plant Cell Physiol. 2010; 51: 1066–78.
40. Nicotra AB, Atkin OK, Bonser SP, Davidson AM, Finnegan EJ and Mathesius U. Plant phenotypic plasticity in a changing climate. J Trends Plant Sci. 2010.15: 684–92.
41. Verhoeven KJF, Jansen JJ, Van Dijk PJ and Biere A. Stress induced DNA methylation changes and their heritability in asexual dandelions. J New Phytol. (2010). 185: 1108–18.
42. Richards CL, Walls RL, Bailey JP, Parameswaran R, George T and Pigliucci M. Plasticity in salt tolerance traits allows for invasion of novel habitat by Japanese knotweed s. l. (Fallopia japonica and F-bohemica, Polygonaceae). Am. J. Bot. 2008. 95: 931–42
43. Feder MA, Benet AF and Huey RB. Evolutionary physiology. Annu. Rev. Ecol. Syst. 2000. 31: 315–341.
44. Gaines SD and Denny MW. The largest, smallest, highest, lowest, longest, and shortest: extremes in ecology. J Ecology. 1993. 74: 1677–92.
45. Chinnusamy, V., and Zhu, J. K. Epigenetic regulation of stress responses in plants. J Curr. Opin. Plant Biol. 2009. 12: 133–39.
46. Westerling AL, Turner MG, Smithwick EAH, Romme WH and Ryan MG. Continued warming could transform Greater Yellowstone fire regimes by mid-21st century. Proc. Natl. Acad. Sci. U.S.A. 2011. 108: 13165–70.
47. Brando PM, Balch JK, Nepstad DC, Morton DC, Putz FE and Coe MT. Abrupt increases in Amazonian tree mortality due to Clonal Growth and Epigenetic Variation drought–fire interactions. Proc. Natl. Acad. Sci. U.S.A. 2014; 111: 6347–52.
48. Bond, WJ and Midgley JM. The persistence niche: ecology of sprouting in woody plants. Trends Ecol. Evol. 2001; 16: 45–51.
49. Latzel V, Zhang Y, Moritz, KK, Fischer M., and Bossdorf, O. Epigenetic variation in plant responses to defence hormones. J Ann. Bot. 2012. 110: 1423–28.
50. Huettel B, Kanno T, Daxinger L, Aufsatz W, Matzke AJM and Matzke M. Endogenous targets of RNA-directed DNA methylation and Pol IV in Arabidopsis. EMBO Journal. 2006. 25: 2828–36.
51. Regulski M, Lu Z and Kendall J. The maize methylome influences mRNA splice sites and reveals widespread paramutation-like switches guided by small RNA. J Genome Research. 2013. 23: 1651–62.
52. Tsuchiya T and Eulgem T. An alternative polyadenylation mechanism coopted to the Arabidopsis RPP7 gene through intronic retrotransposon domestication. Proceedings of the National Academy of Sciences, USA. 2013. 110, E3535–43.
53. Yao Y, Bilichak A, Golubov A and Kovalchuk I. ddm1 plants are sensitive to methyl methane sulfonate and NaCl stresses and are deficient in DNA repair. J Plant Cell Reports. 2012. 31: 1549–61.
54. Mirouze M, Lieberman-Lazarovich M, Aversano R, Bucher E, Nicolet J, Reinders J and Paszkowski J. Loss of DNA methylation affects the recombination landscape in Arabidopsis. Proceedings of the National Academy of Sciences, USA. 2012. 109: 5880–85.
55. Melamed-Bessudo C and Levy AA. Deficiency in DNA methylation increases meiotic crossover rates in euchromatic but not in heterochromatic regions in Arabidopsis. Proceedings of the National Academy of Sciences, USA. 2012. 109: E981–8.
56. Huh JH, Bauer MJ, Hsieh TF and Fischer RL. Cellular programming of plant gene imprinting. J Cell. 2008. 132: 735–44.
57. Cubas P, Vincent C and Coen E. An epigenetic mutation responsible for natural variation in floral symmetry. J. Nature. 1999. 401: 157–61.
58. Soppe WJJ, Jacobsen SE, Alonso-Blanco C, Jackson JP, Kakutani T, Koornneef M and Peeters AJM. The late flowering phenotype of fwa mutants is caused by gain-of-function epigenetic alleles of a homeodomain gene. J Molecular Cell. 2000. 6: 791-802
59. Stam M, Belele C, Dorweiler JE and Chandler VL. Differential chromatin structure within a tandem array 100kb upstream of the maize b1 locus is associated with paramutation. J Genes and Development. 2002. 16: 1906–18.
60. Manning K, Tor M, Poole M, Hong Y, Thompson AJ, King GJ, Giovannoni JJ and Seymour GB. A naturally occurring epigenetic mutation in a gene encoding an SBP-box transcription factor inhibits tomato fruit ripening. J Nature Genetics. 2006. 38: 948–52.
61. Martin A, Troadec C, Boualem A, Rajab M, Fernandez R, Morin H, Pitrat M, Dogimont C and Bendahmane A. A transposon-induced epigenetic change leads to sex determination in melon. J Nature. 2009. 461: 1135–38.
62. Tricker PJ, Gibbings JG, Rodríguez López CM, Hadley P and Wilkinson MJ. Low relative humidity triggers RNA-directed de novo DNA methylation and suppression of genes controlling stomatal development. Journal of Experimental Botany. 2012. 63: 3799–813.
63. Yamamuro C, Miki D, Zheng Z, Ma J, Wang J, Yang Z, Dong J and Zhu JK. Overproduction of stomatal lineage cells in Arabidopsis mutants defective in active DNA demethylation. Nature Communications. 2014. 5, 4062.
64. Hauben M, Haesendonckx B and Standaert E. Energy use efficiency is characterized by an

epigenetic component that can be directed through artificial selection to increase yield. Proceedings of the National Academy of Sciences, USA. 2009. 106: 20109–14.
65. Cortijo S, Wardenaar R and Colomé-Tatché M. Mapping the epigenetic basis of complex traits. J. Science. 2014. 343: 1145–48.
66. Popova OV, Dinh HQ, Aufsatz W and Jonak C. The RdDM pathway is required for basal heat tolerance in Arabidopsis. J Molecular Plant. 2013. 6: 396–410.
67. Mathieu O, Reinders J, Caikovski M, Smathajitt C and Paszkowski J. Transgenerational stability of the Arabidopsis epigenome is coordinated by CG methylation. J Cell. 2007. 130: 851–62.
68. Havecker ER, Wallbridge LM, Fedito P, Hardcastle TJ and Baulcombe DC. Metastable differentially methylated regions within Arabidopsis inbred populations are associated with modified expression of non-coding transcripts. PLoS One. 2012. 7: e45242.
69. Yi H and Richards EJ. Gene duplication and hypermutation of the pathogen resistance gene SNC1 in the Arabidopsis bal variant. J Genetics. 2009. 183: 1227–34.
70. Dowen RH, Pelizzola M, Schmitz RJ, Lister R, Dowen JM, Nery JR, Dixon JE and Ecker JR. Widespread dynamic DNA methylation in response to biotic stress. Proceedings of the National Academy of Sciences, USA. 2012. 109: E2183–91.
71. Boyko A, Kathiria P, Zemp FJ, Yao Y, Pogribny I and Kovalchuk I. Transgenerational changes in the genome stability and methylation in pathogen-infected plants: (virus-induced plant genome instability). J Nucleic Acids Research. 2007; 35: 1714–25
72. Kovarik A, Koukalová B, Bezdek M and Opatrn Z. Hypermethylation of tobacco heterochromatic loci in response to osmotic stress. J Theoretical and Applied Genetics. 1997;95: 301–06.
73. Pecinka A, Mittelsten and Scheid O. Stress-induced chromatin changes: a critical view on their heritability. J Plant and Cell Physiology. 2012. 53: 801–08.
74. Kou HP, Li Y, Song XX, Ou XF, Xing SC, Ma J, Von Wettstein D and Liu B. Heritable alteration in DNA methylation induced by nitrogen-deficiency stress accompanies enhanced tolerance by progenies to the stress in rice (Oryza sativaL.). Journal of Plant Physiology. 2011; 168:1685–93.
75. Zheng X, Chen L, Li M, Lou Q, Xia H, Wang P, Li T, Liu H and Luo L. Transgenerational variations in DNA methylation induced by drought stress in two rice varieties with distinguished difference to drought resistance. PLoS One. 2013; 8: e80253.
76. Steward N, Ito M, Yamaguchi Y, Koizumi N and Sano H. Periodic DNA methylation in maize nucleosomes and demethylation by environmental stress. Journal of Biological Chemistry. 2002;277: 37741–46.
77. González RM, Ricardi MM and Iusem ND. Epigenetic marks in an adaptive water stress-responsive gene in tomato roots under normal and drought conditions. J Epigenetics. 2013; 8: 864–72.
78. Woo H and Richards E. Natural variation in DNA methylation in ribosomal RNA genes of Arabidopsis thaliana. BMC Plant Biology. 2008; 8: 92.
79. Becker C, Hagmann J, Muller J, Koenig D, Stegle O, Borgwardt K and Weigel D. Spontaneous epigenetic variation in the Arabidopsis thaliana methylome. J Nature. 2011; 480: 245–49.
80. Groszmann M, Greaves IK, Albertyn ZI, Scofield GN, Peacock WJ and Dennis ES. Changes in 24-nt siRNA levels in Arabidopsis hybrids suggest an epigenetic contribution to hybrid vigor. Proceedings of the National Academy of Sciences, USA. 2011; 108: 2617–622.
81. Rodríguez-Negrete E, Lozano-Durán R, Piedra-Aguilera A, Cruzado L, Bejarano ER and Castillo AG. Geminivirus Rep protein interferes with the plant DNA methylation machinery and suppresses transcriptional gene silencing. J New Phytologist. 2013; 199: 464–75.
82. To TK, Nakaminami K, Kim J-M, Morosawa T, Ishida J, Tanaka M, Yokoyama S, Shinozaki K and Seki M. Arabidopsis HDA6 is required for freezing tolerance. J Biochemical and Biophysical Research Communications. 2011; 406: 414–19.
83. Zhou C, Zhang L, Duan J, Miki B and Wu K. HISTONE DEACETYLASE19 is involved in jasmonic acid and ethylene signaling of pathogen response in Arabidopsis. J The Plant Cell. 2005;17: 1196–204
84. Lakhotia SC. Long non-coding RNAs coordinate cellular responses to stress. Wiley Interdisciplinary Reviews. 2012;3: 779–96.
85. Di Ruscio A, Ebralidze AK and Benoukraf T. DNMT1-interacting RNAs block gene-specific DNA methylation. J Nature. 2013; 503: 371–76.
86. McClintock B. The significance of responses of the genome to challenge. J Science. 1984. 226: 792–801.
87. Mirouze M and Paszkowski J. Epigenetic contribution to stress adaptation in plants. J Current Opinion in Plant Biology. 2011; 14: 267–74.
88. Fedoroff NV. Transposable elements, epigenetics, and genome evolution. J Science. 2012; 338: 758–67.
89. Lippman Z, May B, Yordan C, Singer T and Martienssen R. Distinct mechanisms determine transposon inheritance and methylation via small interfering RNA and histone modification. PLoS Biology. 2003; 1: e67.
90. Zemach A, Kim MY, Hsieh P-H, Coleman-Derr D, Eshed-Williams L, Thao K, Harmer Stacey L and Zilberman D. The Arabidopsis nucleosome remodeler DDM1 allows DNA methyltransferases to access H1-containing heterochromatin. J Cell. 2013; 153: 193–205.
91. Tittel-Elmer M, Bucher E, Broger L, Mathieu O, Paszkowski J and Vaillant I. Stress-induced activation of heterochromatic transcription. PLoS Genetics. 2010; 6: e1001175.
92. Matsunaga W, Kobayashi A, Kato A and Ito H. The effects of heat induction and the siRNA biogenesis pathway on the transgenerational transposition of ONSEN, a copia-like

retrotransposon in Arabidopsis thaliana. J Plant and Cell Physiology. 2012; 53: 824–33.
93. Johannes F, Porcher E and Teixeira FK. Assessing the impact of transgenerational epigenetic variation on complex traits. PLoS Genetics. 2009;5: e1000530.
94. McCue AD, Nuthikattu S, Reeder SH and Slotkin RK. Gene expression and stress response mediated by the epigenetic regulation of a transposable element small RNA. PLoS Genetics. 201;8: e1002474.
95. Naito K, Zhang F, Tsukiyama T, Saito H, Hancock CN, Richardson AO, Okumoto Y, Tanisaka T and Wessler SR. Unexpected consequences of a sudden and massive transposon amplification on rice gene expression. J. 2009. Nature 461: 1130–34.
96. Cavrak VV, Lettner N, Jamge S, Kosarewicz A, Bayer LM, Mittelsten and Scheid O. How a retrotransposon exploits the plant's heat stress response for its activation. PLoS Genetics. 201;. 10: e1004115.
97. Ito H, Gaubert H, Bucher E, Mirouze M, Vaillant I and Paszkowski J. An siRNA pathway prevents transgenerational retrotransposition in plants subjected to stress. J Nature. 2011; 472: 115–19.
98. Finnegan EJ. Epialleles—a source of random variation in times of stress. Current Opinion in Plant Biology. 200;. 5: 101–06.
99. Lira-Medeiros CF, Parisod C, Fernandes RA, Mata CS, Cardoso MA and Ferreira PCG. Epigenetic variation in mangrove plants occurring in contrasting natural environment. PLoS One 201;. 5: e10326.
100. Teixeira FK, Heredia F and Sarazin A. A role for RNAi in the selective correction of DNA methylation defects. J Science. 200;. 323: 1600–04.
101. Iwasaki M and Paszkowski J. Identification of genes preventing transgenerational transmission of stress-induced epigenetic states. Proceedings of the National Academy of Sciences, USA 2014;111: 8547–52.
102. Singh A, Zubko E and Meyer P. Co-operative activity of DNA methyltransferases for maintenance of symmetrical and non-symmetrical cytosine methylation in Arabidopsis thaliana. J The Plant Journal. 2008. 56: 814–23.
103. Sasaki T, Kobayashi A, Saze H and Kakutani T. RNAi-independent de novo DNA methylation revealed in Arabidopsis mutants of chromatin remodeling gene DDM1. J The Plant Journal. 2012. 70: 750–58.
104. Gentry M and Meyer P. An 11bp region with stem formation potential is essential for de novoDNA methylation of the RPS element. PLoS One. 2013; 8: e63652.
105. Watson M, Hawkes E and Meyer P. Transmission of epi-alleles with MET1- dependent dense methylation in Arabidopsis thaliana. PLoS One. 2014; 9: e105338.

Reviewers of this article

Prof Vinoy kumar Shrivastava

Head and Ex Dean,
Dept of Bioscience,Barkatullah University,Bhopal,madhya Pradesh,India

Prof.Dr.Prapurna Chandra Rao

Assistant Proffessor, KLE University, Belgaum, Karnataka

Prof.Dr.K.Suriaprabha

Asst. Editor , International Journal of Pharma and Bio sciences.

Prof.P.Muthuprasanna

Managing Editor , International Journal of Pharma and Bio sciences.

We sincerely thank the above reviewers for peer reviewing the manuscript