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Review

Oxidative Stress, Ageing and Methods of Seed Invigoration: An Overview and Perspectives

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Abstract: The maintenance of seed quality during the long-term conservation of plant genetic resources is crucial for averting the projected food crises that are linked to the changing climate and rising world population. However, ageing-induced loss of seed vigour and viability during storage remains an inevitable process that compromises productivity in several orthodox-seeded crop species. Seed ageing under prolonged storage, which can occur even under optimal conditions, induces several modifications capable of causing loss of intrinsic physiological quality traits, including germination capacity and vigour, and stand establishment. The problems posed by seed ageing have motivated the development of various techniques for mitigating their detrimental effects. These invigoration techniques generally fall within one of two categories: (1) priming or pre-hydrating seeds in a solution for improved post-harvest performance, or (2) post-storage reinvigoration which often involves soaking seeds recovered from storage in a solution. Seed priming methods are generally divided into classical (hydropriming, osmopriming, redox priming, biostimulant priming, etc.) and advanced (nanopriming, magnetopriming and priming using other physical agents) techniques. With the increasing popularity of seed invigoration techniques to achieve the much-desired enhanced productivity and resilience in the face of a changing climate, there is an urgent need to explore these techniques effectively (in addition to other important practices such as plant breeding, fertilizer application, and the control of pests and diseases). This review aims to provide an overview of ageing in orthodox seeds and invigoration techniques that can enhance desirable agronomic and physiological characters.

Keywords: gene bank; germination; orthodox seeds; priming; reactive oxygen species

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1. Introduction

Given that global food demand is rising, it is necessary to ensure the conservation of genetic resources to preserve ecosystem resilience and to protect plant biodiversity for future agricultural food production [1,2]. Over a billion people are estimated to be added to the already large world population by 2050 [3]. If no pragmatic response is implemented, the challenge of food security will worsen with the increasing impact of hunger and poverty, particularly in developing countries.

The worrisome, widespread drop in crop yield due to a combination of factors, including, but not limited to, soil degradation and drastic changes in the climate [4,5], and the negative crop production projections across the globe [6,7], all point to a need for another Green Revolution with much more yield and better conservation of resources than the first [8,9]. For instance, by the mid-twenty-first century, an increase of up to 60% in food production is estimated to be needed to feed the growing population [10,11]. This

underscores the need to prioritise various approaches and research interventions towards increased crop production.

Attempts being made to address the identified needs include the development of approaches, such as conservation agriculture, sustainable intensification [12,13], and climate-smart agriculture [10,11], with the aim of raising productivity, decreasing emissions and reducing susceptibility to environmental stresses (i.e., improved resilience). The benefits of the application of cutting-edge techniques, based on investigative research efforts, in various areas of agricultural science, including agroecology, ecophysiology, soil science and plant physiology [14], have been recognised. Addressing the identified needs can be achieved using dynamic approaches involving the application of modern biotechnological and physiological research techniques, among others, geared towards addressing low- crop yield-related challenges, often attributable to the low quality of genetic resources, such as seeds—the principal yield determining factor [15], which forms the subject of this study.

Seed, as a genetic resource, may be regarded as the insurance system for world food schemes. The depletion of this resource exposes the schemes to higher risks, which could ultimately lead to catastrophic failure. Without a systematic approach for the conservation of seed genetic and physiological quality, achieving the much-desired increased productivity and greater resilience in the face of the rising world population and changing climate is a mirage. Moreover, seeds are considered the main basis for the sustenance of humans as plants form over 80% of the human diet; promoting high-quality seed delivery is thus essential for enhancing crop production and plant tolerance to environmental challenges [15]. Achieving food security largely depends on the seed security of seed-producing communities in all cropping seasons [15]. The application of advances in plant physiology, particularly the various techniques of pre-hydration treatment (which uses priming technology to invigorate debilitated germplasms), in addition to other important components, such as plant breeding for adaptation to climate change and higher yields, and cultural practices (e.g., irrigation, fertilizer application, and control of pests and diseases), is needed to improve seed performance, crop yields, maximum yield, and to enable planting on less favourable land, by making seeds better able to withstand sub-optimal conditions, thereby reducing crop losses. Accordingly, agriculture in this century and subsequently can be more productive and provide for improved conservation of plant genetic resources compared to previous periods. Heightened efforts in this regard, therefore, will ensure that the prospect of reaching millions of the poor with crop production research benefits is achieved [8].

2. Storage of Orthodox Seeds in Gene Banks

In terms of conserving plant genetic resources, seed capacity for prolonged storage is particularly essential for gene banks. As far back as 1908, Ewart had grouped seed longevity into short-, medium- and long-term, providing insights into the duration of seed storage before severe viability loss [16]. Later, several experiments testing seed longevity were conducted under artificial and natural sowing conditions. The Beal [17–19] and the Vienna [20,21] germination studies include the oldest (over 100 years) seed longevity studies performed under natural conditions [22]. Other pioneering seed longevity studies [23–25] have shown that moisture content, temperature, relative humidity, and oxygen are the critical factors influencing seed viability and vigour during storage; however, genetic factors and pre-storage conditions are also important [16].

At moisture levels as low as 5% (fresh mass basis) or less, and at sub-zero temperatures (usually −18 °C) in dry conditions, the mature seeds of some species classified as orthodox can be stored for long periods [26–29]. This is also the easiest method of conserving most spermatophyte genetic resources in conventional gene banks [30], but seeds do not retain their initial quality with extended storage, gradually deteriorating, and inevitably proceeding towards death [31,32]. For instance, seeds that were initially stored in a gene bank at 5 °C but were later moved to −18 °C and stored for between 15–19 years suffered a decline in germination capacity from 91% to 11% in *Brassica oleracea* and 97% to 2% in

Lactuca sativa [33]. The post-harvest loss of physiological quality of seeds, even when seeds are stored in gene banks, has thus remained a major issue demanding attention for long-term storage [34]. As seeds deteriorate, vigour is first lost, after which comes a loss of viability [35].

Moreover, some species classified as recalcitrant (not covered in this review) have desiccation-sensitive seeds and are not amenable to short or long-term storage under the conditions mentioned above [30]. With the development of cryostorage techniques, involving germplasm storage at ultra-low temperatures (−120 to −196 degrees, [36]), the life span of seeds (including both orthodox and recalcitrant species) can be further extended, but not indefinitely [33,34,37]. This implies that though the degree and rate of deterioration of seeds stored under the enhanced conditions of the conventional seed gene banks can be reduced to an appreciable level [32], seed deterioration cannot be completely halted. Walters et al. (2004) showed, by measuring changes in the viability of seeds of several plant species over 20 years of cryostorage, that cryogenic temperatures could not sufficiently stop seed deterioration. They further suggested that there could be as much as a 300% variation in longevity among species and within accessions stored in these conditions, as the degree of longevity in cryostorage depends on the inherent properties of seeds and seed handling, such as the pre-storage temperature and the year of harvest. This implies that cryostorage temperatures do not completely halt all biological activities; molecules are still sufficiently mobile at these low temperatures to allow ageing reactions to proceed [37].

3. Germination-Related Physiology

Under favourable conditions of moisture, warmth, and oxygen, quiescent but viable seed is vivified, forming an actively metabolising structure, in a process described as germination [38]. The progress of germination can be roughly assessed by measuring respiration or water uptake [39], while the completion of germination can be taken to be when the system no longer depends on its stored food [38], or is visibly marked by the protrusion of the radicle [39]. In instances where the radicle may grow before penetrating the surrounding tissues, germination can be taken to have been completed from the time a sustained increase in seed fresh weight is recognised [39]. So, the initiation of germinative activities gradually and eventually leads to the formation of normal, growing seedlings [38].

In cases where a viable seed fails to germinate under favourable germination conditions, dormancy is said to have set in as such seeds require additional conditions, such as a specific light, or temperature regime, or exposure to chemical or physical treatments [39]. Dormant seeds that have been hydrated undergo almost all the metabolic processes that take place during the germination of nondormant seeds, yet the protrusion of the radicle does not occur [40]. Their germination takes place later when the additional conditions required for release from dormancy are met. Three identified stages of seed germination include water imbibition (first stage), nutrient conversion (second stage), and cell elongation and cell multiplication (third stage) [41]. The events following germination, such as the mobilisation of food reserves from the endosperm, supply the much-needed energy for seedling growth until the seedlings become photoautotrophic [42]. The seed germination pattern usually follows a sigmoid curve whereby a few seeds germinate earlier than the others in a population, followed by a rapid rise in percentage germination, and then relatively late germination of a few seeds is recorded. Seed-germination curves are generally right-skewed as the occurrence of more germinations is recorded in the first half of the germination period than the second. Whilst the shape of the curves are generally similar, notable differences in germination patterns are observed among populations [39].

4. Oxidative Stress in Plants

Oxidative stress is widely described as a physiological state (response) in cells, tissues and organs, as a result of increased pro-oxidative activities (through the generation of reactive oxygen species (ROS)) compared to antioxidative (enzymic and non-enzymic) activities [43]. This is a consequence of aerobic metabolism during which aerobic organ-

isms produce incompletely paired, oxygen-containing radicals formed by the unavoidable leakage of electrons onto molecular oxygen during electron transport in the mitochondria, chloroplast, and cell membranes [44]. The generation of ROS may be triggered by severe abiotic and biotic stress conditions [45]. The physiological responses are often characterised by a gradual accretion of various oxidised biomolecules, such as nucleic acids, proteins, lipids, polysaccharides, and metabolites, causing deleterious changes in normal biochemical, mechanical, and physical functions of cell components [43].

Oxidative stress functions, therefore, as an injurious factor. The main mechanisms involve altering the balance between the levels of generated and quenched ROS owing to the upset of regular cellular metabolism, and ROS biosynthesis as a component of developmental processes such as the signalling responses needed for adaptation and defence or programmed cell death [43,46]. Demidchik and Maathuis [47] stated that plants could employ ROS accumulation for encoding and recognising various stress factors, including xenobiotic stressors such as nanoparticles and herbicides that were not recognised before. Stress factors often engender secondary metabolic effects to be overcome by plant tissues for survival and restoration of growth and development [48]. For instance, salinity [49], drought [44], desiccation [50], light [51], temperature [52], and pathogens [46] can induce oxidative stress by increasing the production of free radicals and reactive oxygen species (ROS). Uncontrolled production of ROS can upset the balance of ROS generated during aerobic events and the antioxidative defence system [53], leading to oxidative stress [54].

4.1. Biochemical Effects of Ageing and Oxidative Stress in Seeds

Oxidative stress has been implicated in the loss of vigour in plant tissues [55]. Loss of vigour in plant tissues is a fundamental physiological phenomenon observed when plant tissues are exposed to environmental stress of any type (abiotic and biotic) under suboptimal external (both agricultural and natural) conditions [56]. It is a pressing global challenge for modern agriculture. Both abiotic and biotic factors can cause oxidative stress [57], which is widely described as the physiological state (response) brought about by increased pro-oxidative activities (through a gradual generation and accumulation of reactive oxygen species (ROS)) over antioxidative (enzymic and non-enzymic) activities [43,58]. In *B. oleracea*, for instance, seed deterioration has been related to changes in the levels of electrolyte leakage [59], proline, proteins, soluble sugars, and phenolic compounds [60]. However, Golovina et al. [61,62] reported no change in protein secondary structure in some seeds of orthodox species stored for 20 to 30 years, including *Allium cepa*, *Raphanus sativus*, *Cucumis melo*, *Capsicum annuum,* and *Brassica napus*, despite the loss of membrane integrity. In *L. sativa*, seed deterioration has been attributed to changes in the levels of lipid hydroperoxides [63] and volatile products such as aldehydes and alcohols [64]. Of these environmental conditions, abiotic stress is recognised to constitute a major drawback to crop farming [65,66], accounting for an approximately 51–82% loss of potential crop yield worldwide [56,67]. In many cases, abiotic stressors engender secondary metabolic effects that need to be overcome by plant tissues for survival and restoration of growth and development [48]. For instance, salinity [49], drought [44], light [51] and temperature [52] induce oxidative stress by increasing the production of free radicals and ROS, thereby offsetting the balance of ROS generated during metabolic events and the defence system [53]. The impacts of oxidative stress are usually expressed in relation to the overall growth of the plant, including vigour, yield, biomass accumulation or primary assimilation events [56], as well as in terms of the quality of seed [68].

Seeds, due to their rich genetic diversity, are considered the most efficient natural means of protecting the variability of genetic material, in comparison to somatic tissues. The challenge of loss of vigour creates a severe threat [69] which risks the conservation of millions of genetic materials kept in several world gene banks [70], making the understanding of loss of vigour, and consequential seed ageing in storage, vital for plant physiologists. Seed ageing has been intimately linked to oxidative stress involving ROS which are highly

reactive, toxic, and capable of causing degradative reactions of several biomolecules over an extended storage period [69,71]. Biological molecules, including carbohydrates, lipids, proteins, and polynucleic acids, such as DNA and RNA, are believed to be the main ROS targets during oxidative stress [43,72]. The physiological lesions that result include loss of membrane integrity (through lipid peroxidation), reduced respiration, enzyme inactivation and degradation, and genetic degradation [31,73,74], leading to severely damaging effects on seed vigour, viability and germinability, especially.

4.2. Oxidation of Major Biological Molecules

4.2.1. Lipids

Among other affected biomolecules, ROS-mediated oxidation of polyunsaturated fatty acids (lipid peroxidation) is the most harmful as it can cause chain reactions involving the formation and spread of ROS [75]. Lipid peroxidation is suggested to be a significant bioindicator of oxidative stress [76]. The damaging effect is irreversible, causing severe degradation of the membrane, inactivation of enzymes, total loss of membrane-bound protein activities, and cell death [43]. Lipid peroxidation has been implicated in the loss of viability during storage of seeds of many crop species [77,78] and has been shown to lead to swelling of mitochondria, increased membrane viscosity and heightened bilayer permeability (measured as increased solute leakage) [31,79]. Products of lipid oxidation can also cause DNA damage and interrupt the normal functioning of several cellular systems [79,80].

With respect to mechanisms, lipid peroxidation can occur via non-enzymic and enzymic processes [75,81]. The non-enzymic, ROS-mediated, process of lipid peroxidation entails an activation (initiation) stage involving ROS generation, a distribution (propagation) stage involving ROS chain reactions, and a termination stage in which non-radical products are formed [75,82]. The peroxidation initiation stage is activated by the removal of a hydrogen atom from a methylene ($-CH_2-$) group, leaving behind $-^{\bullet}CH-$ (lipid radical), by sufficiently reactive species such as alkoxyl ($RO^{\bullet}$), hydroxyl radicals ($HO^{\bullet}$), peroxyl radicals ($ROO^{\bullet}$), hydroperoxyl ($HO_2^{\bullet}$) and peroxynitrite, but not superoxide ($O_2^{\bullet -}$) or hydrogen peroxide (H_2O_2). Saturated and monounsaturated fatty acids, for example, oleic acid, with one double bond and 18 carbon atoms, can be subjected to oxidation reactions but not the chain reaction of lipid peroxidation, as they are less vulnerable [75,83]. However, the polyunsaturated fatty acids of cellular membrane phospholipids contain double bonds, which make them susceptible to peroxidation by the facilitation of hydrogen atom removal [43,82,84]. The lipid radicals formed then trigger O_2-mediated chain reactions involving the formation of lipid peroxyl radicals ($LOO^{\bullet}$), which in turn abstract hydrogen atoms from nearby fatty acid molecules forming a stable intermediate lipid hydroperoxide (LOOH) and another lipid radical in the propagation phase [43,83]. The process is limited by the termination reaction phase producing non-radical products. In addition, non-radical peroxidation of lipids can occur by polyunsaturated fatty acids reacting with singlet oxygen ($^1O_2$) forming LOOH without production of intermediate radicals [85–88]. Though reasonably stable, lipid peroxides may be decomposed by metal complexes in a reaction catalysed by transition metals producing radicals that can reinitiate peroxidation via redox cycling of the metal ions, forming products, such as 4-hydroxy-2-nonenal (4HNE), 4-hydroxyhexenal (4-HHE), and malonaldehyde (MDA), which are useful and extensively studied biomarkers of lipid peroxidation [75,82,83,89]. These aldehydes, in turn, bind with DNA or protein, causing more severe damage [90]. Loss of membrane integrity, breakdown of organelles, oxidation and impairment of DNA, RNA, and proteins result where there is severe lipid peroxidation reaction [76,88]. Of these aldehydes, 4-HNE is considered the key product of the peroxidation of omega-6 fatty acids, such as linoleic acid (C18:2, n-6) and arachidonic acid (C20:4, n-6). The production of 4-HHE, the aldehyde thought to induce the permeability of mitochondrial inner membrane [91] and upset metabolic events [92], has been reported from the peroxidation of omega-3 fatty acids, such as α-linolenic acid (C18:3, n-3) and docosahexaenoic acid (C22:6, n-3) [83].

In enzymic peroxidation, dioxygenases including lipoxygenases (LOX enzymes) are considered the key oxidising enzymes of polyunsaturated fatty acids, with linoleic acids (C18:2 and C18:3) as the major substrates [81,93]. In plants, LOX enzymes can add an oxygen molecule at carbon 9 or 13 of C18-fatty acids [81], forming 9- and 13-hydroperoxyl derivatives of linoleic acid, respectively [94]. The involvement of LOX enzymes in the ageing-induced lipid peroxidation of seeds has been investigated in several species where it was demonstrated that absence or lowering of LOX enzyme activity decreased the levels of MDA (*Zea mays* [95]), MDA and LOOH (*Oryza sativa* seeds [96]) promoted storability and germination (*Oryza sativa* seeds [97]), and improved vigour and viability (*Nicotiana tabacum* [98]).

4.2.2. Proteins

Since reactive oxidants can be indiscriminately generated in cells, especially at a heightened rate during abiotic or biotic stress, proteins are also a major target biomolecule [99,100], as they are abundant and readily reactive with several oxidants [101]. Proteins constitute about 68% of oxidised biomolecules [99,102]; thus, protein oxidation is a useful bioindicator of oxidative stress [100]. Defined as a covalent alteration of proteins by reactive oxidants or oxidative stress spinoffs [100], the ROS-mediated oxidation of proteins has been described extensively [103–106]. Protein oxidation often occurs even under normal physiological circumstances indicating that it is not always an injurious plant process [75,107]. Avery (2011) suggested that some proteins are more vulnerable to oxidation than others due to factors such as more easily oxidised amino acid residue content, metal-binding sites, the localisation of protein within cells, molecular conformation, and degradation rate. It is becoming increasingly clear that newly synthesised proteins are highly susceptible to post-synthesis oxidative degradation, suggesting that attaining and conforming to a stable multimeric protein complex may be protective against oxidative injury [108,109]. The oxidation of protein can facilitate the build-up of toxic non-native proteins capable of inducing programmed cell death in severe cases [43,75]. The production of unstable intermediates and the formation of stable products are useful for the estimation of protein damage [101]. ROS-induced protein injury can vary since protein properties are not all equivalent. The extremely reactive ROS, $HO^{\bullet}$, usually generated from H_2O_2 via the Fenton reaction, often leads to non-specific oxidation, unlike the specific type caused by other ROS [75]. Other ROS causing oxidation of proteins include the radicals of alkoxyl ($RO^{\bullet}$), hydroperoxyl ($HO_2^{\bullet}$), peroxyl ($RO_2^{\bullet}$), superoxide ($O_2^{\bullet -}$) and non-radical species, such as hypochlorous acid (HOCl), hydrogen peroxide (H_2O_2), ozone (O_3), peroxynitrite ($ONOO^-$), and singlet oxygen ($^1O_2$) [75,110]. While the oxidation of certain amino acids (e.g., those containing sulphur) is reversible, most ROS-mediated modifications are characterised by irreversible loss or inactivation of the parent amino acid residue, and catalytic, metabolic, regulatory, structural, or other activities and functions leading to protein damage or elimination [43,101,111]. Irreversible amino acid modifications, such as to arginine and lysine, tryptophan and tyrosine, the production of dityrosine, and protein-to-protein cross-linking, are in most cases accountable for the permanent shutdown of function of the affected proteins which are later degraded [111]. The reversible types of amino acid modification, such as S-nitrosylation and glutathionylation, may play a redox regulatory role protecting cysteine from irreversible oxidation, as well as modulating protein function [100,111]. The main oxidative modifications of proteins are outlined in Table 1.

Table 1. Commonly reported ROS-induced modifications of polyunsaturated fatty acids (PUFA), proteins, carbohydrates, and DNA.

Examples of commonly reported ROS-induced modifications of PUFA [100]	
PUFA	Oxidised product
Linoleic acid (18:2)	4-HNE
Linolenic acid (18:3)	Cyclic oxylipin, hydroxyoctadecatrieonic acid, MDA

Table 1. *Cont.*

Examples of commonly reported ROS-induced modifications of proteins [43,100,112,113]	
Amino acid	Oxidised product
Cysteine	Cysteic acid (cysteine sulfonic acid)
Methionine	Methionine sulfone
Arginine, Lysine, Proline, Threonine	Carbonyls (ketones, aldehydes): aminoadipic semialdehyde, pyrrolidone, acrolein, 4-HNE, MDA, glu γ-semialdehyde, 2-amino-3-ketobutyric acid
Glutamyl (glutathione, glutamine, glutamate)	Pyruvic acid, oxalic acid
Histidine	2-Oxohistidine, 4-HNE, aspartate, asparagine
Phenylalanine	Hydroxyphenylalanines
Tryptophan	Kynurenine
Tyrosine	3-Nitrotyrosine
Examples of commonly reported ROS-induced modifications of carbohydrates [100,114]	
Sugar	Oxidised product
Aldohexose, polyol	Aldopentose, formic acid
Examples of commonly reported ROS-induced modifications of DNA [100]	
DNA	Oxidised product
Purines (e.g., guanine)	8-Hydroxyguanine, FapyGua

The most common mechanisms of ROS-mediated protein damage involve the direct metal-catalysed oxidation (primary carbonylation) of S-containing amino acid residues such as:

(1) cysteine (Cys) to produce cystine (disulfide), which is further oxidised through cysteine sulfenic acid to form cysteine sulfinic acid; these initial stages are reversible until the highest oxidation and damaging level where cysteic acid is irreversibly formed [43,100,111];
(2) methionine (Met) to produce methionine sulfoxide. This stage is also reversible, but the final stage of Met oxidation to sulfone seems to be damaging and irreversible [100]; and
(3) most of the other amino acids, especially arginine (Arg), lysine (Lys), proline (Pro), and threonine (Thr) form stable aldehydes or ketones (carbonyls) in an irreversible reaction [75,100,112] that is not particular to any oxidants [113,115]. Thus, the extent of reactive oxidant-induced modification of proteins is generically measured as protein carbonyl [113,116,117].

Carbonyl formation (protein carbonylation, (PC)) demands higher energy inputs than the oxidation of other AA residues and leads to deleterious alterations of protein structure and function [43]. Secondary carbonylation reactions may occur by the reaction of proteins with aggressive lipid peroxidation products, such as 4HNE and MDA [43,75]. In addition, carbonyl formation can result from protein glycation or glycoxidation [118,119], which may be a confounding factor in using carbonylation as an exclusive oxidation biomarker [113], or by direct protein backbone oxidation forming protein fragments with an N-terminal α-ketoacyl amino acid residue [111]. All these processes severely alter or inhibit the physiological and enzymatic activities of protein [43]. Heightened PC has been reported for several plant oxidative stresses [120] induced by salinity [121,122], dehydration [116,123], heavy metals [124,125], pathogen attack [126,127], and ROS-induced seed ageing [128,129].

4.2.3. Carbohydrates

Studies on the oxidative modification of carbohydrates have received less attention even though carbohydrates are considered more abundant than the other plant biomolecules [43]. As with other biomolecules, the oxidative modification of carbohydrates may be injurious to living systems [43]. Free polyols, such as mannitol, pinitol and sorbitol [130], and sugars, are oxidised by $HO^{\bullet}$, mainly forming formic acid [100]. Miller [131] stated that arabinogalactan, cellulose, pectin, and similar polysaccharides

in the cell wall could be broken down by $HO^{\bullet}$. The auxin-mediated extension of cells induces the generation of ROS, which is used by cell wall-bound peroxidases to produce $HO^{\bullet}$ near scission sites [100,132]. Moreover, cell wall Cu^{2+} reduced to Cu^{+} by $O_2^{\bullet -}$ and ascorbate can produce $HO^{\bullet}$ by reacting with apoplastic H_2O_2 [133]. The $HO^{\bullet}$ formed causes non-enzymatic separation of pectins and xyloglucans, leading to loosening of the cell wall [43,133]. Similar Fenton reactions of H_2O_2 with Cu or Fe might substantially increase under stress conditions, leading to deleterious effects [43,134]. On the other hand, simple sugars, disaccharides [43,135] and some osmoprotectants (e.g., mannitol, sorbitol, proline, and myo-inositol) may be capable of scavenging ROS, such as $HO^{\bullet}$ [130]. Increased levels of carbohydrate, such as mannitol, sucrose, and glucose have been correlated with oxidative stress resistance in several species of plant [136,137]; however, there is a dearth of information on the direct connection between the physiology of plants and the ROS-induced oxidation of carbohydrates [43].

4.2.4. Polynucleotides

The oxidative modification of DNA is often implicated in the ageing of seeds [69,138] and, in some cases, perennial plants [43]. Essentially, ROS attack on DNA causes chemical modification of bases, fragmentation of deoxysugar, and breaking of strands [139]. Again, $HO^{\bullet}$, being the most reactive, are particularly harmful to polynucleic acids (DNA and RNA) [43]. $HO^{\bullet}$ attaches to double bonds of nucleotide bases and abstracts H^{+} from 2′-deoxyribose (resulting in sugar damage) [139] and $-CH_3-$ of thymine [43]. $HO^{\bullet}$ can also oxidise purines, forming products such as 7-hydro-8-oxoguanine (8-oxoG). The formation of 2,6-diamino-4-hydroxy-5-formamidopyrimidine (FapyGua) has also been reported as a product of polynucleic acid oxidation [43,139]. Guanine is often attacked by $^1O_2$, but not $O_2^{\bullet -}$ and H_2O_2, to form 8-Hydroxyguanine [100]. ROS-modification of DNA can be both direct and indirect. Often, MDA (a breakdown product of PUFA) conjugation with guanine leads to the formation of an additional ring [100,140]. DNA impairment has both cytotoxic and genotoxic effects [141]. Besides mutations, DNA oxidation can cause alterations of cytosine methylation required for the regulation of gene expression [100]. Repair mechanisms of the oxidative damage of plant DNA include directly reversing the impairment caused as well as replacing the base or even the entire nucleotide [100,142,143]. A defence system, both in cytosol and organelles, may also be implemented as a form of protection [43]. Under oxidative stress, however, nuclear ROS-scavengers (glutathione and peroxiredoxin) inadequately protect the DNA [100,142]. Enzymes such as catalase and ascorbate peroxidase in the cytosol are required to protect nuclear DNA in such conditions [43,144].

4.3. Cellular Generation of Reactive Oxygen Species (ROS)

Reactive oxygen species are produced at several locations in the cells, such as chloroplasts, mitochondria, the plasma membrane, peroxisomes, apoplast, the endoplasmic reticulum, and the cell wall. Conventionally, it is believed that ROS are unavoidably produced during metabolic processes of aerobic systems [145]. Several possible sources of ROS have been identified in plants, including reactions involving normal plant metabolisms such as photosynthesis [46] and mitochondrial respiration [72]. There are other ROS sources as well, which are produced from abiotic stress-induced pathways. For example, during photorespiration, the oxidation of glycolate by glycolate oxidase in peroxisomes accounts for the majority of ROS, such as hydrogen peroxide [46]. Recently, more plant ROS sources have been recognised, such as plasma membrane-bound peroxidases, amine oxidases, and nicotinamide adenine dinucleotide phosphate (NADPH) oxidases, involved in events such as apoptosis and defence against pathogens [146]. While a low level of cellular ROS is formed under normal growth conditions, ROS formation is heightened under stress conditions [46].

Various enzymes (e.g., oxygenases) and non-enzymic processes “fix” oxygen atoms into various biological molecules [147]. Partly reduced forms of molecular oxygen (O_2),

resulting from O_2 excitation to produce singlet oxygen ($^1O_2$), or from the transfer of one electron to O_2 forming the superoxide radical ($O_2^{\bullet -}$), two electrons to O_2 forming hydrogen peroxide (H_2O_2), or three electrons to O_2 forming the hydroxyl radical ($HO^{\bullet}$) [46], are more readily reactive than atmospheric oxygen. Hence, they are termed reactive oxygen species [58]. These ROS can cause unrestrained oxidation of various biomolecules leading to oxidative cellular damage [46]. Metabolically active organelles, such as the mitochondria, peroxisomes and chloroplasts, processing extremely oxidising reactions, or that have high electron flow rates, are the primary ROS sources within cells [148]. Ubiquinone-cytochrome complexes I and III of the electron transport chain (ETC) are the main sites of $O_2^{\bullet -}$ production in mitochondria, while photosystems I and II are the main sites of $^1O_2$ and $O_2^{\bullet -}$ production in chloroplasts [148].

4.3.1. The Dual Capacity of ROS

Though ROS are harmful when in excess, they are beneficial in cellular processes, such as signalling, cellular differentiation and proliferation [72], ion transport, and gene expression [149], when produced in moderation. While plants can employ the ROS steady-state concentration to monitor stress levels within cells, this must be tightly controlled to avoid over-accretion of ROS that can cause cell death [146]. The ROS-induced death of cells can occur as a result of oxidative modifications of biomolecules, such as enzymes, DNA, RNA, proteins, and membrane lipids (the classical concept). On the other hand, heightened ROS levels can trigger programmed cell death, which has been shown by anti-apoptotic genes suppression of paraquat-induced oxidative stress cell death in *Nicotiana tabacum* [46]. Further, some cell death, earlier believed to be directly caused by oxidative stress, is now regarded as programmed cell death, consistent with the view that ROS can have beneficial effects on plants, promoting physiological function, cellular proliferation, and viability [72]. In essence, plants require a regulatory system to ensure low ROS concentration, and another to allow for the quenching of surplus ROS production [46]. Balancing the different steady-state ROS level and generated ROS types, as driven by the interaction of different ROS-generating and ROS-quenching systems, is also important. The balance may be altered significantly depending on the physiological state of the plant and the combination of various biochemical, developmental, and environmental stimuli [46]. Apart from aggravating cellular impairment, ROS can stimulate the expression of defence genes. ROS, such as $O_2^{\bullet -}$ or H_2O_2, can separately or jointly induce various genes, thereby allowing for more ROS signalling flexibility. Furthermore, reports on plant responses to abiotic stress show that ROS may be involved in regular signalling for adaptation to stress [146].

4.3.2. ROS Scavenging in Plant Cells

The main plant defence system against ROS involves the activities of antioxidants –compounds that can protect cells from oxidative injury even when present in low quantity [149]. These antioxidants can be either enzymatic or non-enzymatic. Major enzymic antioxidants include

(1) Superoxide dismutases (SODs): These are ubiquitous metalloenzymes involved in essential defence against superoxide [149] via a redox cycle where the active site metal is deoxidised by one $O_2^{\bullet -}$ radical and re-oxidised by another [150]. The three (3) forms of identified SOD, defined by the active site metals, are iron-SOD, copper and zinc-SOD, and manganese-SOD [151]. SOD catalyses the dismutation of $O_2^{\bullet -}$ to O_2 and H_2O_2 [147], which can then be broken down by other essential enzymes—the catalases.

(2) Catalases (CATs): These are peroxisome-localised, heme-group-containing enzymes [152], though their presence has also been reported in mitochondria [153]. They are involved in the breakdown of H_2O_2 to H_2O and O_2 [147]. They are recognised as essential defence enzymes against ROS-induced oxidative stress [154,155]. In addition, they are involved in plant defence and metabolism as well as the perception of cellular signals [156].

(3) Glutathione reductases (GRs): These flavoproteins occur mostly in the chloroplasts but have also been reported in the cytosol, mitochondria, and peroxisomes [157]. They are extremely specific and are involved in the reduction of oxidised glutathione (GSSG) back to the reduced form (GSH) using NADPH as the reductant [147], thereby sustaining a high GSH to GSSG ratio [158]. They sustain the reduced state of GSH through the ascorbate–glutathione cycle and are involved in maintaining the –SH group and act as a substrate for glutathione-S-transferases. In conjunction with superoxide dismutase and ascorbate–glutathione pathway enzymes, GRs constitute an important ROS scavenger [158]. They have been demonstrated to enhance oxidative stress tolerance in transgenic *Nicotiana tabacum* [157].

(4) Ascorbate peroxidases (APXs): These heme-containing enzymes are also involved in the decomposition of H_2O_2 using ascorbate as a reductant [159]. Different isoforms have been reported in the cytosol, chloroplast, mitochondria, thylakoid, stroma, and peroxisome [152,159,160]. Increased APX activity has been reported under abiotic stress such as light [161], drought and heat [162], and heavy metal contamination [163].

(5) Glutathione peroxidases (GPXs): These are non-heme-containing antioxidant enzymes [159] using glutathione as a reductant [164]. They are ubiquitous and predicted to be localised in cytosol, chloroplast, endoplasmic reticulum, mitochondria and plastids [165]. They have been demonstrated to play a role in lipid hydroperoxide detoxification, plant defence, and response to biotic [166] and abiotic stresses [164].

A balance between the activities of antioxidant enzymes such as APX, CAT, and SOD is necessary to determine the steady-state ROS (e.g., $O_2^{\bullet -}$ and H_2O_2) level [46]. In addition to metal ion sequestration, this balanced activity is considered crucial to forestalling the production of the extremely toxic $HO^{\bullet}$ through the metal-dependent Fenton or Haber–Weiss reactions [46]. APX and CAT are thought to be of different groups of H_2O_2 scavengers due to their different affinity for H_2O_2 (μM and mM range, respectively). Ascorbate peroxidase can reduce H_2O_2 to very low concentrations and is conceivably involved in ROS modulation for signalling, while the main role of CAT is to scavenge excess ROS under stress [46]. Since CAT is not reductant dependent to play its role, it might not be sensitive to cell redox status, contrary to the other systems [46]. Interestingly, some intricate interactions between the mechanisms generating ROS and those scavenging ROS have been reported in transgenics having repressed ROS-quenching systems. Plants having repressed APX formation have their CAT, GR, and SOD induced to compensate for the absence of APX, while plants having inhibited CAT compensate for it by inducing other antioxidant enzymes, such as GPX and APX, suggesting some level of redundancy [167].

The non-enzymic antioxidants also play vital roles in the antioxidant defence system, which forms a strong basis for their use as indicators of stress [148]. Major non-enzymic antioxidants include:

(1) Ascorbic acid (AA): AA is known to be abundant and one of the most potent antioxidants involved in ROS (e.g., $O_2^{\bullet -}$ [168]) detoxification and prevention [148,149]. This water-soluble antioxidant is found in all cellular compartments and at higher concentrations in photosynthetic cells [46]. AA is mostly present in its reduced form [169]. It is crucial for the maintenance of membrane structure and capable of completely preventing lipid peroxidation initiation, scavenging ROS, such as singlet oxygen, hydroperoxyl radicals, superoxide, and peroxynitrite, and protects other substrates from oxidative impairment [148,149]. In addition, AA has been documented to be involved in ROS scavenging by controlling redox balance in cells [170]. AA has been reported to enhance abiotic stress tolerance [169,171]. AA is involved in the modulation of the synthesis of tocopherol [172] and the regulation of plant defence responses over and above developmental processes [173].

(2) Glutathione (GSH): In addition to AA, GSH is another non-enzymic antioxidant involved in the detoxification of ROS [168]. Both GSH and AA are involved in the ascorbate–glutathione cycle, where ascorbate peroxidase plays a role in the direct removal of H_2O_2 [174], singlet oxygen [148] and hydroxyl radical [175]. AA is most

abundant in its reduced and active form and found in various cellular compartments, including the cytosol, mitochondria, endoplasmic reticulum, vacuole, peroxisomes, apoplast, and chloroplasts [176]. GSH provides a substrate for several reactions forming oxidised glutathione (GSSG). Balanced GSH to GSSG levels are key to maintaining a redox state in cells [177]. A decline in GSH levels during stress often leads to an imbalanced redox state, thereby causing system deterioration [178]. Heightened biosynthesis of GSH in chloroplasts, instead of protecting cells, may cause oxidative impairment, perhaps by adjusting the general redox state of chloroplasts [179]. It has been reported that the ratio of reduced to oxidised antioxidants can signal the modulation of ROS-scavenging mechanisms [46,51]. GSH plays a major role in protection from oxidative attack on biological membranes [149] and participates in various physiological events, including sulphate transport regulation, xenobiotics detoxification, and signal transduction [148]. Heightened GSH level has been linked with plants' ability to withstand oxidative stress [180].

(3) Tocopherol (vitamin E): This lipophilic phenolic compound exists in eight similarly potent forms as alpha(α)-, beta(β)-, gamma(γ)- and delta(δ)- tocotrienols and tocopherols [181]. It forms part of the biological membrane, playing both non-antioxidant and radical-chain-breaker functions [181]. It is regarded as a potential ROS and lipid radical scavenger [182]. Reduction in tocopherol levels following seed ageing suggests that it is involved in protection against oxidative stress-induced impairments [183], thus making it a useful indicator of seed deterioration [149]. Its synthetic analogue, trolox, has also been reported to be similarly capable of preventing oxidative impairment [184]. Trolox has some advantages in being moderately soluble in water [185]. Unlike α-tocopherol, trolox may be integrated directly into both lipid and water parts of cells [184], thus making it suitable for conducting studies involving both living systems and model systems [185]. The antioxidant power of trolox has been reported to be much more than that of α-tocopherol [184]. Other synthesised analogues include Vitamin E acetate, α-tocopherylphosphate, and α-tocopherylsuccinate [181].

(4) β-carotene: Besides tocopherols, carotenoids play an important role in the photoprotection of phototrophs by eliminating surplus energy as heat, directly scavenging reactive oxidants [148], including $^1O_2$ free radicals, and protecting cells from oxidative impairment by suppressing lipid peroxidation [149]. Their antioxidant property is attributed to their extended conjugated double bond system [186]. Low β-carotene levels have been shown to protect membrane lipids from peroxidative reactions [149].

(5) Gallic acid (GA): In plants, GA is a relatively ubiquitous [187] endogenous polyphenolic compound with several biological activities, including reacting with active oxidants, preventing their formation and accumulation [188]. GA occurs in the free or conjugate (as esterified hydrolysable tannins [56]) form in several plants [189]. Though polyphenols, such as quercetin [190], as well as GA [191], may act as prooxidants depending on concentration and condition [192], GA is primarily used as an antioxidant [193] due to its capacity to scavenge ROS, such as H_2O_2 [194].

5. Seed Invigoration Treatments

5.1. A Brief History of Seed Pre-Hydration Treatment

Seeds are continually faced with multiple challenges relating to production, post-harvest storage, and subsequent quality. Moreover, in view of the effects of global warming as a symptom of climate change, different stress factors may cause poor seed performance in terms of reduced germination, uneven seedling emergence, poor seedling establishment, or destructive alteration of root cell architecture, leading to a substantial yield loss [195]. Hence, dating back to the ancient Greeks, concerted efforts towards the improvement of seed performance have led to the development of different pre-sowing treatment techniques that can augment germination and synchronise seedling emergence under different suboptimal growth conditions [196]. Seed pre-hydration was discovered by Theophrastus, Democritus (5th century B.C.), and Mago (4th-3rd century B.C) [197]. It was suggested that

seed pre-hydration treatments in water or milk enhanced the germination of cucumber seeds (Theophrastus, D.H.P. Book VII, 1: 6). Democritus suggested steeping all seeds in some "roof tiles" plant extract before sowing (Plinius, N.H. Book XVIII, XLV: 159). Some other mentions include pre-hydrating almond seeds in a solution of honey or manure according to Carthaginian Mago (N.H. Book XVII, XI: 63), pre-hydrating pulses in "nitre" (Theophrastus, D.H.P. Book II, IV: 2), seed ripening of mistletoe in bird droppings (Plinius, N.H. Book XVI, XCII: 247), and pre-soaking *B. oleracea* seeds in houseleek extract to provide resistance to various insects (Plinius, N.H. Book XIX, LVIII: 180). The need to dry seeds artificially "to make them fertile" was also mentioned by Plinius (Plinius, N.H. Book XIX, XXXVI: 120) [197].

In the 16th century, Olivier de Serres described the steeping of grains (*Hordeum*, *Secale* and *Triticum* spp.) in manure solution for 24 h followed by drying back as a pre-sowing technique for enhanced seedling performance [196,198]. In the 19th century (1855), Charles Darwin [199] experimented with a seawater pre-hydration treatment and reported enhanced germination in treated cress and *L. sativa* seeds. May et al. [200] demonstrated that drying seeds for some time after hydration bestowed beneficial effects leading to increased germination rates under normal and adverse conditions. In 1963, Ells James presented the modern seed priming concept, pointing out the vital parameters of seed pre-hydration treatment and reporting that an increased rate of seedling emergence was observed in tomato seed exposed to the nutrient solution [196,198]. Heydecker [201] recognised the term seed "priming" as used by Malnassy [202], describing it as a seed pre-sowing treatment that can improve performance under suboptimal conditions [203]. Furthermore, Heydecker [201] described seed priming as a pre-hydration treatment in an osmotic solution that permits imbibition in the first germination phase before conversion of nutrient and radical protrusion. Such seeds are sometimes dried back ('hardening' [204]) to their initial moisture level and sown or stored [205]. In addition, the use of specific terms, such as halopriming (imbibing in salt solutions), and osmotic priming (imbibing in other osmotic solutions), were proposed [201] to specify the priming agent. The technique thus far is recognized and widely used to improve seed performance in the field of agriculture [206]. During pre-hydration treatment, the absorption of water is controlled to allow for the activation of pre-germinative metabolism without permitting radicle emergence by limiting the seed moisture content [203,207]. The resultant seedlings assume a physiological (primed) state which enables faster growth and/or better activation of plant defence responses [66,208].

5.2. Seed Pre-Hydration and Pre-Germinative Metabolism

In the 'primed state', the hydration-induced specific metabolic changes are responsible for the ensuing beneficial effects of seed pre-hydration treatments [196]. Upon seed imbibition, major cell functions and processes are activated, such as de novo proteins and nucleic acid biosynthesis, ATP formation, phospholipid and sterol accumulation, DNA repair and antioxidant system activation—the 'pre-germinative metabolism' [196]. Severe oxidative impairment of biomolecules such as lipids, nucleic acids, and proteins may occur in the early germination stages, during maturation on the mother plant, as well as in post-harvest storage, and under various stress conditions [54,78]. For seed vigour to be preserved and germination to be successful, embryonic DNA repair mechanisms must be well preserved. A good repair of impaired DNA allows for the resumption of cell cycle progression and DNA replication, while a defective repair system causes oxidative cell death [54,209]. DNA impairments in seed embryo are repaired during early imbibition and are essential for performance in terms of germination and storability [210]. Thus, DNA repair is a vital part of 'pre-germinative metabolism' triggered during imbibition and accompanied by unrestrained ROS activities [196] capable of causing mutation in the meristematic tissues of the embryo [211]. All major DNA repair pathways, such as base- and nucleotide-excision repair, are triggered at the early imbibition phase for the maintenance of genome integrity [209]. Efficient ligase-dependent re-joining of strand breaks is

key to most DNA repairs, and DNA ligase VI, found only in plants, has been described as a major determining factor of seed quality and storability in *Arabidopsis thaliana* [210].

With regards to the regulatory roles of reactive oxidants in the germination of seeds, Møller et al. [100] suggested that comparatively long-lived oxidants, such as H_2O_2 take the signal to a distant target, whereas short-lived oxidants, such as $HO^{\bullet}$, likely act near their production site; the product of oxidation (acting as a secondary messenger) then takes the signal to the target transcription factors. Besides signalling mediated by ROS, severe lesions to biomolecules can result from ROS activities. Though DNA impairments can be 'addressed' by certain repair functions, RNA is extremely sensitive to ROS-induced oxidative impairment owing to the lack of a specified mechanism of repair [212], while protein damage can be reversible (as in the oxidation of cysteine and methionine) and/or irreversible (as in carbonylation) [75,100,111].

Nevertheless, the enhanced activity of antioxidant (defence) enzymes such as APX, CAT, SOD, and GR allows for the control of ROS levels during imbibition [213,214]. The ROS scavenging antioxidant potential of the seed is critical for the enhancement of germination and stress tolerance [78]. In addition, gene expression profiling encoding antioxidant enzymes is a useful index of seed antioxidant response during germination. These safeguarding functions are triggered during pre-hydration treatments, thereby allowing seeds to undergo major metabolic and physiological pre-germinative phase changes up to the first cellular division, leading to improved germination and increased seedling vigour upon sowing [196].

5.3. The Seed Priming Technology Overview

The priming concept usually refers to several approaches towards seed invigoration, all involving controlled hydration of seeds [215]. The seed priming technique is used to improve the overall post-harvest performance of seed [216,217], including longevity (storability) [218,219], and ability to withstand unfriendly environmental conditions [66,220]. Priming enhances seed germination in three phases (Figure 1) [40]: imbibition, germination, and growth [198]. During the first phase (imbibition), characterised by rapid water uptake owing to low seed water potential, respiratory activity and protein synthesis, through existing DNA and mRNA, are promoted. Phase II (germination) is a lag phase involving the initiation of various physiological functions relating to germination, including protein and mitochondria synthesis, degradation of stored food and reorganisation of cellular membranes, to support radicle protrusion and growth of the seedling, which commences in Phase III (growth phase) [198,221]. The key determinant of seed priming is the controlled uptake of water up to Phase II, prior to radicle emergence [198,221], which allows for vital physiological events, such as damaged DNA and mitochondria repair [40]. Priming duration can vary from less than 24 h [222] to days [223] or weeks [224], depending on cultivars, species, and seed lot [225]. Phase II is more sensitive to environmental factors than Phase III. Hence, primed seeds that have undergone Phase II may be able to germinate better than unprimed seeds under suboptimal conditions [198].

In many cases, primed seeds are dried back to a particular moisture level and stored [226] or sown by the conventional method [227,228]. Seed drying back is thought to confer a 'hardening' effect [229]. In the hardening technique, multiple (two to three soakings with drying back) cycles, are suggested to yield a better result, although one cycle is enough for most species [226,230,231]. Seed hardening induced by pre-sowing treatments is attributed to some cytoplasmic, physico-chemical changes, such as decreased lipophilic and increased hydrophilic colloids, greater protoplasmic elasticity and viscosity, increased hydration of colloids, increased bond water level, and increased protein coagulation temperature [231]. However, there have been reports of delayed germination and/or emergence in primed seeds that are dried back, relative to primed but not dried back seeds, owing to the extra time needed for rehydration, though other beneficial effects of priming are conserved [203,232]. Additionally, deterioration of seeds in storage has been reported when primed seeds were dried back in different species, such as *Lycopersicon esculentum* [233], *Cichorium endivia* [234],

and *L. sativa* [235]. Tarquis and Bradford [236] stated that though pre-hydration treatments caused an increased germination rate, drying back predisposed *L. sativa* seeds to loss of storability. This effect varies depending on initial seed quality [203]. Thus, it has been suggested that the storage of primed seeds cannot extend beyond a few weeks as mechanisms for repair of impaired DNA become reduced [237]. In a study on *Mimosa bimucronata*, Brancalion et al. [238] added that priming benefits were partly lost in dried back seeds, recording lower performance in terms of percentage germination, seedling vigour, uniformity and germination speed index, and higher electrical conductivity relative to primed but not dried back seeds.

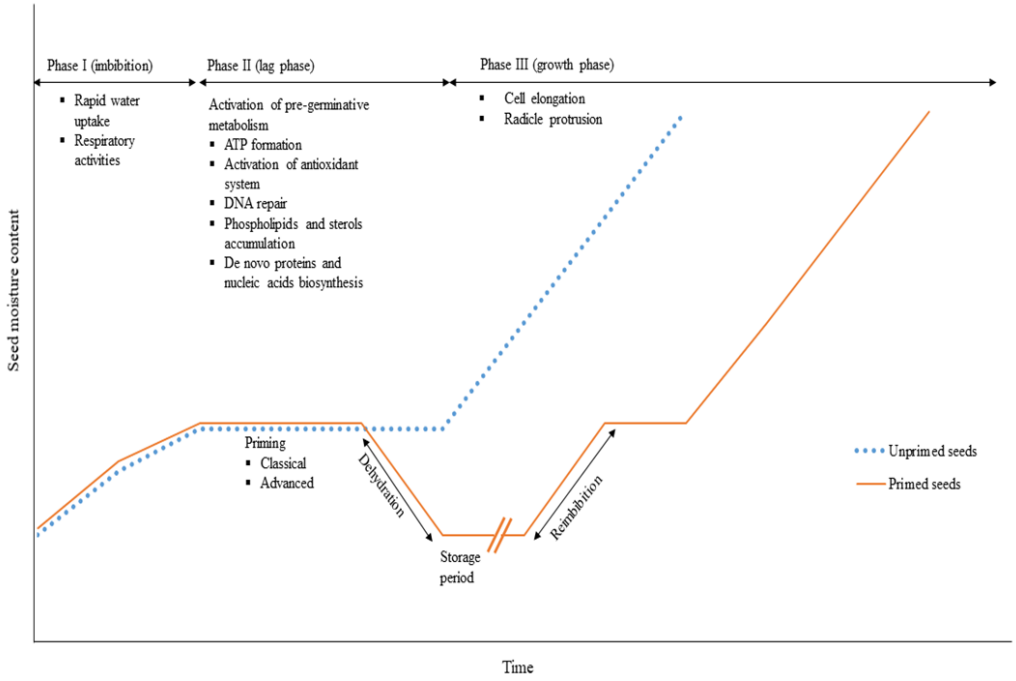

Figure 1. Imbibition curves showing the three phases of germination in unprimed and primed seeds.

5.4. Seed Priming Methods

Seed priming methods are generally divided into classical (hydropriming, osmopriming, redox priming, hormonal priming, cellular chemical priming, nutrient priming, and plant biostimulant biopriming [66,198,207]), and advanced (nanopriming [239], magnetopriming, irradiation with microwaves or ionising radiations and some other physical priming agents [240]) techniques, some of which are described below.

5.4.1. Classical Seed Priming Techniques

Hydropriming

Hydropriming is an age-old seed invigoration method popular with farmers as it is simple and economical. Hydropriming is of two types: drum-priming and on-farm priming [207]. Drum-priming involves seed hydration by water vapour generated from a gentle rotation of a drum at a particular temperature [204]. In on-farm priming, seeds are pre-soaked in water for a period before sowing [207,241]. The hydropriming technique is particularly useful under stressful conditions, such as high heat and salinity and water deficit stress, as seed hydration and water uptake efficiency in these conditions are enhanced [198]. However, maintaining optimum humidity and temperature is critical to preventing radicle protrusion, as hydropriming can allow for uncontrolled water uptake [242]. In contrast to unprimed (direct) sowing, the benefits of hydropriming have been demonstrated in several studies, including a 3–4 times increase in biomass allocation and seedling length of *Cicer arietinum* under drought stress conditions [243], rapid emergence and increased seedling vigour in rice seeds subjected to water-stress [227], and increased

germination of three-years-stored seeds of napa cabbage (*Brassica rapa*) which correlated with decreased electrical leakage, as well as enhanced antioxidant enzymes (superoxide dismutase and peroxidase) activities, and soluble sugar level [244].

Osmopriming

In this pre-sowing treatment method, seeds are subjected to controlled hydration in an osmotic solution of low water potential generated from the addition of osmotica such as polyethylene glycol, sorbitol, glycerol, and mannitol to priming water [66,207]. The low water potential of the osmotic solution is a crucial factor enabling seeds to be partially hydrated for pre-germinative metabolism but inhibited protrusion of the radicle [245]. In addition, the use of various salt solutions (halopriming) has been widely reported, and their beneficial effects elucidated [246]. For instance, Singh et al. [247] osmoprimed *Vigna unguiculata* seeds with KNO_3 solution and reported improved germination, plant height, and biomass accumulation compared with unprimed and hydroprimed seeds. Fatokun et al. [248] reported enhanced seedling emergence, photosynthetic and growth parameters of *Pisum sativum* and *Cucurbita pepo* seeds aged to 50% viability after priming with a mixture of $CaCl_2$ and $MgCl_2$ solutions relative to the unprimed seeds. Priming of *B. oleracea* seeds using varying levels (1%, 2% and 3%) of inorganic salts, such as KCl, KH_2PO_4, KNO_3, $MgCl_2$, $MgSO_4$, and NaCl, significantly increased germination, seedling vigour, biomass accumulation and reduced mean germination time [249]. Priming of artificially deteriorated *Brassica napus* seeds with $CaCl_2$ promoted seedling vigour [250]. Carrozzi et al. [251] reported that priming with $MgSO_4$ increased germination of *L. sativa* seeds stored for a year. Osmopriming is a low-cost priming option and allows for better water conservation [252].

Redox Priming

This seed invigoration method refers to priming with antioxidative compounds [66]. Plant cell redox state is key to the regulation of growth, development, and stress tolerance [253,254]. Plant redox status is disturbed in response to external stimuli, and the severity of disturbance is determined by the kind of stimulus, the amount, and the duration of, tissue exposure [66]. Maintaining an appropriate redox environment [255] is thought to help in minimising the severity of stress-induced damage [66]. During oxidative stress, antioxidants are well-known redox buffers capable of reacting with ROS and functioning as a metabolic interface that moderate the proper induction of acclimation responses or programmed cell death [256]. Among the compounds of major importance in the antioxidant pathway of plants, glutathione plays a significant role in the cellular redox signalling networks influencing growth, development, and defence [66,178]. Glutathione and tocopherol used as seed pre-hydration treatments resulted in increased seedling length in *Helianthus annuus* [257], gallic acid improved seedling vigour in *B. oleracea* [258], and trolox enhanced photosynthetic rate in *L. sativa* [259]. In addition, pre-hydration treatment of seeds with other antioxidant solutions has been reported to improve seed performance in several species. For instance, pre-hydration with ascorbic acid solution improved agronomic and biochemical vigour of *Pisum sativum* seeds [260] and improved germinability and tolerance to deterioration of *Elymus sibiricus* artificially aged for 48 h [261]. As mentioned by Afzal et al. [262], seed pre-hydration treatment with AA and tocopherol enhanced vigour and storability of *H. annuus* [263], maize, mustard [264] and *Oryza sativa* [265].

Plant Biostimulant Priming

Biostimulants are substances sourced from biological materials, e.g., microbial bioeffectors and extracts from plants and animals. They are diverse, ranging from single isolated compounds to complex matrices with various groups of biologically active constituents. The application of biostimulants to offset abiotic stress effects is well-established and represents one of the most promising techniques for attenuating stress impact in plants. It has attracted much interest both in research and commerce [266]. In addition to improving

plant tolerance against several abiotic stressors, this eco-friendly innovation enhances nutrient use efficiency, plant growth, and crop productivity [267]. The stimulatory effects of plant biostimulants, such as smoke-water on *Sceletium tortuosum* seeds [268], karrikinolide on *Lactuca sativa* [269] and *Aristolochia debilis* [270] seeds, commercial brown seaweed extract (Kelpak®) on *Abelmoschus esculentus* [271] and *Ceratotheca triloba* [272] seeds, and yeast extract on *Oryza sativa* seeds [273], have been reported.

5.4.2. Advanced Seed Priming Techniques

Nanopriming

The use of nanomaterials in agriculture is somewhat recent relative to their application in biomedical and industrial sectors [239], and it is considered a promising approach that can transform food production and agriculture [274,275] to meet the demand for food security in view of the envisaged rise in world population [276,277]. Nanotechnology employs not more than 100 nm size of biocompatible nanoparticles [198], often synthesised with plant extracts of desirable phytochemical properties as the nanopriming agents (phytosynthesised nanoparticles) [239]. For example, Mahakham et al. [239] primed *Oryza sativa* seeds stored for three years using phytosynthesised silver particles obtained from silver nitrate ($AgNO_3$) solution mixed with *Citrus hystrix* leaves extract (as reducing and stabilising agents). They reported enhanced performance in terms of germination and seedling vigour. Further, they proposed the mechanisms of action of nanopriming-induced invigoration of seed to include nanopore formation for the enhancement of water uptake, optimising ROS/antioxidant systems in seeds, production of $HO^{\bullet}$ for loosening of the cell wall and weakening of endosperm to enhance seed germination as well as nanocatalyst-enhanced hydrolysis of starch. In another study, Sundaria et al. [277] demonstrated increased germination and shoot length in IITR26 and WL711 wheat (*Triticum aestivum*) genotypes, respectively, using iron oxide synthesised nanoparticles as a priming agent. Further, they demonstrated and proposed nanopriming for wheat grain biofortification with iron, which is a potential strategy for overcoming iron deficiency in humans.

Seed Priming with Physical Agents

Thus far, many studies have shown that plant metabolic and developmental processes are sensitive to magnetic fields [278–280]. Magnetic fields are now being used for the invigoration of seeds and enhancement of agricultural productivity [207,281]. Several beneficial effects of magnetopriming (priming with the magnetic field) have been documented in various studies for different plant species. For instance, Baby et al. [282] reported improved germination, vigour, seedling biomass, the performance index of chlorophyll a fluorescence, and reduced level of $O_2^{\bullet -}$ in leaves of *Glycine max* seeds primed with a static magnetic field. Besides increased germination and germination speed, field emergence, vigour and seedling biomass, other beneficial effects, such as improved membrane integrity and reduced electrolyte leakage, were reported in *Helianthus annuus* seeds subjected to magnetopriming [283]. Further, they ascribed high germination rates and vigour to magnetopriming-induced rise in α-amylase, protease, and dehydrogenase activities.

Gamma radiation [240,284], UV radiation [285,286], X-rays [287,288], and microwaves [289,290] are some other commonly used physical priming agents [195,198].

6. Conclusions

There is little doubt that ageing-induced loss of crop seed vigour and viability is a serious threat to food security, particularly in countries where farmers are dependent on seed storage. Seed deterioration during long-term storage also poses a significant threat to germplasm conservation. Seed ageing, therefore, represents a challenge for the agri-food sector and seed industry, threatening the world's ability to meet global food demand. With increasing populations, especially in the developing nations, crop production losses owing to poor seed vigour have already resulted in market instability and enormous pressure on governments. This is expected to worsen when combined with the effects of climate

change and human mass migrations. As a result, the United Nations (UN), in the 2030 Agenda for Sustainable Development, placed great emphasis on food security, improved nutrition, and sustainable agriculture [291]. Many resources have also been invested in research on seed storage, priming, and invigoration, to increase crop production.

In this regard, slowing down the deterioration of seeds and enhancing seed viability and vigour have become crucial for seed preservation, given the inevitability of seed viability loss even under enhanced storage conditions in gene banks. Seed treatments before storage for enhancing ageing resistance are useful and urgently needed, especially where long-term storage facilities are not available, or seeds are stored using poor and/or ageing infrastructure. Focused research involving the use of state-of-the-art techniques on seed invigoration will be useful in elucidating the mechanisms of ageing-induced loss of seed vigour and promising invigorative methods.

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