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Review

Stressful ‘‘memories’’ of plants: Evidence and possible mechanisms

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Abstract

A history of exposure to a range of different types of stress alters subsequent plant responses. The process of priming or hardening involves prior exposure to a biotic or an abiotic stress factor making a plant more resistant to future exposure. This feature, in higher plants, indicates some capacity for ‘‘memory’’. However, the molecular mechanism(s) by which this plant memory works must be entirely different from the memory in animals which is dependent on the nervous system. We therefore use the term ‘‘stress imprint’’ in this review to describe this plant-based phenomenon. Sustained alterations in levels of key signalling metabolites or transcription factors could provide an explanation for how plant metabolism is altered by exposure to various stresses. Alternatively epigenetic changes could play a role by enabling long-term changes in gene expression. Exposure to a priming agent could activate a gene or set of genes but instead of reverting to the transcriptionally silent state once the stimulus is removed, an epigenetic mark could perhaps be left, keeping the region in a ‘permissive’ state, facilitating quicker and more potent responses to subsequent attacks. Future research is needed to establish the molecular mechanism by which plants store information on stress exposure because biotic and abiotic stresses limit agricultural production and stress responses often lead to down-regulation of yield determining processes such as photosynthesis.

Keywords: Memory; Stress imprint; Biotic and abiotic stresses; Epigenetic change; Priming

Contents

1. Introduction

Higher plants have intricate mechanisms enabling them to respond to environmental changes, most likely established over a long period of evolution as sessile organisms [1,2]. They need to be able to respond and adapt to recurring biotic and abiotic stresses as they cannot move away from them. These plant responses are controlled at the molecular level by changes in gene expression and many genes are involved in such stress responses [3–6]. Signalling pathways involving the plant hormones jasmonic acid, salicylic acid, ethylene, abscisic acid, giberellic acid, nitric oxide and auxin play a central role in integrating and coordinating whole plant stress responses [2,7]. A common theme underlying responses to a range of biotic and abiotic stresses is the phenomenon of priming whereby previous exposure makes a plant more resistant to future exposure as illustrated in Fig. 1. Primed plants display either faster and, or stronger, activation of the various defence

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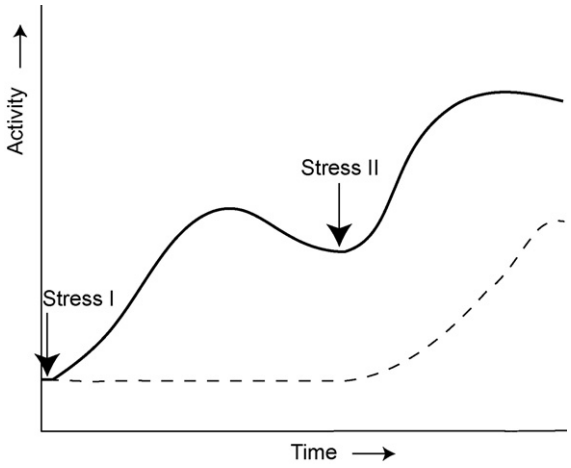

Fig. 1. Comparison of the activity of a stress-responsive gene in a primed plant (–) and an unprimed plant (- -). The primed plant is exposed to Stresses I and II whereas the unprimed plant is only exposed to Stress II. Expression levels of the stress-responsive gene are higher on exposure to the stress event in the primed plant. This is a simplified generic diagram and more complicated permutations can occur in reality.

responses that are induced following attack by either pathogens or insects, or in response to abiotic stress [8]. The advantage to the plant in being primed for particular stress responses is in facilitating a more rapid response if the stress recurs [9]. It provides the benefit of enhanced protection without the costs associated with constitutive expression of stress related genes [10,11]. There are commonalities in responses to biotic and abiotic stresses but different terminology is used in the literature. Enhanced responses to biotic stresses come under the category of induced defence but in primed plants the defence response is only switched on when an attack occurs after the priming event [12]. Altered responses to abiotic stresses are referred to as acclimation or hardening and these responses can also be enhanced by priming treatments. Priming can be elicited by exogenous application of chemical treatments as well as by exposure to the stress cues themselves [13–17].

2. Plant exposure to stress and evidence for stress imprint effects

The time interval that occurs between the priming event and the subsequent stress exposure in which the altered plant response is realised is of considerable interest. There appears to be a mechanism for storing information from previous exposure that indicates that some parts of plant responses to stress are more complicated than mere signalling cascades set off by stress signals. The implication is that plants have the capacity for some form of ‘‘memory’’ here termed ‘‘stress imprint’’ to avoid anthropomorphic connotations associated with the word memory and so as not to imply that plants are cognisant. We define a plant ‘‘stress imprint’’ as a genetic or biochemical modification of a plant that occurs after stress exposure that causes future responses to future stresses to be different. Priming (against biotic stresses) or hardening (against abiotic stresses) is the process by which such stress imprints are made. A number of experiments have shown that priming effects can last for several days at least. It is quite possible that priming lasts longer than this because many experiments have been restricted to time lags of less than one week for pragmatic reasons. Pioneering research by Baldwin and Schmelz [18] demonstrated an ‘‘immunological memory’’ of induced nicotine accumulation in *Nicotiana sylvestris* whereby plants with prior induction with methyl jasmonate showed increases in their nicotine pools two days earlier when exposed again compared with plants without prior induction. This was with a 6-day period between inductions that allowed nicotine levels to fall back to the pre-induction level. This altered response suggests that tobacco plants can store information on previous induction for at least 6 days. Stress imprint functions related to repeated exposure to stressful concentrations of the phytohormone abscisic acid (ABA) that lasted for at least 3 days have also been demonstrated with *Arabidopsis* plants [19]. Priming with sub-lethal oxidative stress (1.0 μM paraquat) induces increases in levels of six antioxidant related enzymes in the horseweed *Conyza bonariensis* which confer resistance to acute oxidant stress [20]. Pre-treated plants exposed to 1.0 mM paraquat showed significantly enhanced recovery after 3 days. In *Arabidopsis* previous encounters with either osmotic or oxidative stress can markedly alter subsequent osmotic stress-induced Ca^{2+} responses suggesting that there is an imprint of previous stress encounters [21].

There is a sizeable literature on seed priming in which long lasting effects occur after germination. For example, soaking wheat seed in saline solution has been shown to prime plants germinating from the treated seed so that they are more resistant to salt stress for the whole growing season [22]. In some cases, the effects of seed priming are stronger in more advanced growth stages as was shown with tomato [23]. Some stress imprint effects in plants have even been shown to be perpetuated to the next generation: Molinier et al. [24] used ultraviolet radiation and flagellin, a bacterially derived elicitor, as stress factors and observed genomic changes (hyper-recombination) in the somatic tissue of not only the treated plants but also their progeny. Thus, stress exposure of parent plants can even lead to stress imprints that are carried forward to the next generation of unstressed plants, a phenomenon that is different from the priming effect discussed above but in some ways even more interesting. This transgenerational stress imprint effect was also observed in wild radish, *Raphanus raphanistrum*, responses to herbivore damage (*Pieris rapae*) and treatment with jasmonic acid [25]. Progeny of treated plants were more resistant to herbivory than control plants were. In another example the increased sensitivity of grapevines, *Vitis vinifera*, to ozone in consecutive years indicated a stress imprint effect for ozone exposure in previous years [26] but this was not an adaptive mechanism as the stress imprint was deleterious to the plant. Increased sensitivity is the opposite of the increased resistance associated with priming. Even where priming increases resistance to stress factors overall plant performance can be compromised by trade-offs such as down-regulation of photosynthesis.

3. Possible mechanisms

The existence of a stress imprint effect in plant responses to a variety of biotic and abiotic stresses raises the question of how such effects occur and what the underlying mechanism is. Conrath et al. [8] state that the molecular mechanisms responsible for priming are not well understood but propose two potential mechanisms one involving accumulation of signalling proteins and the other involving accumulation of transcription factors. Here we also propose an epigenetic mechanism (Fig. 2).

3.1. Accumulation of signalling proteins or transcription factors

Priming could involve accumulation of signalling proteins in an inactive configuration that are activated upon exposure to stress, perhaps by a protein kinase being triggered by changes in calcium levels. Activation of heat-shock proteins might also occur in a similar way. The *Arabidopsis ibs1* mutant is affected in a cyclin-dependent kinase like protein and this mutant cannot acquire BABA-induced priming for salicylate-dependent defences [15]. The protein IBS1 appears to function as a BABA-induced accelerator of the salicylate-dependent defence pathway.

It has also been suggested that there could be accumulation of transcription factors in primed plants that enhance defence gene transcription after stress recognition [8]. Forty stress inducible transcription factor genes have been found in *Arabidopsis* [27], which could perhaps support this potential mechanism. The potential importance of transcriptional regulation of gene expression has also been highlighted by Yamaguchi-Shinozaki and Shinozaki [28] in their review of plant responses to dehydration and cold stresses. One such stress-induced transcription factor gene is *HOS10*, which encodes an R2R3-type MYB transcription factor that is essential for cold acclimation that appears to affect dehydration stress tolerance in plants by controlling stress-induced ABA biosynthesis [29]. A transcription factor AtERF7 plays an important role in ABA responses and hence plant drought stress responses [30]. AtERF7 acts as a repressor of gene transcription, and guard cells of plants overexpressing this factor had reduced sensitivity to ABA and increased transpirational water loss. There could also be more complicated responses to the ratio of levels of more than one factor.

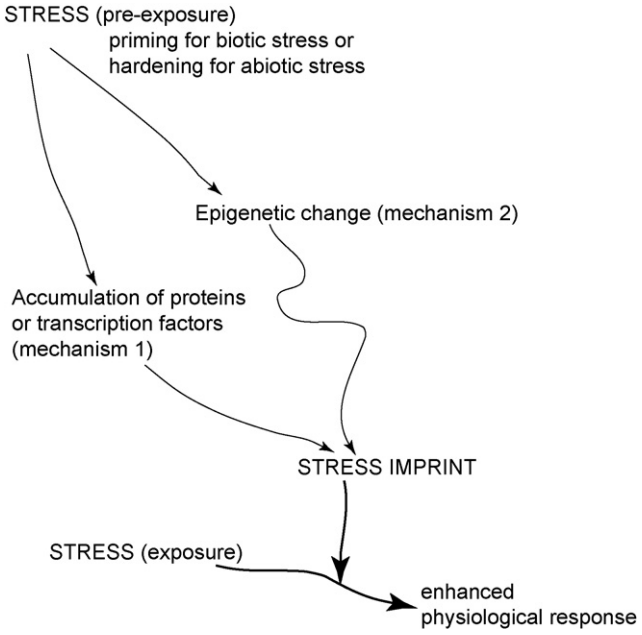

Fig. 2. Summary of the process of stress-imprint formation: the stress imprint leads to an enhanced physiological response when the plant is exposed again. Imprints could be formed by accumulation of proteins or transcription factors (mechanism 1) or by epigenetic change (mechanism 2) or indeed by both mechanisms or by a mechanism not yet discovered. Elicitor treatments can be used as a surrogate for stress exposure.

3.2. Epigenetic changes

Another intriguing possibility is that priming effects could also occur as a result of epigenetic changes. These changes involve modification of DNA activity by methylation, histone modification or chromatin remodelling without alteration of the nucleotide sequence [31]. Such a mechanism would enable longer term stress imprints to be left in the plant than with the metabolite accumulation models described which probably mediate more transient or short-term effects. The involvement of epigenetic mechanisms in plant memory of transient events during development, such as vernalisation, organisation of shoot and root apical meristems, seed development and repression of endosperm development before fertilisation, has been well documented [32]. Epigenetic events should be viewed as: "the structural adaptation of chromosomal regions so as to register, signal or perpetuate altered activity states" [33]. Molecular mechanisms underpinning this include modifications of the DNA by cytosine methylation and/or alteration of the nucleosome core histones (H2A, H2B, H3, H4) through acetylation, methylation, phosphorylation and ubiquitinylation [34]. These changes in chromatin structure determine gene expression by activation or silencing [35]. Epigenetics plays an important role in vernalisation in *Arabidopsis* [36]. During vernalisation the long-term exposure over winter to low temperatures is 'memorised' and is essential for the plant to acquire the competence to flower in the following spring. The molecular basis for this 'memory' is a change in the active chromatin structure of the *FLOWERING LOCUS C* (*FLC*) gene from an active state into mitotically stable repressive heterochromatin. *FLC* inhibits the transition to flowering when it is transcribed but *FLC* transcript levels are down-regulated after cold treatment and epigenetic down-regulation of *FLC* is a major target of the vernalisation pathway. This process is mediated by several *VERNALISATION* (*VRN1*, *VRN2* and *VRN3*) genes that are hypothesised to be involved in the deacetylation and methylation (H3 Lys9 and Lys27) of histones in the *FLC* gene region. These histone modifications are thought to promote heterochromatin formation, thereby rendering the *FLC* gene inaccessible to transcription, which ultimately leads to the removal of the factor repressing

flowering. Epigenetic changes have also been shown to play key roles in other aspects of plant development including organisation of shoot and root apical meristems [37], seed development [38] and repressing endosperm development before fertilisation [39,40].

Epigenetic control of transcription could provide a means for altering gene expression after stress events and there is already evidence that this occurs in some circumstances. Several recent reports have shown that different environmental stresses lead to altered methylation status of DNA as well as modification of nucleosomal histones. When maize seedlings were exposed to cold stress, genome-wide 5-methylcytosine demethylation occurred predominantly at the nucleosome core regions in root tissue [41]. A notable feature was that, even after the seedlings were returned to normal growth conditions, the decreased methylation level did not recover. It was hypothesised [41] that for re-methylation to occur, *de novo* methyltransferase activity would be necessary, which might be absent in quiescent cells such as root tissue and that therefore the methylation status cannot be restored. Histone modification also occurs after plant exposure to biotic and abiotic stress factors and histone acetylation can directly enhance gene expression, allowing increased expression of stress-responsive genes. Increased H3 acetylation was found after submergence of rice plants which led to increasing expression of two stress-responsive genes *ADH1* and *PDC1* [42]. The depletion of oxygen during submergence of rice seedlings led to the acetylation of the histone H3 and to the conversion of di-methyl H3-K4 to tri-methyl H3-K4 at two submergence-inducible genes, *ADH1* and *PDC1*. Both modifications are associated with active transcription and were reversible after the removal of the stress. For plant responses to biotic stress, genes involved in jasmonic acid and ethylene signalling are often crucial and a role for histone deacetylation in the expression of these has been shown [43]. These authors discovered that a histone deacetylase, HDA19, was involved in the *Arabidopsis* jasmonic acid, ethylene and pathogen response and that *HDA19*-overexpressing plants showed an increased resistance to the pathogen *Alternaria brassicicola*. Although deacetylation of histones is usually regarded as a transcriptionally repressive event, creating localised regions of repressed chromatin [44], it may, in certain cases, also activate transcription by preventing binding of repressors, as has been demonstrated in yeast [45]. Expression of *HDA19* was shown to be induced by attack by *A. brassicicola* or by wounding. HDA19 also synergises the AtERF7 transcription factor mentioned earlier [30]. Another histone deacetylase, AtHD2C, has a role in enhancing plant tolerance to salt and drought stresses by modulating ABA responsive gene expression [46]. Overexpression of the *AtHD2C* gene created an ABA insensitive phenotype.

Taken together, these very recent studies show that epigenetic modifications of chromatin, both at the level of DNA and nucleosomes, are implicated in plant stress responses. The role of chromatin remodelling in the transcription of stress-responsive genes is presumably to allow modifications that switch on gene expression when stress is sensed and then reinstate repression, once the stress stimulus is removed. It has been suggested that this dynamic behaviour could leave behind a record of gene activity in so-called 'memory' marks, which indicate either dynamic activity, memory of activity or poising for future activity [47]. In the yeast *Saccharomyces cerevisiae* it has been shown that di-methylation of H3-K4 seems to correlate with a 'permissive' state, in which genes are either active or potentially active, whereas tri-methylation of H3-K4 is linked to on-going transcription [48]. Furthermore, hyper-methylation within the mRNA coding regions of H3-K4 persists for up to 5 h after transcriptional inactivation as is Set1 (yeast histone H3-lysine 4 (H3-K4) methylase) dissociation from chromatin, indicating that H3-K4 hypermethylation can provide a molecular memory of recent transcriptional activity [49]. Such findings could be related to the priming phenomenon. We hypothesise that exposure to a priming agent could activate a gene or set of genes but instead of reverting to the transcriptionally silent state once the stimulus is removed, an epigenetic mark (such as histone acetylation) could be left, keeping the region in a 'permissive' state perhaps facilitating quicker and more potent responses to subsequent attacks.

4. Conclusions

There is evidence that plants are adept at altering their physiology and metabolism in response to prior experience. However, much still remains to be learnt about the mechanism by which plants store information from previous exposure. The mechanisms suggested in this review are largely hypothetical although there are specific examples of stress responses where particular mechanisms have been elucidated. The mechanism(s) that pertain to one plant stress response could well be different from the mechanism(s) that pertain(s) to another one. It is likely that the epigenetic mechanism underpins more longer lasting effects than the other suggested mechanisms that involve metabolite accumulation. Although the molecular basis for all stress imprinting in plants need not be the same as the one for vernalisation or even the same mechanism for different types of stress, we suggest that the involvement of epigenetic control is a hypothesis worth testing especially as it would enable long-term marking of stress exposed plants.

The ''memory'' that occurs in plants certainly is different from memory in animals because plants rely more on adaptive biochemical changes rather than on cognisant processes. However, it would be of great interest to more fully establish the mechanisms by which plants store information on exposure to stress because biotic and abiotic stresses limit agricultural production. This should be a fruitful area for future research both in terms of new science and in terms of applied value.

Exposure to low levels of certain volatile compounds can elicit stress responses in plants. These elicitors can thus be surrogates and allow the formation of ''stress imprints'' even in the absence of exposure to real stress. Beneficial organisms such as mycorrhizal fungi can also switch on plant stress response genes. Furthermore, internal signalling within the plant can occur. A particularly intriguing possibility is that siRNAs, which have been shown to induce epigenetic changes through RNA-dependent DNA methylation (RdDM) and

related chromatin modifications [50], could function as systemically transported priming signals by causing specific epigenetic modifications. Better information on plant stress imprinting and associated signalling would facilitate the development of priming treatments for crops to enhance yields under conditions of stress. If we could discover how to use priming or stress imprinting processes to switch on genes we could manipulate expression of plant defence genes such as (*E*)-β-farnesene synthase [51,52]. Conversely, under conditions where stress is absent, alterations in plant physiology imprinted by previous stress events could compromise aspects of plant productivity, for example by down-regulation of photosynthesis. Thus, it could be valuable to discover ways of deleting such imprints or preventing such imprints from initially occurring.

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