Epigenetic responses to stress: triple defense?
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Stressful conditions for plants can originate from numerous physical, chemical and biological factors, and plants have developed a plethora of survival strategies including developmental and morphological adaptations, specific signaling and defense pathways as well as innate and acquired immunity. While it has become clear in recent years that many stress responses involve epigenetic components, we are far from understanding the mechanisms and molecular interactions. Extending our knowledge is fundamental, not least for plant breeding and conservation biology. This review will highlight recent insights into epigenetic stress responses at the level of signaling, chromatin modification, and potentially heritable consequences.

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Introduction
Plants in the field are permanently exposed to stress. Limitations originate from many different factors: too high or too low temperatures/water supply/light intensity, non-optimal mineral composition or soil contamination, mechanic inhibition, pathogen infestation, lack of symbiotic partners, interactions with other plants, parasites or herbivores. These conditions can activate defense responses by which plants can minimize detrimental consequences of stressful conditions for their survival, growth, or propagation. These mechanisms allow plants to occupy even extreme habitats, despite their sedentariness. Under field conditions, different stress types usually occur concomitantly, like heat and drought, and the molecular responses can be difficult to separate. Response to one stress type can be also antagonistic to another, as adaptation to nutrient-poor substrates might reduce stress by competition. Therefore, mechanistic studies of stress effects are mostly performed under controlled laboratory conditions, applying one stress type and distinguishing biotic from abiotic stress. While this resulted in information about signaling cascades, transcription factors, and defense compounds, interference of stress with epigenetic factors became evident only recently. Stress effects on the epigenetic level are expected to allow more permanent changes of gene expression and potentially long-term adaptation that could have evolutionary impact, as chromatin modifications can be mitotically or meiotically heritable. This review will focus on the connection of stress with epigenetic regulation, separated in three levels.

Level 1: epigenetic components of stress signaling
Investment into stress defense, alongside constitutive morphological and metabolic survival equipment and seasonal adaptations, expends plants' general resources and therefore should be restricted to the actual occurrence of stressful situations. Plants use a range of different sensing and signaling mechanisms to induce dynamic stress responses only when challenged. Signaling includes mainly the hormones salicylic acid (SA), jasmonic acid (JA) and ethylene upon biotic stress, and abscisic acid (ABA) in case of abiotic stress (reviewed in [1]). Nevertheless, there is growing evidence that noncoding and siRNAs and the proteins generating or binding them are involved in stress-signaling and can subsequently induce transcriptional or posttranscriptional gene silencing (TGS or PTGS, respectively). These principles are described in the review by Wierzbicki (this issue).

There are many examples of differential siRNA, miRNA or ncRNA expression upon stress [2]. The recent identification of a mutated *NRPD2* gene responsible for constitutive overexpression of a SA-inducible gene provided a mechanistic link between stress signaling and elements of the RNA directed DNA methylation (RdDM) pathway, excluding only PolIV [3•]. Some RdDM mutants had a compromised immune response to pathogenic fungi correlated with a lack of gene induction by JA. In contrast, resistance to a bacterial pathogen was increased, corresponding with elevated levels of salicylic acid-related defense genes and enriched activating chromatin marks H3K4me3 and H3K9ac at the promoter of the SA-inducible gene *PR-1*. This argues for the overlap between targets of RdDM and SA-signaling and a role of RdDM to relay stress signals to the nucleus.

Stress can certainly induce the production of siRNAs, either via antisense transcription of protein-coding or non-protein-coding sequences, or from inverted repeats.

An early example of naturally occurring antisense was the discovery that induction of the Arabidopsis *SRO5* expression under salt stress creates a transcript partially complementary to that of the constitutively expressed *P5CDH*, resulting in dsRNA as a substrate for Dicer-like proteins and siRNAs that are only present and effective under stress [4]. Among 76 long non-protein-coding RNAs (npcRNA) in the Arabidopsis genome, 26 had altered expression levels upon low phosphate, salt or drought stress [5]. Some of them gave rise to 24 bp siRNAs, and npc536 conferred improved root growth under salt stress. By now, many pairs of potential antisense transcripts are identified [6], and natural cis-antisense siRNAs (nat-siR-NAs) are defined as a separate class of the small RNA family. Many of them are found exclusively or enriched upon specific stress conditions [7]. One example role of siRNAs in extreme stress tolerance is a dehydration- and ABA-inducible retroelement-derived siRNA that regulates an adaptive response in the resurrection plant *Craterostigma plantagineum* [8].

Applying tailor-made transcripts of inverted repeats (IRs) is a routine technique to interfere with transcription of target genes with homology to the resulting siRNAs, but similar siRNA can also originate from transcripts of endogenous IRs. Two Arabidopsis repeats, IR71 and IR2039 [9••], produce siRNA of different size classes, of which some can silence a GFP-reporter *in trans*. Endogenous IRs are highly variable in the genome of different ecotypes of Arabidopsis, indicating fast evolution and rapid adaptive changes [9••]. However, the actual response of IR-derived siRNA to environmental factors and contribution to stress-adaptation is yet to be demonstrated.

Responses of transposable elements (TE) to stress are the topic of reviews by Lisch (this issue) and Bucher et al. (this issue), but TE activation raises the interesting potential of TE-derived small RNAs that target stress-related protein-coding genes [10] and thereby represent an indirect stress signaling pathway.

Level 2: stress etching on chromatin

Stress signaling leads to stress-adapted gene expression and also affects chromatin structure at responsive genes, directly or indirectly [11–14]. The changes can affect DNA methylation, histone tail modifications, exchange of histone variants, or nucleosome occupancy and larger chromatin configuration, as documented in the following examples (Figure 1).

DNA methylation is an important defense strategy against infections by DNA-viruses. Double-stranded viral transcripts can induce methylation at homologous sequences, which led to the discovery of the RdDM phenomenon [15]. Arabidopsis RdDM mutants are hypersensitive to gemini viruses infection [16]. Some siRNAs associated with virus infection can be complementary to regions of the host genome, especially to regulatory sequences containing retro-elements evolved from ancient integration events, and can exert stable gene silencing on such 'off-targets'. The same principle could apply to other endogenous siRNAs that match with promoter regions and could affect stress-related genes [17]. For example, repeat elements in the promoter of a sodium transporter gene are methylation targets and determine salt stress tolerance [18]. A plausible correlation is also the DNA methylation of two genes controlling stomata development, their transcriptional repression, and a reduced number of stomata in Arabidopsis plants grown under low humidity [19••]. Methylation changes that appear non-targeted or not yet associated with stress gene targets are induced by salt stress in Arabidopsis [20], or in roots of rice plants, to different degree depending on the salt sensitivity of the cultivars [21]. These could be secondary effects due to stress-induced activity changes of epigenetic regulators. Multiple methylation changes were also observed associated with herbivore and pathogen attack in dandelions [22], and between mangroves or rubber trees growing in different habitats [23,24]. Higher resistance to pathogens of Arabidopsis mutants with defects in DNA methyltransferases inspired a genome-wide analysis after infection with a bacterial pathogen, revealing methylation changes in all sequence contexts and at multiple loci, including defense-related genes, transposons and repeats [25•].

There are several reports of stress-induced histone tail modifications and altered stress resistance in mutants lacking histone-modifying enzymes. Installation of activating marks can result in increased transcription levels of stress genes but may also simply poise target genes for faster or stronger response upon a more serious attack later [26,27]. ABA and salt change H3 phosphorylation and H4 acetylation in cell cultures of tobacco and Arabidopsis [28]. Activation of a gene for an immune receptor protein (R) gene in Arabidopsis depends on a histone methyltransferase installing H3K36me3 [29•]. A prominent target is also the *PR-1* (Pathogen-Related 1) gene involved in initiating defense or stress adaptation where H3K4me2, H3K4me3 and H3ac levels increase upon SA treatment or pathogens [30,31]. *PR1* expression is negatively regulated by two WRKY transcription factors, which both interact with histone deacetylase 19 (HDA19) [32]. Deacetylation reduces active marks, and the induction of HDA19 upon bacterial infection could be a chromatin-based way to weaken the plant defense system, among many other strategies [13]. Another histone deacetylase, HDA6, is required for freezing tolerance in Arabidopsis, although the link to histone acetylation at a specific target gene is missing [33,34]. A histone ubiquitin ligase determines the resistance to necrotrophic fungi [35], and the growing number of identified modification types makes it likely that more of them will also be connected with stress regulation.

Figure 1
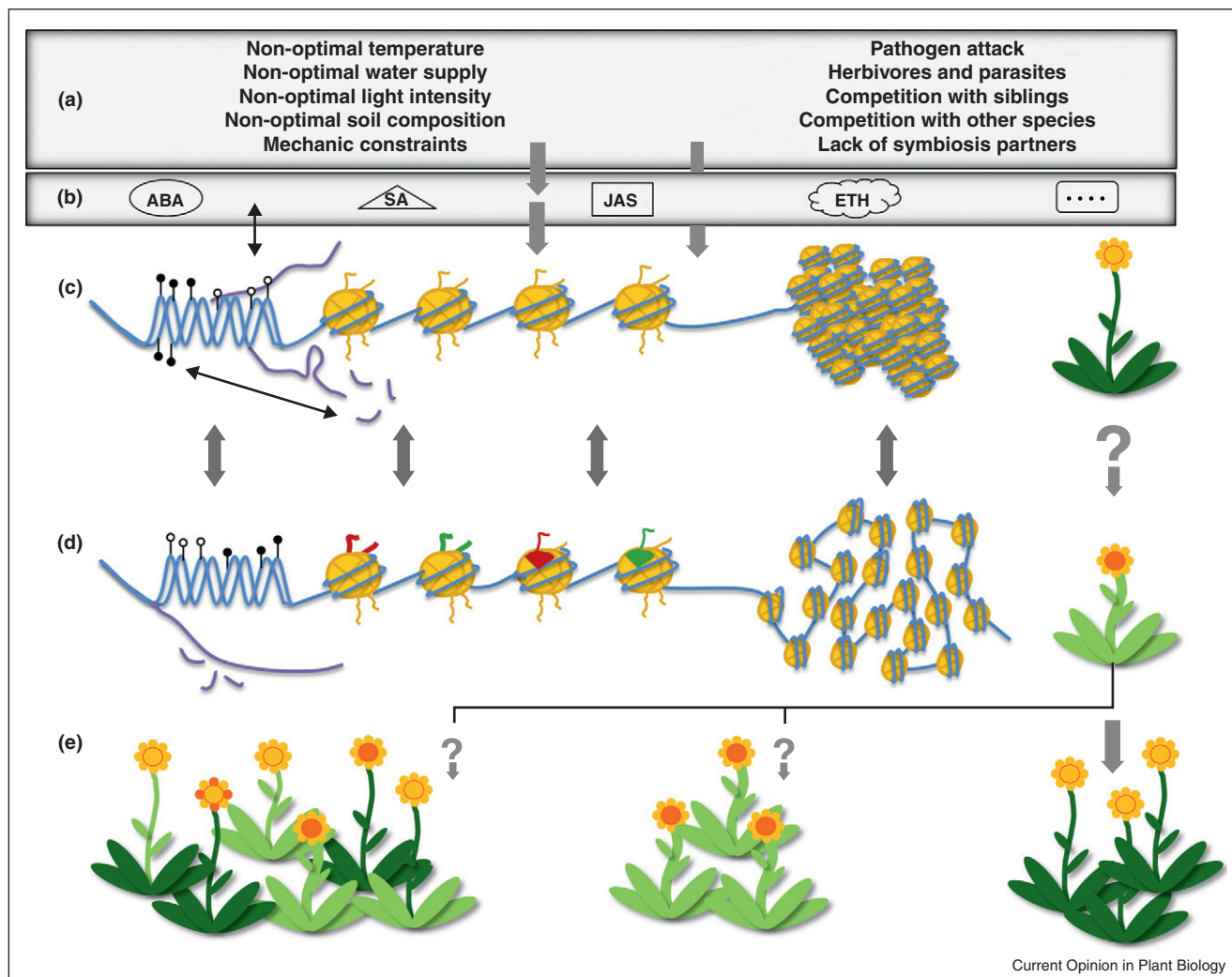
Abiotic and biotic stress conditions **(a)** can change gene expression with and without involving plant stress hormones **(b)**. Transcription changes, or stress factors directly, can affect chromatin via DNA methylation, histone tail modifications, histone variant replacements, or nucleosome loss and chromatin de-condensation (**c, d**). These changes are largely reversible but can modify metabolic or morphologic plant features under stress conditions. Usually, the new phenotypes are not transmitted to progeny. However, chromatin-associated changes have the potential to be heritable and might result in uniform maintenance of new features or new combination and epigenetic diversity **(e)**.

The histone methylation at H3K27 under control of the Polycomb/Trithorax complexes and its response to environmental signals are described in detail elsewhere [36]. MSI1, a member of the PRC2 as well as the CAF1 complex, contributes to drought stress resistance by controlling the expression of ABA-responsive genes and proline synthesis [37]. Lower H3K27me3 levels at abscisic acid-responsive genes upon downregulation of the cell cycle regulator RETINOBLASTOMA-RELATED (RBR), and induction of several stress-related genes before deregulation of the cell cycle machinery [38,39] indicate a tight and complex connection between stress factors that modify chromatin features and developmental regulation. Three other histone variants are so far also connected with stress responses. Histone variant H2A.Z associated with the 5′end of many genes is relevant for repression of pathogen response, as mutants lacking subunits of the SWR1 complex that installs H2A.Z show increased resistance to bacterial infections [40]. A clear connection of this variant also with abiotic stress is evident from its eviction by heat exposure [41••]. Lack of ARP6, one SWR1 subunit, mimics the transcriptional changes even at ambient temperatures. Further, a plant-specific linker histone H1 variant is expressed under drought stress in Arabidopsis, and its downregulation in tomato revealed its importance in water stress response [42]. DNA damaging stress is followed by incorporation of H2A.X, which becomes phosphorylated and attracts

DNA repair complexes and transcriptional activators [43]. However, many more chromatin components might be involved in enabling DNA repair and recombination.

Lastly, the association of nucleosomes with DNA can be modified in response to stress. Indirect evidence came from analyzing mutants with defects in putative or proven chromatin remodeling factors in response to stress (e.g. [44]). A protein phosphatase 2C and ABA co-receptor, HYPERSENSITIVE TO ABA1 (HAB1) can interact with a subunit of the ATP-dependent chromatin remodeling complex SWI3B, and *swi3b* mutants show reduced ABA responses [45]. The deregulation of epigenetically controlled genes and repetitive sequences upon heat stress [46–48] is associated with transient loss of DNA-bound nucleosomes at transcribed and non-transcribed genomic regions, and with substantial heterochromatin de-condensation [47]. A role for nucleosome occupancy is further suggested by delayed re-silencing of heat stress-activated repeats in mutants with impaired CHROMATIN ASSEMBLY FACTOR 1 (CAF-1) subunits [47]. Nucleosome eviction, together with histone tail modifications were also observed at target genes of stress-induced transcription factors in the green alga Chlamydomonas [49•].

Level 3: potential for lasting adaptation
Extreme stress can result in a complete growth arrest, but, after limited damage, plants can recover and resume growth and development. This is in part exerted by activation of replication- and cell cycle checkpoints that prohibit amplification and transmission of damaged DNA [50]. It is tempting to propose the existence of a similar epigenetic checkpoint control that senses detrimental alterations in the epigenome and halts growth until these alterations are repaired. However, plants tolerate loss of DNA damage checkpoint components much better than animals [50]. Similarly, epigenetic mutations with lethal consequences in animals are often less severe in plants, which could indicate a less stringent 'quality' control of epigenetic integrity. Many plants proliferate partially or even exclusively by vegetative propagation, circumventing epigenetic reprogramming during gamete formation and sexual reproduction as observed in mammals. However, if such 'resetting' occurs in plants, it seems to be at least not as extensive [51], and mitotic and meiotic transmission of stress-induced epigenetic disruptions is therefore conceivable. Indeed, there is growing interest in 'memory' effects in stressed plants. These could even be beneficial if they make exposed plants and/or their progeny more resistant upon recapitulated challenges. The concept of priming plants with a certain stress, resulting in a faster and more pronounced response later, is exemplified by Systemic Acquired Resistance (SAR), in which soluble and/or volatile signaling molecules can spread within an individual or be transmitted between plants. Several stress types were reported to induce a priming effect that can be assayed also in subsequent generations [52–57]. In some cases, the lack of epigenetic components, mainly in the RdDM pathway, was shown to reduce or eliminate the transmission to the progeny [52,54] or mimic the primed state [53]. However, the effects did not last more than one or two generations, and concomitant chromatin changes were connected only in one case with the primed state of stress-specific genes [53]. Seemingly transgenerational responses can also originate from parental stress affecting embryo development, seed germination or early growth of the progeny, independent or dependent from chromatin-regulated components.

Although a role of chromatin in priming is widely assumed [58–60], proof of strong causal epigenetic changes affecting traits with adaptive values, without concomitant genetic changes or long-living signaling components transmitted through the cytoplasm of gametes, would be desirable [51,61]. This is not meant to discourage further studies: on the contrary, the wealth of interesting phenomena that could indicate transgenerational epigenetic inheritance [62,63], together with the growing toolbox for thorough genome-wide genetic and epigenetic analysis, make it a very interesting field of research. Analysis of DNA methylation in individuals, between generations or populations from different habitats indicates a range of epigenetic diversity that might by far exceed genetic diversity, as described in the contribution of Becker and Weigel (this issue). But it is also clear that many epigenetic changes are triggered by genetic mutations, especially transposon movements, in the vicinity. So far, these genetically triggered epigenetic changes are those with the most relevant and drastic consequences, as exemplified by the development of genetic incompatibility [64•]. On the other hand, many transposons are under epigenetic control, as reviewed by Bucher et al. (this issue), which makes the question of a direct or indirect epigenetic stress memory an interesting, but difficult-to-investigate chicken-and-egg problem.

Conclusions
Stress and epigenetic regulation meet at many different levels from which only a few aspects are documented so far. Current information about the connection resembles a few fragments of a jigsaw puzzle for which neither the number of parts nor the dimensions of the picture are known. It is very likely that epigenetic variation contributes to the adaptation potential of plants and, like genetic diversity, is under selection by environmental conditions. Whether epigenetic responses to stress can serve as adaptive traits remains a matter of debate. Surprisingly few primary publications have addressed the issue, while many reviews discuss this possibility. Adding another one will not shift this imbalance but we hope to stimulate more work in the field.

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