NRC Research Press

MINIREVIEW

Does variable epigenetic inheritance fuel plant evolution?

Mark A.A. Minow and Joseph Colasanti

Abstract: Epigenetic changes influence gene expression and contribute to the modulation of biological processes in response to the environment. Transgenerational epigenetic changes in gene expression have been described in many eukaryotes. However, plants appear to have a stronger propensity for inheriting novel epialleles. This mini-review discusses how plant traits, such as meristematic growth, totipotency, and incomplete epigenetic erasure in gametes promote epiallele inheritance. Additionally, we highlight how plant biology may be inherently tailored to reap the benefits of epigenetic metastability. Importantly, environmentally triggered small RNA expression and subsequent epigenetic changes may allow immobile plants to adapt themselves, and possibly their progeny, to thrive in local environments. The change of epigenetic states through the passage of generations has ramifications for evolution in the natural and agricultural world. In populations containing little genetic diversity, such as elite crop germplasm or habitually self-reproducing species, epigenetics may provide an important source of heritable phenotypic variation. Basic understanding of the processes that direct epigenetic shifts in the genome may allow for breeding or bioengineering for improved plant traits that do not require changes to DNA sequence.

Key words: small RNA, meristems, plant development, epigenetics, evolution.

Résumé : Les modifications épigénétiques influencent l'expression génique et contribuent à moduler les processus biologiques en réponse à l'environnement. Des changements épigénétiques trans-générationnels de l'expression génique ont été décrits chez plusieurs eucaryotes. Cependant, les plantes semblent avoir une propension plus grande pour la transmission d'épiallèles inédits. Dans cette mini-synthèse, les auteurs discutent comment certaines propriétés des plantes, telles que la croissance des méristèmes, la totipotence et l'effacement épigénétique incomplète chez les gamètes, contribuent la transmission des épiallèles. De plus, les auteurs soulignent de quelle manière la biologie des plantes est adaptée pour récolter les bénéfices d'une méta-stabilité épigénétique. De manière importante, l'expression induite par l'environnement de petits ARN et les changements épigénétiques subséquents peuvent permettre aux plantes, par ailleurs immobiles, de s'adapter elles-mêmes, ainsi que possiblement leur progéniture, pour prospérer dans des environnements locaux. Le changement d'états épigénétiques au cours des générations a des conséquences en matière d'évolution tant dans le contexte naturel qu'agricole. Chez des populations contenant peu de diversité génétique, telles que les variétés élites ou des espèces à reproduction autogame, l'épigénétique procure possiblement une source importante de variation phénotypique héritable. Une compréhension des processus qui mènent à des changements épigénétiques dans le génome pourrait permettre une sélection ou une ingénierie de caractères améliorés qui ne nécessitent aucun changement à la séquence d'ADN. [Traduit par la Rédaction]

Mots-clés : petits ARN, méristèmes, développement de la plante, épigénétique, évolution.

Introduction

Plants differ from animals in several fundamental ways. In addition to being typically autotrophic and immobile, plants react to their environment through developmental plasticity. Plant cells are totipotent and, unlike most other eukaryotes, plant development continues after embryogenesis and throughout the life of the individual. This is exemplified in plants through developmental flexibility, where cells arise from stem cell precursors, become part of the soma, but maintain the potential to dedifferentiate back into stem cells. This impermanence in cell fate requires reprogramming of gene expression, which in part relies upon re-writing epigenetic states in somatic cells. Although many changes are temporary during development, some epigenetic changes persist transgenerationally across passage through gametes. This paper focuses on how perpetual development and

Received 8 November 2019. Accepted 10 February 2020.

M.A.A. Minow and J. Colasanti.* Department of Molecular and Cellular Biology, University of Guelph, 50 Stone Road East, Guelph, ON N1G 2W1, Canada.

Corresponding author: Joseph Colasanti (email: jcolasan@uoguelph.ca).

*Joseph Colasanti currently serves as an Editor; peer review and editorial decisions regarding this manuscript were handled by Jianping Xu.

other plant traits relate to the inheritance of variable epigenetic allele states, and how epigenetics may drive plant evolution.

Epigenetics was originally used to explain how cells within the same organism with identical genomes can produce incredibly diverse biological structures (Waddington 1957). The term epigenetics was repurposed later to denote heritable changes in gene expression that do not arise from changes in DNA sequence (Holliday 1987). Molecular studies have revealed that the same epigenetic biochemical marks facilitate both development and transgenerational changes, although it remains unclear how development changes caused by epigenetic changes overlap with classical forms of gene regulation. In eukaryotes, most epigenetic marks involve alterations to histone tail modifications, DNA methylation, and small RNA (sRNA) expression (Bender 2004). Throughout the genome these epigenetic marks interact in myriad ways. Generally, epigenetic marks promote the recruitment of other marks promoting the same state; i.e., a repressive mark tends to promote the accumulation of other repressive marks, and an active mark tends to promote other active epigenetic changes (Krogan et al. 2003; Henikoff and Shilatifard 2011; Sadeghi et al. 2015). This provides a level of redundancy and stability for any given epigenetic state.

However, epigenetic states are somewhat unstable and can change over time. If a new state persists, it can form a new epiallele which is an allele of distinct function but equivalent DNA sequence to related alleles. The level of epigenetic stability at a locus is influenced by several factors, including exogenous stresses (Kashkush et al. 2003; Wibowo et al. 2016). Genetics also contributes to epigenetic instability. For example, DNA sequences close to a gene, notably transposon insertions, often promote epigenetic instability (Kinoshita et al. 2007; Saze and Kakutani 2007; Fujimoto et al. 2008; Hollister and Gaut 2009). Additionally, in plants, unlinked regions of the genome can act in *trans* to alter epigenetic states. *Trans* interactions between homologous alleles (*trans*-homolog interactions) can result in heritable changes in allele expression (Hollick 2017). Accordingly, sequences with partial homology to a gene or its DNA regulatory elements can influence epigenetic changes and gene expression (Matzke et al. 1989; Van Blokland et al. 1994). Epigenetic *trans* regulation is often mediated by sRNA, which use homology to repress genes and transposons (Fultz et al. 2015; Bousios and Gaut 2016). This sRNA repression takes place at both the transcriptional level, via RNA-directed DNA methylation (RdDM), and at post-transcriptional level, through mRNA repression and cleavage (Borges and Martienssen 2015). sRNA-mediated transcriptional silencing interacts with histone tail modifying enzymes to establish and maintain repressive heterochromatin (Deleris et al. 2010; Enke et al. 2011; Greenberg et al. 2013). Likewise, heterochromatin-associated histone marks and DNA methylation recruit the RdDM machinery to help perpetuate silencing (Law et al. 2013; Johnson et al. 2014; Liu et al. 2014). Although both plants and animals have demonstrated inheritance of novel epigenetic states, plant genes seem particularly prone to epi-allelism (Quadrana and Colot 2016). Here we examine some of the possible mechanisms, consequences, and advantages of genomic epigenetic shifts within plant lineages.

Meristematic growth facilitates the accumulation of stable epimutants

The shoot apical meristem (SAM) produces the above ground vegetative and reproductive structures. Therefore, unlike mammals, plants do not set aside an embryonic germline and the next plant generation arises from the same set of stem cells that produces the somatic shoot tissues. Moreover, in many plants, somatic cells can dedifferentiate and form new shoot meristems in the course of normal development and in response to trauma. This provides a route for somatic events to be passed on to the next generation. Thus, because of the nature of meristematic growth, plants are predisposed to accumulate epigenetic mutations.

Epimutants typically are associated with changes in DNA (cytosine) methylation. Plants methylate DNA in three different cytosine contexts: CG, CHG, and CHH (where H is A, C, or T). DNA methylation is associated with a heterochromatic DNA environment that is less accessible to gene regulators. When DNA methylation occurs over a DNA regulatory element, (e.g., promoter, distal enhancer, splice sites) it is often associated with repression of that DNA regulatory element (Haring et al. 2010; Osabe et al. 2017; Philips et al. 2019; Zicola et al. 2019). However, plant DNA methylation frequently occurs over the coding sequences of expressed genes in a process known as gene body methylation. The function of gene body methylation remains enigmatic, as loss of the gene body methylation did not have an overt effect on gene expression (Bewick et al. 2016; Zilberman 2017). However, the unknown function of gene body methylation comes at the cost of elevated rates of mutation over methylated genes (Kiefer et al. 2019).

Plants do not undergo genome wide epigenetic erasure in germinal cells to the same extent as mammals, and plant DNA methylation patterns can persist in the next generation (Kawashima and Berger 2014). During male gametogenesis, lowered DNA methyltransferase activity coupled with cell divisions dilutes CHH cytosine methylation patterns without explicit erasure (Calarco et al. 2012). In tandem, unique sRNA driven epigenetic reprogramming occurs during gametogenesis and embryogenesis (Mosher et al. 2009; Slotkin et al. 2009; Ibarra et al. 2012; Vu et al. 2013; Martinez et al. 2018). Moreover, plants can specifically demethylate DNA through DNA glycosylation (Law and Jacobsen 2010).

However, directed DNA demethylation affects only a few genes and many methylation patterns are maintained across the passage of reproduction (Choi et al. 2002; Penterman et al. 2007; Quadrana and Colot 2016). The combination of meristematic growth and the lack of widespread epigenetic erasure in germ cells fosters the passage of somatic epimutations to the next generation.

An early example of plant epiallelism was observed at the *Zea mays* (maize) gene *Pericarp color1* (*P1*), which exemplifies features common to other epialleles (Das and Messing 1994). A functional *P1* maternal gene causes red colouration of the cob and kernels. An epimutation at the *P1* locus gave rise to a somatic sector that encompassed the developing inflorescence (Das and Messing 1994). This sector produced transgenerationally stable epialleles that exhibited tissue-specific loss of expression (with stochastic gain of expression late in corn kernel development). Loss of expression was due to increased DNA methylation of repeats at the 5′ and 3′ end of the gene. The stochastic re-pigmentation correlated to loss of DNA methylation later in development that reactivates expression in the kernel. Levels of methylation fluctuated through the passage of generations and somatic reversions to the unmethylated state occasionally produced fully pigmented kernels. The process of reversion to the previous epigenetic state is often observed in epialleles, and although the frequency varies, reversion is usually rare. Similarity in methylation patterns and *P1* transcript levels were highest between siblings and more different when compared with distant relatives (Das and Messing 1994). This supports the notion that epigenetic states "drift" from the parental state upon the passage of generations; that is, at any given locus, the next generation possesses an epigenetic state similar, but not identical, to the parental state. The *P1* epiallele exhibits conserved characteristics of plant epialleles (Jacobsen and Meyerowitz 1997; Cubas et al. 1999; Manning et al. 2006; Zhang et al. 2012). Specifically, genesis of an epiallele in a somatic lineage, drift in expression away from the parental state after reproduction, and occasional reversion to the ancestral allele expression state are frequent hallmarks of epigenetic instability.

Plant life strategies may benefit from epigenetic instability

Do epigenetically unstable alleles confer any benefit to an organism? Many selective advantages come at a tradeoff and are only advantageous under certain conditions. Unlike genetic mutations, epimutations can revert to a previous expression state (Catoni et al. 2017). Thus, a plant population can have both expressed and silenced epialleles. This instability allows individuals to express a beneficial phenotype, without completely altering the ancestral genetic state for possible expression in its progeny. The relative stability of these epigenetic changes allows for selection that favours a specific expression state. After reproduction, epigenetic drift creates diversity in progeny by producing a range of expression states distributed around the parental level (Das and Messing 1994). This drift from the parental epigenetic state in offspring is consistent with classical observations of the maize *R* locus after paramutation, which is a type of *trans* homolog interaction wherein an allelic interaction transgenerationally changes expression (Styles and Brink 1966). Styles and Brink (1966) observed that expression at the *R* locus was progressively influenced by different parental states in otherwise genetically identical individuals. Intriguingly, the influence of the parental epigenetic state on progeny epigenetics and expression has also been demonstrated in mice (Surani et al. 1990). Since drift in epigenetic state is relative to the pre-existing state, new epigenetic states will be close to that found in the previous reproductively successful generation. This slight epigenetic drift would produce a range of progeny phenotypes like that found in the parent.

Plant developmental plasticity in different environments is well documented and the processes controlling plant environmental response could also contribute to direct gradual epigenetic changes. There is evidence of histone changes influencing phytohormone and circadian rhythm pathways which, in turn, regulate many aspects of plant growth and development in response to the environment (Hao et al. 2016; Yamamuro et al. 2016; Chen and Mas 2019). Emerging evidence suggests that several of these histone modifying enzymes may directly or indirectly regulate DNA methyltransferases, possibly changing DNA methylation at important downstream gene regulators (Saze et al. 2008; Deleris et al. 2010; Liu et al. 2012; Stroud et al. 2013; Ng et al. 2014; Hristova et al. 2015; Zhang et al. 2015; Yu et al. 2017; Forestan et al. 2018). If this interaction elicits stable methylation changes, it could bridge environmental plasticity and transgenerationally stable phenotype adaptations, fostering a sort of "epigenetic assimilation". In this model, repeated exposure to a particular environmental stimulus would repeatedly recruit the same histone modifying enzymes to a response gene, repeatedly re-enforcing an open or closed chromatin state. If these repetitive chromatin remodelling events gradually change DNA methylation it could result in an epiallele with a more constitutive expression state.

Any new range of epiallele expression could undergo stabilizing selection that favours the parental expression state, or directional selection that favours a more extreme expression state. Transgenerational metastability has the potential to provide a continuous range of gene expression, thus creating continuous phenotypic variation (plasticity/polyphenism) without requiring a change in genotype (Cubas et al. 1999). This phenotypic flexibility may be of considerable value in genetically narrow populations. Many plants habitually self-fertilize, or reproduce clonally, so somatic epimutation could provide

an important source of functional diversity to supplant genetic changes. Indeed, the importance of epigenetic variation has been suggested by studies of asexual *Taraxacum officinale* (dandelion) (Verhoeven et al. 2010; Verhoeven and van Gurp 2012). Increased variation through epigenetic changes may also contribute to the process of bet hedging, wherein organisms produce offspring with variable phenotypes above and below a fitness optimum (Herman et al. 2014). This increased variability, although disadvantageous in the current environment, leaves the progeny poised to exploit changes in the environment and is especially important in unstable ecosystems. Additionally, plants often produce high numbers of potential offspring. This high fecundity, combined with strong seedling selection, may allow increased tolerance to deleterious epigenetic variants in offspring. Maladaptive epigenetic changes in a few seeds may not constrain individual reproduction due to the volume of seed produced and the paucity of individuals that reach maturity.

Seed production from many meristems increases the diversity of heritable epigenetic states

Unlike metazoans, plants can have multiple cell lineages that give rise to progeny since multiple shoot meristems arising from the soma give rise to flowers and seeds. The ability to produce offspring from divergent somatic lineages further compounds the advantages provided by epigenetic instability by providing a larger pool of cells in which variants can arise and be passed to offspring. Developmentally late epigenetic events are unlikely to affect all seed producing cell lineages, as the resulting clonal sectors will not encompass all stem cell niches. Therefore, any "epigenetic experimentation", or slight alterations to the ancestral epigenetic state, is expected to be variable even within the same parent.

The production of seed from different meristems relates to the genetic mosaicism hypothesis, which posits that different mutations accumulate in separate meristems, resulting in a mosaic of diverse genetic chimeras within the same plant. The genetic mosaicism hypothesis was originally developed to reconcile how long-lived perennials can compete in a molecular arms race with their short-lived pathogens (Whitham 1981; Gill 1986). In the decades since this idea was proposed there has been little sustained research into the genetic mosaicism hypothesis (Simberloff and Leppanen 2019). However, some evidence exists to support the genetic mosaicism hypothesis in both polycarpic (plants with bodies formed from repeating physiological units) and clonal monocarpic (plants with bodies of one physiological unit) plants (Gill et al. 1995). Genetic differences have been observed in distal somatic tissues in one *Quercus robur* (oak) tree and several populations of *Thuja plicata* (western cedar) (O'Connell and Ritland 2004; Schmid-Siegert et al. 2017). More genome-wide studies, ideally conducted on several individuals, are needed to establish the frequency of somatic (epi)mutation and if this frequency is related to stressful growth conditions.

Intra-organism competition between perennial buds for shared resources like water, nutrients, and light (energy) weeds out maladaptive somatic variants before reproduction. Likewise, competition between different shoot branches promotes any changes that benefit shoot growth. For example, somatic selection favours branches resistant to biotic stresses (Gill 1986; Gill et al. 1995). Indeed, recent studies of polycarpic *Eucalyptus melliodora* and *Tsuga canadensis* (eastern hemlock) have supported a role for mosaicism in providing somatic variability in insect pest susceptibility (Padovan et al. 2013; Simberloff and Leppanen 2019). Somatic variability has also caused the genetic evolution of herbicide resistance in obligate asexual populations of *Hydrilla verticillate* three independent times (Michel et al. 2004). Of the few studies investigating the genetic mosaic hypothesis none measure transposon changes, despite their established importance in endogenous mutagenesis and their ability to change the epigenetic environment. Like genetic mosaicism, epigenetic states can arise in different meristems, thus increasing intra-organism variability. Somatic intra-organism competition can help select the most advantageous variants even before seeds are set, helping epigenetic instability produce beneficial variants. The resulting (epi)genetic mosaic of meristems will produce abundant and variable seedlings, which will compete in the next generation to produce the fittest variants.

Regimented cell divisions within meristems poses a conundrum for the genetic mosaicism hypothesis in that different cell layers consistently give rise to the same somatic tissues. For example, the subepidermal (L2) layer produces the eudicot germline (Satina et al. 1940; Stewart and Burk 1970; Dermen and Stewart 1973; Dawe and Freeling 1990). Consistent with the genetic mosaicism hypothesis, somatic mutations that occur in the L2 layer of the meristem have the potential to be selected upon somatically and passed to the next generation. However, beneficial mutations occurring outside of the L2 layer can provide an advantage in somatic competition but are not heritable. It has been demonstrated in somatic tissues that mobile sRNA can cause vegetatively stable changes in DNA methylation to modify gene expression (Melnyk et al. 2011*a*; Yu et al. 2018). This mobility of sRNA potentially sidesteps the conundrum caused by the heritability of advantageous changes outside of the L2 layer. That is, if epigenetic changes produce cell-to-cell mobile sRNA, an epigenetic change initiated in one meristematic cell layer could spread to encompass the entire stem cell niche. This would allow a beneficial epigenetic change initiated outside of the L2 layer, such as in epidermal initials, to influence the germline. In this way, intra-organism selection could act on epigenetic variation originating in meristem layers other than the L2 and

pass these traits to the germline, despite these tissues not contributing to the gametes genetically.

Plant sRNA machinery could promote adaptive epigenetic changes

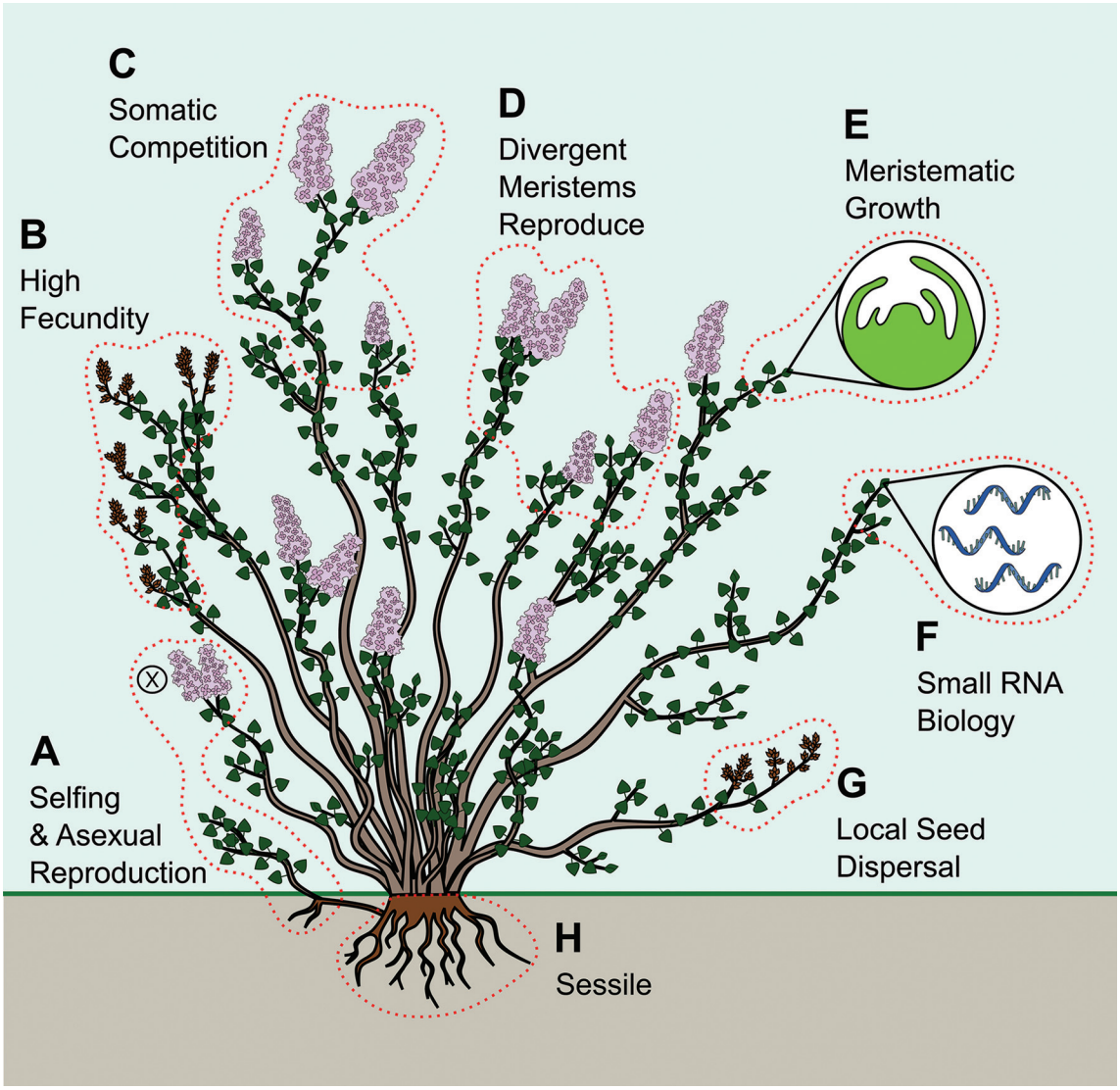
The environment of the maternal parent has been observed to influence the phenotype of its progeny to improve offspring fitness in a similar environment (Sultan 1996; Galloway 2005; Galloway and Etterson 2007). Although the molecular mechanism behind this environmental adaptation remains undiscovered, it is thought that some environmentally initiated epigenetic changes could adapt the phenotype of any progeny to life to that environment (Bruce et al. 2007; Chinnusamy and Zhu 2009; Boyko and Kovalchuk 2011). Indeed, large scale *Arabidopsis* population studies have correlated DNA methylation to climate and the methylation of a newly discovered epiallele might contribute to environmental adaptation (Kawakatsu et al. 2016; He et al. 2018). Likewise clonal poplar populations exhibit global DNA methylation differences that have been related to environmental conditions and drought exposure (Raj et al. 2011). Environmentally induced epigenetic change, if mitotically stable, could contribute to individual polyphenism, where different phenotypes are exhibited by the same genotype in different environments.

Furthermore, if environmentally induced epigenetic shifts are stable across generations, they could facilitate adaptive evolution to better suit progeny for that environment. This form of adaptation is reminiscent of the inheritance of acquired characteristics popularized by Lamarck (Lamarck 1809; however, see Burkhardt 2013) which persisted throughout the 19th century. Darwin suggested hypothetical "gemmules" would carry information about the environment to the developing germline to prime the next generation for success in that environment (Darwin 1868). Although Lamarckian evolution has been largely discredited in animals (however, see Wang et al. 2017), mobile plant sRNAs have been proposed as possible analogs to Darwin's gemmules (Martienssen 2008, 2010; Brosnan and Voinnet 2011). Several properties of sRNA make it a good candidate as a potential gemmule-like molecule: (*i*) sRNA can move via the shared plant cytoplasm (symplasm) and has been detected in the plant vasculature, indicating that sRNA moves systemically (Melnyk et al. 2011*b*); (*ii*) plants contain unique RNA-directed RNA polymerases that can elicit secondary sRNA production to amplify a weak signal into a strong response (Mourrain et al. 2000; Allen et al. 2005); (*iii*) sRNA-induced DNA methylation is transgenerationally stable due to a silencing feedback loop (Matzke and Mosher 2014); (*iv*) unlike proteinaceous transcription factors, sRNA rapidly evolves new sequences that can target any region of the genome for epigenetic changes via homology. If sRNA can act as gemmule-like molecules, sensory organs (leaves and roots) could perceive environmental signals, elicit sRNA expression (in the phloem), which would then move via the vasculature to the SAM to epigenetically regulate meristem gene expression. Since the same meristem gives rise to both the above-ground somatic tissues and germ cells, environmentally triggered sRNA expression could facilitate epigenetic changes that alter growth in this generation and the next. Although phloem-mobile plant sRNA has been shown to enter root apical meristems and late development flowers, phloem-derived sRNA entry into the SAM remains undocumented in the literature (Melnyk et al. 2011*a*; Zhang et al. 2014). Phloem-to-SAM sRNA transport would provide a channel for environmental sensing to directly influence meristem gene expression. Due to the immobile nature of plants, local environmental adaptation is of utmost importance and plants may have evolved mechanisms to facilitate environmentally triggered epigenetic adaptation for individuals and their offspring. However, stable adaptive epigenetic changes elicited by environmental stress have yet to be reported.

Plants are immobile and must endure stresses at all costs

Plants, because they are sessile and cannot behave like animals to avoid stresses, must endure environmental extremes or perish. To live through stressful conditions, adequate perception and subsequent physiological adaption must occur. High levels of stress have been shown to trigger "genome shock". McClintock (1984) describes genome shock as the way an organism responds to stress it is unprepared for by invoking genomic rearrangements, duplications, and transposon activation. Epigenetic changes regularly accompany transposon activation and therefore it seems likely that epigenetic variation accompanies genome shock. Accordingly, genome shock might produce a burst of novel (epi)genetic variation that leads to changes that provide adaption to an otherwise lethal stress, which offsets any deleterious (epi)genetic changes. For example, in *Linum usitatissimum* (flax), growth in differing environments elicits genome shock and causes large genetic changes that alter plant phenotype to potentially adapt to that new environment (Cullis 1987; Schneeberger and Cullis 1991). In polycarpic plants, if any part of the plant adapts it may allow the organism to survive. Meanwhile, the modular nature of polycarpic plants would restrict any maladaptive changes to portions of the plant, further ameliorating the risks of genome shock. In a monocarpic species, if one individual in a population evolves to tolerate a stress, it may allow that species to persist in that environ. High fecundity would allow for a few newly stress-tolerant individuals to rapidly expand in abundance. Seed dispersal is geographically tied to the maternal parent and any beneficial changes affecting the female germ cells would provide novel adaptions to the local environment. In this way, both polycarpic and monocarpic plants may be more tolerant than other higher eukaryotes to the negative effects of genome shock. Tolerance to genomic changes may allow plants to respond to local climate extremes and condition

Fig. 1. Plant traits poise individuals and populations to benefit from epigenetic changes to the genome. (A) Epigenetic changes within selfing and asexual reproducing populations provide functional diversity despite genetic similarity. (B) The potentially deleterious effects of epimutation are offset by the production of a high number of potential progenies, as few offspring must survive for the plant to reproduce successfully. (C) Competition between branches for limited resources can select for fit epigenetic changes that arise during vegetative growth before seed production. (D) Production of seed from divergent meristems produces numerous variations on the parental epigenetic state, further increasing the phenotypic diversity produced from an individual genotype. (E) Meristematic growth, and incomplete erasure of epigenetic marks during reproduction, allows somatic epigenetic changes to pass to the next generation. (F) Plant possess small RNA molecular machinery that could facilitate adaptive epigenetic changes in response to environmental sensing. (G) Most seed are dispersed close to the female parent. Any environmentally adaptive epigenetic changes produced will be relevant to the next generation. (H) Plant sessility prevents stress avoidance and stresses must be weathered to survive. Extreme stresses trigger large-scale genetic and epigenetic changes as a final effort to endure.



their progeny to adapt and thrive in a changing world. Rapid plant adaptation to environmental change will be invaluable during rampant climate change in the 21st century.

Perspectives

The possible benefits conferred by epigenetic adaptation may provide parallel advantages to augment plant genetic evolution. Various components of plant biology leave plants poised to exploit epigenetic evolution (Fig. 1). Understanding the processes underpinning plant epigenome evolution will allow for better manipulation of plant evolution without the need for DNA sequence changes. This could become useful in modern breeding programs where elite germplasm harbours very little genetic diversity due to years of artificial selection. Several preliminary breeding experiments have supported selection of epigenetic states in an array of plants, although the stability of these changes has

varied (de la Rosa Santamaria et al. 2014; Yang et al. 2015; Raju et al. 2018; Schmid et al. 2018). A better understanding of the epigenetic mechanisms could allow breeders to create variants through the manipulation of endogenous epigenetic responses to a chosen stress. Thereafter, stable epigenetic diversity could be substituted for genetic diversity to fuel artificial selection for tolerance of this stress. Indeed, if plants direct their epigenetic evolution, why should we not direct them towards our own goals?

Acknowledgements

Research supported by the Natural Sciences and Engineering Research Council of Canada (NSERC). We would like to thank Victoria Lesy and Jacob Van Vloten for critical readings of this manuscript during the writing process, and two anonymous reviewers for insightful comments and suggestions. M.A.A.M. is a recipient of an NSERC PGS-D scholarship.

References

Allen, E., Xie, Z., Gustafson, A.M., and Carrington, J.C. 2005. microRNA-directed phasing during trans-acting siRNA biogenesis in plants. Cell, **121**(2): 207–221. doi:10.1016/j.cell.2005.04.004. PMID:15851028.

Bender, J. 2004. DNA methylation and epigenetics. Annu. Rev. Plant Biol. **55**: 41–68. doi:10.1146/annurev.arplant.55.031903.141641. PMID:15725056.

Bewick, A.J., Ji, L., Niederhuth, C.E., Willing, E.M., Hofmeister, B.T., Shi, X., et al. 2016. On the origin and evolutionary consequences of gene body DNA methylation. Proc. Natl. Acad. Sci. USA. **113**(32): 9111–9116. doi:10.1073/pnas.1604666113. PMID:27457936.

Borges, F., and Martienssen, R.A. 2015. The expanding world of small RNAs in plants. Nat. Rev. Mol. Cell Biol. **16**(12): 727–741. doi:10.1038/nrm4085. PMID:26530390.

Bousios, A., and Gaut, B.S. 2016. Mechanistic and evolutionary questions about epigenetic conflicts between transposable elements and their plant hosts. Curr. Opin. Plant Biol. **30**: 123–133. doi:10.1016/j.pbi.2016.02.009. PMID:26950253.

Boyko, A., and Kovalchuk, I. 2011. Genome instability and epigenetic modification-heritable responses to environmental stress? Curr. Opin. Plant Biol. **14**(3): 260–266. doi:10.1016/j.pbi.2011.03.003. PMID:21440490.

Brosnan, C.A., and Voinnet, O. 2011. Cell-to-cell and long-distance siRNA movement in plants: mechanisms and biological implications. Curr. Opin. Plant Biol. **14**(5): 580–587. doi:10.1016/j.pbi.2011.07.011. PMID:21862389.

Bruce, T.J.A., Matthes, M.C., Napier, J.A., and Pickett, J.A. 2007. Stressful "memories" of plants: evidence and possible mechanisms. Plant Sci. **173**(6): 603–608. doi:10.1016/j.plantsci.2007.09.002.

Burkhardt, R.W. 2013. Lamarck, evolution, and the inheritance of acquired characters. Genetics, **194**(4): 793–805. doi:10.1534/genetics.113.151852. PMID:23908372.

Calarco, J.P., Borges, F., Donoghue, M.T.A., Van, Ex, F., Jullien, P.E., Lopes, T., et al. 2012. Reprogramming of DNA methylation in pollen guides epigenetic inheritance via small RNA. Cell, **151**(1): 194–205. doi:10.1016/j.cell.2012.09.001. PMID:23000270.

Catoni, M., Griffiths, J., Becker, C., Zabet, N.R., Bayon, C., Dapp, M., et al. 2017. DNA sequence properties that predict susceptibility to epiallelic switching. EMBO J. **36**(5): 617–628. doi:10.15252/embj.201695602. PMID:28069706.

Chen, Z.J., and Mas, P. 2019. Interactive roles of chromatin regulation and circadian clock function in plants. Genome Biol. **20**(1): 62. doi:10.1186/s13059-019-1672-9.

Chinnusamy, V., and Zhu, J.K. 2009. Epigenetic regulation of stress responses in plants. Curr. Opin. Plant Biol. **12**(2): 133–139. doi:10.1016/j.pbi.2008.12.006. PMID:19179104.

Choi, Y., Gehring, M., Johnson, L., Hannon, M., Harada, J.J., Goldberg, R.B., et al. 2002. DEMETER, a DNA glycosylase domain protein, is required for endosperm gene imprinting and seed viability in Arabidopsis. Cell, **110**(1): 33–42. doi:10.1016/S0092-8674(02)00807-3. PMID:12150995.

Cubas, P., Vincent, C., and Coen, E. 1999. An epigenetic mutation responsible for natural variation in floral symmetry. Nature, **401**(6749): 157–161. doi:10.1038/43657. PMID:10490023.

Cullis, C.A. 1987. The generation of somatic and heritable variation in response to stress. Am. Nat. **130**: S62–S73. doi:10.1086/284692.

Darwin, C. 1868. The variation of animals and plants under domestication. John Murray, London, U.K. doi:10.5962/bhl.title.87899.

Das, O.P., and Messing, J. 1994. Variegated phenotype and developmental methylation changes of a maize allele originating from epimutation. Genetics, **136**(3): 1121–1141. PMID:8005419.

Dawe, R.K., and Freeling, M. 1990. Clonal analysis of the cell lineages in the male flower of maize. Dev. Biol. **142**(1): 233–245. doi:10.1016/0012-1606(90)90167-H. PMID:2172058.

de la Rosa Santamaria, R., Shao, M.R., Wang, G., Nino-Liu, D.O., Kundariya, H., Wamboldt, Y., et al. 2014. MSH1-induced nongenetic variation provides a source of phenotypic diversity in Sorghum bicolor. PLoS ONE, **9**(10): e108407. doi:10.1371/journal.pone.0108407. PMID:25347794.

Deleris, A., Greenberg, M.V.C., Ausin, I., Law, R.W.Y., Moissiard, G., Schubert, D., and Jacobsen, S.E. 2010. Involvement of a Jumonji-C domain-containing histone demethylase in DRM2-mediated maintenance of DNA methylation. EMBO Rep. **11**(12): 950–955. doi:10.1038/embor.2010.158. PMID:21052090.

Dermen, H., and Stewart, R.N. 1973. Ontogenetic study of floral organs of peach (*Prunus persica*) utilizing cytochimeral plants. Am. J. Bot. **60**(3): 283–291. doi:10.1002/j.1537-2197.1973.tb10229.x.

Enke, R.A., Dong, Z., and Bender, J. 2011. Small RNAs prevent transcription-coupled loss of histone H3 lysine 9 methylation in Arabidopsis thaliana. PLoS Genet. **7**(10): e1002350. doi:10.1371/journal.pgen.1002350. PMID:22046144.

Forestan, C., Farinati, S., Rouster, J., Lassagne, H., Lauria, M., Ferro, N.D., and Varotto, S. 2018. Control of maize vegetative and reproductive development, fertility, and rRNAs silencing by HISTONE DEACETYLASE 108. Genetics, **208**(4): 1443–1466. doi:10.1534/genetics.117.300625. PMID:29382649.

Fujimoto, R., Kinoshita, Y., Kawabe, A., Kinoshita, T., Takashima, K., Nordborg, M., et al. 2008. Evolution and control of imprinted FWA genes in the genus Arabidopsis. PLoS Genet. **4**(4): e1000048. doi:10.1371/journal.pgen.1000048. PMID:18389059.

Fultz, D., Choudury, S.G., and Slotkin, R.K. 2015. Silencing of active transposable elements in plants. Curr. Opin. Plant Biol. **27**: 67–76. doi:10.1016/j.pbi.2015.05.027. PMID:26164237.

Galloway, L.F. 2005. Maternal effects provide phenotypic adaptation to local environmental conditions. New Phytol. **166**(1): 93–100. doi:10.1111/j.1469-8137.2004.01314.x. PMID:15760354.

Galloway, L.F., and Etterson, J.R. 2007. Transgenerational plasticity is adaptive in the wild. Science, **318**(5853): 1134–1136. doi:10.1126/science.1148766. PMID:18006745.

Gill, D.E. 1986. Individual plants as genetic mosaics: ecological organisms versus evolutionary individuals. *In* Plant ecology. *Edited by* M.J. Crawley. Blackwell Scientific Publications. pp. 321–343.

Gill, D.E., Chao, L., Perkins, S.L., and Wolf, J.B. 1995. Genetic

mosaicism in plant and clonal animals. Annu. Rev. Ecol. Syst. **26**(40): 423–444. doi:10.1146/annurev.es.26.110195.002231.

Greenberg, M.V.C., Deleris, A., Hale, C.J., Liu, A., Feng, S., and Jacobsen, S.E. 2013. Interplay between active chromatin marks and RNA-directed DNA methylation in *Arabidopsis thaliana*. PLoS Genet. **9**(11): e1003946. doi:10.1371/journal.pgen.1003946. PMID:24244201.

Hao, Y., Wang, H., Qiao, S., Leng, L., and Wang, X. 2016. Histone deacetylase HDA6 enhances brassinosteroid signaling by inhibiting the BIN2 kinase. Proc. Natl. Acad. Sci. USA. **113**(37): 10418–10423. doi:10.1073/pnas.1521363113. PMID:27562168.

Haring, M., Bader, R., Louwers, M., Schwabe, A., Van, Driel, R., and Stam, M. 2010. The role of DNA methylation, nucleosome occupancy and histone modifications in paramutation. Plant J. **63**(3): 366–378. doi:10.1111/j.1365-313X.2010.04245.x. PMID:20444233.

He, L., Wu, W., Zinta, G., Yang, L., Wang, D., Liu, R., et al. 2018. A naturally occurring epiallele associates with leaf senescence and local climate adaptation in Arabidopsis accessions. Nat. Commun. **9**(1): 460. doi:10.1038/s41467-018-02839-3. PMID:29386641.

Henikoff, S., and Shilatifard, A. 2011. Histone modification: cause or cog? Trends Genet. **27**(10): 389–396. doi:10.1016/j.tig.2011.06.006. PMID:21764166.

Herman, J.J., Spencer, H.G., Donohue, K., and Sultan, S.E. 2014. How stable "should" epigenetic modifications be? Insights from adaptive plasticity and bet hedging. Evolution, **68**(3): 632–643. doi:10.1111/evo.12324. PMID:24274594.

Hollick, J.B. 2017. Paramutation and related phenomena in diverse species. Nat. Rev. Genet. **18**(1):5–23. doi:10.1038/nrg.2016.115. PMID:27748375.

Holliday, R. 1987. The inheritance of epigenetic defects. Science, **238**(4824): 163–170. doi:10.1126/science.3310230. PMID:3310230.

Hollister, J.D., and Gaut, B.S. 2009. Epigenetic silencing of transposable elements: a trade-off between reduced transposition and deleterious effects on neighboring gene expression. Genome Res. **19**(8): 1419–1428. doi:10.1101/gr.091678.109. PMID:19478138.

Hristova, E., Fal, K., Klemme, L., Windels, D., and Bucher, E. 2015. HISTONE DEACETYLASE6 controls gene expression patterning and DNA methylation-independent euchromatic silencing. Plant Physiol. **168**(4): 1298–1308. doi:10.1104/pp.15.00177. PMID:25918117.

Ibarra, C.A., Feng, X., Schoft, V.K., Hsieh, T.F., Uzawa, R., Rodrigues, J.A., et al. 2012. Active DNA demethylation in plant companion cells reinforces transposon methylation in gametes. Science, **337**(6100): 1360–1364. doi:10.1126/science.1224839. PMID:22984074.

Jacobsen, S.E., and Meyerowitz, E.M. 1997. Hypermethylated *SUPERMAN* epigenetic alleles in *Arabidopsis*. Science, **277**(5329): 1100–1103. doi:10.1126/science.277.5329.1100. PMID:9262479.

Johnson, L.M., Du, J., Hale, C.J., Bischof, S., Feng, S., Chodavarapu, R.K., et al. 2014. SRA- and SET-domain-containing proteins link RNA polymerase V occupancy to DNA methylation. Nature, **507**(7490): 124–128. doi:10.1038/nature12931. PMID:24463519.

Kashkush, K., Feldman, M., and Levy, A.A. 2003. Transcriptional activation of retrotransposons alters the expression of adjacent genes in wheat. Nat. Genet. **33**(1): 102–106. doi:10.1038/ng1063. PMID:12483211.

Kawakatsu, T., Huang, S.C., Jupe, F., Sasaki, E., Schmitz, R.J.J., Urich, M.A.A., et al. 2016. Epigenomic diversity in a global collection of *Arabidopsis thaliana* accessions. Cell, **166**(2): 492–505. doi:10.1016/j.cell.2016.06.044. PMID:27419873.

Kawashima, T., and Berger, F. 2014. Epigenetic reprogramming in plant sexual reproduction. Nat. Rev. Genet. **15**(9): 613–624. doi:10.1038/nrg3685. PMID:25048170.

Kiefer, C., Willing, E.M., Jiao, W.B., Sun, H., Piednoël, M., Hümann, U., et al. 2019. Interspecies association mapping links reduced CG to TG substitution rates to the loss of gene-body methylation. Nat. Plants, **5**(8): 846–855. doi:10.1038/s41477-019-0486-9. PMID:31358959.

Kinoshita, Y., Saze, H., Kinoshita, T., Miura, A., Soppe, W.J.J., Koornneef, M., and Kakutani, T. 2007. Control of FWA gene silencing in *Arabidopsis thaliana* by SINE-related direct repeats. Plant J. **49**(1): 38–45. doi:10.1111/j.1365-313X.2006.02936.x. PMID:17144899.

Krogan, N.J., Dover, J., Wood, A., Schneider, J., Heidt, J., Boateng, M.A., et al. 2003. The Paf1 complex is required for histone H3 methylation by COMPASS and Dot1p: linking transcriptional elongation to histone methylation. Mol. Cell, **11**: 721–729. doi:10.1016/s1097-2765(03)00091-1. PMID:12667454.

Lamarck, J.B. 1809. Philosophie zoologique, ou exposition des considérations relatives à l'histoire naturelle des animaux ; à la diversité de leur organisation et des facultés qu'ils en obtiennent ; aux causes physiques qui maintiennent en eux la vie et donnent lieu aux mouvem. Baillière, Paris.

Law, J.A., and Jacobsen, S.E. 2010. Establishing, maintaining and modifying DNA methylation patterns in plants and animals. Nat. Rev. Genet. **11**(3): 204–220. doi:10.1038/nrg2719. PMID:20142834.

Law, J.A., Du, J., Hale, C.J., Feng, S., Krajewski, K., Palanca, A.M.S., et al. 2013. Polymerase IV occupancy at RNA-directed DNA methylation sites requires SHH1. Nature, **498**(7454): 385–389. doi:10.1038/nature12178. PMID:23636332.

Liu, X., Yu, C.W., Duan, J., Luo, M., Wang, K., Tian, G., et al. 2012. HDA6 Directly interacts with DNA methyltransferase MET1 and maintains transposable element silencing in Arabidopsis. Plant Physiol. **158**(1): 119–129. doi:10.1104/pp.111.184275. PMID:21994348.

Liu, Z.W., Shao, C.R., Zhang, C.J., Zhou, J.X., Zhang, S.W., Li, L., et al. 2014. The SET domain proteins SUVH2 and SUVH9 are required for Pol V occupancy at RNA-directed DNA methylation loci. PLoS Genet. **10**(1): e1003948. doi:10.1371/journal.pgen.1003948. PMID:24465213.

Manning, K., Tör, M., Poole, M., Hong, Y., Thompson, A.J., King, G.J., et al. 2006. A naturally occurring epigenetic mutation in a gene encoding an SBP-box transcription factor inhibits tomato fruit ripening. Nat. Genet. **38**(8): 948–952. doi:10.1038/ng1841. PMID:16832354.

Martienssen, R. 2008. Great leap forward? Transposable elements, small interfering RNA and adaptive Lamarckian evolution. New Phytol. **179**(3): 570–572. doi:10.1111/j.1469-8137.2008.02567.x. PMID:18715321.

Martienssen, R. 2010. Small RNA makes its move. Science, **328**(5980): 834–835. doi:10.1126/science.1190510. PMID:20466910.

Martinez, G., Wolff, P., Wang, Z., Moreno-Romero, J., Santos-González, J., Conze, L.L., et al. 2018. Paternal easiRNAs regulate parental genome dosage in Arabidopsis. Nat. Genet. **50**(2): 193–198. doi:10.1038/s41588-017-0033-4. PMID:29335548.

Matzke, M.A., and Mosher, R.A. 2014. RNA-directed DNA methylation: an epigenetic pathway of increasing complexity. Nat. Rev. Genet. **15**(8): 570. doi:10.1038/nrg3794.

Matzke, M.A., Primig, M., Trnovsky, J., and Matzke, A.J.M. 1989. Reversible methylation and inactivation of marker genes in sequentially transformed tobacco plants. EMBO J. **8**(3): 643–649. doi:10.1002/j.1460-2075.1989.tb03421.x. PMID:16453872.

McClintock, B. 1984. The significance of responses of the genome to challenge. Science, **226**(4676): 792–801. doi:10.1126/science.15739260. PMID:15739260.

Melnyk, C.W., Molnar, A., Bassett, A., and Baulcombe, D.C. 2011*a*. Mobile 24 nt small RNAs direct transcriptional gene silencing in the root meristems of *Arabidopsis thaliana*. Curr. Biol. **21**(19): 1678–1683. doi:10.1016/j.cub.2011.08.065. PMID:21962713.

Melnyk, C.W., Molnar, A., and Baulcombe, D.C. 2011*b*. Intercel-

lular and systemic movement of RNA silencing signals. EMBO J. **30**(17): 3553–3563. doi:10.1038/emboj.2011.274. PMID: 21878996.

Michel, A., Arias, R.S., Scheffler, B.E., Duke, S.O., Netherland, M., and Dayan, F.E. 2004. Somatic mutation-mediated evolution of herbicide resistance in the nonindigenous invasive plant hydrilla (*Hydrilla verticillata*). Mol. Ecol. **13**(10): 3229–3237. doi:10.1111/j.1365-294X.2004.02280.x. PMID:15367135.

Mosher, R.A., Melnyk, C.W., Kelly, K.A., Dunn, R.M., Studholme, D.J., and Baulcombe, D.C. 2009. Uniparental expression of PolIV-dependent siRNAs in developing endosperm of *Arabidopsis*. Nature, **460**(7252): 283–286. doi:10.1038/nature08084. PMID:19494814.

Mourrain, P., Béclin, C., Elmayan, T., Feuerbach, F., Godon, C., Morel, J.B., et al. 2000. Arabidopsis SGS2 and SGS3 genes are required for posttranscriptional gene silencing and natural virus resistance. Cell, **101**(5): 533–542. doi:10.1016/S0092-8674(00)80863-6. PMID:10850495.

Ng, D.W.K., Miller, M., Yu, H.H., Huan, T.Y., Kim, E.D., Lu, J., et al. 2014. A role for CHH methylation in the parent-of-origin effect on altered circadian rhythms and biomass heterosis in *Arabidopsis* intraspecific hybrids. Plant Cell, **26**(6): 2430–2440. doi:10.1105/tpc.113.115980. PMID:24894042.

O'Connell, L.M., and Ritland, K. 2004. Somatic mutations at microsatellite loci in western redcedar (*Thuja plicata*: Cupressaceae). J. Hered. **95**(2): 172–176. doi:10.1093/jhered/esh024. PMID:15073234.

Osabe, K., Harukawa, Y., Miura, S., and Saze, H. 2017. Epigenetic regulation of intronic transgenes in *Arabidopsis*. Sci. Rep. **7**: 45166. doi:10.1038/srep45166. PMID:28338020.

Padovan, A., Lanfear, R., Keszei, A., Foley, W.J., and Külheim, C. 2013. Differences in gene expression within a striking phenotypic mosaic Eucalyptus tree that varies in susceptibility to herbivory. BMC Plant Biol. **13**(1): 29. doi:10.1186/1471-2229-13-29. PMID:23421644.

Penterman, J., Zilberman, D., Huh, J.H., Ballinger, T., Henikoff, S., and Fischer, R.L. 2007. DNA demethylation in the Arabidopsis genome. Proc. Natl. Acad. Sci. USA. **104**(16): 6752–6757. doi:10.1073/pnas.0701861104. PMID:17409185.

Philips, J.G., Dudley, K.J., Waterhouse, P.M., and Hellens, R.P. 2019. The rapid methylation of T-DNAs upon agrobacterium inoculation in plant leaves. Front. Plant Sci. **10**: 312. doi:10.3389/fpls.2019.00312. PMID:30930927.

Quadrana, L., and Colot, V. 2016. Plant transgenerational epigenetics. Annu. Rev. Genet. **50**(1): 467–491. doi:10.1146/annurev-genet-120215-035254. PMID:27732791.

Raj, S., Bräutigam, K., Hamanishi, E.T., Wilkins, O., Thomas, B.R., Schroeder, W., et al. 2011. Clone history shapes *Populus* drought responses. **108**(30): 12521–12526. doi:10.1073/pnas.1103341108. PMID:21746919.

Raju, S.K.K., Shao, M.R., Sanchez, R., Xu, Y.Z., Sandhu, A., Graef, G., and Mackenzie, S. 2018. An epigenetic breeding system in soybean for increased yield and stability. Plant Biotechnol. J. **16**(11): 1836–1847. doi:10.1111/pbi.12919. PMID: 29570925.

Sadeghi, L., Prasad, P., Ekwall, K., Cohen, A., and Svensson, J.P. 2015. The Paf1 complex factors Leo1 and Paf1 promote local histone turnover to modulate chromatin states in fission yeast. EMBO Rep. **16**(12): 1673–1687. doi:10.15252/embr.201541214. PMID:26518661.

Satina, S., Blakeslee, A.F., and Avery, A.G. 1940. Demonstration of the three germ layers in the shoot apex of datura by means of induced polyploidy in periclinal chimeras. Am. J. Bot. **27**(10): 895–905. doi:10.1002/j.1537-2197.1940.tb13952.x.

Saze, H., and Kakutani, T. 2007. Heritable epigenetic mutation of a transposon-flanked Arabidopsis gene due to lack of the chromatin-remodeling factor DDM1. EMBO J. **26**(15): 3641–3652. doi:10.1038/sj.emboj.7601788. PMID:17627280.

Saze, H., Shiraishi, A., Miura, A., and Kakutani, T. 2008. Control of genic DNA methylation by a jmjC domain-containing protein in *Arabidopsis thaliana*. Science, **319**(5862): 462–465. doi:10.1126/science.1150987. PMID:18218897.

Schmid, M.W., Heichinger, C., Coman Schmid, D., Guthörl, D., Gagliardini, V., Bruggmann, R., et al. 2018. Contribution of epigenetic variation to adaptation in *Arabidopsis*. Nat. Commun. **9**(1): 4446. doi:10.1038/s41467-018-06932-5.

Schmid-Siegert, E., Sarkar, N., Iseli, C., Calderon, S., Gouhier-Darimont, C., Chrast, J., et al. 2017. Low number of fixed somatic mutations in a long-lived oak tree. Nat. Plants, **3**(12): 926–929. doi:10.1038/s41477-017-0066-9. PMID:29209081.

Schneeberger, R.G., and Cullis, C.A. 1991. Specific DNA alterations associated with the environmental induction of heritable changes in flax. Genetics, **128**(3): 619–630. PMID:1678726.

Simberloff, D., and Leppanen, C. 2019. Plant somatic mutations in nature conferring insect and herbicide resistance. Pest Manage. Sci. **75**(1): 14–17. doi:10.1002/ps.5157. PMID:30066358.

Slotkin, R.K., Vaughn, M., Borges, F., Tanurdžić, M., Becker, J.D., Feijó, J.A., and Martienssen, R.A. 2009. Epigenetic reprogramming and small RNA silencing of transposable elements in pollen. Cell, **136**(3): 461–472. doi:10.1016/j.cell.2008.12.038. PMID:19203581.

Stewart, R.N., and Burk, L.G. 1970. Independence of tissues derived from apical layers in ontogeny of the tobacco leaf and ovary. Am. J. Bot. **57**(8): 1010–1016. doi:10.1002/j.1537-2197.1970.tb09902.x.

Stroud, H., Greenberg, M.V.C., Feng, S., Bernatavichute, Y.V., and Jacobsen, S.E. 2013. Comprehensive analysis of silencing mutants reveals complex regulation of the Arabidopsis methylome. Cell, **152**(1–2): 352–364. doi:10.1016/j.cell.2012.10.054. PMID:23313553.

Styles, E., and Brink, R. 1966. The metastable nature of paramutable *R* alleles in maize. I. Heritable enhancement in level of standard R^r Action. Genetics, **54**(2): 433–439. PMID: 17248320.

Sultan, S.E. 1996. Phenotypic plasticity for offspring traits in *Polygonum persicaria*. Ecology, **77**(6): 1791–1807. doi:10.2307/2265784.

Surani, M.A., Allen, N.D., Barton, S.C., Fundele, R., Howlett, S.K., Norris, M.L., and Reik, W. 1990. Developmental consequences of imprinting of parental chromosomes by DNA methylation. Philos. Trans. R. Soc. B Biol. Sci. **326**(1235): 313–327. doi:10.1098/rstb.1990.0014. PMID:1968667.

Van Blokland, R., Van, der, Geest, N., Mol, J.N.M., and Kooter, J.M. 1994. Transgene mediated suppression of chalcone synthase expression in *Petunia hybrida* results from an increase in RNA turnover. Plant J. **6**(6): 861–877. doi:10.1046/j.1365-313X.1994.6060861.x.

Verhoeven, K.J.F., and van Gurp, T.P. 2012. Transgenerational effects of stress exposure on offspring phenotypes in apomictic dandelion. PLoS ONE, **7**(6): e38605. doi:10.1371/journal.pone.0038605. PMID:22723869.

Verhoeven, K.J.F., Jansen, J.J., van Dijk, P.J., and Biere, A. 2010. Stress-induced DNA methylation changes and their heritability in asexual dandelions. New Phytol. **185**(4): 1108–1118. doi:10.1111/j.1469-8137.2009.03121.x. PMID:20003072.

Vu, T.M., Nakamura, M., Calarco, J.P., Susaki, D., Lim, P.Q., Kinoshita, T., et al. 2013. RNA-directed DNA methylation regulates parental genomic imprinting at several loci in *Arabidopsis*. Development, **140**(14): 2953–2960. doi:10.1242/dev.092981. PMID:23760956.

Waddington, C.H. 1957. The strategy of the genes: a discussion of some aspects of theoretical biology. Allen & Unwin, London, U.K.

Wang, Y., Liu, H., and Sun, Z. 2017. Lamarck rises from his grave: parental environment-induced epigenetic inheritance in

model organisms and humans. Biol. Rev. Camb. Philos. Soc. **92**(4): 2084–2111. doi:10.1111/brv.12322. PMID:28220606.

Whitham, T.G. 1981. Individual trees as heterogeneous environments: Adaption to herbivory or epigenetic noise? *In* Insect life history patterns: habitat and geographic variation. *Edited by* R.F. Denno and H. Dingle. Springer-Verlag, New York. pp. 9–27.

Wibowo, A., Becker, C., Marconi, G., Durr, J., Price, J., Hagmann, J., et al2016. Hyperosmotic stress memory in *Arabidopsis* is mediated by distinct epigenetically labile sites in the genome and is restricted in the male germline by DNA glycosylase activity. Elife, **5**: e13546. doi:10.7554/eLife.13546. PMID:27242129.

Yamamuro, C., Zhu, J.K., and Yang, Z. 2016. Epigenetic modifications and plant hormone action. Mol. Plant, **9**(1): 57–70. doi:10.1016/j.molp.2015.10.008. PMID:26520015.

Yang, X., Kundariya, H., Xu, Y.Z., Sandhu, A., Yu, J., Hutton, S.F., et al. 2015. Muts HOMOLOG1-derived epigenetic breeding potential in tomato. Plant Physiol. **168**(1): 222–232. doi:10.1104/pp.15.00075. PMID:25736208.

Yu, C.W., Tai, R., Wang, S.C., Yang, P., Luo, M., Yang, S., et al. 2017. HISTONE DEACETYLASE6 acts in concert with histone methyltransferases SUVH4, SUVH5, and SUVH6 to regulate transposon silencing. Plant Cell, **29**(8): 1970–1983. doi:10.1105/tpc.16.00570. PMID:28778955.

Yu, N., Cao, L., Yuan, L., Zhi, X., Chen, Y., Gan, S., and Chen, L. 2018. Maintenance of grafting-induced epigenetic variations in the asexual progeny of *Brassica oleracea* and *B. juncea* chimera. Plant J. **96**(1): 22–38. doi:10.1111/tpj.14058. PMID:30086201.

Zhang, L., Cheng, Z., Qin, R., Qiu, Y., Wang, J.L., Cui, X., et al2012. Identification and characterization of an Epi-allele of FIE1 reveals a regulatory linkage between two epigenetic marks in rice. Plant Cell, **24**(11): 4407–4421. doi:10.1105/tpc.112.102269. PMID:23150632.

Zhang, S., Zhan, X., Xu, X., Cui, P., Zhu, J.K., Xia, Y., and Xiong, L. 2015. Two domain-disrupted hda6 alleles have opposite epigenetic effects on transgenes and some endogenous targets. Sci. Rep. **5**: 17832. doi:10.1038/srep17832. PMID:26666962.

Zhang, W., Kollwig, G., Stecyk, E., Apelt, F., Dirks, R., and Kragler, F. 2014. Graft-transmissible movement of inverted-repeat-induced siRNA signals into flowers. Plant J. **80**(1): 106–121. doi:10.1111/tpj.12622. PMID:25039964.

Zicola, J., Liu, L., Tänzler, P., and Turck, F. 2019. Targeted DNA methylation represses two enhancers of FLOWERING LOCUS T in *Arabidopsis thaliana*. Nat. Plants, **5**(3): 300–307. doi:10.1038/s41477-019-0375-2. PMID:30833712.

Zilberman, D. 2017. An evolutionary case for functional gene body methylation in plants and animals. Genome Biol. **18**(1): 17–19. doi:10.1186/s13059-017-1230-2. PMID:28486944.