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Review

Unraveling Molecular and Genetic Studies of Wheat (*Triticum aestivum* L.) Resistance against Factors Causing Pre-Harvest Sprouting

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Abstract: Pre-harvest sprouting (PHS) is one of the most important factors having adverse effects on yield and grain quality all over the world, particularly in wet harvest conditions. PHS is controlled by both genetic and environmental factors and the interaction of these factors. Breeding varieties with high PHS resistance have important implications for reducing yield loss and improving grain quality. The rapid advancements in the wheat genomic database along with transcriptomic and proteomic technologies have broadened our knowledge for understanding the regulatory mechanism of PHS resistance at transcriptomic and post-transcriptomic levels. In this review, we have described in detail the recent advancements on factors influencing PHS resistance, including grain color, seed dormancy, α-amylase activity, plant hormones (especially abscisic acid and gibberellin), and QTL/genes, which are useful for mining new PHS-resistant genes and developing new molecular markers for multi-gene pyramiding breeding of wheat PHS resistance, and understanding the complicated regulatory mechanism of PHS resistance.

Keywords: wheat; pre-harvest sprouting; seed dormancy; abscisic acid; gibberellin; QTL/genes

1. Introduction

Pre-harvest sprouting (PHS) refers to the germination of grains in mature cereal spikes before harvest under continuous wet weather conditions [1]. PHS has adverse impacts on wheat quality and yield [2,3] and reduces baking quality of dough by making it porous, sticky, and off-color. The price of sprouted grain is decreased by 20–50% and is unacceptable for human food if it contains more than 4% sprouted grains [4]. The decreased bread and noodle quality is due to increased activity of lipases, amylases, and proteases, enzymes which degrade lipids, starch, and proteins in sprouting grains [5,6]. Global yield and quality losses due to PHS have a financial impact estimated at $1 billion annually [7]. PHS occurred frequently in many major wheat producing areas of the world, including China, USA, Japan, Canada, Australia, and also in Europe [8]. In China, PHS is a major problem, especially in the northern spring wheat region, Yangtze River Valley, and northeastern spring wheat region which are characterized by heavy rainfall and high humidity before harvest [9]. In recent years, it has also become a serious problem in the Yellow and Huai Valleys' wheat region due to climate

changes. Therefore, improving PHS resistance is a major breeding objective to mitigate the risk of PHS and increase the production of high-quality wheat.

PHS resistance is associated with several developmental, physiological, and morphological features of the spike and seed, which includes seed coat (pericarp) color and permeability, seed dormancy, α-amylase activity, and levels of plant growth hormones (abscisic acid, gibberellin and auxin) [1,10–18]. Other factors, such as waxiness, hairiness, ear morphology, and germination-inhibitory compounds produced in bracts surrounding the grains have also been linked with PHS resistance [19,20]. Among them, seed dormancy is the major genetic factor controlling PHS resistance, therefore, much attention has been paid to understand the molecular mechanism of seed dormancy as a means to improve PHS resistance in wheat breeding programs.

PHS resistance is a typical quantitative trait controlled by numerous QTL/genes. Many quantitative trait loci (QTL) have been identified for PHS resistance in wheat [1,14,18,21–37]. Several candidate genes for PHS resistance have also identified, including *TaSdr*, *TaPHS1*, *TaMFT*, *TaVp-1*, *Tamyb10*, and *TaMKK3-A* [38–46]. These QTL/genes are valuable for gene pyramiding in breeding programs. However, the regulatory mechanisms of PHS remain unclear, which is why progress in improving wheat PHS resistance is limited.

To understand the regulatory mechanism of PHS resistance and provide valuable information for developing PHS resistant wheat varieties, this review summarizes recent advances of several major factors affecting PHS resistance, including grain color, seed dormancy, α-amylase activity, and plant growth hormones.

1.1. Grain Color

Grain color (GC) is an important genetic factor affecting the brightness of flour and is also associated with seed dormancy and PHS resistance. It is controlled by the *R-1* gene series distally located on long arms of chromosomes 3A, 3B, and 3D [47]. Dominant *R-1* alleles confer red grain color and are denoted by *R-A1b*, *R-B1b*, and *R-D1b* whereas the recessive alleles contribute white grain color and are named as *R-A1a*, *R-B1a*, and *R-D1a*, respectively. For dominant *R-A1b*, *R-B1b*, and *R-D1b* alleles, only one allele is enough for red color, while redness increases in a gene dosage-dependent manner [48]. The *R* genes act as transcriptional activators of flavonoid synthesis genes and are positioned in the same region as Myb-type transcription factor loci (*Tamyb10-A1*, *Tamyb10-B1*, and *Tamyb10-D1*) [49]. Himi et al. [40] confirmed the three *Tamyb10-1* genes on chromosomes 3AL, 3BL, and 3DL as candidate genes underlying the *R-1* loci for wheat grain color.

The red pigment in the testa of plant grains is composed of catechin, and proanthocyanidins (PA) that are produced in the flavonoid biosynthesis pathway and synthesized by different enzymes such as dihydroflavonol-4-reductase (DFR), chalcone flavanone isomerase (CHI), flavanone 3-hydroxylase (F3H), and chalcone synthase (CHS) [50–52] (Figure 1). These enzymes are expressed only in immature red grains and are almost completely repressed in the grains of white wheat [49]. The above Myb-type *Tamyb10-1* transcription factors control anthocyanin production and the red pigment of wheat grain by up-regulating the structural genes encoding DFR, CHI, F3H, and CHS in the flavonoid biosynthesis pathway.

In general, red-grained genotypes are more resistant to PHS compared to white-grained genotypes [53,54]. Himi et al. [53] observed the effect of *R* genes on grain dormancy by using near-isogenic red grained ANK lines and white grained mutant (EMS-AUS) lines and found that the level of dormancy conferred by *R* genes decreased rapidly in ANK lines during the after-ripening stage whereas reduction in the white grained mutant (EMS-AUS) line was not large indicating that *R* genes might play a minor role in seed dormancy. Groos et al. [1] detected four QTL for both PHS resistance and GC using a recombinant inbred line (RIL) population from a cross between Renan (red-grained) and Récital (white-grained). Three of these QTLs were close to *R* genes, and one was mapped on chromosome 5AS. Lin et al. [55] reported the genetic architecture of GC and PHS and genetic relationship of these two traits in a panel of 185 U.S. elite breeding lines and cultivars using

a genome-wide association study (GWAS). These results showed that GC genes (*Tamyb10-A1* and *Tamyb10-D1*) had a significant effect on PHS resistance, but *Tamyb10-B1* was significant only for GC and not for PHS resistance. In addition, a novel QTL for GC was also identified on chromosome 1B. Zhou et al. [37] identified three main QTLs for PHS resistance by GWAS, including a novel locus on chromosome 5D and two loci co-located with *Tamyb10-1* genes on chromosomes 3A and 3D. Furthermore, 32 GC-related QTLs (GCR-QTL) were also detected, and a strong correlation was observed between the number of GCR-QTL and seed germination rate. The above results imply that GC is significantly associated with PHS resistance, and might be controlled jointly by many QTLs in addition to he *Tamyb10-1* gene. Of these, some QTLs are for both GC and PHS resistance; others are for GC only and not for PHS resistance. Therefore, it should be possible to breed PHS-resistant white wheat by using the gene-editing technology known as CRISPR/Cas9 to alter the GC-related genes keeping in view the other dormancy-related QTLs besides those provided by the *R-1* genes of the red grained parent used for such editing.

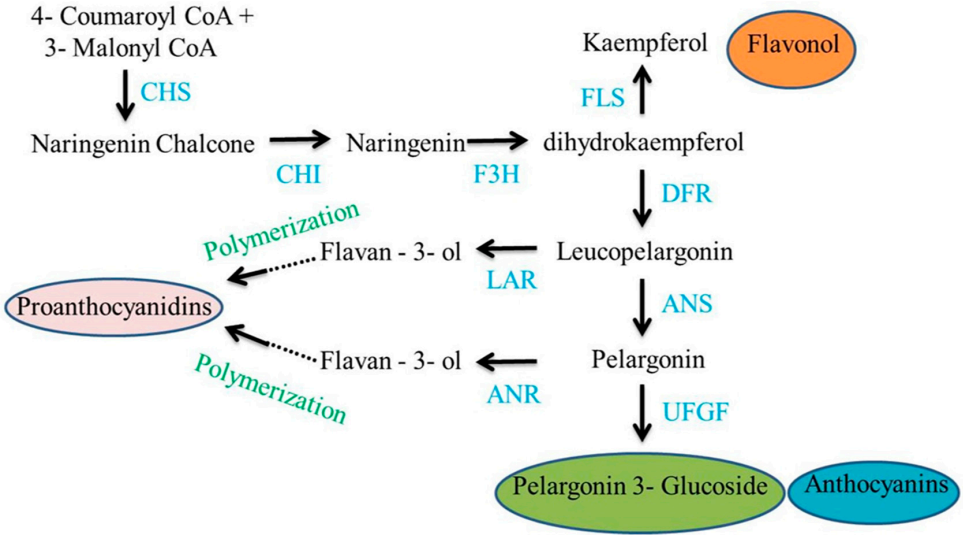

Figure 1. Schematic representation of flavonoid biosynthesis pathway in plants. Enzymes are shown in blue while intermediates are shown in black. End products are indicated in colored shapes. Dotted arrows represent multiple steps. CHS, chalcone synthase; CHI, chalcone isomerase; F3H, flavanone 3-hydroxylase; DFR, dihydroflavonol 4-reductase; ANS, anthocyanidin synthase; UFGT, UDP-glucose flavonoid 3-O glucosyltransferase; FLS, flavonol synthase; LAR, leucoanthocyanidin reductase; ANR, anthocyanidin reductase.

1.2. Seed Dormancy

Dormancy is the inhibition of germination of morphologically ripe and healthy seeds even under optimum conditions of light, moisture, and temperature [56,57]. Initiation and maintenance of dormancy is affected by both genetic and environmental factors [58]. Dormancy is regarded as a major genetic component of PHS resistance [59–61]. Seed dormancy in wheat is a complex phenomenon and can be divided into seed coat-imposed and embryo-imposed dormancy [62,63]. Seed coat inhibitory compounds are associated with seed coat-based dormancy [53], whereas crosstalk of phytohormones, such as abscisic acid (ABA), gibberellin (GA), and auxin, are involved in embryo-imposed dormancy [64,65]. Seed coat-imposed dormancy in particular is involved in the seed survival mechanism of several species [66]. The seed coat exerts its germination-restrictive action by its mechanical resistance to radicle protrusion or being impermeable to water and/or oxygen. These properties are positively correlated with seed coat color due to phenolic compounds in diverse species. In wheat, red-grained genotypes exhibit a wide range of seed dormancy and are more resistant to PHS because they contained dominant alleles in their trigenic series, whereas white-grained cultivars lack seed dormancy at maturity and are susceptible to PHS [63,67–69].

It is widely known that abscisic acid (ABA) is the major mediator for seed dormancy because it plays a significant role in inducing and maintaining dormancy during seed development as well as in imbibed seeds [70,71]. Many genes, like *TaPHS1* (a *TaMFT*-like gene), *TaCYP707A1*, and *TaDOG1*, have been identified for seed dormancy and are also involved in ABA synthesis and its signal transduction [41,43,72–74]. Until now, *TaPHS1/TaMFT*, *TaSdr*, *PM19-A1/A2*, and *TaMKK3-A* are the cloned genes involved in controlling seed dormancy and PHS resistance in wheat. *TaMFT* (*Mother of FT and TFL1*) is a homologue of the *Arabidopsis MFT* gene which controls seed dormancy and also regulates ABA and GA signal transduction. These studies indicated that wheat and *Arabidopsis* share the same regulatory mechanism of seed dormancy [41,43,72]. An SNP in the promoter region (at position –222) of *TaMFT* has identified which may increase *MFT* expression and likely contributes to increase seed dormancy [41]. Another gene *TaPHS1* (homolog of wheat *MFT* gene) involved in the regulation of seed dormancy and PHS resistance was identified on wheat chromosome 3A while the mutations at +646 and +666 positions of the coding region of *TaPHS1* gene resulted in PHS susceptibility [42,43]. It has been reported that *Sdr* gene plays an intermediate role in inhibiting germination and promoting dormancy in rice [75]. In wheat, three *TaSdr* genes *TaSdr-A1*, *TaSdr-B1*, and *TaSdr-D1* have been cloned and are involved in seed dormancy, among them; *TaSdr-B1* on chromosome 2B was observed to play a vital role in regulating seed dormancy [46,76]. Barrero et al. [44] identified two candidate genes *PM19-A1* and *PM19-A2* which positively regulate seed dormancy. They also demonstrated that *PM19-A1* highly expressed in dormant genotypes during grain maturation while *PM19-A2* showed sequence variations between non-dormant and dormant genotypes. In wheat, another gene *MKK3-A* (mitogen-activated protein kinase kinase 3), also called *TaMKK3-A*, has been identified on chromosome 4AL as a candidate gene of the *Phs-A1* locus which is associated with the length of seed dormancy [45]. Despite the multi-genic control of seed dormancy, a few major loci, including *Phs-A1* on chromosome 4AL and *TaPHS1/TaMFT* on chromosome 3A, have also shown to involve in sprouting resistance and account for a significant proportion of natural variations in diverse mapping populations [77]. Based on the strong effect, *Phs-A1* has been identified in at least 15 multi-parent and bi-parental mapping populations developed from diverse germplasm originated in the United Kingdom, Australia, China, Japan, Mexico, Europe, Canada, and Africa [44,78–81]. Shorinola et al. [77] studied the physiological evaluation of *Phs-A1* during seed after-ripening and observed that it delayed the rate of loss in dormancy when plants were grown across a wide temperature range of 13–22 °C. In another study, Shorinola et al. [82] conducted a haplotype analysis of the *Phs-A1* locus and found that *TaMKK3-A*, but not *PM19*, was the causal gene underlying variation in sprouting associated with *Phs-A1* in diverse Asian, North American, European, and Australian germplasm.

In addition to the PHS-resistant genes identified in wheat, maize could also act as a model to improve PHS resistance in wheat, e.g., the maize *viviparous-1* (*Vp1*) gene. McCarty et al. [83] reported that *Vp1* gene encodes a transcription factor that plays a significant role in the regulation of late embryogenesis in maize and late embryo development in bread wheat. *TaVp1* genes were extensively studied in wheat and were linked with seed dormancy and PHS resistance [38,39,61,84–89]. The *TaVp1* genes were mapped about 30 cM from *R* loci on homologous group 3Lchromosomes [86]. Six *TaVp-1B* alleles, *TaVp-1Ba*, *TaVp-1Bb*, *TaVp-1Bc*, *TaVp-1Bd*, *TaVp-1Be*, and *TaVp-1B* were identified in wheat [38,39,89,90]. Based on this allelic variation, the STS marker (*Vp1B3*) was developed for seed dormancy and it was observed that *TaVp-1Bb* and *TaVp-1Bc* alleles were linked with higher PHS tolerance [38]. Another STS marker (*Vp1A3*) was also developed for PHS tolerance by observing greater PHS resistance in various combinations of allelic variations, like *TaVp-1Agm/TaVp-1Ba*, *TaVp-1Agm/TaVp-1Bb*, *TaVp-1Aam/TaVp-1Bb*, and *TaVp-1Aim/TaVp-1Bb* [61]. Moreover, genetic analyses identified other genes such as *DOG1* involved in seed germination and dormancy [91–94]. The DELAY OF GERMINATION1 (*DOG1*) gene was first identified in *Arabidopsis* as a major QTL involved in increased seed dormancy [95]. The length of seed dormancy was estimated by the amount of expression of *DOG1* protein in freshly-harvested seeds, which indicated that the *DOG1* gene is a timer for the release from dormancy [96]. In a recent study, Nishimura et al. [97] demonstrated that the

DOG1 and *AHG1* genes interact with multiple environmental factors as well as the PYL/RCAR ABA receptor-mediated regulatory system to establish an important regulatory mechanism for control of seed dormancy and germination. Recent advances in genome sequencing and whole genome assembly of hexaploid wheat will trigger progress in identifying more seed dormancy and PHS resistance genes [98–102].

1.3. α-Amylase Activity

The α-amylase (amy) enzyme is involved in many plant physiological processes such as cold tolerance and germination rate and can hydrolyze α-1,4 -glycosidic bonds in saccharides [103,104]. The expression of amy enzyme is strictly controlled by the phytohormones ABA and gibberellin. ABA inhibits the amy expression during grain development. However, in genetic defect wheat, a high level of high pI amy genes could be expressed, resulting in poor grain quality during late grain development which is referred to as late maturity α-amylase (LMA) [105]. An elevated level of GA promotes amy expression during seed germination [106]. Alpha-amylase activity and PHS resistance are associated with each other possibly due to the fact that increased α-amylase activity upon water absorption promotes seed germination [107,108]. A remarkable difference was found in α-amylase activity between PHS-resistant and -susceptible cultivars in wheat [108]. Of three PHS traits, falling number (FN) [109] was found to indirectly measure the α-amylase enzyme activity that degrades starch in germinating seeds and is an important factor in quantifying PHS [110]. Breakdown of starch due to increased α-amylase activity results in a decreased FN value and is an indirect sign of low seed dormancy and low PHS resistance. Four isozymes of α-amylase affecting PHS have been identified in wheat, including malt-α-amylase (α-amylase-1) on homologous chromosomes 6, green-α-amylase (α-amylase-2) on homologous chromosomes 7, α-amylase-3 on homologous chromosomes 5, and α-amylase-4 has two members on homologous chromosomes 2 and 3 [111,112]. The wheat B genome contains genes for α-amylase-1 and α-amylase-2 among all the three genomes. Promoters of α-amylase-1 gene contains GA responsive complex that consists of a GA-responsive element (CAATAAA), pyrimidine box (CCTTTT), and TATCCAT/C box [112]. GA3 seemed to be involved in regulation of expression level of α-amylase-1 and α-amylase-2 [113]. The α-amylase-1 activity seemed to be significantly correlated with seed dormancy and contributed about 84% to seed germination [111].

In addition to α-amylase variation, α-amylase subtilisin inhibitors (ASI) were also identified in wheat, rice, rye, and barley by limiting α-amylase activity to restrain seed germination [114,115]. Moreover, ten ASI isomerides were identified through monoclonal antibody immune imprinting and isoelectric focusing electrophoresis techniques [116,117]. Yuan et al. [118] reported that PHS tolerance can be increased by reducing the α-amylase activity through combing α-amylase-1 and ASI complex. The α-amylase quantity and activity is very low in dormant seeds and increases after seed germination, therefore, it is necessary to identify the regulatory factors interacting with α-amylase, which can contribute to understand the complicated molecular mechanism of α-amylase regulating PHS tolerance.

1.4. Plant Growth Hormones

Previous studies have described the significance of plant hormones in metabolic and signaling aspects and their probable role in the maintenance and release of dormancy in seeds of cereal crops [43,119,120]. Among plant growth hormones, abscisic acid (ABA) and gibberellin (GA) play important roles in regulation of dormancy and germination, ABA induces dormancy and GA stimulates seed germination [121,122]. A change in balance between ABA and GA levels in seed constitutes a regulatory mechanism that results in maintenance or release of seed dormancy [120,123]. Several studies have reported the regulatory mechanisms of other hormones like ethylene, jasmonate, brassinosteroids, and auxin in controlling seed dormancy, germination and PHS resistance [43,121,124,125]. Environmental

factors, such as light and temperature, also affect the dormancy and germination by disturbing the balance between ABA and GA levels in cereal crops [126,127].

Numerous mutants have been developed to understand the regulatory role of plant growth hormones in seed germination and dormancy. Recent advances in the genomics of cereal crops have led to identify many genes involved in metabolic and signaling pathways of plant hormones for regulating seed germination and dormancy. The levels of plant growth hormones are noticeably different in PHS resistant and susceptible varieties; therefore, PHS resistance can be improved by identifying more genes involved in the expression and regulation of plant growth hormones.

1.4.1. Abscisic Acid

Abscisic acid (ABA) is an essential hormone that promotes seed dormancy, seed maturation and tolerance to desiccation [128]. Dormant wheat ABA levels increases by up to 2.5-fold during imbibition but remains unchanged in non-dormant grains [129]. ABA level in seeds/tissues is regulated by its synthesis and catabolism [130]. ABA biosynthesis is catalyzed by numerous enzymes like NCED (9-cis-epoxycarotenoid dioxygenase) that acts as a key regulator of ABA biosynthesis during seed maturation. During ABA biosynthesis, oxidative cleavage of violaxanthin and 9-cis-neoxanthin by NCED is regarded as rate-limiting step [131], whereas ABA catabolism is triggered by ABA 8′-hydroxylase enzyme (ABA8′OH) encoded by *CYP707A* genes that induce ABA hydroxylation at the 8′ position [132–134] (Figure 2). Therefore, the *NCED* and *CYP707A* genes play important roles in germination and dormancy by controlling the ABA level in seeds.

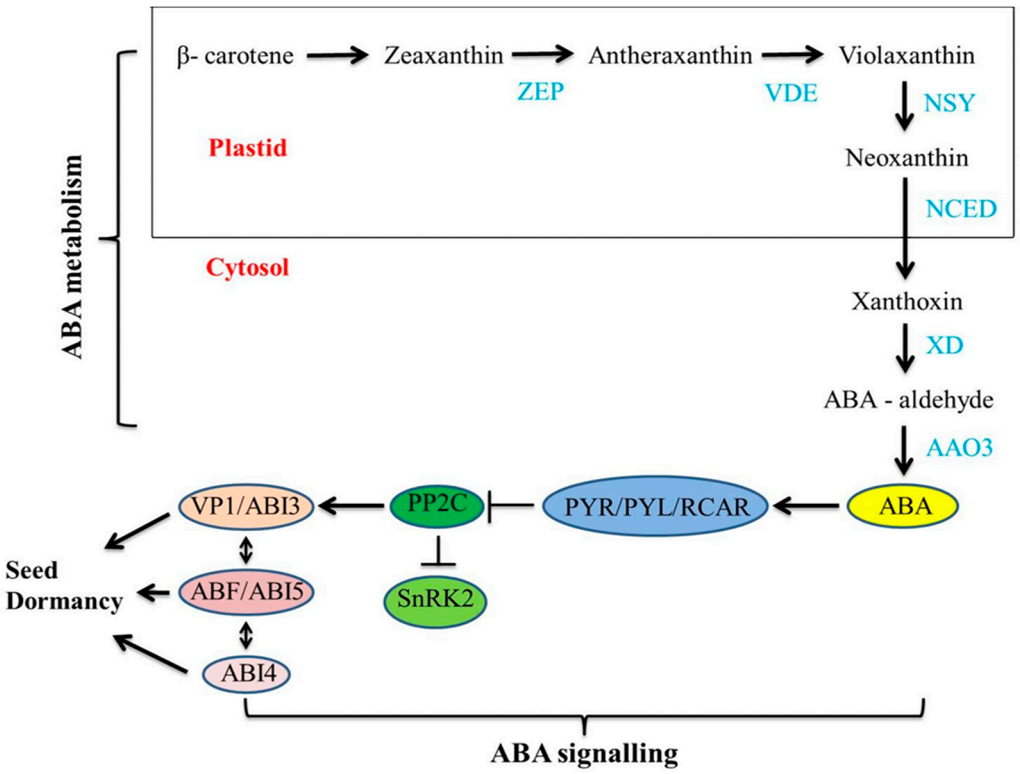

Figure 2. Schematic representation of ABA metabolism and signaling pathway in plants. Enzymes are shown in blue while intermediates are shown in black. The end product is indicated in the yellow circule; ZEP, zeaxanthin epoxidase; VDE, violaxanthin de-epoxidase; NSY, neoxanthin synthase; NCED, 9-cisepoxycarotenoid dioxygenase; XD, xanthoxin dehydrogenase; AAO3, abscisic aldehyde oxidase; PYR/PYL/RCAR, pyrabactin resistance/pyrabactin-like/regulatory components of ABA receptors; PP2C, protein phosphatase 2C; SnRK2, SNF1-related protein kinase2; ABI3, abscisic acid insensitive 3; ABI4, abscisic acid insensitive 4; ABI5, abscisic acid insensitive 5; VP1, viviparous 1; ABF, ABRE binding factor.

Seed development in wheat is characterized by two peaks of ABA accumulation that occur during the mid and late maturation phases. The first peak arises around 25 days after pollination (DAP) while the second peak arises around 35 DAP and extended up to 40 DAP in dormant wheat seeds [135,136], indicating the significance of ABA in inducing embryo dormancy [137]. Moreover, imbibed dormant wheat seeds have shown 3.8-fold higher expression of *TaNCED2* than non-dormant imbibed seed, while non-dormant seeds exhibit 2.5-fold higher expression of *TaABA8′OH1* (a wheat homolog of *CYP707A*) than dormant seeds in both imbibed and dry conditions [138]. Mutational analysis of the two homologs of *TaABA8′OH1* (*TaABA8′OH1A* and *TaABA8′OH1D*) showed an increase in embryonic ABA contents during mid and late stages (40–60 DAA) of seed development resulting in a higher level of seed dormancy [139] and highlighting the importance of higher embryonic ABA levels in inducing seed dormancy during the seed maturation phase in wheat.

ABA has been involved in the regulation of several seed developmental processes like deposition of storage reserves and primary dormancy induction that are evident from the observation of ABA mutants or deficient plants in maize and *Arabidopsis* [140]. A large number of mutants with reduced ability of synthesizing ABA have been developed in various crops, like the *aba1* mutant in *Nicotiana plumbaginifolia*, viviparous (*Vp*) mutants *Vp5*, *Vp7*, *Vp10/Vp13*, *Vp14*, and *Vp15* in maize; *aba1*, *aba2*, and *aba3* mutants in *Arabidopsis*, and *sit*, *flc*, and *not* mutants in tomato [91,131]. Several knock-out mutations are available for most wheat genes that provide an invaluable resource for characterizing the gene function. The resource of Targeting Induced Local Lesions In Genome (TILLING) mutants, like Kronos (tetraploid) and Cadenza (hexaploid), have been developed in wheat. The exome sequences of 1535 Kronos and 1200 Cadenza mutants have been resequenced using Illumina next-generation sequencing that can be used to screen for mutations in pre-harvest sprouting and dormancy related genes [141].

ABA biosynthetic mutants failed to induce seed dormancy and revealed a wilty vegetative phenotype, e.g., the *aba1* mutant in *Arabidopsis* and *aba2* mutant in tobacco were not able to produce zeaxanthin epoxidase (ZEP), the first identified ABA biosynthetic enzyme [142]. Another ABA-deficient mutant *aba4* was identified in *Arabidopsis* during a screening of paclobutrazol resistance germination and has known to be impaired in neoxanthin synthase (NSY) enzyme [143]. The *vp14* (*viviparous14*) mutant in maize and the notabilis mutant in tomato have shown impairment for NCED, which acts as a catalyst for oxidative cleavage of 9′-cis neoxanthin and/or xanthophylls, 9-cis-violaxanthin, and produces xanthoxin, as shown in Figure 2 [144,145]. The *vp10* and *vp15* mutants in maize, *sitiens* and *flacca* mutant in tomato and *aba2* and *aba3* mutants in *Arabidopsis* have also shown the impairment in later steps during ABA biosynthetic pathway in the cytosol [146,147].

The role of ABA in seed dormancy of wheat has already been described [16,43]. Nambara et al. [130] reported three core components of ABA signaling in seeds such as protein phosphatase 2Cs (PP2Cs), SNF1-related protein kinase2s (SnRK2s) and pyrabactin resistance/pyrabactin like/regulatory components of ABA receptors (PYR/PYL/RCAR), as shown in Figure 2. ABA forms a complex by binding with its receptor PYR/PYL/RCAR which then interacts with PP2Cs to inhibit its function. The PP2Cs negatively regulate ABA signaling by repressing the SnRK2s activity, which is a positive regulator of downstream targets. Inhibition of PP2Cs causes de-repression of SnRK2s, which in turn phosphorylates and activates down-stream transcriptional factors including ABI3 (B3 type protein), ABI4 (AP2 type transcription factor), ABI5 (abscisic acid insensitive 5), and ABFs (bZIP-type transcription factors). These transcriptional factors are important for the expressional regulation of ABA-responsive genes of seeds [130]. In the absence of ABA, PP2Cs becomes activated and, in turn, dephosphorylate and deactivate the SnRK2s. The molecular components involved in the ABA signaling pathway seem to be conserved in seeds of both monocot and dicot species [148].

The current understanding of signaling elements like ABA that control seed dormancy and germination mainly results from genetic analysis. In wheat, QTL and mutational analysis have revealed the importance of ABA sensitivity in regulating seed dormancy [149,150]. Dormant wheat seeds show more ABA sensitivity than non-dormant seeds [151,152]. *Vp1* was the first gene cloned

in maize against ABA response [83]. Expression of *Vp1* in wheat embryos was positively correlated with ABA sensitivity and degree of seed dormancy [87,153]. Splicing of the *Vp1* gene in wheat and rice counterpart resulted in susceptibility to PHS in both species [88,154]. *ABI3* is the ortholog of *Vp1* in *Arabidopsis* and the seeds containing *Vp1* or *ABI3* alleles exhibited similar phenotypes including ABA insensitivity, desiccation intolerance, and premature activation of the shoot apical meristem [155]. Mutational analysis of the *ABI4* and *ABI5* loci in *Arabidopsis* showed similar quantitative effects as *ABI3* on ABA sensitivity and seed development, but *ABI3* null mutations were more destructive than *ABI4* and *ABI5* [156]. These studies may help to explain the role of ABA in inducing wheat seed dormancy and to understand the molecular mechanisms underlying the regulation of ABA metabolism in inducing dormancy during seed maturation.

1.4.2. Gibberellin

Gibberellin (GA) is another major plant hormone that plays an important role in regulation of seed dormancy and germination [64]. GA breaks the seed dormancy and promotes germination by balancing the primitive endogenous inhibitors [157]. It also regulates the expression of α-amylase synthesis genes involved in seed germination and hydrolyzing the starch in the endosperm. In *Arabidopsis*, leafy cotyledon 2 (*lec2*) and fusca3 (*fus3*) could up-regulate GA activity resulting in germination of seeds before maturity [158,159].

The bioactive concentration of GA in plants is regulated by the balance between its synthesis and inactivation, that are mainly controlled by the genes *GA2ox* (encoding GA 2-oxidase), *GA3ox* (encoding GA 3-oxidase), and *GA20ox* (encoding GA 20-oxidase), respectively [160] (Figure 3). Many genes encoding these enzymes have been identified in a range of crop species including wheat, rice, and barley [160,161] and their expression plays significant roles in dormancy and germination by regulating the GA level in seeds.

Variations at the transcriptional level of these genes orthologs due to after-ripening and in non-dormant and dormant cereal crop seeds indicated the role of GA in regulating the seed germination and dormancy. For instance, dormancy loss in imbibed after-ripened barley and wheat seeds has shown to be linked with increased expression of the *TaGA3ox* and *TaGA20ox* genes and a higher level of bioactive GA_1 [43,126,162]. Moreover, transcriptional activation of *GA20ox* gene orthologs induced an increase in the level of GA_4 in non-dormant embryos of sorghum seeds, whereas up-regulation of *GA2ox* gene orthologs led to a decrease in the GA_4 level [163]. Mutational studies of these genes will provide further information regarding the molecular mechanisms of GA in regulating seed germination and dormancy. Genetic studies in rice have identified other candidate genes, such as *OsGA20ox2* and *OsGA2ox3*, responsible for regulating seed germination [164,165], while mutational analysis of *OsGA20ox2* showed greater dormancy due to reduction in the GA levels in seeds [164].

GA signals in plants are perceived by the soluble receptor protein gene *GID1* (Gibberellin insensitive dwarf 1), that was first mapped in rice. Mutational analysis of *GID1* in rice showed repression of α-amylase production and had no inhibitory effect on seed germination [166]. Orthologs of *GID1* protein have also been identified in wheat but further characterization of functions analysis of *GID1* orthologs in seed dormancy is required [167].

DELLA proteins in plants are another important element of GA signaling pathway, which function as a GA repressor and are broken down by ubiquitination induced by GA [168]. GA binds with GIDI and triggers the formation of the GA-GID1-DELLA complex which then interacts with F-box protein (the principal component of SCFSLY 1/GID2 E3 ubiquitin ligase) to degrade the DELLA protein through the ubiquitin-26S proteasome pathway [169–171] (Figure 3). In *Arabidopsis*, five DELLA proteins including RGL1 (RGA like1), RGL2 (RGA like2), RGL3 (RGA like3), GA1 (GA insensitive), and RGA (Repressor of GA1) were identified [172], among them RGL2 is known to be an important seed germination repressor [173]. The DELLA proteins in cereals such as RHT (reduced height) in wheat, SLN1 (slender1) in barley and SLR1 (slender rice1) in rice are transcribed by single

genes [174–176]. Chandler [177] observed that DELLA mutant seeds of barley were non-dormant and exhibited higher α-amylase activity in the aleurone layer.

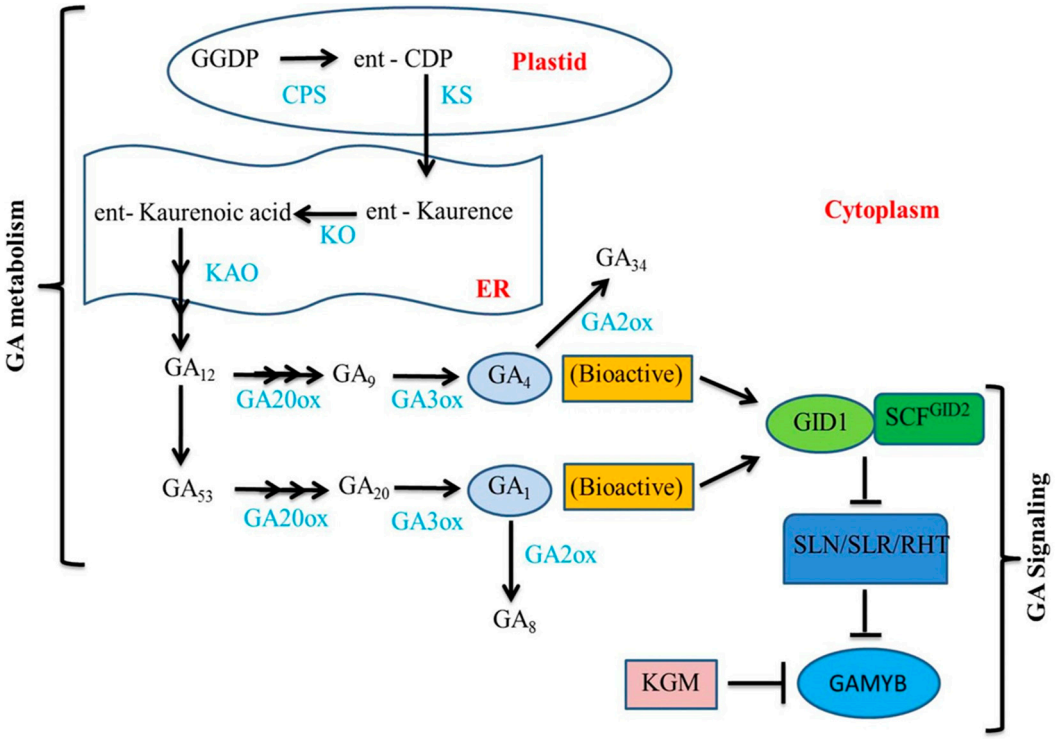

Figure 3. Schematic representation of GA metabolism and signaling pathway in plants. Enzymes are shown in blue while intermediates are shown in black. Multiple arrows represent multiple steps. GGDP, geranylgeraryl diphosphate; CPS, ent- ent- Copalyl diphosphate synthase; KS, ent-Kaurene synthase; KO, ent-Kaurene oxidase; KAO, ent- Kaurenoic acid oxidase; GA20ox,GA20 oxidase; GA3ox,GA3 oxidase; GA2ox,GA2 oxidase; GID1, gibberellin insensitive dwarf 1; GID2, gibberellin insensitive dwarf 2; SLN, slender1 in barley; SLR1, slender rice1; RHT, reduced height; GAMYB, GA regulated MYB transcriptional regulator; KGM, kinase associated with GAMYB.

In addition to DELLA proteins, other GA-regulated MYB transcriptional regulators (GAMYB) also play a significant role in the GA signaling pathway in aleurone cells of cereal crops [178]. In cereal aleurone, GAMYB triggers the transcriptional activation of GA and regulates hydrolytic enzymes especially α-amylase by directly binding to GA responsive elements in promoter regions [178]. The GAMYB function in cereal aleurone cells was repressed by another GA downstream signaling component named KGM1 (KINASE ASSOCIATED WITH GAMYB1) [179]. Mutational analysis of *GAMYB* orthologs in rice showed repression of α-amylase gene expression but had no effect on seed germination [180]. In wheat, whole seed transcriptional analysis showed no transcriptional differences in orthologs of *GID1*, *RHT*, *GAMYB*, and *KGM1* represented in the GeneChip Wheat Genome Array between after-ripened and dormant seeds. However, transcription of GA responsive genes encoding cell wall hydrolases and amalyses are induced in response to after-ripening [43]. These results might indicate that wheat seeds responsiveness to GA is controlled by post-transcriptional mechanisms or functions without these GA signaling elements. Although these studies demonstrate the role of GAMYB in germination and dormancy, but need further studies to identify and characterize more genes that interact with GAMYB and increase our understanding about the role of downstream GA signaling elements in controlling germination and seed dormancy.

1.4.3. Other Plant Hormones

Previous studies have described the importance of some other plant hormones, like ethylene, jasmonate (JA), auxin, and brassinosteroid (BR), in regulating seed dormancy and PHS resistance [121,124,125]. Transcriptomic analysis of dormant wheat seeds showed that imbibition triggered variations in expression level of several orthologous genes encoding key enzymes involved in ethylene, JA, auxin, and BR biosynthesis and their metabolic pathways due to after-ripening, indicating roles in regulating seed germination and dormancy [43,119]. Jacobsen et al. [181] reported that JA promotes dormancy release in dormant wheat seeds and perform antagonistically to ABA. Transcriptional activation of biosynthetic gene orthologs, such as *KAT3* (3-ketoacyl coenzyme a thiolase3), *LOX5* (lipoxygenase5), and *AOS* (allene oxide synthase) involved in JA biosynthesis were detected in imbibed after-ripened seeds. The production of a huge amount of jasmonate-isoleucine along with other orthologs of the biosynthetic pathway during after-ripened seed imbibition revealed the role of JA in controlling wheat seed dormancy. It has been reported that methyl jasmonate decreased the level of seed dormancy in wheat, but its role was regulated by variation in expression levels of the *ABA8´OH* and *NCED1* genes and ABA concentration [181]. Xu et al. [182] studied the role of JA and other hormones in the stratification of wheat dormant seeds and found that JA formation is necessary for seed germination induced by cold stratification. They also concluded that an increase in JA synthesis promoted a reduction in ABA concentration in cold-stratified grain embryos. However, the mechanism of JA in release of seed dormancy to allow germination is still not clear.

Transcriptomic analysis of after-ripened dormant wheat seeds revealed imbibition-mediated activation of BR biosynthetic ortholog genes such as *DET2* (De-etiolated 2) and *DWF4* (Dwarf 4) [119]. Transcriptional activation of these BR biosynthetic and signaling components in imbibed after-ripened wheat seeds have shown to be associated with transcriptional stimulation of BR responsive orthologs, such as *BBE* (BR enhanced expression) and *PRE* (paclobutrazol resistance), that control cell elongation, a process essential for seed germination [183,184]. These results indicated that BR plays a significant role in controlling seed dormancy and germination in wheat.

In *Arabidopsis*, previous studies have revealed that BR plays a significant role in the regulation of seed dormancy along with ABA and also increased the synthesis of ethylene, which has a regulatory role in seed dormancy of monocot species, such as wild oat [185–187]. During ethylene biosynthesis, BR mediates post-transcriptional activation of the ethylene biosynthetic enzymes such as ACC (aminocyclopropane-1- carboxylic acid) synthase (ACS) which acts as a catalyst during the first step [188]. In addition to ACS, ethylene biosynthesis is also mediated by another enzyme ACO (ACC oxidase). In wheat, ACO orthologs are involved in up-regulation in whole imbibed after-ripened seeds compared to dormant seeds [119]. Transcriptional activation of these enzymes and other ethylene receptor orthologs in wheat, such as ERS1 (ethylene response sensor 1), have revealed the role of ethylene in controlling seed germination and dormancy. In other cereal crops like rice and barley, ethylene promotes germination in non-dormant seed but is not involved in the loss of dormancy [189,190]. At present, the role of ethylene in regulating seed dormancy is not clear and needs further investigation of its regulatory role in seed dormancy in different crops.

Recent studies have revealed the role of auxin in maintaining seed dormancy. For instance, exogenous application of auxin increased seed dormancy in wheat [191,192] and *Arabidopsis* [193,194] through ABA activation. Liu et al. [43] studied the temporal expression patterns of metabolic and signaling genes of ABA, GA, IAA and jasmonate in both dormant and after-ripening dry and imbibed wheat seeds and observed that after-ripening mediated developmental switch from dormancy to germination seems to be linked with declines in seed sensitivity to ABA and IAA and repression of auxin signaling. Mutational analysis of wheat ERA8 (Enhance Response To ABA8) mutant showed an increase in dormancy due to increased level of embryonic jasmonate and aleurone IAA [195]. Metabolomic profiling of two water imbibed wheat cultivars Sukang (dormant) and Baegjoong (non-dormant) detected variable amounts of many auxin-related compounds in the 48h samples of Baejoong and found that indoleacetate abundance was not changed in the Sokang sample but

showed an abrupt reduction in Baegjoong at 48h water imbibition. Three catabolites of IAA including indole-3-carboxylate were also detected that showed similar of IAA at 48 h but with two other metabolites, such as indoleacetyl-aspartate and 2-oxindole-3-acetate, showed much higher levels at 48h in Baegjoong [196]. It also regulates several plant growths and developmental processes through the auxin signaling pathway mediated by aux/indole-3-acetic acid (IAA), transport inhibitor response1 (TIR1)/additional F box protein (AFB), and auxin response factors (ARFs) [197,198]. The ARFs are responsible for regulating the expression of a large number of auxin-responsive genes by binding with promoters of auxin response elements (AuxREs) [199,200]. Auxin inhibits seed germination and promotes dormancy through ABA-mediated response by regulating seed specific signaling components of ABA like *ABI3*, *ABI4*, and *ABI5*. Among them, *ABI3* is the only transcription factor involved in the regulation of seed dormancy [201–203]. Liu et al. [65] observed that auxin and ARF10/16 are involved in the regulation of *ABI3* expression which in turn inhibits seed germination and promotes seed dormancy in after-imbibed seeds. The function of ARF10 and ARF16 as positive regulators during the ABA signaling pathway contributes in developing a map of integrated hormone signaling during plant growth and development [204,205]. Auxin-induced seed dormancy seems to be an evolutionary mechanism that inhibits seed germination during unfavorable conditions and might be important for conservation of diversity and evolution in seed plant species [65]. Whether auxin is directly involved in seed dormancy is not clear, and its mechanism in controlling seed dormancy is also still unknown.

1.5. Environmental Factors Affecting PHS Resistance

Environmental factors such as rainfall, temperature and high relative humidity during the grain filling and maturation stages play an important role in the regulation of dormancy and sprouting in wheat. Temperature is one of the most important environmental factors for maintaining dormancy during seed development and for inducing dormancy during seed imbibition [206]. During seed development, low temperatures between 10 and 15 °C can induce high and prolonged dormancy while low temperature during germination breaks dormancy of freshly-harvested wheat seeds [207,208]. At low temperatures the MOTHER OF FT AND TFL1 (*MFT*) gene is involved in enhancing the dormancy during seed development in wheat [41]. It has also been reported that rainfall 10–20 days before harvest causes little or no sprouting but may influence the crop to be highly susceptible for sprouting at later rainfall [209]. Lunn et al. [210] studied the relationship between mean temperature and period of seed dormancy during the grain development and found that shorter dormancy periods occur after high mean temperatures.

Environmental factors such as temperature fluctuations, salinity and seed moisture content seem to promote ABA synthesis in plants with consequent effects on seed dormancy [211]. Footitt et al. [212] reported that the depth of seed dormancy and gene expression patterns were linked with seasonal variations in soil temperature. They also explained that ABA signaling was linked with deep dormancy during winter while its repression was linked with relief from dormancy during spring. ABA-signaling repression was concurrent with increased DELLA repression of germination. During winter, the expression of *NCED6* (ABA synthesis) and *GA2ox2* (GA catabolism) genes were found to be increased resulting in increased seed dormancy due to the decrease in soil temperature whereas, during spring, the endogenous ABA biosynthesis was found to be decreased while the expression of *CYP707A2* (ABA catabolism) and *GA3ox1* (GA synthesis) genes was increased resulting in declined seed dormancy in *Arabidopsis* [212]. In another study, Kashiwakura et al. [162] used two PHS-tolerant varieties, OS38 (highly dormant) and Gifu-komugi (Gifu, moderately dormant) to characterize the mechanisms of both dormancy maintenance and breakage at low temperatures. They observed that Gifu grains were germinated after imbibition at 15 °C whereas OS38 grains remained dormant. Imbibition of Gifu grains at low temperature caused a reduction in ABA levels in dormant embryos primarily due to the expression of *TaABA8′OH1* and *TaABA8′OH2* (ABA catabolism) and *TaGA3ox2* (GA synthesis) genes resulting in increased GA levels. On the other hand, imbibition of extremely dormant OS38 grains at a low temperature increased ABA levels by inducing the expression of *TaNCED* (ABA biosynthesis)

gene and suppressing *TaGA3ox2* and *TaABA8′OH2* genes. In a recent study, Izydorczyk et al. [127] observed a delay in germination of non-dormant imbibed wheat seeds under supra-optimal and suboptimal temperatures which was due to the expression of ABA signaling genes; ABI3, ABI5, PYL5, and SnRK2 in the embryo tissues resulted in enhanced ABA sensitivity. These studies explained the role of ABA and GA in dormancy and germination but needs further genetic studies to understand the physiological role of metabolic genes of ABA and GA in the regulation of seed dormancy in cereal crops.

1.6. QTL/genes Identified for PHS Resistance

The genetics of PHS resistance is controlled by both epistatic and additive effects which are affected by environmental conditions. The interaction between QTL epistasis (Q × Q) and the environment (Q × E, Q × Q × E) for PHS resistance has been studied to understand the complex genetic structure of QTL [213–215]. In wheat, PHS resistance is controlled jointly by multiple QTLs located an almost 21 chromosomes (1A, 1B, 2A, 2B, 2D, 3A, 3B, 3D, 4A, 4B, 5B, 5D, 6A, 6B, 6D, 7A, 7B and 7D) [1,14,18, 21–37,216–220] (Table 1).

In addition, several candidate genes for PHS resistance have also been identified based on comparative genomics or transcriptomic analysis, such as *TaSdr-A1* and *TaSdr-B1* on 2AS and 2BS [46,76], *TaPHS1* and *TaMFT* on 3AS [16,41,42], *TaVp-1* and *Tamyb10* on group 3 chromosomes [38–40,84,86], and *PM19-A1/A2* and *TaMKK3-A* on 4AL chromosome [44,45] (Table 2).

The interaction of genes with different PHS resistant QTL is different. QTL positioned on chromosome 4A may interact with *R* gene controlling red seed color to affect the PHS tolerance. Another QTL for PHS resistance was identified on wheat chromosome 5D independently of an *R* gene [29]. PHS is a typical quantitative trait controlled by multiple QTL and genes. A major QTL was mapped on the chromosome 4A by using various mapping populations which controlled about 40% of the phenotypic variation in PHS resistance in wheat [3,27,227]. These studies were conducted using SSR markers for the construction of genetic map and QTL mapping. Due to cost-effective and rapid innovations in sequencing technologies, thousands of molecular markers especially SNPs (such as wheat 820K, 660K, and 90K arrays) have been developed in wheat, which are useful for fine-mapping of QTLs and for cloning of candidate genes in the target regions. Moreover, recent advances in genome sequencing and whole genome assembly of hexaploid wheat will provide the bases for rapid identification of various PHS resistance genes [98–102].

Table 1. QTLs for PHS and related traits identified in wheat.

Trait	QTL	Chromosome	Nearest Marker	QTL name	Material	Reference
PHS and GC	5	3AL 3BL 3DL 5AS	Xffb293 Xgwm403, Xbcd131 Xgwm3 Xbcd1871	-	RILs	[1]
PHS and GC	3	1BS 4BL 7AS	Xpsp3000 Xpsp3030-Xpsp3078 Xpsp3050	-	RILs	[22]
PHS and SD	3	3A 3A 3A	Xpsr394-Xgwm5 Xcdo345 Xcdo345-Xbcd141	*taVp1* *QPhs.ocs-3A.1* *QPhs.ocs-3A.2*	RILs	[23]
PHS and SD	2	4AL 3AL	Xksuf8a-Xbcd402b Xpsr903b-XATPased	-	RILs	[217]
PHS	1	3AL	Xwmc153-Xgwm155	*QPhs.ccsu3A.1*	RILs	[24]
SD	1	4A	Xgwm397-Xgwm269-Xbarc170	-	DHLs	[3]
PHS and SD	1	3AL	Xbarc310-Xbcd907	*QPhs.ocs-3A.1*	RILs	[25]
PHS and SD	1	3AS 3AS	Xbarc310 Xbarc321	*QPhs-3AS* *QPhs.pseru-3AS*	RILs	[26]
PHS	3	2B 2B	Xdup398-Xbarc54 Xbarc105-Xbarc334	*QPhs.pseru-2B.1* *QPhs.pseru-2B.2*	RILs	[221]
PHS	1	2DS	Xgwm261-Xgwm484	*Qphs.sau-2D*	F_2 and F_6	[28]
PHS	4	2B 2D 3D 6D	Xbarc55-Xwmc474 Xwmc111-WxPt-999 7Xbarc1161 Xcfd37-Xbarc196	*QPhs.cnl-2B.1* *QPhs.cnl-2D.1* *QPhs.cnl-3D.1* *QPhs.cnl-6D.1*	DHLs	[14]
PHS	3	2AL 3AL 3BL	Xgwm1045-Xgwm296 Xgwm153-Xgwm155 Xgwm1005–Xgwm980	*QPhs.ccsu-2A.5* *QPhs.ccsu-3A.1* *QPhs.ccsu-3B.6*	RILs	[32]
PHS and GC	5	3B 3D 3A 5D 3D	Xbarc77–Xwmc30 7Xwmc552–Xwmc533 Xcfa2193–Xwmc594 Xgwm469–Xcfd10 Xwmc11–Xcfd223	*QGi.crc-3B* *QGi.crc-3D* *QSi.crc-3A* *QSi.crc-5D* *QCl.crc-3D*	DHLs	[29]

Table 1. *Cont.*

Trait	QTL	Chromosome	Nearest Marker	QTL name	Material	Reference
PHS and SD	1	3BL	Xwmc527-Xgwm77	-	DHLs	[31]
PHS	1	5D	XCFD40-XBARC1097	*qPhs5D.1*	DHLs	[33]
PHS and SD	5	2A 2B 3A 4A 7B	521-2A 521-2B 521-3A 521-4A 521-7B	-	Single chromosome substitution lines	[218]
PHS	3	1A 2A 7B	Xwmc611-Xwmc333 Xgwm515-Xgwm425 Xgwm297-Xwmc532	*QPhsd.spa.-1A.1* *QPhsd.spa.-2A.1* *QPhsd.spa.-7B.1*	RILs	[35]
PHS	4	3B 4A 7B 7D	19 SNPs flanking the QTL 12 SNPs flanking the QTL 10 SNPs flanking the QTL 04 SNPs flanking the QTL	*QSi.crc-3B* *QGi.crc-4A* *QSi.crc-7B* *QFn.crc-7D*	DHLs	[80]
PHS	5	1A 1B 5B 7A 7B	wPt-6274 Xwmc191 wPt-6910-wPt-7400 Xcfa2174 Xwmc606	*QPhs.spa-1A* *QPhs.spa-1B* *QPhs.spa-5B* *QPhs.spa-7A* *QPhs.spa-7B*	DHLs	[222]
PHS and SD	1	2B	Xwmc477-Xbarc55	*Sdr2B*	RILs	[76]
PHS and SD	1	4A	wsnp_Ex_c66324_64493429 - CD920298	*4A-1*	RILs	[44]
PHS and SD	4	4A 4B 5A 5B	GBS212432-GBS10994 7Xbarc20-Xwmc238 TTM_199619-TTM_1259 7Xbarc346-2-TTM_62137_50	*Qphs.pseru-4A.1* *Qphs.pseru-4B.1* *Qphs.pseru-5A.1* *Qphs.pseru-5B.1*	RILs	[223]
PHS and GC	6	3AL 3AL 3AL 3DL 3DL 1A/1D/3A/5B	Xwmc559-1 Tamyb10-A1-66 Tamyb10-A1-74 BS00067163_51 Tamyb10-D1-93 Xbarc148	-	RILs	[55]

Table 1. *Cont.*

Trait	QTL	Chromosome	Nearest Marker	QTL name	Material	Reference
PHS	6	3A 4A 1B 7B 4A 6B	TaMFT cfa2256 Xbarc181 UCW99 cfa2256 Xwmc397	*QDor-3A* *QDor-4A* *QDor-1B* *QHt-7B* *QAwn-4A* *QAwn-6B*	RILs	[224]
PHS and SD	1	2A	Xgwm95-Xgwm372	*Sdr2A*	RILs	[46]
PHS and SD PHS PHS	3 2 3	2D 3D 3D 1B 1B 3A 3D 5D	Xwmc503 Xcfd22 Vp1-4 tPt-7980 wPt-645 7AX-111578083 3 DArT-seq and 5 SNPs AX-109028892	*QDor-2D* *QDor-3D* *TaVp1* - *QTL1* *QTL2* *QTL3*	Back crosspopulation 86 Chinesegermplasm 717 Chinese wheat landraces	[225] [226] [37]
PHS	5	1A 4D 5A 5D 7B	wPt-6654-wPt-7030 wPt-0710-Rht-D1 gwm186-P7560-439 P7551-267-wmc574 P7455-236-P7553-711	-	RIL	[220]

Note: PHS-pre-harvest sprouting; DHLs-doubled haploid lines; RILs-recombinant inbred lines; GC-grain color; SD-seed dormancy.

Table 2. Genes for PHS and related traits identified in wheat.

Wheat Gene	Chromosomes	Gene Function	Homologs/Orthologs Gene	Experimental Methodology	References
TaSdr-A1	2AS	SD	Rice *OsSdr4* orthologs	Comparative genomics approach	[46]
TaSdr-B1	2BS	SD	Rice *OsSdr4* orthologs	homologous cloning approach	[76]
TaMFT	3AS	SD	Wheat *TaMFT* homolog	Transcriptomic approach	[41]
TaPHS1	3AS	SD	Wheat *MFT* homolog	comparative fine mapping and map-based cloning	[16,42]
TaVp-1	Group 3 Chromosomes	SD and PHS	Maize *Vp1* and rice *OsVp1* orthologs	Genomic southern analysis	[38,39,84,86]
Tamyb10	Group 3 Chromosomes	GC	*Arabidopsis TT2* and Rice *OsMYB3* orthologs	Cloning approach	[40]
PM19-A1/A2	4AL	SD	-	Transcriptomic approach	[44]
TaMKK3-A	4AL	SD	-	Map-based approach	[45]

Note: SD-seed dormancy; PHS-pre-harvest sprouting; GC-grain color.

PHS resistance is controlled by genotype, environment and their interaction [58]. The use of molecular markers for marker-assisted selection (MAS) could be helpful for direct identification of favorable or deleterious alleles in diverse groups of genotypes [228]. Iyer-Pascuzzi and McCouch [229] reported that MAS can be used for indirect selection of desired traits with considerable reduction in cost and time. Many gene-specific markers, such as SSRs (Xgwm15, Xgwm894, and Xgwm937), STMS markers (wmc104, Xwmc397, and Xwmc468), and STS markers (Vp1-B2 and Vp1B3), were developed for the *Vp1* gene and can be used for identification of PHS resistance in diverse genotypes [15,27,34,38,230–233]. Ogbonnaya et al. [27] found that Xgwm894 and Xgwm937 markers are significantly associated with PHS resistance and could be used for improving PHS resistance in wheat breeding programs. Liu et al. [16] developed an SNP marker named *TaPHS1-SNP1* that can be used as diagnostic marker for identifying the resistance allele of *TaPHS1* in breeding. Based on SNP flanking sequences on chromosomes 3B, 4A, 7B, and 7D, 18 KASP markers were developed that can be used for PHS resistance in future genetic studies and might also be useful for evaluating the PHS in breeding as well as germplasm materials [80]. In a study, Rasheed et al. [234] developed five KASP based assays of functional markers for four genes, including SDR_SNP for *TaSdr-B1*, TaMFT-1617R for *TaMFT-A1*, TaMFT-721J for *TaMFT-A1*, Vp1B1-83_IND for *TaVp-1B*, and Vp1B1-193_IND for *TaVp-1B*. These four genes may have different pathways to induce PHS tolerance; therefore, these KASP assays could provide an excellent opportunity to combine beneficial alleles for PHS tolerance in breeding programs. Wang et al. [235] developed STS (sequence tagged site) marker Tamyb10D for the *Tamyb10D1* gene and showed that it can be used as an efficient marker for evaluating the depth of seed dormancy in bread wheat genotypes. Moreover, the CAPS (cleaved amplified polymorphism sequence) marker Sdr2A has also been developed which is positioned on 2.9 cM intervals between Xgwm95 and Xgwm372 markers and can be used for identifying PHS resistant genotypes [46]. With the advancements in sequencing technologies, more than 124,000 gene loci have been annotated, [98,101], which provides a huge base for identifying more genes for PHS resistance and for the development of functional markers linked with PHS resistance, that can be used for developing PHS resistant varieties during wheat molecular breeding programs.

2. Conclusions and Future Prospects

PHS is a complex trait and determined by various endogenous and exogenous factors. Development of PHS resistant varieties is desirable in wheat growing areas especially having long wet weather conditions during harvest. Only a few PHS-resistant cultivars are commercially available in the field, and the grain quality of those cultivars needs to be improved. Therefore, selecting and inserting new resources could also be helpful in developing PHS resistant cultivars.

Understanding the genetics of various factors affecting PHS resistance is necessary for improving PHS resistance in wheat cultivars. The combination of both genetic and genomic technologies should be used to deeply study the temporal and spatial transcription of the genes involved directly or indirectly in controlling PHS resistance. Genomic and post-genomic data will broaden our knowledge about various factors affecting PHS resistance.

Construction of mutant libraries is important for future studies. Map-based cloning and mutant analysis of the genes underlying PHS resistance will provide new insights in improving PHS resistance of crop species. It is also necessary to use the available crop genome database that will trigger the progress in this field. Furthermore, rapid advancements in molecular technologies, like next-generation sequencing (NGS) technologies [236], and ongoing chromosomal-based and wheat whole genome sequencing projects (International Wheat Genome Sequencing Consortium, IWGSC) [98–102] will provide new opportunities for identification and functional analysis of the candidate genes controlling PHS resistance.

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