antioxidants

MDPI

Review

Beyond the Oxidative Window: Revisiting ROS Signaling in Seed Germination Under Abiotic Stress

Christophe Bailly [1,2,*] and Xiujie Mu [1,2]

[1] Development, Adaptation and Ageing, Dev2A, Sorbonne Université, CNRS, Inserm, F-75005 Paris, France; xiujie.mu@sorbonne-universite.fr
[2] Institut de Biologie Paris-Seine, IBPS, Sorbonne Université, CNRS, Inserm, F-75005 Paris, France
* Correspondence: christophe.bailly@sorbonne-universite.fr

Abstract

Reactive oxygen species (ROS) play essential roles in seed dormancy alleviation and germination, acting as both signaling molecules and potential sources of oxidative damage. The oxidative window concept proposes that successful germination requires ROS accumulation within a permissive range. However, emerging evidence indicates that this framework needs to be reconsidered under environmental stress. This review synthesizes physiological, molecular and transcriptomic evidence on ROS regulation during seed germination under abiotic constraints. A comparative analysis of transcriptomic datasets across plant species reveals recurrent ROS-associated transcriptional signatures, supporting the involvement of redox-related regulatory networks in stress responses. Importantly, environmental stresses do not systematically increase ROS accumulation, and germination inhibition may also result from insufficient oxidative signaling. We propose an extended oxidative window framework incorporating three complementary dimensions: the temporal dynamics of ROS accumulation, defined as oxidative trajectories; the spatial organization of ROS signaling across seed tissues and cellular compartments; and oxidative memory, through which maternal environmental conditions may influence subsequent seed redox responses. Together, these concepts suggest that germination depends not only on ROS abundance but also on the timing and localization of oxidative signals. This framework provides new perspectives for understanding seed germination and improving seed performance under environmental stress.

Keywords: abiotic stress; maternal environment; oxidative window; oxidative memory; reactive oxygen species; redox signaling; seed germination; ROS spatial dynamics

Check for updates

Academic Editor:
Marta Jańczak-Pieniążek

Received: 1 November 2026
Revised: 22 November 2026
Accepted: 23 November 2026
Published: 25 November 2026

Copyright: © 2026 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license.

1. Introduction

Abiotic environmental stresses affect multiple stages of the plant life cycle and climate change is intensifying many of these constraints with consequences for plant development and agricultural production [1], including seed development and subsequent germination, posing challenges to seed quality and crop establishment [2]. Seed development encompasses the processes occurring from fertilization to the formation of a mature seed, including embryogenesis, reserve accumulation, acquisition of desiccation tolerance and maturation drying [3]. Germination, in contrast, begins with water uptake by a dry seed and ends with radicle protrusion through the surrounding seed tissues [4,5]. Environmental conditions can shape seed performance both through their effects during seed formation and through their direct action during germination [6].

Maternal environmental cues, including temperature, photoperiod, drought, light quality and nutrient availability, influence seed dormancy, vigour, quality and stress tolerance, contributing to offspring developmental plasticity and adaptation [7–10]. These maternal effects may persist across generations, although their classification as intergenerational or transgenerational memory depends on the duration of their persistence and the generations directly exposed to stress [11,12]. Subsequent seed germination is an actively regulated developmental process whose timing reflects both the physiological state of the seed and the environmental conditions it encounters [5]. The timing and uniformity of germination have important agricultural consequences. In crops, rapid and uniform germination contributes to timely emergence and the formation of a homogeneous stand, whereas delayed or heterogeneous germination can compromise establishment. Although germination alone does not determine final yield, seed vigour and successful emergence provide an essential foundation for crop productivity [13]. The ability to germinate is influenced by genetic background, dormancy status and seed physiological quality, while progression towards germination completion depends strongly on water availability, temperature, oxygen and, in some species, light [5,13]. These environmental cues are integrated through regulatory networks in which abscisic acid (ABA) and gibberellins (GA) play central, generally antagonistic roles. Interactions with other hormones, including ethylene, further modulate embryo growth and the weakening of surrounding tissues [14,15]. Germination also involves distinct developmental programmes in the embryo and endosperm, as demonstrated by compartment-resolved transcriptomic analyses in *Arabidopsis* [16].

In plants, reactive oxygen species (ROS) held a central position at the interface between metabolism, development and environmental responses. Their functions extend far beyond their capacity to cause oxidative damage since regulated ROS production contributes to cellular signaling and to the coordination of physiological responses [17]. Their regulatory roles in seeds have progressively become established through studies demonstrating that ROS contribute to dormancy alleviation and germination completion. Pioneering studies established a regulatory role for ROS in seed dormancy alleviation and germination [18–20]. ROS influence several processes required for germination completion, including the regulation of ABA catabolism and GA biosynthesis, as shown for hydrogen peroxide in *Arabidopsis* [21]. They also contribute to the mechanical changes enabling radicle protrusion, notably through hydroxyl radical-mediated cell wall loosening during endosperm weakening and embryo elongation in cress seeds [22]. The dual role of ROS in seed physiology was formalized through the concept of the "oxidative window for germination", which proposes that germination-associated processes occur within a permissive range of ROS levels, limited by insufficient oxidative signaling at the lower limit and damaging oxidation at the upper limit [23]. This concept provides a useful framework for interpreting the requirement for ROS alongside their potential toxicity. The spatial and temporal regulation of ROS during seed germination, together with evidence for compartment-specific redox regulation, indicates that the biological consequences of redox changes depend on where and when they occur [20,24,25]. A central question remains on whether abiotic stresses can prevent germination by promoting damaging oxidation, restricting the ROS-dependent processes required for growth, or disrupting the coordination of redox events across tissues and organelles.

In this review, we synthesize current knowledge of the roles of ROS and redox regulation during seed germination under abiotic stress, integrating physiological, biochemical, genetic and transcriptomic evidence. Beyond this synthesis, we examine how the oxidative window might be refined to incorporate ROS identity, cellular origin, tissue distribution and temporal dynamics. We also explore endosperm–embryo redox communication and the possibility that environmental conditions during seed development establish persistent

changes in subsequent seed redox responses. These emerging perspectives are presented as hypotheses requiring mechanistic validation.

2. Seed Germination in Challenging Environments

In natural and agricultural environments, seeds frequently encounter abiotic conditions that restrict germination. These include for example low water availability, salinity, supraoptimal or suboptimal temperatures, and oxygen limitation associated with waterlogging or poorly aerated soils. Depending on their intensity, duration and timing, these constraints can delay germination or fully prevent its completion. Their effects involve interacting changes in hydration, energy metabolism, ion homeostasis and hormonal regulation [2,13]. Stress-induced inhibition can also arise through regulated developmental responses. For example, high temperature promotes ABA biosynthesis and restricts GA action in *Arabidopsis* seeds, while salinity shifts ABA and GA biosynthesis towards germination inhibition in soybean [26,27]. These mechanisms emphasize that failure to germinate under stress cannot automatically be attributed to irreversible cellular damage. Delayed radicle protrusion, reversible inhibition and loss of seed viability represent distinct outcomes that must be distinguished experimentally [13,28]. Kranner et al. [28] described three successive phases of stress response: alarm, resistance and, under severe or prolonged stress, exhaustion. Stress does not necessarily cause irreversible damage, as seeds can activate protective mechanisms to maintain their physiological functions. ROS and redox regulation play important roles in these responses, contributing to stress adaptation but also to cellular damage when stress becomes excessive.

In addition, abiotic stress during reproduction and seed development affects both yield formation and the properties of the resulting seeds. Temperature stress can impair pollen development and fertilization, reducing seed set, while drought and heat during seed filling can restrict assimilate supply, alter reserve deposition and shorten the filling period. Consequently, stress can reduce seed number, individual seed mass and harvestable yield, as well as modify seed composition and physiological quality [29,30]. Beyond these effects on production, environmental conditions during seed maturation influence progeny dormancy, vigour, germination requirements and stress tolerance through changes in seed provisioning, developmental regulation and embryo-surrounding tissue [2,8,9]. Such maternal environmental effects are increasingly recognised as important components of developmental plasticity, potentially allowing offspring responses to reflect maternal environmental cues. Considerable progress has been made in understanding temperature effects, particularly in *Arabidopsis*, where the seed developmental environment influences ABA metabolism, chromatin organisation and the expression of dormancy regulators [31]. Water, stress, photoperiod, light quality and nutrient availability can also modify seed developmental programmes and subsequent germination behaviour [7,10,31]. In sunflower, drought during seed development reduced dormancy and enhanced germination under water deficit, salinity, low temperature and hypoxia [32]. These effects are context dependent and may either impair progeny performance or favour germination under particular conditions. To understand seed responses to environmental stress and the role of redox regulation, it is important to consider both the conditions experienced during seed development and those encountered during germination.

3. The Redox Landscape of Seed Development and Germination

Developing seeds undergo profound physiological and metabolic changes, including embryogenesis, reserve accumulation, acquisition of desiccation tolerance and progressive metabolic quiescence. These transitions are likely accompanied by changes in ROS production and antioxidant metabolism, yet the extent to which ROS actively participate in

their regulation remains largely unexplored and only a limited number of studies have examined ROS dynamics during seed development. In sunflower seeds, Bailly et al. [33] reported a progressive decrease in hydrogen peroxide (H_2O_2) content during seed maturation, accompanied by changes in catalase activity and expression, suggesting that ROS homeostasis is developmentally regulated. Similarly, a subsequent study in sunflower [34] described changes in ROS accumulation and antioxidant enzyme activities during seed development. In maize, Huang and Song [35] investigated oxidative metabolism during embryo development and showed that changes in H_2O_2 content and antioxidant activities were associated with the acquisition of desiccation tolerance. Wu et al. [36] further reported modifications in mitochondrial metabolism and ROS-scavenging enzyme activities during maize embryo maturation, suggesting that adjustments in mitochondrial redox metabolism may accompany the establishment of desiccation tolerance. In *Arabidopsis*, Cairns et al. [37] demonstrated that glutathione biosynthesis within the embryo is essential for successful seed maturation, indicating that the maintenance of cellular redox metabolism is critical during late seed development. These studies indicate that ROS concentrations and antioxidant systems undergo substantial changes during seed development. However, most of the available evidence remains descriptive or correlative, and the physiological significance of these changes has not been clearly established. In particular, it remains uncertain whether changes in ROS homeostasis actively regulate developmental processes such as embryo differentiation, reserve accumulation, acquisition of desiccation tolerance or the establishment of dormancy, or whether they primarily reflect the metabolic adjustments associated with seed maturation and dehydration. Finally, whether environmental conditions experienced by the mother plant influence ROS homeostasis in developing seeds, and whether such changes contribute to subsequent seed physiological quality, remains largely unknown. Establishing whether ROS act as genuine developmental signals, rather than simply reflecting changes in metabolic activity or oxidative protection, represents an important challenge for understanding how seed maturation determines subsequent seed performance.

Upon imbibition, the metabolism of mature dry seeds is rapidly reactivated, resulting in the production of reactive oxygen species (ROS), including superoxide ($O_2^{\bullet-}$) and hydrogen peroxide (H_2O_2), as by-products of aerobic metabolism [23]. Unlike photosynthetic tissues, in which chloroplasts represent major sites of ROS production, germinating seeds rely predominantly on non-photosynthetic sources of ROS, particularly during the early stages of imbibition, before the establishment of photosynthetic activity. In these conditions, chloroplast-derived ROS production associated with photosynthetic electron transport is absent or negligible, and ROS originate from several intracellular compartments and the apoplast (Figure 1). Mitochondrial metabolism resumes rapidly following imbibition and represents an important source of ROS during early germination [24,25]. Electron leakage from the mitochondrial electron transport chain (ETC), particularly at complexes I and III, results in the reduction of molecular oxygen to $O_2^{\bullet-}$, which can subsequently be converted to the more stable H_2O_2 (Figure 1). Pharmacological disruption of mitochondrial function alters ROS production and delays *Arabidopsis* seed germination, supporting a role for mitochondrial ROS during germination [25]. The alternative oxidase (AOX) pathway provides an additional means of controlling mitochondrial redox status by maintaining electron flow and limiting over-reduction of the ETC. Accordingly, perturbation of AOX activity affects ROS accumulation and germination, particularly under adverse environmental conditions [38]. In the cytosol, xanthine dehydrogenase (XDH), involved in purine catabolism, may generate $O_2^{\bullet-}$ and H_2O_2 under particular biochemical conditions. Cytosolic polyamine oxidase (PAO) isoforms can also produce H_2O_2 through polyamine oxidation. Lipoxygenases (LOXs) catalyse the oxygenation of polyunsaturated fatty acids, generating lipid hydroperoxides that may contribute indirectly to oxidative signaling and

lipid oxidation [39]. Peroxisomes may provide another important source of ROS as reserve mobilization becomes established during germination. Peroxisomes and glyoxysomes generate H_2O_2 through several oxidative metabolic pathways, including fatty acid β-oxidation catalysed by acyl-CoA oxidases (ACXs), glycolate oxidation by glycolate oxidase (GOX), polyamine catabolism by polyamine oxidases (PAOs), purine degradation involving urate oxidase (UOX), and sulfite oxidation by sulfite oxidase (SO) [40]. These pathways may contribute to intracellular redox homeostasis during seed imbibition, reserve mobilization and responses to environmental stress. Acyl-CoA oxidases (ACXs) catalyse the first step of fatty acid β-oxidation and produce H_2O_2 (Figure 1). The *Arabidopsis acx1-2 acx2-1* double mutant shows reduced germination, supporting a role for peroxisomal β-oxidation during germination [41]. ROS are also generated at the plasma membrane by NADPH oxidases, or respiratory burst oxidase homologues (RBOHs) (Figure 1). Pharmacological inhibition of NADPH oxidase activity restricts germination, while *rbohD* seeds display enhanced dormancy [20]. RBOH-derived apoplastic ROS may facilitate radicle elongation and endosperm weakening through ROS-mediated cell-wall loosening, including oxidative cleavage of cell-wall polysaccharides [42–44]. In the apoplast and cell wall, additional ROS-producing systems include copper-containing amine oxidases (CuAOs) and apoplastic PAOs, which generate H_2O_2 through polyamine catabolism, and class III peroxidases (PRXs), which can either consume or promote ROS production depending on their catalytic cycle and substrate availability. Oxalate oxidases (OXOs) constitute an additional, species-dependent source of apoplastic H_2O_2. ROS homeostasis during seed germination results from a dynamic balance between ROS production and scavenging, involving both enzymatic and non-enzymatic antioxidant systems. Enzymatic mechanisms include superoxide dismutase (SOD), catalase (CATs) and enzymes of the ascorbate–glutathione cycle, while non-enzymatic antioxidants, such as ascorbate, glutathione and tocopherols, contribute to maintaining cellular redox balance. The regulation of these antioxidant systems, which has been extensively investigated in seeds (see [45]), is therefore as important as ROS production in determining ROS accumulation and availability for signaling. Consequently, changes in ROS levels during germination reflect the coordinated regulation of ROS-producing and ROS-scavenging pathways, rather than changes in ROS production alone.

The effect of ROS on seed germination is strongly dose-dependent, as illustrated by the oxidative window concept proposed by Bailly et al. [23]. Germination occurs within a permissive range of ROS levels: insufficient ROS may fail to sustain germination-related cell signaling, whereas excessive accumulation leads to oxidation of proteins, lipids and nucleic acids and ultimately compromises seed viability. This dose dependence has recently been demonstrated in *Arabidopsis* using exogenous H_2O_2. Low H_2O_2 concentrations promoted germination, whereas concentrations above 2 mM became progressively inhibitory, with 10 mM H_2O_2 causing oxidative damage in radicle cells [46]. These concentrations refer to exogenous H_2O_2 treatments rather than endogenous ROS thresholds, but they illustrate the different effects of ROS depending on their level. Research over the past decade shows that the oxidative window cannot be defined solely from the total amount of ROS present in the seed. ROS production changes during imbibition and is spatially heterogeneous. In *Arabidopsis*, dormancy release is accompanied by ROS accumulation predominantly in the embryo axis and radicle, while their intracellular distribution also changes as imbibition proceeds. This spatial organization is consistent with the involvement of several ROS-producing systems during germination. Mitochondrial respiration, peroxisomal metabolism and plasma membrane RBOHs generate ROS in different cellular compartments and are associated with different metabolic or developmental processes. The ROS detected at the whole-seed level therefore represents the combined contribution of several spatially distinct ROS pools, potentially masking local changes associated with germination.

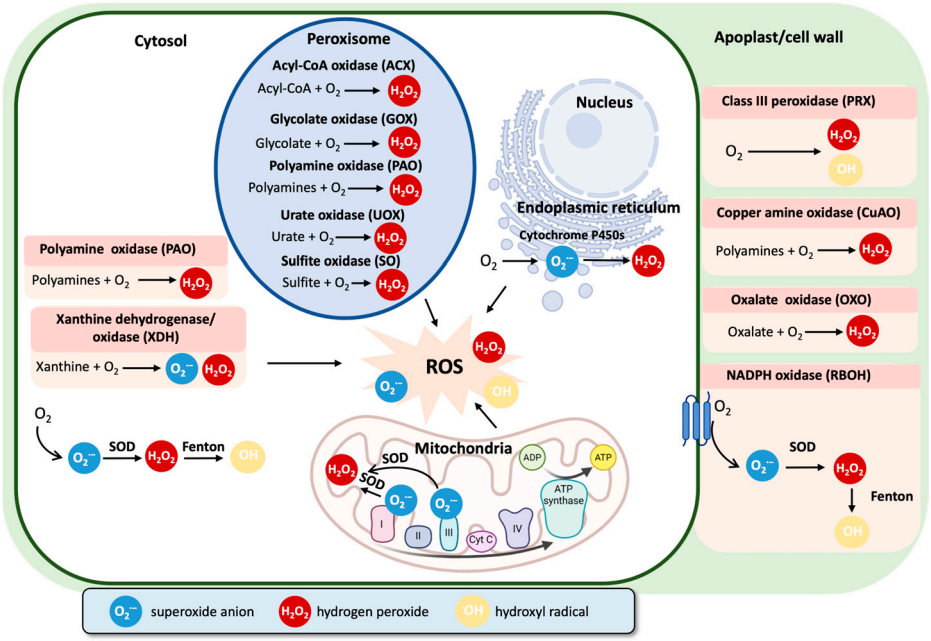

Figure 1. Subcellular sources of reactive oxygen species (ROS) in embryonic seed cells. Schematic representation of the major enzymatic systems and subcellular compartments involved in ROS production in non-photosynthetic plant cells potentially involved during seed germination. ROS production occurs in several cellular compartments, including mitochondria, peroxisomes, the endoplasmic reticulum, cytosol, plasma membrane and apoplast. Hydroxyl radicals (•OH) can also be generated through non-enzymatic Fenton reactions involving H_2O_2 and redox-active transition metals, particularly Fe^{2+}. The relative contributions of these ROS-producing systems depend on the tissue, developmental stage, and environmental conditions. Abbreviations: ACX, acyl-CoA oxidase; ADP, adenosine diphosphate; ATP, adenosine triphosphate; CuAO, copper-containing amine oxidase; GOX, glycolate oxidase; OXO, oxalate oxidase; PAO, polyamine oxidase; PRX, class III peroxidase; RBOH, respiratory burst oxidase homolog; ROS, reactive oxygen species; SOD, superoxide dismutase; SO, sulfite oxidase; UOX, urate oxidase; XDH, xanthine dehydrogenase; $O_2^{\bullet-}$, superoxide anion radical; H_2O_2, hydrogen peroxide; •OH, hydroxyl radical.

ROS coordinate with phytohormones to regulate seed germination. This interaction is particularly well documented for ABA and GA, which have opposing effects on germination. ABA generally limits ROS accumulation, and components of ABA signaling can directly regulate ROS homeostasis. In Arabidopsis, under salt stress, ABI4 directly activates *RBOHD* expression while repressing *VTC2*, thereby promoting ROS accumulation and inhibiting germination [47]. In parallel, ABI5 has been shown to directly activate *CAT1* transcription, linking ABA signaling to catalase-dependent regulation of ROS homeostasis during seed germination [48]. Conversely, exogenous H_2O_2 application influenced germination by modulating the ABA/GA balance in Arabidopsis seeds [21,49]. GA itself can stimulate ROS production, further illustrating the reciprocal relationship between hormonal and redox regulation [50,51]. A similar interaction occurs with ethylene. In sunflower, ethylene promotes dormancy release through NADPH oxidase-dependent ROS production [52], while interactions between ethylene and ROS have also been reported during germination in soybean and *Arabidopsis* [25,53]. Therefore, the interaction between ROS and phytohormones forms a complex network that coordinates germination process.

4. ROS and Abiotic Stress During Seed Germination

ROS accumulation and oxidative damage are widely associated with plant responses to abiotic stress. In vegetative tissues, drought, salinity and temperature extremes frequently

enhance ROS production, potentially leading to oxidative damage when ROS generation exceeds cellular antioxidant capacity [54–56]. This has also been widely applied to germinating seeds. However, because ROS production is itself required for germination, stress-induced changes in seed ROS cannot necessarily be interpreted in the same way as in vegetative tissues.

ROS metabolism is actively modified when seeds germinate under stress. Table 1 provides a non-exhaustive review of different studies having demonstrated that abiotic stress was likely to alter ROS homeostasis in germinating seeds of various species. However, in Table 1, we did not take into account the diversity of analytical approaches used to assess ROS accumulation. Fluorescent probes, histochemical staining and biochemical assays differ in their specificity, sensitivity and spatial resolution, and therefore do not necessarily provide directly comparable measurements. Moreover, whole-seed measurements may fail to capture tissue-specific and subcellular ROS dynamics that are particularly relevant to germination regulation.

Table 1. Involvement of ROS in responses of seeds to abiotic stresses during germination.

Species	Stress	Stress Level/ Treatment	Germination Sampling	ROS/Oxidative Markers	Antioxidant/ Redox Measurements	ROS/Redox Manipulation or Genes	Reference
Arabidopsis thaliana	NaCl	High salinity	Germinating seeds	H_2O_2	Antioxidant capacity/oxidative damage	SA; sid2; NahG; ascorbate	[57]
Arabidopsis thaliana	NaCl	High salinity	Germinating seeds	H_2O_2/ROS	—	SA; ABA/GA relationship	[58]
Arabidopsis thaliana	NaCl	100–200 mM NaCl	Germinating seeds	H_2O_2, NO	—	ACC/ethylene; SNP/NO; cPTIO; ein3-1	[59]
Arabidopsis thaliana	NaCl	100 mM NaCl	Dry seed to 36 h imbibition; ~50% germination	H_2O_2	Mitochondrial thiol-redox system	AtTrxo1 knockout; AtbZIP9; AZF2	[60]
Arabidopsis thaliana	NaCl	Salinity	Germination	ROS	NADPH/redox metabolism	Cytosolic G6PD; NADPH oxidase	[61]
Arabidopsis thaliana	Cadmium	Cd exposure	Germination assays	ROS	SOD/oxidative status	Allantoin metabolism	[62]
Arabidopsis thaliana	H_2O_2/oxidative stress	0–10 mM H_2O_2	Germinating seeds	H_2O_2, $O_2^{\bullet-}$ spatial distribution	ROS homeostasis	SDH2-3; RHD2/RBOH; PRXs; dose manipulation	[46]
Oryza sativa	Heat + chilling	Imbibitional temperature extremes	Early imbibition/germination	H_2O_2, $O_2^{\bullet-}$, TBARS, protein carbonyls	SOD, CAT, APX, GR; thiols	Exogenous H_2O_2	[63]
Oryza sativa	High temperature + drought	Combined HT and water deficit	Germinating seeds	H_2O_2, $O_2^{\bullet-}$, MDA; H_2O_2 flux	SOD, POX, CAT, APX	ABA; Ca^{2+}; OsNCED3; OsCATc; OsAPX2; OsCu/ZnSOD	[64]
Oryza sativa	NaCl + PEG	Salt and osmotic stress	Germination phase	ROS; lipid peroxidation	SOD, CAT, APX, GPX; AsA, GSH	Hydro-, halo- and UV-B priming	[65]
Oryza sativa	PEG/osmotic stress	−1.5 MPa PEG	Imbibed embryos; before germination completion	Endogenous ROS	—	NADPH oxidase genes; GA/ABA; aleurone experiments	[66]
Oryza sativa	PEG drought	PEG-induced drought	Germinating seeds	H_2O_2, $O_2^{\bullet-}$, MDA	SOD, POD, CAT, APX; GSH, AsA	Selenite treatment	[67]

Table 1. *Cont.*

Species	Stress	Stress Level/ Treatment	Germination Sampling	ROS/Oxidative Markers	Antioxidant/ Redox Measurements	ROS/Redox Manipulation or Genes	Reference
Helianthus annuus	Water deficit	Approx. −0.6 MPa	15 h imbibition, before radicle protrusion	H_2O_2/ROS, MDA	CAT, SOD, GR	Contrasting drought-tolerant and drought-sensitive hybrids	[68]
Medicago truncatula	PEG + NaCl	100 g L^{-1} PEG/100 mM NaCl	First hours of imbibition	ROS accumulation	Redox/ metabolic context	Trehalose pathway genes	[69]
Zea mays	NaCl	Salt stress following pretreatment	Germinating seeds	H_2O_2	APX, CAT, GPX	H_2O_2 pretreatment	[70]
Zea mays	Heat + heavy metals	Heat + Cu/Cd/Hg	Germination	H_2O_2	Antioxidant response	H_2O_2 + DMTU; NADPH oxidase	[71]
Zea mays	Low temperature	Cold germination	Germinating seeds	Oxidative status	SOD/ antioxidant metabolism	Contrasting genotypes	[72]
Hordeum vulgare	High temperature	35 °C germination ± 0 °C pretreatment	Germinating seeds	MDA	SOD, APX, CAT, GR	Temperature cross-acclimation	[73]
Hordeum vulgare	NaCl	Salinity	Germination	ROS/oxidative damage	SOD, CAT, GR, GPX, GSH	Nanopriming	[74]
Triticum aestivum	NaCl	Salinity	Seed pretreatment + germination	H_2O_2; oxidative damage	Antioxidant system	H_2O_2 pretreatment	[75]
Triticum aestivum	Drought	Water deficit	Germinating seeds	H_2O_2 signalling	CAT, POD, SOD, GR	ZnO nanopriming; H_2O_2	[76]
Triticum aestivum	NaCl	150 mM NaCl	24 and 72 h germinated seeds	H_2O_2, $^{\bullet}OH$, $O_2^{\bullet-}$, MDA	NAD(P)H/ $NAD(P)^+$; antioxidant defence	Tea polyphenols	[77]
Glycine max	PEG + NaCl + combined	Drought/ salinity	Germination	ROS, MDA	SOD, POD, CAT, APX	Contrasting cultivars	[78]
Glycine max	Drought	Osmotic/water deficit	Early germination	H_2O_2	Antioxidant capacity	GmRbohC2; ABA; NAA	[79]
Chenopodium quinoa	NaCl	Salinity	Seed germination stage	H_2O_2 + NO	Metabolic/re-dox effects	H_2O_2; SNP; $CaCl_2$ treatments	[80].
Chenopodium quinoa	NaCl	Various salinities	Germina-tion/embryo	Oxidative stress	Multiple antioxidant enzymes	Contrasting genotypes/ betalains	[81]
Fagopyrum tataricum	NaCl	50 mM NaCl	Germination	Oxidative damage/MDA	CAT, SOD	5–10 mM H_2O_2 pretreatment	[82]
Brassica oleracea var. botrytis	NaCl	Salt stress	Germination dynamics	ROS	Antioxidant defence	H_2O_2 priming vs. GA_3/NaCl priming	[83]
Brassica napus	NaCl	0.1 M NaCl	24 and 48 h germination	H_2O_2, TBARS	Total antioxidant capacity	H_2S/NaHS priming	[84]
Brassica rapa	NaCl	200 mM NaCl	Germination monitored	H_2O_2, MDA	SOD, CAT, POD, APX	Se priming; antioxidant genes	[85]

Table 1. *Cont.*

Species	Stress	Stress Level/ Treatment	Germination Sampling	ROS/Oxidative Markers	Antioxidant/ Redox Measurements	ROS/Redox Manipulation or Genes	Reference
Cucumis sativus	Heat	35 °C vs. 28 °C	24 and 48 h germination	H_2O_2	Non-protein thiols, antioxidant enzymes	Biostimulant manipulation	[86]
Solanum lycopersicum	Heat	High-temperature treatment	Germination + recovery	H_2O_2, $O_2^{\bullet-}$	Antioxidant response	SlIAA9 mutants; HSFA9; HSP70; ABA/ethylene genes	[87]
Limonium stocksii/ *Suaeda fruticosa*	NaCl	Salinity	Germination sensu stricto	H_2O_2, MDA	AsA/GSH; SOD, CAT, APX, GPX, GR	—	[88]
Anadenanthera peregrina/ *Myracrodruon urundeuva*	As + Zn	Arsenic/zinc toxicity	Germinating seeds	H_2O_2, MDA	SOD, CAT, APX, GR	—	[89]
Capsicum annuum	NaCl	0–200 mM NaCl	Germination	H_2O_2, MDA	Multiple antioxidant enzymes + osmolytes	—	[90]
Abelmoschus esculentus	NaCl × temperature	Combined salinity/temperature	Germination	MDA/oxidative stress	SOD, POD, CAT	—	[91]
Cucumis melo	NaCl	Salt stress	Germination	ROS/redox inferred	Antioxidant response	H_2O_2 treatment	[92]
Sorbus pohuashanensis	Salt–alkali stress	Na_2CO_3; PEG treatment	Isolated embryo germination	ROS + reactive nitrogen species	Redox physiological markers	PEG modulation; hormones; ROS/NO	[93]

Abiotic stress is often associated with increased ROS accumulation in germinating seeds, and excessive ROS production has been linked to oxidative damage and impaired germination (Table 1). However, contrary to the prevailing view, this relationship is not consistent across species and stress conditions. Increased ROS content, for example, is not always accompanied by oxidative damage. In sunflower, two hybrids with contrasting responses to water stress differed in H_2O_2 accumulation and antioxidant activities, but changes in H_2O_2 did not parallel lipid peroxidation [68]. An increase in ROS under stress therefore does not necessarily indicate that the upper boundary of the oxidative window has been exceeded. Conversely, stress-induced inhibition of germination can also occur with reduced ROS accumulation. Salt stress has been reported to delay *Arabidopsis* germination while decreasing H_2O_2 levels [60], while under polyethylene glycol (PEG)-induced water stress, the more sensitive indica rice material showed lower H_2O_2 accumulation and NADPH oxidase activity than the more tolerant material [66]. Recent work indicates that water stress can delay *Arabidopsis* seed germination by preventing the establishment of the ROS signaling required to initiate growth rather than by inducing excessive ROS accumulation [94]. In rice, inadequate H_2O_2 accumulation can contribute to impaired germination at an unfavorable temperature [95] and ROS availability contributes positively to germination under low water potential, potentially through GA biosynthesis [66]. In maize, under salt stress, omethoate treatment improved seed germination and increased H_2O_2 content relative to salt treatment alone [96]. Therefore, even though most studies assume that environmental stresses increase ROS production, evidence accumulated during the last decade suggests that this paradigm is overly too simplistic for seeds. Depending on the nature, intensity and timing of the stress,

ROS production may either increase or decrease, and alterations in ROS signaling may occur in the absence of detectable oxidative damage. Although the oxidative deficit hypothesis is supported by observations linking reduced ROS accumulation to impaired germination under abiotic stress, direct evidence remains limited. Importantly, our recent study on *Arabidopsis* germination under water stress [94] provides strong experimental support for this concept by combining ROS measurements, compartment-specific redox analyses, and transcriptomic profiling. Nevertheless, while these findings support the existence of an oxidative deficit under water stress, further genetic and biochemical investigations are needed to establish its causal contribution to germination inhibition and determine its relevance across species and environmental conditions.

5. ROS-Associated Transcriptional Footprints Reveal the Integration of Redox-Related Pathways into Seed Stress Responses

To go further and explore the involvement of ROS-associated regulatory networks in seed germination under abiotic stress, we performed a comparative analysis of publicly available transcriptomic datasets from seeds of different plant species exposed to abiotic stresses [94,97–101] using the ROS-wheel framework [102] (Supplemental Table S1). To account for differences in the magnitude of transcriptional responses, the number of genes assigned to ROS-wheel clusters was normalized to the total number of differentially expressed genes (DEGs) in each comparison. Remarkably, ROS-associated transcriptional signatures were consistently detected across species and stress conditions, generally representing approximately 3–8% of the identified DEGs (Figure 2A). This recurrent transcriptional footprint suggests that ROS-related regulatory networks constitute a common component of seed responses to environmental constraints during germination, despite differences in species, stress treatments and experimental conditions. This analysis revealed that different ROS-wheel clusters were selectively recruited, suggesting that stress-induced ROS-associated transcription is not associated with a uniform oxidative stress response but rather with a coordinated regulation of distinct signaling pathways (Figure 2B). In particular, the recurrent representation of the high-light-early cluster suggests the recruitment of conserved stress-responsive transcriptional programmes, even in non-photosynthetic germinating tissues. Furthermore, the detection of GUN-associated and RBOHF-associated transcriptional signatures suggests potential links with organellar retrograde signaling and ROS-dependent signaling pathways. Together, these patterns point towards the integration of ROS homeostasis, intercompartmental communication and stress acclimation during germination. Interestingly, the relative representation of ROS-wheel genes did not necessarily increase with the overall magnitude of the transcriptional response. This suggests that ROS-associated pathways are selectively regulated within broader stress-responsive networks rather than simply reflecting the extent of transcriptional reprogramming. Their contribution may therefore depend on the timing and coordination of specific redox-related processes rather than the absolute number of responsive genes, which provides cross-species transcriptomic evidence that ROS-associated regulatory networks are recurrently involved in seed responses to abiotic stress. They support the concept that ROS participate in the integration of environmental signals with the metabolic and developmental processes governing germination. Although these transcriptional footprints do not establish direct ROS-dependent regulation, they provide a molecular framework that is consistent with physiological and genetic evidence identifying ROS as important regulators of germination under environmental constraints.

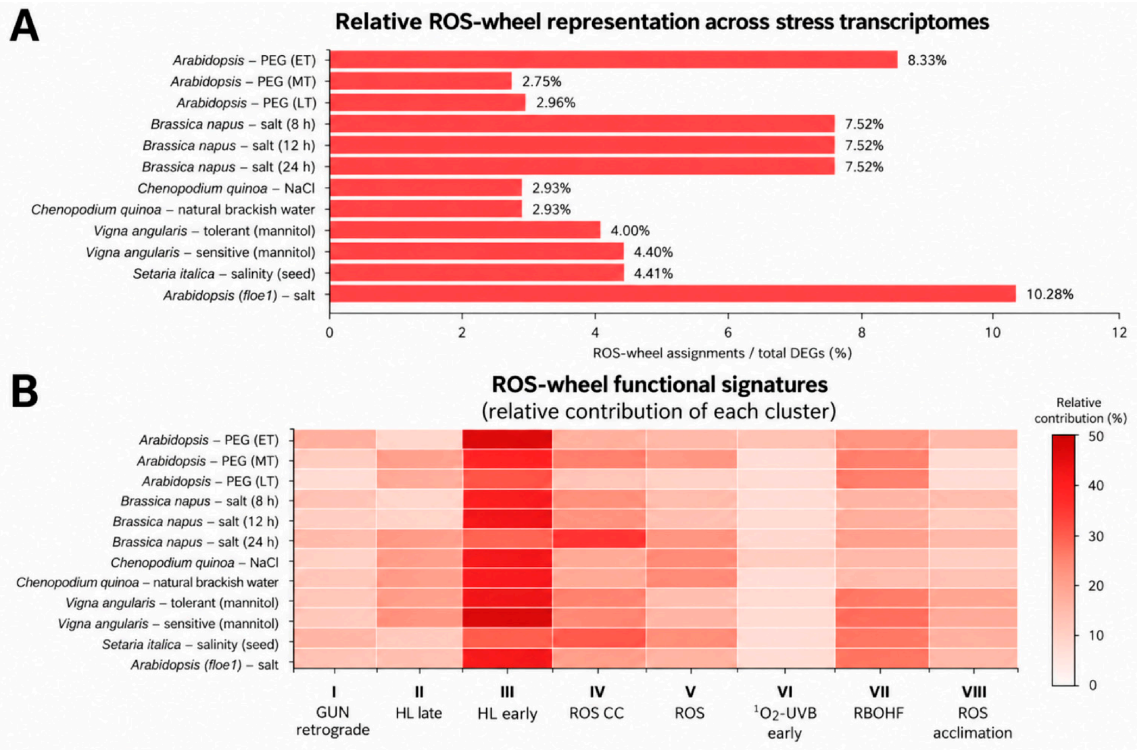

Figure 2. Representation and functional distribution of ROS-related genes across seed germination transcriptomes under abiotic stress. (**A**) Relative representation of ROS-wheel genes among differentially expressed genes. Horizontal bars indicate the proportion of DEGs assigned to ROS-wheel clusters relative to the total number of DEGs identified in each transcriptomic comparison, expressed as a percentage. Datasets include *Arabidopsis thaliana* seeds exposed to polyethylene glycol (PEG)-induced water stress at early (ET), middle (MT) and late (LT) germination stages; *Brassica napus* seeds exposed to salt stress for 8, 12 or 24 h; *Chenopodium quinoa* seeds exposed to NaCl or natural brackish water; *Vigna angularis* genotypes differing in tolerance to mannitol-induced osmotic stress; *Setaria italica* seeds exposed to salinity; and *Arabidopsis* seeds exposed to salt stress (floe1 mutant comparison). Percentages represent the relative contribution of ROS-wheel-associated genes to the overall transcriptional response rather than the absolute number of ROS-related DEGs. (**B**) Functional distribution of ROS-wheel-associated genes across transcriptomic comparisons. The heatmap shows the relative contribution (%) of each of the eight ROS-wheel functional clusters to the total number of ROS-wheel assignments within each comparison. Clusters correspond to (I) GUN retrograde signaling, (II) high-light late response (HL late), (III) high-light early response (HL early), (IV) ROS-related cell-cycle regulation (ROS CC), (V) general ROS responses (ROS), (VI) early singlet oxygen/UV-B response ($^1O_2$–UVB early), (VII) RBOHF-associated responses (RBOHF) and (VIII) ROS acclimation. Colour intensity indicates the relative representation of each cluster, ranging from white (0%) to dark red (50%). Values are normalized independently for each comparison, allowing the functional composition of ROS-associated transcriptional responses to be compared across species, stress treatments and germination stages. Method of analysis is described in Supplementary Information.

6. The Spatial Dimension of ROS Signaling in Seed Stress Responses

The spatial organization of ROS metabolism within seeds may represent an important but largely unexplored component of germination responses to environmental stress, both at the tissue and cellular levels. In endospermic seeds, germination requires the coordination of embryo growth with the weakening of the surrounding endosperm, which constitutes a mechanical constraint to radicle protrusion [15,42]. ROS have been implicated in these processes, notably through the involvement of hydroxyl radicals in cell-wall loosening in cress [42] and the accumulation of superoxide and H_2O_2 in the micropylar endosperm of lettuce during germination [43]. These findings provide evidence that ROS

production is spatially regulated within germinating seeds and can contribute to distinct physiological processes in the endosperm and in the embryo. However, the endosperm is not only a mechanical barrier but also an active regulatory tissue that integrates environmental and hormonal signals to control embryo growth. In *Arabidopsis*, the endosperm contributes to the maintenance of dormancy through RGL2-dependent ABA production and release, which inhibits embryo growth [103]. This hormonal communication involves specific ABA transporters that mediate ABA movement between the endosperm and embryo [104]. Furthermore, tissue-specific light responses demonstrate that the endosperm can perceive environmental cues and translate them into hormonal signals that regulate germination [103]. Together, these findings establish the endosperm as a key regulatory interface between environmental conditions and embryo developmental activity. Beyond its local contribution to cell-wall loosening in the endosperm, ROS metabolism may also participate in these regulatory functions, although its involvement in environmental sensing and intertissue signaling remains largely unexplored. Bailly [45] proposed that the spatiotemporal regulation of ROS production contributes to the perception and transduction of environmental signals during germination, acting in concert with hormonal pathways to control cellular expansion. Evidence for tissue-specific ROS production in germinating seeds [43,105], together with the involvement of ROS in ABA and GA metabolism and signaling [47,105], provides a basis for considering a possible regulatory role of endosperm redox metabolism.

Extending this concept to the endosperm–embryo interface, we therefore propose that the endosperm may constitute an important site of ROS-dependent environmental sensing during germination. Abiotic constraints, such as water deficit, salinity or temperature extremes, could modify ROS production and antioxidant metabolism in the endosperm, thereby altering its redox status. These changes could subsequently influence ABA biosynthesis, catabolism, signaling or transport, as well as other hormonal pathways, ultimately modifying the hormonal environment experienced by the embryo and its capacity to resume growth. In this model, ROS would not necessarily act as mobile signals between tissues but could function locally within the endosperm to regulate the production or release of hormonal and metabolic signals controlling embryo growth.

Although direct experimental evidence for such a mechanism under abiotic stress is currently lacking, we propose that the endosperm redox status may act as a regulatory checkpoint linking environmental perception to hormonal communication and the control of embryo growth. This hypothesis extends the concept of spatiotemporal ROS signaling proposed by Bailly [45] by suggesting that the redox state of one seed tissue may influence the developmental activity of another. Resolving ROS and redox dynamics separately in the endosperm and embryo, together with tissue-specific analyses of hormonal metabolism and transport, will be essential to determine whether endosperm redox homeostasis plays a causal role in controlling germination under adverse environmental conditions.

If ROS homeostasis within the endosperm participates to the sensing of environment, ROS dynamics within the embryo is also likely to play a role in germination completion. Bailly [45] showed that a ROS signal was propagating along the embryonic axis in germinating seeds which ultimately could lead to cell elongation in the hypocotyl area. The participation of ROS in long-distance communication has been characterized in vegetative tissues through the so-called ROS wave, a self-propagating signal involving successive activation of ROS production in neighbouring cells, primarily through the NADPH oxidase RBOHD [106,107]. Vascular tissues play a major role in this process, allowing rapid transmission of stress signals and activation of systemic acquired acclimation [108]. However, this mechanism has only been characterized only in vegetative tissues, and its occurrence during seed development or germination remains largely unexplored.

Interestingly, our recent ROS imaging experiments revealed that ROS-associated fluorescence in germinating seeds was predominantly localized in the radicle meristem and along the embryonic vasculature, consistent with previous observations [45]. In contrast, non-germinating seeds imbibed for the same duration under water stress imposed by PEG exhibited weaker fluorescence, largely restricted to the meristematic region (Figure 3A,B). Although these observations suggest that vascular-associated ROS accumulation may contribute to spatial coordination within the germinating embryo, they provide only static information on ROS distribution and do not demonstrate the temporal propagation of a ROS signal. Establishing whether a genuine ROS wave operates during seed germination will require time-resolved imaging and functional analyses of the molecular mechanisms underlying ROS production and signal propagation.

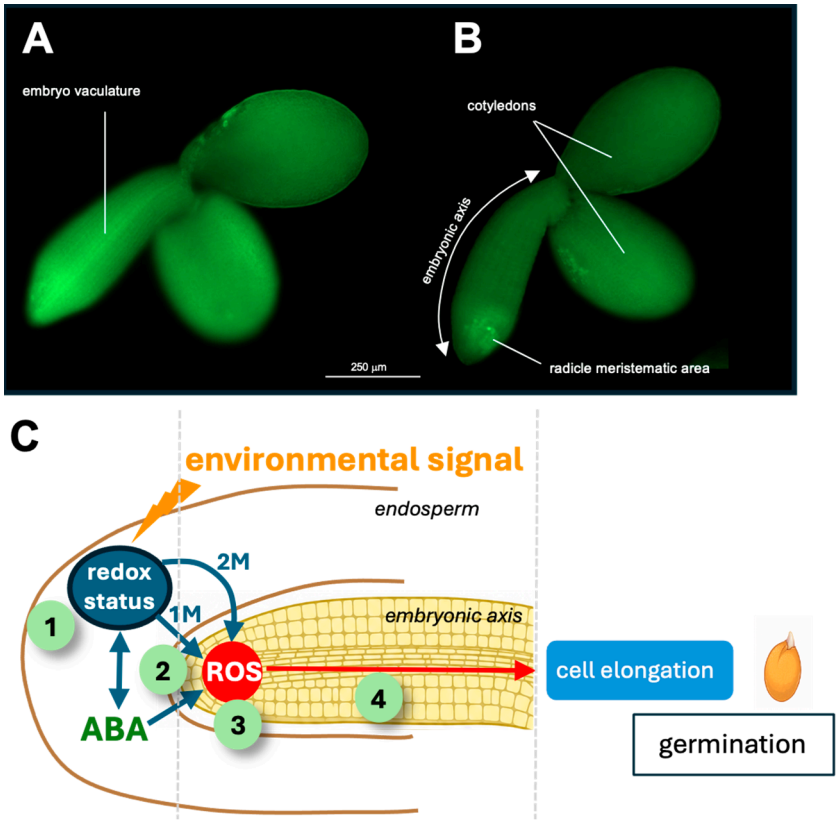

Figure 3. Proposed spatial ROS signaling during seed germination. (**A**,**B**) ROS detection in embryos of non-dormant *Arabidopsis thaliana* seeds after 24 h of imbibition at 15 °C on water (**A**) or on a polyethylene glycol (PEG) solution at −0.9 MPa (**B**). Seeds complete germination within 2 days on water, compared with 6 days under PEG-induced water stress [93]. ROS accumulation was visualized by fluorescence using 5-(and-6)-chloromethyl-2′,7′-dichlorodihydrofluorescein diacetate (CM-H_2DCFDA) and fluorescence microscopy using a Zeiss Axio ZoomV16 macroscope equipped with an ApoTome imaging system. (**C**) Proposed model of spatial ROS signaling during seed germination. Environmental cues modify redox homeostasis in the endosperm, potentially affecting ABA metabolism and signaling (1). Redox signals may then be transmitted to the embryonic axis either through the direct diffusion of H_2O_2, acting as a primary messenger (1M), or through mobile oxidized molecules (e.g., proteins or other secondary messengers), together with changes in ABA signaling (2). Under favourable environmental conditions, ROS production is initiated near the radicle meristematic region (3), and the signal is subsequently proposed to propagate along the embryonic vasculature towards the hypocotyl elongation zone, where cell expansion contributes to radicle protrusion and the completion of germination. Method of ROS imaging is described in Supplementary Information.

These observations suggest that ROS distribution during germination is spatially organized rather than uniform throughout the embryo, raising the possibility that vascular tissues may contribute to coordinating redox signaling during this developmental transition. This finding is particularly intriguing in light of the work of Topham et al. [109], who identified a spatially organized decision-making centre in the embryonic radicle of *Arabidopsis*. Their study demonstrated that ABA and GA responses are distributed among distinct cell populations, with GA signaling associated with vascular cells and ABA responses enriched in outer radicle tissues. Communication between these spatially separated hormonal signaling domains was proposed to contribute to the integration of environmental information and the decision to germinate. The preferential localization of ROS within the embryonic vasculature therefore raises the possibility that redox signaling may contribute to the spatial coordination of hormonal and developmental responses controlling germination. It also suggests a potential parallel with the vascular-associated ROS signaling described in vegetative tissues. Nevertheless, ROS localisation alone does not demonstrate signal propagation, nor does it establish a functional interaction with the ABA–GA decision-making network. Whether ROS signals propagate along the embryonic vascular axis, interact with spatially distinct hormonal signaling domains, or contribute to communication between embryo tissues remains to be determined. Figure 3C presents a proposed integrated model of ROS dynamics during seed germination, incorporating redox sensing in the endosperm and putative signal propagation from the root meristem to the hypocotyl elongation zone through the embryonic vasculature.

Spatial regulation of ROS signaling also occurs at the subcellular level, where different organelles may generate distinct redox signals contributing to the regulation of seed germination. In this context, retrograde signaling provides a mechanism through which changes in organellar metabolism and ROS homeostasis can influence nuclear gene expression and developmental transitions. Direct evidence for this process has been obtained in *Arabidopsis*, where mitochondrial ROS production participates in ethylene-induced dormancy release through activation of a retrograde signaling pathway involving AOX1a and ANAC013 [25]. Subsequently, Jurdak et al. [51] demonstrated that dormancy release induced by light, cold stratification or gibberellins is associated with temporally regulated intracellular ROS accumulation, initially in mitochondria and subsequently in nuclei. Interestingly, the mitochondrial retrograde pathway identified in response to ethylene was not similarly activated by these other dormancy-releasing treatments, suggesting that distinct environmental and hormonal cues may recruit different organellar redox signaling mechanisms.

Beyond mitochondria, peroxisomes emerge as particularly interesting candidates for integrating metabolic and redox signals during germination. Peroxisomal β-oxidation is required for normal germination, with functions extending beyond the mobilization of stored lipids [110,111]. Peroxisomes are also major sites of H_2O_2 production, notably through acyl-CoA oxidase activity, while catalases and other antioxidant systems regulate its accumulation. Their metabolic activity could therefore generate redox signals reflecting changes in cellular metabolic status. Interestingly, genetic analyses have shown that mutations affecting peroxisomal functions can modify germination responses to light and ABA, suggesting interactions between peroxisomal metabolism and environmental or hormonal regulation. Furthermore, Jurdak et al. [51] reported an increase in peroxisome abundance during dormancy release induced by light, cold stratification or gibberellins, accompanied by changes in the expression of genes involved in peroxisomal ROS metabolism. These findings indicate that peroxisomal organization and redox-related metabolism are dynamically associated with the transition from dormancy to germination, although they do not establish that peroxisomes directly perceive environmental signals. An emerging hypothesis is that peroxisomes may function as metabolic–redox signaling hubs linking environmental con-

straints to germination control. In vegetative tissues, peroxisomal ROS metabolism has been implicated in cellular stress responses and communication with the nucleus [112,113], raising the possibility of similar mechanisms during seed germination. Environmental stresses, such as water deficit, salinity or temperature extremes, could alter peroxisomal metabolic activity, ROS production and antioxidant capacity, thereby modifying organelle-specific redox homeostasis. These changes could potentially influence nuclear transcriptional programmes involved in hormone metabolism, stress responses and embryo growth. However, direct evidence for peroxisome-derived ROS-dependent retrograde signaling during germination is currently lacking. Moreover, the effects of altered peroxisomal metabolism on germination may arise from changes in β-oxidation, energy supply or hormone-related metabolic pathways rather than ROS signaling itself. Distinguishing these possibilities remains essential to establish whether peroxisomes actively participate in environmental signal transduction or primarily respond to metabolic changes associated with germination.

The nucleus may represent another important component of this spatially organized redox network. Seed maturation is accompanied by nuclear size reduction and chromatin condensation, which are reversed during germination [114]. Chromatin remodelling contributes to the developmental transition from quiescence to active growth, notably through the regulation of embryonic identity and hormonal responses. For example, the chromatin-remodelling factor PICKLE (PKL) participates in the repression of embryonic developmental programmes and modulates ABA-responsive gene expression, including ABI3 and ABI5, during germination under osmotic stress [115,116]. Interestingly, Jurdak et al. [51] observed that nuclear ROS accumulation during dormancy release coincides with nuclear expansion and heterochromatin decompaction in embryonic-axis cells. They also showed that exogenous H_2O_2 can promote chromatin relaxation in young seedlings, suggesting that ROS may influence higher-order chromatin organization. However, a direct causal relationship between nuclear ROS accumulation and chromatin remodelling during germination has not been demonstrated. In particular, whether nuclear ROS influence histone modifications, DNA methylation, chromatin accessibility or the activity of redox-sensitive chromatin regulators remains largely unknown.

Together, these findings suggest that the spatial organization of ROS metabolism may provide an additional level of regulation during germination, connecting organellar metabolic activity, nuclear responses and developmental control. This perspective also extends the oxidative window concept. ROS measurements at the whole-seed level integrate contributions from different tissues and subcellular compartments, which may differ in their ROS production, scavenging capacity and sensitivity to redox signals. Consequently, changes in ROS homeostasis within specific organelles may remain undetected when only total seed ROS content is measured, particularly under abiotic stress. The oxidative window may therefore need to be considered not only in terms of the amount of ROS accumulated, but also where and when ROS are produced and how compartment-specific redox signals contribute to germination control. In this novel framework, we propose that peroxisomes may represent an important, yet largely unexplored, component of the signaling network connecting environmental constraints to nuclear regulation of germination. Determining whether peroxisomal ROS participate in retrograde signaling, potentially influencing nuclear redox status, chromatin organization and hormonal responses, represents a promising direction for future research.

7. Oxidative Memory: Linking Maternal Environmental Conditions to ROS Signaling During Germination

Maternal environmental conditions can influence the redox properties of developing seeds, potentially shaping their subsequent responses to abiotic stress. Here, we define ox-

idative memory as a form of intergenerational memory arising from maternal environmental conditions experienced during seed development only, distinct from transgenerational epigenetic inheritance, which refers to the transmission of environmentally induced changes in gene regulation across successive generations. Environmental constraints experienced during seed production may result in distinct redox-related physiological states in mature seeds. In barley, drought imposed during grain filling induced transcriptional and metabolic reprogramming involving ROS metabolism, flavonoid biosynthesis and antioxidant pathways [117]. Similarly, Nagel et al. [118] reported that maternal drought and elevated temperature modified the antioxidant-related metabolite profile of barley embryos, including a decrease in γ-tocopherol under drought conditions. Interestingly, these changes were not accompanied by significant treatment-induced differences in malondialdehyde content, suggesting that maternal stress can modify antioxidant metabolism without necessarily increasing oxidative damage. Further evidence indicates that maternal environmental constraints can alter the redox properties of the resulting seeds, although the responses depend on stress intensity and duration. In barley, salinity imposed during maternal plant development modified seed glutathione content and redox status, with short-term exposure improving subsequent germination and seedling vigour, whereas prolonged exposure reduced seed quality [119]. In *Lallemantia* species, maternal water limitation increased H_2O_2 accumulation and lipid peroxidation while generally reducing antioxidant enzyme activities, including superoxide dismutase, catalase and ascorbate peroxidase, with responses influenced by species and mycorrhizal inoculation [120]. Azad et al. [121] reported that high temperature and high light during *Arabidopsis* seed production altered the abundance of stored transcripts associated with oxidative stress responses and antioxidant defence, including *CAT2*, *CAT3*, *DHAR1* and genes of the glyoxalase system. Comparison of their differentially expressed genes with the ROS wheel [102] revealed changes across several ROS-associated transcriptional clusters, including Cluster III, GUN retrograde, ROS core and RBOHF-associated clusters. These observations suggest that maternal environmental conditions can influence the redox-related transcriptional landscape of mature seeds, potentially affecting their capacity to respond to subsequent environmental challenges. However, changes in stored transcript abundance do not necessarily reflect changes in ROS production or redox status, particularly in dry seeds where metabolic activity is strongly restricted. Whether these transcriptional differences contribute to subsequent redox regulation during germination therefore remains to be determined.

The consequences of maternal environmental history may become particularly apparent when progeny seeds encounter stress during germination. Recent findings in *Arabidopsis* indicate that maternal drought can improve germination at low water potential while modifying the temporal organization of ROS-related responses during imbibition [95]. Transcriptomic analyses of isolated endosperm and embryo fractions revealed distinct tissue-specific responses. In seeds produced under maternal drought, the endosperm exhibited early transcriptional changes involving genes associated with superoxide detoxification, oxidized protein repair and ROS homeostasis. In contrast, changes in the embryo occurred later and involved antioxidant metabolism, including APX1, MDAR2 and DHAR2. These observations suggest that maternal drought influences not only the redox-related properties established during seed development but also the timing and tissue-specific regulation of oxidative metabolism during subsequent germination.

Collectively, these observations suggest that maternal environmental conditions can influence seed redox biology at two interconnected levels: by shaping antioxidant metabolism and ROS-related molecular profiles during seed development, and by modifying the temporal regulation of ROS metabolism during subsequent germination. Such effects may contribute to a form of environmental priming in which the conditions experienced during

seed production influence the physiological and molecular responses of progeny seeds to later stress. However, whether these changes constitute a functional oxidative memory remains an open question. In particular, the extent to which maternal stress modifies the spatial and subcellular organization of ROS signaling, rather than simply its overall accumulation, remains largely unexplored. The tissue-specific responses observed in *Arabidopsis* raise the possibility that maternal environmental history influences the oxidative window by altering the coordination of redox metabolism between the endosperm and embryo. Determining whether early endosperm redox changes actively regulate subsequent embryo ROS production and growth, or instead reflect parallel responses to maternal environmental history, will be essential for understanding how maternal stress shapes ROS-dependent germination responses.

8. Conclusions and Futures Perspectives

ROS are central regulators of seed germination, integrating environmental signals with metabolic and developmental processes. Recent findings extend the oxidative window concept by highlighting that germination depends not only on ROS abundance but also on their temporal dynamics, spatial distribution and physiological context. Importantly, abiotic stress does not invariably lead to excessive ROS accumulation: under certain conditions, particularly water limitation, reduced ROS production may contribute to delayed or inhibited germination by restricting ROS-dependent signaling. The emerging concept of oxidative memory adds a further dimension to this framework. Environmental conditions experienced during seed development can influence the redox properties and antioxidant machinery of mature seeds, potentially shaping their subsequent responses to stress. However, the mechanisms underlying the persistence of these maternal effects and their contribution to germination performance remain poorly understood. Establishing causal relationships between maternally induced redox modifications and subsequent stress tolerance represents an important challenge.

Altogether these observations call for a reassessment of the oxidative window concept, incorporating not only ROS concentration but also temporal and spatial dynamics (Figure 4). We propose that the oxidative trajectory, i.e., the ability to reach or to exceed the oxidative window, determine the ability of the kinetics of seed germination. In addition, we also incorporate the concept of the oxidative window, which can modulate seed oxidative trajectory and allow faster germination, providing the oxidative window is not exceeded.

Another major perspective concerns the spatial organisation of ROS signaling. ROS produced in distinct cellular compartments and seed tissues may fulfil specific functions that cannot be resolved through whole-seed measurements. In particular, understanding how local ROS signals are coordinated between the embryo and endosperm, and potentially propagated through embryonic tissues, could reveal new mechanisms controlling germination. Whether processes resembling the systemic ROS wave described in vegetative tissues also operate in seeds remains an open question.

Addressing these novel questions will require the development of spatially and temporally resolved approaches to investigate ROS dynamics in living seeds. Genetically encoded fluorescent sensors, such as roGFP2-Orp1, Grx1-roGFP2 and HyPer7, combined with high-resolution imaging, tissue-specific expression and genetic approaches, offer promising opportunities to monitor ROS and redox changes within individual organelles, cells and tissues [122]. A major objective will be to understand how ROS-related signals are generated, transmitted and perceived across cellular compartments and tissues, and how these processes contribute to the coordination of germination. However, despite recent advances in ROS imaging, monitoring ROS dynamics in living seeds remains challenging due to difficulties in expressing genetically encoded biosensors in specific tissues, particularly the

endosperm, and to optical scattering and limited light penetration through the seed coat and surrounding tissues. Overcoming these limitations will require improved tissue-specific biosensor expression systems and advanced imaging approaches capable of resolving ROS dynamics at cellular and subcellular scales without disrupting seed integrity.

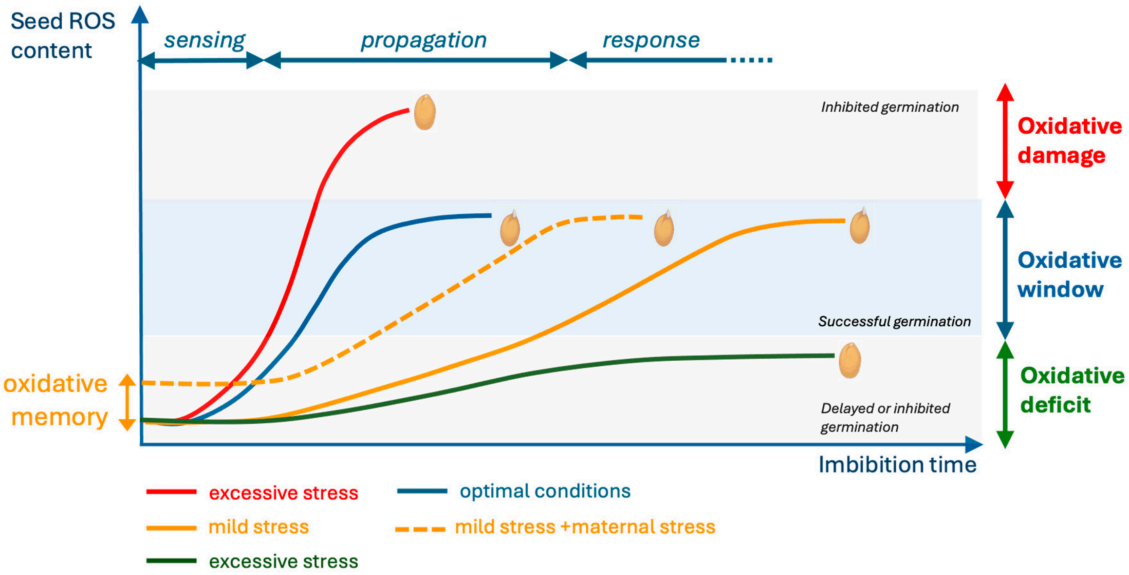

Figure 4. A proposed extended oxidative window model integrating seed oxidative trajectories. The figure illustrates the changes in seed ROS content during imbibition under different environmental and physiological conditions. Under optimal conditions (blue curve), ROS progressively accumulate and reach a range compatible with successful germination, corresponding to the oxidative window permissive for germination. Excessive stress (red curve) induces rapid ROS accumulation beyond this window, resulting in oxidative damage and inhibition of germination. Conversely, severe environmental stress or conditions that restrict metabolic activation during imbibition (green curve), such as water deficit, may impair ROS production and accumulation, preventing seeds from reaching the oxidative window required for germination. This oxidative deficit may compromise ROS-dependent signaling and delay or inhibit germination, even in the absence of excessive oxidative damage. Under mild stress (orange curve), ROS accumulation is delayed, resulting in slower germination without necessarily compromising its completion. The dashed orange curve illustrates the proposed contribution of maternal stress-induced oxidative memory, whereby modifications in the redox properties of progeny seeds may facilitate ROS accumulation and improve germination performance under subsequent stress. ROS signaling includes sensing, propagation and responses as defined in Figure 3C.

Beyond these fundamental questions, a better understanding of ROS regulation offers promising opportunities for the development of seed technologies aimed at improving germination and seedling establishment under adverse environmental conditions. Seed priming, including hydropriming, chemical priming and emerging nanopriming approaches, can modify antioxidant metabolism, ROS homeostasis and stress-related signaling pathways. Recent studies illustrate this potential. In rapeseed, nanopriming with selenium-doped carbon dots improved germination under salinity while enhancing antioxidant enzyme activities and respiratory metabolism [123]. In cotton, carbon-dot nanopriming promoted germination under salt stress through an RBOH-associated H_2O_2 signaling pathway involved in sodium homeostasis [124]. Similarly, ZnO-based seed treatments improved antioxidant responses and drought tolerance in wheat seedlings [125]. These findings suggest that the effectiveness of seed treatments may depend not simply on limiting ROS accumulation but on establishing an appropriate balance between oxidative signaling and antioxidant protection. Nevertheless, the efficacy, dose dependence, environmental safety, and persistence of these effects require further evaluation across species and growing condi-

tions, particularly given the strong species- and seed-lot-dependent variability and limited reproducibility of seed-priming outcomes that remain major bottlenecks in agricultural applications. The frameworks of oxidative trajectories and oxidative memory proposed here may help explain these variations by considering how differences in ROS dynamics during germination and maternal environment-induced changes in seed redox properties influence the response to priming treatments, potentially guiding the development of more targeted and reproducible priming strategies.

In conclusion, the oxidative window, oxidative memory and spatial organisation of ROS signaling provide complementary perspectives for understanding how seeds integrate environmental information to regulate germination. A major challenge will be to move beyond measurements of overall ROS accumulation towards a mechanistic understanding of their production, compartmentalization and intercellular communication. In the context of increasingly frequent and severe environmental stresses associated with climate change, such knowledge will be essential not only for advancing fundamental seed biology but also for designing innovative seed treatments that improve germination reliability, seedling establishment and crop resilience.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/antiox15121552/s1, Table S1: transcriptome analyses according to ROS wheel model; Supplementary Information: Protocols.

Author Contributions: Conceptualization, writing, C.B.; investigation, data curation, formal analysis, X.M. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Data Availability Statement: The transcriptomic datasets used on this work are available in the NCBI under accession numbers GSE338026, PRJNA237675, PRJNA795723, PRJNA577173, PRJNA649876 and GSE167280.

Acknowledgments: The authors thank Nicole Chaumont for providing the images of ROS fluorescence. During the preparation of this manuscript, the authors used GPT-6, OpenAI, accessed October 2026, to assist with English language editing and with preparing the layout of Figure 2. The authors have reviewed and edited the output and take full responsibility for the content of this publication.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. AR6 Synthesis Report: Climate Change 2023. Available online: https://www.ipcc.ch/report/ar6/syr/ (accessed on 15 October 2026).
2. Bailly, C.; Gomez Roldan, M.V. Impact of Climate Perturbations on Seeds and Seed Quality for Global Agriculture. *Biochem. J.* **2023**, *480*, 177–196. [CrossRef] [PubMed]
3. Leprince, O.; Pellizzaro, A.; Berriri, S.; Buitink, J. Late Seed Maturation: Drying without Dying. *J. Exp. Bot.* **2017**, *68*, 827–841. [CrossRef] [PubMed]
4. Bewley, J.D. Seed Germination and Dormancy. *Plant Cell* **1997**, *9*, 1055–1066. [CrossRef] [PubMed]
5. Finch-Savage, W.E.; Leubner-Metzger, G. Seed Dormancy and the Control of Germination. *New Phytol.* **2006**, *171*, 501–523. [CrossRef] [PubMed]
6. Donohue, K. Completing the Cycle: Maternal Effects as the Missing Link in Plant Life Histories. *Phil. Trans. R. Soc. B Biol. Sci.* **2009**, *364*, 1059–1074. [CrossRef] [PubMed]
7. Galloway, L.F. Maternal Effects Provide Phenotypic Adaptation to Local Environmental Conditions. *New Phytol.* **2005**, *166*, 93–100. [CrossRef] [PubMed]
8. Herman, J.J.; Sultan, S.E. Adaptive Transgenerational Plasticity in Plants: Case Studies, Mechanisms, and Implications for Natural Populations. *Front. Plant Sci.* **2011**, *2*, 102. [CrossRef] [PubMed]

9. Bezodis, W.; Penfield, S. Maternal Environmental Control of Progeny Seed Physiology: A Review of Concepts, Evidence and Mechanism. *Seed Sci. Res.* **2024**, *34*, 48–55. [CrossRef]
10. Donohue, K.; Heschel, M.S.; Butler, C.M.; Barua, D.; Sharrock, R.A.; Whitelam, G.C.; Chiang, G.C.K. Diversification of Phytochrome Contributions to Germination as a Function of Seed-Maturation Environment. *New Phytol.* **2008**, *177*, 367–379. [CrossRef] [PubMed]
11. Lämke, J.; Bäurle, I. Epigenetic and Chromatin-Based Mechanisms in Environmental Stress Adaptation and Stress Memory in Plants. *Genome Biol.* **2017**, *18*, 124. [CrossRef] [PubMed]
12. Brunel-Muguet, S.; Baránek, M.; Fragkostefanakis, S.; Sauvage, C.; Lieberman-Lazarovich, M.; Maury, S.; Kaiserli, E.; Segal, N.; Testillano, P.S.; Verdier, J. Maternal Environmental Effects and Climate-Smart Seeds: Unlocking Epigenetic Inheritance for Crop Innovation in the Seed Industry. *Plant J.* **2025**, *123*, e70407. [CrossRef] [PubMed]
13. Finch-Savage, W.E.; Bassel, G.W. Seed Vigour and Crop Establishment: Extending Performance beyond Adaptation. *J. Exp. Bot.* **2016**, *67*, 567–591. [CrossRef] [PubMed]
14. Holdsworth, M.J.; Bentsink, L.; Soppe, W.J.J. Molecular Networks Regulating *Arabidopsis* Seed Maturation, After-ripening, Dormancy and Germination. *New Phytol.* **2008**, *179*, 33–54. [CrossRef] [PubMed]
15. Linkies, A.; Müller, K.; Morris, K.; Turečková, V.; Wenk, M.; Cadman, C.S.C.; Corbineau, F.; Strnad, M.; Lynn, J.R.; Finch-Savage, W.E.; et al. Ethylene Interacts with Abscisic Acid to Regulate Endosperm Rupture during Germination: A Comparative Approach Using *Lepidium sativum* and *Arabidopsis thaliana*. *Plant Cell* **2009**, *21*, 3803–3822. [CrossRef] [PubMed]
16. Dekkers, B.J.W.; Pearce, S.; van Bolderen-Veldkamp, R.P.; Marshall, A.; Widera, P.; Gilbert, J.; Drost, H.-G.; Bassel, G.W.; Müller, K.; King, J.R.; et al. Transcriptional Dynamics of Two Seed Compartments with Opposing Roles in *Arabidopsis* Seed Germination. *Plant Physiol.* **2013**, *163*, 205–215. [CrossRef] [PubMed]
17. Mittler, R. ROS Are Good. *Trends Plant Sci.* **2017**, *22*, 11–19. [CrossRef] [PubMed]
18. Schopfer, P.; Plachy, C.; Frahry, G. Release of Reactive Oxygen Intermediates (Superoxide Radicals, Hydrogen Peroxide, and Hydroxyl Radicals) and Peroxidase in Germinating Radish Seeds Controlled by Light, Gibberellin, and Abscisic Acid. *Plant Physiol.* **2001**, *125*, 1591–1602. [CrossRef] [PubMed]
19. Oracz, K.; Bouteau, H.E.-M.; Farrant, J.M.; Cooper, K.; Belghazi, M.; Job, C.; Job, D.; Corbineau, F.; Bailly, C. ROS Production and Protein Oxidation as a Novel Mechanism for Seed Dormancy Alleviation. *Plant J.* **2007**, *50*, 452–465. [CrossRef] [PubMed]
20. Leymarie, J.; Vitkauskaite, G.; Hoang, H.H.; Gendreau, E.; Chazoule, V.; Meimoun, P.; Corbineau, F.; El-Maarouf-Bouteau, H.; Bailly, C. Role of Reactive Oxygen Species in the Regulation of *Arabidopsis* Seed Dormancy. *Plant Cell Physiol.* **2011**, *53*, 96–106. [CrossRef] [PubMed]
21. Liu, Y.; Ye, N.; Liu, R.; Chen, M.; Zhang, J. H2O2 Mediates the Regulation of ABA Catabolism and GA Biosynthesis in *Arabidopsis* Seed Dormancy and Germination. *J. Exp. Bot.* **2010**, *61*, 2979–2990. [CrossRef] [PubMed]
22. Müller, K.; Linkies, A.; Vreeburg, R.A.M.; Fry, S.C.; Krieger-Liszkay, A.; Leubner-Metzger, G. In Vivo Cell Wall Loosening by Hydroxyl Radicals during Cress Seed Germination and Elongation Growth. *Plant Physiol.* **2009**, *150*, 1855–1865. [CrossRef] [PubMed]
23. Bailly, C.; El-Maarouf-Bouteau, H.; Corbineau, F. From Intracellular Signaling Networks to Cell Death: The Dual Role of Reactive Oxygen Species in Seed Physiology. *Comptes Rendus Biol.* **2008**, *331*, 806–814. [CrossRef] [PubMed]
24. Nietzel, T.; Mostertz, J.; Ruberti, C.; Née, G.; Fuchs, P.; Wagner, S.; Moseler, A.; Müller-Schüssele, S.J.; Benamar, A.; Poschet, G.; et al. Redox-Mediated Kick-Start of Mitochondrial Energy Metabolism Drives Resource-Efficient Seed Germination. *Proc. Natl. Acad. Sci. USA* **2020**, *117*, 741–751. [CrossRef] [PubMed]
25. Jurdak, R.; Launay-Avon, A.; Paysant-Le Roux, C.; Bailly, C. Retrograde Signalling from the Mitochondria to the Nucleus Translates the Positive Effect of Ethylene on Dormancy Breaking of *Arabidopsis thaliana* Seeds. *New Phytol.* **2021**, *229*, 2192–2205. [CrossRef] [PubMed]
26. Toh, S.; Imamura, A.; Watanabe, A.; Nakabayashi, K.; Okamoto, M.; Jikumaru, Y.; Hanada, A.; Aso, Y.; Ishiyama, K.; Tamura, N.; et al. High Temperature-Induced Abscisic Acid Biosynthesis and Its Role in the Inhibition of Gibberellin Action in *Arabidopsis* Seeds. *Plant Physiol.* **2008**, *146*, 1368–1385. [CrossRef] [PubMed]
27. Shu, K.; Qi, Y.; Chen, F.; Meng, Y.; Luo, X.; Shuai, H.; Zhou, W.; Ding, J.; Du, J.; Liu, J.; et al. Salt Stress Represses Soybean Seed Germination by Negatively Regulating GA Biosynthesis While Positively Mediating ABA Biosynthesis. *Front. Plant Sci.* **2017**, *8*, 1372. [CrossRef] [PubMed]
28. Kranner, I.; Minibayeva, F.V.; Beckett, R.P.; Seal, C.E. What Is Stress? Concepts, Definitions and Applications in Seed Science. *New Phytol.* **2010**, *188*, 655–673. [CrossRef] [PubMed]
29. Zinn, K.E.; Tunc-Ozdemir, M.; Harper, J.F. Temperature Stress and Plant Sexual Reproduction: Uncovering the Weakest Links. *J. Exp. Bot.* **2010**, *61*, 1959–1968. [CrossRef] [PubMed]
30. Sehgal, A.; Sita, K.; Siddique, K.H.M.; Kumar, R.; Bhogireddy, S.; Varshney, R.K.; HanumanthaRao, B.; Nair, R.M.; Prasad, P.V.V.; Nayyar, H. Drought or/and Heat-Stress Effects on Seed Filling in Food Crops: Impacts on Functional Biochemistry, Seed Yields, and Nutritional Quality. *Front. Plant Sci.* **2018**, *9*, 1705. [CrossRef] [PubMed]

31. Penfield, S.; MacGregor, D.R. Effects of Environmental Variation during Seed Production on Seed Dormancy and Germination. *J. Exp. Bot.* **2017**, *68*, 819–825. [CrossRef] [PubMed]
32. Vancostenoble, B.; Blanchet, N.; Langlade, N.B.; Bailly, C. Maternal Drought Stress Induces Abiotic Stress Tolerance to the Progeny at the Germination Stage in Sunflower. *Environ. Exp. Bot.* **2022**, *201*, 104939. [CrossRef]
33. Bailly, C.; Leymarie, J.; Lehner, A.; Rousseau, S.; Côme, D.; Corbineau, F. Catalase Activity and Expression in Developing Sunflower Seeds as Related to Drying. *J. Exp. Bot.* **2004**, *55*, 475–483. [CrossRef] [PubMed]
34. Thakur, A.; Bhatla, S.C. A Probable Crosstalk between Ca+2, Reactive Oxygen Species Accumulation and Scavenging Mechanisms and Modulation of Protein Kinase C Activity during Seed Development in Sunflower. *Plant Signal Behav.* **2014**, *9*, e27900. [CrossRef] [PubMed]
35. Huang, H.; Song, S. Change in Desiccation Tolerance of Maize Embryos during Development and Germination at Different Water Potential PEG-6000 in Relation to Oxidative Process. *Plant Physiol. Biochem.* **2013**, *68*, 61–70. [CrossRef] [PubMed]
36. Wu, J.-H.; Wang, W.-Q.; Song, S.-Q.; Cheng, H.-Y. Reactive Oxygen Species Scavenging Enzymes and Down-Adjustment of Metabolism Level in Mitochondria Associated with Desiccation-Tolerance Acquisition of Maize Embryo. *J. Int. Plant Biol.* **2009**, *51*, 638–645. [CrossRef] [PubMed]
37. Cairns, N.G.; Pasternak, M.; Wachter, A.; Cobbett, C.S.; Meyer, A.J. Maturation of *Arabidopsis* Seeds Is Dependent on Glutathione Biosynthesis within the Embryo. *Plant Physiol.* **2006**, *141*, 446–455. [CrossRef] [PubMed]
38. Ruan, M.; He, W.; He, R.; Wang, X.; Wei, J.; Zhu, Y.; Li, R.; Jiang, Z.; Na, X.; Wang, X.; et al. Alternative Oxidase 2 Influences *Arabidopsis* Seed Germination under Salt Stress by Modulating ABA Signalling and ROS Homeostasis. *Env. Exp. Bot.* **2024**, *217*, 105568. [CrossRef]
39. Bailly, C.; Bogatek-Leszczynska, R.; Côme, D.; Corbineau, F. Changes in Activities of Antioxidant Enzymes and Lipoxygenase during Growth of Sunflower Seedlings from Seeds of Different Vigour. *Seed Sci. Res.* **2002**, *12*, 47–55. [CrossRef]
40. Corpas, F.J.; González-Gordo, S.; Palma, J.M. Plant Peroxisomes: A Factory of Reactive Species. *Front. Plant Sci.* **2020**, *11*, 853. [CrossRef] [PubMed]
41. Pinfield-Wells, H.; Rylott, E.L.; Gilday, A.D.; Graham, S.; Job, K.; Larson, T.R.; Graham, I.A. Sucrose Rescues Seedling Establishment but Not Germination of *Arabidopsis* Mutants Disrupted in Peroxisomal Fatty Acid Catabolism. *Plant J.* **2005**, *43*, 861–872. [CrossRef] [PubMed]
42. Müller, K.; Carstens, A.C.; Linkies, A.; Torres, M.A.; Leubner-Metzger, G. The NADPH-Oxidase AtrbohB Plays a Role in *Arabidopsis* Seed after-Ripening. *New Phytol.* **2009**, *184*, 885–897. [CrossRef] [PubMed]
43. Zhang, Y.; Chen, B.; Xu, Z.; Shi, Z.; Chen, S.; Huang, X.; Chen, J.; Wang, X. Involvement of Reactive Oxygen Species in Endosperm Cap Weakening and Embryo Elongation Growth during Lettuce Seed Germination. *J. Exp. Bot.* **2014**, *65*, 3189–3200. [CrossRef] [PubMed]
44. Yang, X.; Zhang, F.; Yang, M.; He, Y.; Li, Z.; Yang, J.; Wang, X. The NADPH-Oxidase LsRbohC1 Plays a Role in Lettuce (*Lactuca sativa*) Seed Germination. *Plant Physiol. Biochem.* **2020**, *154*, 751–757. [CrossRef] [PubMed]
45. Bailly, C. The Signalling Role of ROS in the Regulation of Seed Germination and Dormancy. *Biochem. J.* **2019**, *476*, 3019–3032. [CrossRef] [PubMed]
46. Wang, Y.; Liu, X.; Sun, X.; Mao, X.; Wang, Z.; Peng, J.; Yang, Z.; Ali, F.; Wang, Z.; Li, F. The Promotive and Repressive Effects of Exogenous H_2O_2 on *Arabidopsis* Seed Germination and Seedling Establishment Depend on Application Dose. *Physiol. Plant* **2025**, *177*, e70098. [CrossRef] [PubMed]
47. Luo, X.; Dai, Y.; Zheng, C.; Yang, Y.; Chen, W.; Wang, Q.; Chandrasekaran, U.; Du, J.; Liu, W.; Shu, K. The ABI4-RbohD/VTC2 Regulatory Module Promotes Reactive Oxygen Species (ROS) Accumulation to Decrease Seed Germination under Salinity Stress. *New Phytol.* **2021**, *229*, 950–962. [CrossRef] [PubMed]
48. Bi, C.; Ma, Y.; Wu, Z.; Yu, Y.-T.; Liang, S.; Lu, K.; Wang, X.-F. Arabidopsis ABI5 Plays a Role in Regulating ROS Homeostasis by Activating CATALASE 1 Transcription in Seed Germination. *Plant Mol. Biol.* **2017**, *94*, 197–213. [CrossRef] [PubMed]
49. Cheng, M.; Guo, Y.; Liu, Q.; Nan, S.; Xue, Y.; Wei, C.; Zhang, Y.; Luan, F.; Zhang, X.; Li, H. H_2O_2 and Ca^{2+} Signaling Crosstalk Counteracts ABA to Induce Seed Germination. *Antioxidants* **2022**, *11*, 1594. [CrossRef] [PubMed]
50. Cembrowska-Lech, D.; Koprowski, M.; Kępczyński, J. Germination Induction of Dormant *Avena fatua* Caryopses by KAR_1 and GA_3 Involving the Control of Reactive Oxygen Species (H_2O_2 and O^{2-}) and Enzymatic Antioxidants (Superoxide Dismutase and Catalase) Both in the Embryo and the Aleurone Layers. *J. Plant Physiol.* **2015**, *176*, 169–179. [CrossRef] [PubMed]
51. Jurdak, R.; Rodrigues, G.d.A.G.; Chaumont, N.; Schivre, G.; Bourbousse, C.; Barneche, F.; Bou Dagher Kharrat, M.; Bailly, C. Intracellular Reactive Oxygen Species Trafficking Participates in Seed Dormancy Alleviation in *Arabidopsis* Seeds. *New Phytol.* **2022**, *234*, 850–866. [CrossRef] [PubMed]
52. El-Maarouf-Bouteau, H.; Sajjad, Y.; Bazin, J.; Langlade, N.; Cristescu, S.M.; Balzergue, S.; Baudouin, E.; Bailly, C. Reactive Oxygen Species, Abscisic Acid and Ethylene Interact to Regulate Sunflower Seed Germination. *Plant Cell Environ.* **2015**, *38*, 364–374. [CrossRef] [PubMed]

53. Ishibashi, Y.; Koda, Y.; Zheng, S.-H.; Yuasa, T.; Iwaya-Inoue, M. Regulation of Soybean Seed Germination through Ethylene Production in Response to Reactive Oxygen Species. *Ann. Bot.* **2013**, *111*, 95–102. [CrossRef] [PubMed]
54. Mittler, R. Oxidative Stress, Antioxidants and Stress Tolerance. *Trends Plant Sci.* **2002**, *7*, 405–410. [CrossRef] [PubMed]
55. Choudhury, F.K.; Rivero, R.M.; Blumwald, E.; Mittler, R. Reactive Oxygen Species, Abiotic Stress and Stress Combination. *Plant J.* **2017**, *90*, 856–867. [CrossRef] [PubMed]
56. Hasanuzzaman, M.; Bhuyan, M.H.M.B.; Zulfiqar, F.; Raza, A.; Mohsin, S.M.; Mahmud, J.A.; Fujita, M.; Fotopoulos, V. Reactive Oxygen Species and Antioxidant Defense in Plants under Abiotic Stress: Revisiting the Crucial Role of a Universal Defense Regulator. *Antioxidants* **2020**, *9*, 681. [CrossRef] [PubMed]
57. Lee, S.; Kim, S.-G.; Park, C.-M. Salicylic Acid Promotes Seed Germination under High Salinity by Modulating Antioxidant Activity in *Arabidopsis*. *New Phytol.* **2010**, *188*, 626–637. [CrossRef] [PubMed]
58. Lee, S.; Park, C.-M. Modulation of Reactive Oxygen Species by Salicylic Acid in *Arabidopsis* Seed Germination under High Salinity. *Plant Sign Behav.* **2010**, *5*, 1534–1536. [CrossRef] [PubMed]
59. Lin, Y.; Yang, L.; Paul, M.; Zu, Y.; Tang, Z. Ethylene Promotes Germination of *Arabidopsis* Seed under Salinity by Decreasing Reactive Oxygen Species: Evidence for the Involvement of Nitric Oxide Simulated by Sodium Nitroprusside. *Plant Physiol. Biochem.* **2013**, *73*, 211–218. [CrossRef] [PubMed]
60. Ortiz-Espín, A.; Iglesias-Fernández, R.; Calderón, A.; Carbonero, P.; Sevilla, F.; Jiménez, A. Mitochondrial *AtTrxo1* Is Transcriptionally Regulated by AtbZIP9 and AtAZF2 and Affects Seed Germination under Saline Conditions. *J. Exp. Bot.* **2017**, *68*, 1025–1038. [CrossRef] [PubMed]
61. Yang, L.; Wang, X.; Chang, N.; Nan, W.; Wang, S.; Ruan, M.; Sun, L.; Li, S.; Bi, Y. Cytosolic Glucose-6-Phosphate Dehydrogenase Is Involved in Seed Germination and Root Growth Under Salinity in *Arabidopsis*. *Front. Plant Sci.* **2019**, *10*, 182. [CrossRef] [PubMed]
62. Nourimand, M.; Todd, C.D. There Is a Direct Link between Allantoin Concentration and Cadmium Tolerance in *Arabidopsis*. *Plant Physiol. Biochem.* **2019**, *135*, 441–449. [CrossRef] [PubMed]
63. Bhattacharjee, S. Heat and Chilling Induced Disruption of Redox Homeostasis and Its Regulation by Hydrogen Peroxide in Germinating Rice Seeds (*Oryza sativa* L., Cultivar Ratna). *Physiol. Mol. Biol. Plants* **2013**, *19*, 199–207. [CrossRef] [PubMed]
64. Liu, J.; Hasanuzzaman, M.; Wen, H.; Zhang, J.; Peng, T.; Sun, H.; Zhao, Q. High Temperature and Drought Stress Cause Abscisic Acid and Reactive Oxygen Species Accumulation and Suppress Seed Germination Growth in Rice. *Protoplasma* **2019**, *256*, 1217–1227. [CrossRef] [PubMed]
65. Sen, A.; Puthur, J.T. Influence of Different Seed Priming Techniques on Oxidative and Antioxidative Responses during the Germination of *Oryza sativa* Varieties. *Physiol. Mol. Biol. Plants* **2020**, *26*, 551–565. [CrossRef] [PubMed]
66. Kawaguchi, R.; Suriyasak, C.; Matsumoto, R.; Sawada, Y.; Sakai, Y.; Hamaoka, N.; Sasaki, K.; Yamane, K.; Kato, Y.; Bailly, C.; et al. Regulation of Reactive Oxygen Species and Phytohormones in Osmotic Stress Tolerance during Seed Germination in Indica Rice. *Front. Plant Sci.* **2023**, *14*, 1186960. [CrossRef] [PubMed]
67. Wang, Y.; Zhu, Q.; Luo, X.; Dai, G.; Hou, J.; Yu, F.; Zhang, L. Selenite Alleviates PEG-Induced Drought Stress during Rice Seed Germination through Antioxidant Regulation and Osmotic Adjustment. *Plant Soil. Environ.* **2026**, *72*, 271–283. [CrossRef]
68. Saux, M.; Ponnaiah, M.; Langlade, N.; Zanchetta, C.; Balliau, T.; El-Maarouf-Bouteau, H.; Bailly, C. A Multiscale Approach Reveals Regulatory Players of Water Stress Responses in Seeds during Germination. *Plant Cell Environ.* **2020**, *43*, 1300–1313. [CrossRef] [PubMed]
69. Macovei, A.; Pagano, A.; Cappuccio, M.; Gallotti, L.; Dondi, D.; De Sousa Araujo, S.; Fevereiro, P.; Balestrazzi, A. A Snapshot of the Trehalose Pathway During Seed Imbibition in Medicago Truncatula Reveals Temporal- and Stress-Dependent Shifts in Gene Expression Patterns Associated With Metabolite Changes. *Front. Plant Sci.* **2019**, *10*, 1590. [CrossRef] [PubMed]
70. Gondim, F.A.; Gomes-Filho, E.; Lacerda, C.F.; Prisco, J.T.; Azevedo Neto, A.D.; Marques, E.C. Pretreatment with H_2O_2 in Maize Seeds: Effects on Germination and Seedling Acclimation to Salt Stress. *Braz. J. Plant Physiol.* **2010**, *22*, 103–112. [CrossRef]
71. Deng, B.; Yang, K.; Zhang, Y.; Li, Z. Can Heavy Metal Pollution Defend Seed Germination against Heat Stress? Effect of Heavy Metals (Cu^{2+}, Cd^{2+} and Hg^{2+}) on Maize Seed Germination under High Temperature. *Environ. Pollut.* **2016**, *216*, 46–52. [CrossRef] [PubMed]
72. Meng, A.; Wen, D.; Zhang, C. Maize Seed Germination Under Low-Temperature Stress Impacts Seedling Growth Under Normal Temperature by Modulating Photosynthesis and Antioxidant Metabolism. *Front. Plant Sci.* **2022**, *13*, 843033. [CrossRef] [PubMed]
73. Mei, Y.; Song, S. Response to Temperature Stress of Reactive Oxygen Species Scavenging Enzymes in the Cross-Tolerance of Barley Seed Germination. *J. Zhejiang Univ. Sci. B* **2010**, *11*, 965–972. [CrossRef] [PubMed]
74. Cembrowska-Lech, D.; Rybak, K. Nanopriming of Barley Seeds—A Shotgun Approach to Improve Germination under Salt Stress Conditions by Regulating of Reactive Oxygen Species. *Plants* **2023**, *12*, 405. [CrossRef] [PubMed]
75. Wahid, A.; Perveen, M.; Gelani, S.; Basra, S.M.A. Pretreatment of Seed with H_2O_2 Improves Salt Tolerance of Wheat Seedlings by Alleviation of Oxidative Damage and Expression of Stress Proteins. *J. Plant Physiol.* **2007**, *164*, 283–294. [CrossRef] [PubMed]
76. Rai-Kalal, P.; Tomar, R.S.; Jajoo, A. H_2O_2 Signaling Regulates Seed Germination in ZnO Nanoprimed Wheat (*Triticum aestivum* L.) Seeds for Improving Plant Performance under Drought Stress. *Environ. Exp. Bot.* **2021**, *189*, 104561. [CrossRef]

77. Ma, H.; Tao, Y.; He, L.; Wang, F.; Wang, Z.; Yang, Y. Regulation of Redox Homeostasis and Proline Metabolism by Tea Polyphenols Ameliorates Salinity Tolerance during Wheat Seed Germination and Early Seedling Growth. *BMC Plant Biol.* **2026**, *26*, 306. [CrossRef] [PubMed]
78. Begum, N.; Hasanuzzaman, M.; Li, Y.; Akhtar, K.; Zhang, C.; Zhao, T. Seed Germination Behavior, Growth, Physiology and Antioxidant Metabolism of Four Contrasting Cultivars under Combined Drought and Salinity in Soybean. *Antioxidants* **2022**, *11*, 498. [CrossRef] [PubMed]
79. Xing, X.; Cao, C.; Li, S.; Wang, H.; Xu, Z.; Qi, Y.; Tong, F.; Jiang, H.; Wang, X. α-Naphthaleneacetic Acid Positively Regulates Soybean Seed Germination and Seedling Establishment by Increasing Antioxidant Capacity, Triacylglycerol Mobilization and Sucrose Transport under Drought Stress. *Plant Physiol. Biochem.* **2023**, *201*, 107890. [CrossRef] [PubMed]
80. Hajihashemi, S.; Skalicky, M.; Brestic, M.; Pavla, V. Cross-Talk between Nitric Oxide, Hydrogen Peroxide and Calcium in Salt-Stressed *Chenopodium quinoa* Willd. At Seed Germination Stage. *Plant Physiol. Biochem.* **2020**, *154*, 657–664. [CrossRef] [PubMed]
81. Causin, H.F.; Bordón, D.A.E.; Burrieza, H. Salinity Tolerance Mechanisms during Germination and Early Seedling Growth in *Chenopodium quinoa* Wild. Genotypes with Different Sensitivity to Saline Stress. *Env. Exp. Bot.* **2020**, *172*, 103995. [CrossRef]
82. Yao, X.; Zhou, M.; Ruan, J.; Peng, Y.; Yang, H.; Tang, Y.; Gao, A.; Cheng, J. Pretreatment with H_2O_2 Alleviates the Negative Impacts of NaCl Stress on Seed Germination of Tartary Buckwheat (*Fagopyrum tataricum*). *Plants* **2021**, *10*, 1784. [CrossRef] [PubMed]
83. Ellouzi, H.; Oueslati, S.; Hessini, K.; Rabhi, M.; Abdelly, C. Seed-Priming with H_2O_2 Alleviates Subsequent Salt Stress by Preventing ROS Production and Amplifying Antioxidant Defense in Cauliflower Seeds and Seedlings. *Sci. Hort.* **2021**, *288*, 110360. [CrossRef]
84. Bazvand, F.; Wojtyla, Ł.; Adamiec, M.; Garnczarska, M. Comparative Effects of Hydropriming and NaHS-Priming on Salinity Tolerance in *Brassica napus* L. Seedlings. *Plants* **2026**, *15*, 551. [CrossRef] [PubMed]
85. Hussain, S.; Ahmed, S.; Akram, W.; Li, G.; Yasin, N.A. Selenium Seed Priming Enhanced the Growth of Salt-Stressed *Brassica rapa* L. through Improving Plant Nutrition and the Antioxidant System. *Front. Plant Sci.* **2023**, *13*, 1050359. [CrossRef] [PubMed]
86. Campobenedetto, C.; Grange, E.; Mannino, G.; van Arkel, J.; Beekwilder, J.; Karlova, R.; Garabello, C.; Contartese, V.; Bertea, C.M. A Biostimulant Seed Treatment Improved Heat Stress Tolerance During Cucumber Seed Germination by Acting on the Antioxidant System and Glyoxylate Cycle. *Front. Plant Sci.* **2020**, *11*, 836. [CrossRef] [PubMed]
87. Rahmat, B.P.N.; Elfakhriano, I.F.; Carsono, N.; Damayanti, F.; Mubarok, S.; Ken, H.; Ezura, H.; Kang, S.W. *SlIAA9* Mutation Enhances Tomato Seed Resilience to Heat Stress. *Plant Physiol. Biochem.* **2026**, *232*, 111103. [CrossRef] [PubMed]
88. Hameed, A.; Rasheed, A.; Gul, B.; Khan, M.A. Salinity Inhibits Seed Germination of Perennial Halophytes *Limonium stocksii* and *Suaeda fruticosa* by Reducing Water Uptake and Ascorbate Dependent Antioxidant System. *Environ. Exp. Bot.* **2014**, *107*, 32–38. [CrossRef]
89. Gomes, M.P.; Carneiro, M.M.L.C.; Nogueira, C.O.G.; Soares, A.M.; Garcia, Q.S. The System Modulating ROS Content in Germinating Seeds of Two Brazilian Savanna Tree Species Exposed to As and Zn. *Acta Physiol. Plant* **2013**, *35*, 1011–1022. [CrossRef]
90. Sarkar, A.K.; Ghosh, S.; Oraon, S.; Ball, K.; Sadhukhan, S. Salinity-Triggered Modulation of Osmolytes and Antioxidant Defence in *Capsicum annuum* L. cv. Bullet during Seed Germination. *Discov. Plants* **2026**, *3*, 287. [CrossRef]
91. Han, G.; Hu, Z.; Zhao, Z.; Zhu, B.; Chen, J. Synergistic Effects of Temperature and Salt Stress on Seed Germination and the Antioxidant Enzyme System of Okra. *Front. Plant Sci.* **2026**, *17*, 1822950. [CrossRef] [PubMed]
92. Limão, M.A.R.; Rodrigues, M.H.B.S.; Santos, A.d.S.; Barbosa, L.d.S.; Lopes, K.P.; Dias, D.C.F.d.S. Hydrogen peroxide as a mitigator of salt stress for melon seed germination. *Com. Sci.* **2022**, *13*, e3798. [CrossRef]
93. Wang, X.; Shen, H.; Yang, L. The Response of Hormones, Reactive Oxygen Species and Nitric Oxide in the Polyethylene-Glycol-Promoted, Salt–Alkali-Stress-Induced Embryo Germination of *Sorbus pohuashanensis*. *Int. J. Mol. Sci.* **2024**, *25*, 5128. [CrossRef] [PubMed]
94. Mu, X.; Vidal, A.; Wagner, S.; Meyer, A.J.; Barneche, F.; Bailly, C. Integrating Redox Dynamics and Gene Networks Reveals ROS-Mediated Control of *Arabidopsis* Seed Germination under Water Stress. *bioRxiv* **2026**. [CrossRef]
95. Mu, Q.; Liu, Y.; Wu, X.; Chen, M.; Hu, J.; Qiao, F.; Hu, W.; Song, W.; Guan, Y. *OsSAP3* Enhances Rice Seed Vigor by Modulating Hydrogen Peroxide Homeostasis. *Rice* **2026**, *19*, 45. [CrossRef] [PubMed]
96. Yang, K.; Zhang, Y.; Zhu, L.; Li, Z.; Deng, B. Omethoate Treatment Mitigates High Salt Stress Inhibited Maize Seed Germination. *Pestic. Biochem. Physiol.* **2018**, *144*, 79–82. [CrossRef] [PubMed]
97. Long, W.; Zou, X.; Zhang, X. Transcriptome Analysis of Canola (*Brassica napus*) under Salt Stress at the Germination Stage. *PLoS ONE* **2015**, *10*, e0116217. [CrossRef] [PubMed]
98. Yan, H.; Nie, Y.; Cui, K.; Sun, J. Integrative Transcriptome and Metabolome Profiles Reveal Common and Unique Pathways Involved in Seed Initial Imbibition Under Artificial and Natural Salt Stresses During Germination of Halophyte Quinoa. *Front. Plant Sci.* **2022**, *13*, 853326. [CrossRef] [PubMed]

99. Zhu, Z.; Chen, H.; Xie, K.; Liu, C.; Li, L.; Liu, L.; Han, X.; Jiao, C.; Wan, Z.; Sha, A. Characterization of Drought-Responsive Transcriptome During Seed Germination in Adzuki Bean (*Vigna angularis* L.) by PacBio SMRT and Illumina Sequencing. *Front. Genet.* **2020**, *11*, 996. [CrossRef] [PubMed]
100. Dorone, Y.; Boeynaems, S.; Flores, E.; Jin, B.; Hateley, S.; Bossi, F.; Lazarus, E.; Pennington, J.G.; Michiels, E.; Decker, M.D.; et al. A Prion-like Protein Regulator of Seed Germination Undergoes Hydration-Dependent Phase Separation. *Cell* **2021**, *184*, 4284-4298.e27. [CrossRef] [PubMed]
101. Zhang, D.; He, S.; Fu, Y.; Yu, R.; Gao, X.; Wang, Z.; Liu, Z.; Guo, Y.; Chen, M. Transcriptome Analysis Reveals Key Genes in Response to Salinity Stress during Seed Germination in *Setaria italica*. *Env. Exp. Bot.* **2021**, *191*, 104604. [CrossRef]
102. Willems, P.; Mhamdi, A.; Stael, S.; Storme, V.; Kerchev, P.; Noctor, G.; Gevaert, K.; Van Breusegem, F. The ROS Wheel: Refining ROS Transcriptional Footprints. *Plant Physiol.* **2016**, *171*, 1720–1733. [CrossRef] [PubMed]
103. Lee, K.P.; Piskurewicz, U.; Turečková, V.; Strnad, M.; Lopez-Molina, L. A Seed Coat Bedding Assay Shows That RGL2-Dependent Release of Abscisic Acid by the Endosperm Controls Embryo Growth in *Arabidopsis* Dormant Seeds. *Proc. Nat. Acad. Sci. USA* **2010**, *107*, 19108–19113. [CrossRef] [PubMed]
104. Kang, J.; Yim, S.; Choi, H.; Kim, A.; Lee, K.P.; Lopez-Molina, L.; Martinoia, E.; Lee, Y. Abscisic Acid Transporters Cooperate to Control Seed Germination. *Nat. Commun.* **2015**, *6*, 8113. [CrossRef] [PubMed]
105. Ishibashi, Y.; Kasa, S.; Sakamoto, M.; Aoki, N.; Kai, K.; Yuasa, T.; Hanada, A.; Yamaguchi, S.; Iwaya-Inoue, M. A Role for Reactive Oxygen Species Produced by NADPH Oxidases in the Embryo and Aleurone Cells in Barley Seed Germination. *PLoS ONE* **2015**, *10*, e0143173. [CrossRef] [PubMed]
106. Mittler, R.; Vanderauwera, S.; Suzuki, N.; Miller, G.; Tognetti, V.B.; Vandepoele, K.; Gollery, M.; Shulaev, V.; Van Breusegem, F. ROS Signaling: The New Wave? *Trends Plant Sci.* **2011**, *16*, 300–309. [CrossRef] [PubMed]
107. Fichman, Y.; Mittler, R. Rapid Systemic Signaling during Abiotic and Biotic Stresses: Is the ROS Wave Master of All Trades? *Plant J.* **2020**, *102*, 887–896. [CrossRef] [PubMed]
108. Zandalinas, S.I.; Fichman, Y.; Mittler, R. Vascular Bundles Mediate Systemic Reactive Oxygen Signaling during Light Stress. *Plant Cell* **2020**, *32*, 3425–3435. [CrossRef] [PubMed]
109. Topham, A.T.; Taylor, R.E.; Yan, D.; Nambara, E.; Johnston, I.G.; Bassel, G.W. Temperature Variability Is Integrated by a Spatially Embedded Decision-Making Center to Break Dormancy in *Arabidopsis* Seeds. *Proc. Natl. Acad. Sci. USA* **2017**, *114*, 6629–6634. [CrossRef] [PubMed]
110. Footitt, S.; Marquez, J.; Schmuths, H.; Baker, A.; Theodoulou, F.L.; Holdsworth, M. Analysis of the Role of COMATOSE and Peroxisomal Beta-Oxidation in the Determination of Germination Potential in *Arabidopsis*. *J. Exp. Bot.* **2006**, *57*, 2805–2814. [CrossRef] [PubMed]
111. Pan, R.; Liu, J.; Hu, J. Peroxisomes in Plant Reproduction and Seed-Related Development. *J. Int. Plant Biol.* **2019**, *61*, 784–802. [CrossRef] [PubMed]
112. Su, T.; Li, W.; Wang, P.; Ma, C. Dynamics of Peroxisome Homeostasis and Its Role in Stress Response and Signaling in Plants. *Front. Plant Sci.* **2019**, *10*, 705. [CrossRef] [PubMed]
113. Molina-Moya, E.; Rodríguez-González, A.; Peláez-Vico, M.A.; Sandalio, L.M.; Romero-Puertas, M.C. Peroxisomal-Dependent Signalling and Dynamics Modulate Plant Stress Responses: Reactive Oxygen and Nitrogen Species as Key Molecules. *J. Exp. Bot.* **2025**, *76*, 3706–3721. [CrossRef] [PubMed]
114. van Zanten, M.; Koini, M.A.; Geyer, R.; Liu, Y.; Brambilla, V.; Bartels, D.; Koornneef, M.; Fransz, P.; Soppe, W.J.J. Seed Maturation in *Arabidopsis thaliana* Is Characterized by Nuclear Size Reduction and Increased Chromatin Condensation. *Proc. Natl. Acad. Sci. USA* **2011**, *108*, 20219–20224. [CrossRef] [PubMed]
115. Li, H.-C.; Chuang, K.; Henderson, J.T.; Rider, S.D., Jr.; Bai, Y.; Zhang, H.; Fountain, M.; Gerber, J.; Ogas, J. PICKLE Acts during Germination to Repress Expression of Embryonic Traits. *Plant J.* **2005**, *44*, 1010–1022. [CrossRef] [PubMed]
116. Perruc, E.; Kinoshita, N.; Lopez-Molina, L. The Role of Chromatin-Remodeling Factor PKL in Balancing Osmotic Stress Responses during *Arabidopsis* Seed Germination. *Plant J.* **2007**, *52*, 927–936. [CrossRef] [PubMed]
117. Hong, Y.; Ni, S.-J.; Zhang, G.-P. Transcriptome and Metabolome Analysis Reveals Regulatory Networks and Key Genes Controlling Barley Malting Quality in Responses to Drought Stress. *Plant Physiol. Biochem.* **2020**, *152*, 1–11. [CrossRef] [PubMed]
118. Nagel, M.; Arc, E.; Rajjou, L.; Cueff, G.; Bailly, M.; Clément, G.; Sanchez-Vicente, I.; Bailly, C.; Seal, C.E.; Roach, T.; et al. Impacts of Drought and Elevated Temperature on the Seeds of Malting Barley. *Front. Plant Sci.* **2022**, *13*, 1049323. [CrossRef] [PubMed]
119. Ferchichi, S.; Jeddi, K.; Wasli, H.; Mejri, M.; Msaada, K.; Siddique, K.H.M.; Hessini, K. Effects of Salinity Stress during Plant Development in Barley (*Hordium velgare* L.) on Subsequent Seed Quality and Redox State. *Russ. J. Plant Physiol.* **2024**, *71*, 142. [CrossRef]
120. Paravar, A.; Farahani, S.M.; Rezazadeh, A.; Adetunji, A.E.; Farooq, M. Moisture Content and Mycorrhizal Fungi in Maternal Environment Influence Performance and Composition of *Lallemantia* Species Offspring. *Heliyon* **2024**, *10*, e31334. [CrossRef] [PubMed]

121. Azad, S.M.; Gil-Villar, D.; Yordán, E.; Mestre, P.; Forment, J.; Tárraga, S.; González, C.; Niñoles, R.; Gadea, J. Maternal Light and Temperature Modulate Seed Longevity in *Arabidopsis*. *bioRxiv* **2025**. [CrossRef]
122. Ugalde, J.M.; Meyer, A.J. Genetically Encoded Biosensors as Gateways to Retrograde Redox Signalling in Live Plants. *J. Exp. Bot.* **2025**, *76*, 3722–3737. [CrossRef] [PubMed]
123. Nauman Khan, M.; Fu, C.; Liu, X.; Li, Y.; Yan, J.; Yue, L.; Li, J.; Khan, Z.; Nie, L.; Wu, H. Nanopriming with Selenium Doped Carbon Dots Improved Rapeseed Germination and Seedling Salt Tolerance. *Crop J.* **2024**, *12*, 1333–1343. [CrossRef]
124. Zhang, H.; Gao, W.; Wang, N.; Wang, X.; Ma, X.; Chen, J.; Tang, Q.; Zhang, J. Nanopriming with Carbon Dots Enhances Cotton Seed Germination and Salt Tolerance by Activating Salt-Induced ROS Signaling to Modulate Na+ Homeostasis. *Plant Nano Biol.* **2026**, *15*, 100225. [CrossRef]
125. El-Shazoly, R.M.; Othman, A.A.; Zaheer, M.S.; Al-Hossainy, A.F.; Abdel-Wahab, D.A. Zinc Oxide Seed Priming Enhances Drought Tolerance in Wheat Seedlings by Improving Antioxidant Activity and Osmoprotection. *Sci. Rep.* **2025**, *15*, 3863. [CrossRef] [PubMed]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.