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Epigenetic Regulation of Stress-Induced Extracellular Matrix Remodeling in Plants: Mechanisms, Memory, and Applications

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ABSTRACT

The plant extracellular matrix (ECM), mostly comprised of the cell wall and connected polymers, offers an active interface between growth and defense as well as environmental alterations. While ECM architecture remodeling is characteristic for stress responses, mechanistic relationships between nuclear epigenetic regulation and extracellular remodeling are not clearly defined. This work aims to construct a mechanistic framework correlating chromatin-level epigenetics with stress-induced ECM remodeling. By incorporating recent evidence and mechanistic dissection, four hierarchically organized layers of epigenetic regulation are defined: (i) DNA methylation, as a switch for cellulose and lignin biosynthetic genes; (ii) histone modifications, such as H3K4me3, H3K27me3, and histone acetylation, governing transcriptional activation and repression of wall-associated loci; (iii) chromatin remodeling complexes such as SWI/SNF, governing promoter accessibility of cell wall genes; and (iv) small RNAs and epitranscriptomic marks (e.g., m^6A), governing transcript integrity and translation of ECM biosynthetic pathways. These pathways all impinge upon transcription factor – epigenetic modifier complexes (e.g., NAC/MYB, WRKY – BRAHMA), with receptor kinases such as WAKs and FERONIA enforcing ECM-derived inputs upon receptor kinase-mediated outputs by changing nuclear chromatin states. Stress-specific strategies such as DNA hypermethylation under drought stress, histone acetylation under salinization stress, and chromatin priming by pathogen or cold stress create both transient defenses and long-term stress memories such that some modifications are passed through multiple generations. Together, these findings make epigenetic regulation out to be a molecular switchboard balancing immediate plasticity with heritable resilience. This integrated conceptual framework not only clarifies knowledge about nuclear but also provides indications for translational potential by breeding climatically resilient crops through specified epigenetic priming and selection.

KEYWORDS

Plant extracellular matrix (ECM); Epigenetic regulation; ECM-nucleus feedback loops; Climate resilience and crop improvement

1 Introduction

Plant extracellular matrix (ECM), made up mainly of the cell wall and associated polymers, is an actively interacting interface with the environment and takes major roles in growth and defense [1-2]. Far from a passive scaffold, the cell wall is actively reshaped in response to stress. Such remodeling – such as in lignin reinforcement, in the formation of callose, and in the adjustment of cellulose and pectin – is a common defense response against both abiotic and biotic stress [3-4]. Stiffening or altering the ECM (e.g. via lignification or via closure of plasmodesmata via callose) facilitates toughening of the first defense barrier against pathogens and in altering mechanical properties in the presence of drought, salinity, or other stress [5-6]. Such alterations in the cell wall are critical for integrity and function maintenance in the presence of unfavorable conditions as they promote physical defense and can provoke subsequent stress signaling [7].

Epigenetic processes, DNA methylation, histone post-translational modifications, chromatin remodeling, and non-coding RNAs, which provide a complementary system of gene regulation not involving change in the DNA sequence but permitting rapid adjustment in gene expression in response to external stimuli. These epigenetic modifications are in most cases reversible and often intersect with stress hormone signaling (e.g., abscisic acid) to enable rapid activation or inactivation of stress-responsive genes [8]. Significantly, epigenetic regulation allows plants to "prime" their responses: after an initial exposure to stress, chromatin rearrangements can predispose particular genes for rapid or increased reactivation on a repeated stress exposure, an effect described as stress memory [9]-10]. Such epigenetic stress memories, for example, prolonged histone H3K4 methylation at defense gene locations or stress-responsive small RNAs accumulation, enable a rapid or stronger defense on a repeated stress exposure [11]. Hence, epigenetics provides the plant with a rapid and flexible system of transcriptional regulation intimately linked to stress signaling networks.

However, a major gap remains in our understanding of how such nuclear epigenetic reprogramming events are converted into physical remodeling of the ECM in stress adaptation. While epigenetic modifications are regularly invoked to explain environmental stress memories, such causally clear examples are relatively few in number, and such memory's duration and molecular targets remain partially unknown [11]. It is particularly unclear at the chromatin-level how such modifications are ultimately converted into structural cell wall modifications – for instance, does stress-induced DNA methylation or histone acetylation directly change gene expression for cell wall enzymes or polymers, in turn modifying

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wall composition? Or where small RNAs and other epigenetic regulators meet the cell wall integrity sensing circuits? Such questions indicate a major gap in our present understanding at the interface of nuclear regulation and extracellular consequences [11]. Filling such a gap is major, since such a gap revealed and filled, it would explain in detail how plants put gene expression together with cell wall flexibility in stress, relating genome to external protection barrier.

We theorize that epigenetic regulation tunes the characteristics of the ECM to serve as a molecular governor to reconcile short-term plasticity against inherited memory in stress responses in plants. Here, stress epigenetic marks could provoke cell wall–related genes for immediate reinforcement of the ECM such that the incomplete erasure of the marks upon stress exposure yields a type of memory priming the cell wall for later encounters. Through modulation of the extent and duration of remodeling of the cell wall, epigenetic processes may allow for optimization of trade-offs versus rapid defense activation and growth maintenance or future preparation [11]. Here, we present the current evidence for such crosstalks between epigenetic regulators and properties of the cell wall in a bid to shed light on communication between the nucleus and cell wall under stress and through such communication why plants are resilient.

2 Mechanistic layers of epigenetic control in ECM remodeling

Epigenetic processes control the gene networks of the plant extracellular matrix (ECM) determining cell wall composition and remodeling via four main layers.

2.1 DNA methylation as a regulatory switch

Coordinate regulation of cellulose and lignin biosynthesis gene clusters via dynamic cytosine methylation. Switch from dormancy to growth in poplar is associated with DNA hypomethylation within gene promoters for the up-regulation of cell wall biosynthesis [12]. It induces hypermethylation of NAC master regulators (NST1/SND1) at elevated temperatures in birch, inhibiting expression and reducing lignin accumulation [13]. Experimental demethylation of NAC promoters induces secondary wall formation and xylem initiation [12]. Activating demethylation on CCoAOMT1 and PRX25-type loci during tracheary element differentiation establishes DNA methylation as a tunable switch for ECM gene networks [14]. Such promoter methylation–demethylation switches are represented in Panel A, where DNA methylation directly modulates NAC regulators of secondary wall thickening (Figure 1).

2.2 Histone modifications in cell wall gene regulation

H3K4me3 and histone acetylation activate cell wall gene transcription, while H3K27me3 maintains repression [12]. Lignin biosynthetic enzymes CAD2/CAD3 show H3K4me3 enrichment in developing Eucalyptus wood [12]. Salt-stressed maize roots exhibit expansin and xyloglucan endotransglucosylase up-regulation with elevated H3K9ac [12, 15]. H3K27me3 removal under stress de-represses targets: salt-responsive HKT1 activation requires H3K27me3 stripping in Arabidopsis [16]. Cold exposure decreases H3K27me3 at silenced loci while increasing H3K4me3 at others [16-17]. This H3K4me3/H3K27me3 antagonism provides bistable switching for cell wall gene regulation.

2.3 Chromatin remodeling and accessibility of ECM genes

SWI/SNF complexes of BRAHMA utilize ATP hydrolysis to displace nucleosomes, regulating cell wall gene promoter accessibility [18]. Thousands of Arabidopsis sites are bound by BRM, targeting modifying enzymes of the cell wall such as pectin methylesterase inhibitors (PMEIs) [18]. SWI/SNF disruption triggers overall cell wall gene misregulation [18]. BRM acts on both promoters and on terminator regions, impacting antisense RNA synthesis and chromatin structure in invertase/PMEI superfamily genes [18].

2.4 Epitranscriptomic regulation of cell wall biosynthesis

N^6-methyladenosine [m^6A] modifications alter mRNA stability and translation. Drought-stressed apple trees have methyltransferases of MdMTA raising m^6A contents on transcripts of lignin biosynthesis, stabilizing mRNAs and up-regulating translation for increased deposit of lignin [19]. m^6A writers/erasers in Arabidopsis modify stress tolerance through secondary wall formation transcripts.

2.5 Small RNA-mediated post-transcriptional control

MicroRNA miR397 suppresses laccase gene targets, reducing accumulation of lignin. Enhanced activity of miR397 in wild rice correlates with reduced laccase expression and flexible, elongated cell walls [20]. Lack of miR397 results in enhanced laccase gene expression and hyper-lignification [20-21]. Further small RNAs are directed against expansins, peroxidases, and glycosylases.

2.6 Summary

NAC and MYB transcriptional masters accept epigenetic inputs and output to direct downstream expression. H3K4 methyltransferase ATX1 deposits H3K4me3 on NAC locations (SND1/NST1), boosting and maintaining secondary wall activation [22]. H3K4 demethylase JMJ14 works in concert with NAC factors (ANAC050/052), co-repressing genes to fine-tune wall deposition time.

A three-protein complex with GCN5 acetyltransferase and ADA2b adaptor in poplar, controlled by AREB/ABF, triggers hyperacetylation and high-level transcription of xylem differentiation master regulators. Early differentiation activates chromatin remodeling and DNA demethylation in opening NAC/MYB promoters; expressed TFs subsequently turn on structural downstream genes and recruit histone modifiers to finalize the secondary wall program.

Such multi-level regulation necessitates secondary cell wall biosynthesis in the appropriate cells and at the proper time, yet is responsive to both internal cueing and environmental stress. Through the integration of DNA methylation, histone modification, chromatin remodeling, and RNA-dependent mechanisms with master transcription factors, it is possible to achieve rigorous control of cell wall genes in a way such that plants can build, modify, or strengthen cell walls in a coordinated manner strategic for growth and accommodation.

This mechanistic link between chromatin modifiers and ECM gene networks is concisely represented in Panel A, where transcriptional control directly interfaces with wall biosynthesis pathways (Figure 1).

3 Epigenetic strategies tailored to specific stress conditions

Plants employ stress-tailored epigenetic mechanisms to regulate cell wall genes. These divergent yet convergent chromatin strategies ultimately remodel the ECM, as depicted in the multi-layered wall cross-section of Panel B (Figure 1).

3.1 Drought and salinity-induced epigenetic reprogramming

Drought induces lignin production via DNA hypermethylation in the regions of lignin biosynthesis, stiffening the walls and reducing water loss in water-tolerant varieties such as rice [23-24]. Salinity induces histone acetylation. HAT GCN5 acetylates H3K9/K14 in the promoters of the cellulose synthase and remodeling genes (CTL1, PGX3), maintaining cell wall integrity [25]. It is uncovered in Arabidopsis gcn5 mutants, with a brittle root and hypersensitivity to salt, revealing such a mechanism [25]. Both are examples of varied yet similar tactics (DNA methylation and histone acetylation) strengthening the cell wall.

3.2 Temperature extremes and epigenetic memory

Heat stress primes wall-remodeling genes (XTH, expansins) for fast re-induction in recurrence with stable H3K4me3 and acetylation [26]. Arabidopsis Heat Shock Protein loci are prototypical for such heat memory. Cold also induces H3 acetylation in antifreeze protein and extensin promoters, defining cold memory. Marks are not indelible, being reset through demethylases and deacetylases to balance growth with preparation [27]. Temperature stress thus identifies H3K4me3 and acetylation as epigenetic bookmarks for previous exposure-future resilience associations.

3.3 Pathogen-triggered epigenetic modulation

Pseudomonas syringae infection induces genome-wide DNA hypomethylation in Arabidopsis, particularly in regions surrounding transposons and defense genes, activating immunity and wall reinforcement [28-29]. Callose synthase genes are correlated with H3K4me3 and H3K9ac enrichment [30-31]. RdDM-null nrpe1 mutants are stronger in defense, and hypermethylated ros1 mutants are weakened in defense, defining DNA demethylation's role in ensuring comprehensive defense [29]. Fine-tuning also occurs: JMJ14 demethylase removes H3K4me3 in some locations to avert overactivation [26]. Hence, infection response integrates demethylation with active histone marks to enhance the wall.

3.4 Herbivory-induced chromatin dynamics

Insect foraging activates JA signaling, triggering lignification and suberin formation. At the epigenetic level, chromatin accessibility increases at JA-responsive promoters, and defense genes are primed with increased basal H3K4me3 upon earlier herbivory for rapid activation [32]. Certain marks are transgenerational such that offspring of damaged plants possess inherited resistance through differential DNA methylation and prolonged acetylation [33]. Candidate targets are polyphenolic cross-linking and callose formation genes in favor of wall reinforcement.

3.5 Mechanical stress and epigenetic mechanoadaptation

Long-term contact (thigmomorphogenesis) enhances walls through histone and DNA methylation modifications. TCH Arabidopsis genes are induced with H3K4me3 formation and H3K27me3 removal, generating open chromatin. SDG8 (H3K36me3 depositing) mutants are impair in responses, linking histone methylation to mechanoadaptation [34]. Plants

possess short-term memory, with cell wall genes epigenetically primed for initial early re-induction subsequent to sequential bending or wind [35].

3.6 Summary

Stress-specific mechanisms focus on chromatin regulation of wall genes. H3K4me3 is a priming universal mark in both abiotic as well as biotic stresses [26,31]. Abiotic stresses emphasize DNA methylation and histone acetylation, yet biotic stresses are more relying on DNA demethylation and chromatin opening [25,28]. DNA methylation and histone modifications hence converge, stress-type-tuned. Such a flexible system enables cells within a water stress-experiencing plant to reform the cell walls through priming universal memory marks and stress-specialized signatures in a way to remain resistant against water stress, salinity, heat and chill extremes, pathogens, herbivory, as well as mechanical forces.

By placing these diverse stress responses into a structural context, Panel B demonstrates how chromatin-level changes translate into visible alterations of wall architecture (Figure 1).

4 Regulatory network integration and feedback mechanisms

Transcription factors (TFs) and epigenetic modifiers integrate extracellular and hormonal signals with chromatin dynamics, ensuring precise yet flexible regulation of cell wall genes. Wall integrity sensors and receptor kinases exemplify the ECM-to-nucleus signaling loops illustrated in Panel C, showing how extracellular perturbations feed back into chromatin remodeling (Figure 1).

4.1 Transcription factor–epigenetic complexes in cell wall control

Stress-responsive TFs often cooperate with chromatin remodelers. In Arabidopsis, WRKY12/13 fine-tune lignification by regulating NAC and MYB master regulators. These WRKYs physically interact with the SWI/SNF remodeler (BRAHMA) to open lignin gene promoters under drought or pathogen stress. Similarly, R2R3-MYB factors in flavonoid biosynthesis recruit histone methyltransferases (HMTs) to deposit activating marks at target loci [36]. NAC regulators (e.g. SND1/NST1) illustrate epigenetic feedback: reduced DNA methylation at a ramie NAC homolog correlates with its elevated expression and thicker fiber walls [37]. Thus, TF–epigenetic coupling allows environmental cues to be imprinted on chromatin states governing wall biosynthesis.

4.2 ECM Sensing and nucleus-directed feedback loops

Cell wall integrity sensors transduce extracellular changes to the nucleus. Wall-associated kinases (WAKs) perceive pectin fragments released upon wounding or pathogen attack and activate MAPKs MPK3/6, leading to defense gene induction [38]. FERONIA, another receptor-like kinase, senses wall loosening under salt stress and triggers Ca^{2+} signaling, modulating chromatin accessibility at downstream loci [39]. These sensors establish a feedback loop: wall perturbation → kinase signaling → nuclear chromatin remodeling, ensuring rapid activation of cell wall fortification pathways.

4.3 Hormonal crosstalk with the epigenome

Plant hormones modulate chromatin at wall-related genes. Auxin promotes cell expansion by enabling ARF TFs to recruit histone acetylation machinery at expansin loci [36]. Jasmonate (JA) relieves repression by JAZ proteins, activating MYC2 and recruiting histone acetyltransferases/demethylases, thereby elevating defensive metabolites and reinforcing the wall [39]. Gibberellin (GA) induces DELLA degradation, enhancing histone acetylation at growth-related wall genes, while brassinosteroids (BR) activate BES1/BZR1, which interact with chromatin modifiers to stimulate expansin, cellulose, and xylan synthase expression [36]. GA–BR convergence at DELLA–BZR1 nodes exemplifies hormone crosstalk embedded in chromatin states. Collectively, hormones integrate growth–defense trade-offs through epigenetic regulation of cell wall programs.

4.4 Chromatin-based stress memory mechanisms

Transient stress often leaves stable chromatin signatures. Elevated H3K4me3 at defense and wall gene promoters persists in Arabidopsis, priming faster transcription upon re-stress [36]. DNA methylation also underlies long-term memory: drought and salt induce new methylation patterns transmitted to progeny, correlating with altered wall composition and tolerance [36]. In rice, pathogen infection increases H3K9ac at WRKY TF loci, activating defense responses while establishing heritable chromatin marks [40]. These memory mechanisms operate on multiple timescales: immediate priming (histone marks), mitotic stability (DNA methylation), and even transgenerational inheritance, collectively "bookmarking" cell wall fortification pathways.

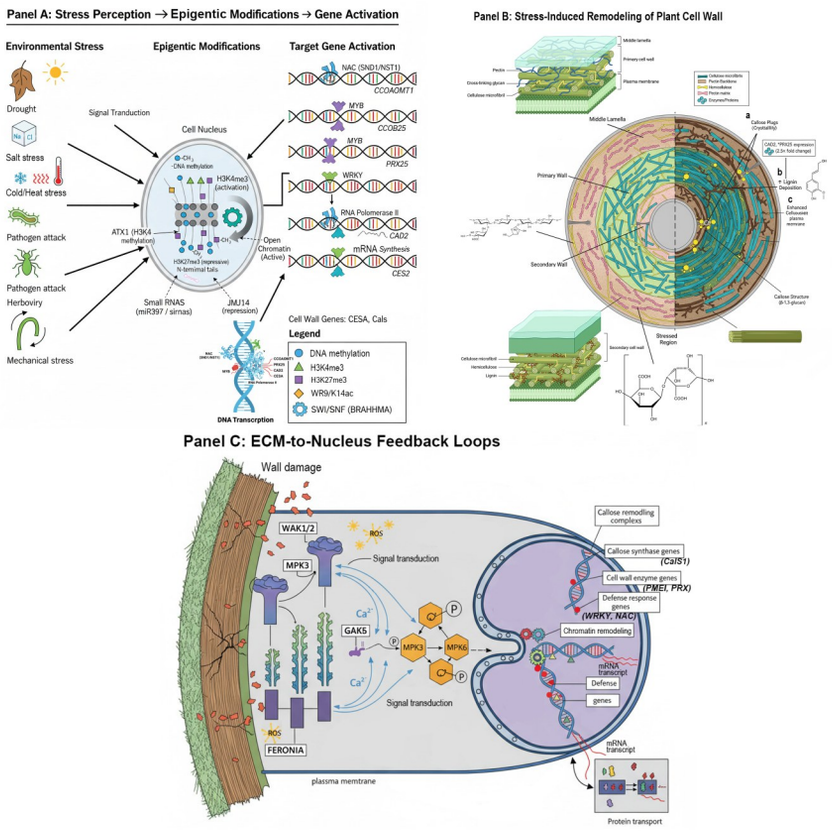

Figure 1 Epigenetic regulation of stress-induced plant extracellular matrix remodeling. (A) Environmental stressors such as drought, salinity, temperature extremes, pathogen attack, herbivory, and mechanical forces initiate signaling cascades that converge on nuclear chromatin. DNA methylation, histone modifications (H3K4me3, H3K27me3), SWI/SNF remodeling, and small RNAs regulate transcription of NAC, MYB, and cell wall biosynthetic genes (CESA, Cals).(B) Structural cross-section of the cell wall shows stress-induced remodeling, including lignification, callose deposition, and cellulose rearrangements across middle lamella, primary, and secondary walls. Enzymes such as CCoAOMT1, laccases, and peroxidases mediate polymer reinforcement.(C) Cell wall perturbations are sensed by receptor kinases (WAK1/2, FERONIA), activating MAPK and Ca^{2+} pathways that feed back to the nucleus, promoting transcription of CALS and defense-related genes (PME, PRX). Together, these panels illustrate a bidirectional loop between chromatin remodeling and ECM plasticity, enabling plants to integrate defense, growth, and memory.

4.5 Summary

Together, TF – epigenetic complexes, ECM sensors, hormone crosstalk, and chromatin-based memory form a multi-layered feedback network. Signals from walls or hormones converge on chromatin, dynamically balancing growth and defense while encoding experience into heritable states. Across species from Arabidopsis to rice, this integrative framework demonstrates how plants remodel their walls through stress-responsive chromatin reprogramming, ensuring both rapid adaptability and long-term resilience. As shown in Panel C, ECM-derived cues are not passive outcomes but active regulators that shape nuclear states through receptor-mediated feedback, establishing a self-reinforcing regulatory loop (Figure 1).

5 Discussion

Epigenetic control framework of ECM gene networks is characterized by three overriding controversies. First, mark specificity vs. pleiotropy: drought induces genome-wide remodeling of methylation, raising the possibility of whether cell wall control is targeted or incidental. While general stress responses suggest pleiotropy, specific instances such as H3K4me3 enrichment at callose synthase genes or demethylation of pectinase genes in support of semi-specific reprogramming. Secondly, reversibility vs. persistence of marks: the majority of stress-triggered changes are rapidly eliminated upon recovery in accordance with plasticity, while others are resilient and are the basis for stress memory and even multi-generational persistence [41]. Thirdly, the extent of transgenerational inheritance remains in contention. In rice, inherited methylation increases the tolerance of plants for water shortage, but opposite results lacked replicability or assumed hidden genetic variation for explanation [42]. Whether prolonged stress or an acute severe stimulus underlies long-term epigenetic memory remains uncertain and has major implications for adaptation and breeding.

5.1 Critical research gaps

Different gaps hinder advances. Causality is lacking: the bulk of the evidence is correlative, employing mutants or chemical perturbations targeting the whole plant rather than targeted real-time assays [41]. Poor temporal resolution also exists: frozen methylomes and ChIP-seq snapshots obscure how fast chromatin movements precede events of transcription and wall remodeling [41]. Cell-type specificity is similarly obscure: bulk tissue profiles mask the heterogeneity between epidermis/cortex/xylem, even though single-cell ATAC-seq and RNA-seq now reveal such dynamics . Integration across layers is also limited: DNA methylation, histone marks, chromatin access or ncRNAs are assayed individually in the bulk of research studies while overlooking cross-talk such as DNA methylation congealing H3K27me3 or m6A feedback circuits. Trade-offs and negative effects also need studies: 5-azacytidine remedies induce resilience but also cause the activation of transposons or stunt growth [40]. Finally, correlations of epigenetic marks and wall traits (e. g., H3K27me3 enrichment of expansin genes corresponding to rigidity) are not accompanied by matching chemistry or mechanics of the wall. Eliminating these lacunae requires multi-dimension high-resolution approaches correlating chromatin states and ECM phenotypes.

6 Future directions

Different strategies promise breakthroughs. High-level mapping by CRISPR/dCas9 editing enables causal testing by adding or deleting defined marks (e.g., promoter demethylation of pectinase genes, inducible acetylation at expansins). Locus-specific interventions move beyond correlation to permit exact "epimutations" for probing functions. Single-cell and live-cell epigenetics will resolve heterogeneity and kinetics: sorting salt-stressed cells from roots should reveal cell-type – specific chromatin changes, and biosensors for histone acetylation or chromatin compaction would track stress responses in real time. Spatial epigenomics will put changes in context in situ, showing for example epidermal cells at fungal entry points shedding H3K27me3 across callose synthase genes while distant cells are unaffected.

On the applied side, crop improvement potentially could employ epigenetic priming (under mild stress or chemicals) to establish desirable heritable states. Epigenomic selection by biomarkers like methylation at discrete loci potentially could accelerate breeding for stress robustness [41]. Regulatory circuit design is a new frontier too: synthetic factors potentially could elicit wall strengthening genes while recruiting chromatin modifiers for a stress memory, or tissue-specific HDAC tuning potentially might regulate elasticity during water shortage with lesser pleiotropic side-effects. Sustainability and environment implications are also significant: microbes and the soil itself can dictate host epigenomes and so potentially agronomic practices can use natural epigenetic modulators to enhance resilience [42].

7 Conclusion and outlook

Epigenetic regulation offers plants balance in plasticity and memory and a molecular switchboard for the environment and extracellular architecture. Future progression is dependent upon the combination of precision editing and temporal and spatial profiling and translational approaches for breeding. Such advancements will resolve fundamental debates, establish causality, and yield useful tools for sustainable production in a variable climate.

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