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FACTORS AFFECTING PRE-HARVEST SPROUTING RESISTANCE IN WHEAT (*TRITICUM AESTIVUM* L.): A REVIEW

X. Gao[1], C. H. Hu[1], H. Z. Li, Y. J. Yao, M. Meng, J. Dong, W. C. Zhao, Q. J. Chen and X. Y. Li

College of Agronomy, Northwest A&F University, Yangling, Shaanxi Province 712100, People's Republic of China
Corresponding author E-mail: xiaoyanli@nwsuaf.edu.cn

ABSTRACT

Pre-harvest sprouting (PHS) is one of the most important factors to affect the yield and quality of crops worldwide especially in wet harvest period. Breeding PHS-resistant cultivars has important implications to improve the wheat quality and production. The PHS is determined by environmental conditions, inner factors and interaction between these factors. We here reviewed recent advances influencing factors of PHS, including seed dormancy, seed coat permeability and color, á-amylase activities, endogenous hormones levels, genes and QTLs. The present article will provide basic materials for mining new genes and developing new molecular makers to improve the tolerance of PHS in wheat as well as increase wheat production.

Key words: wheat, pre-harvest sprouting (PHS), influencing factors, review, breeding.

INTRODUCTION

Continuous rains after seed maturity may induce the grain sprouting when it is still on the ear before harvest (Groos *et al*., 2002). The pre-harvest sprouting (PHS) has been recognized one of the main factors that decreases the yield and quality of crops worldwide especially in wet harvest period. The tolerance of PHS could be induced by environmental conditions, genotypes, quantitative trait loci (QTLs) and the interaction between these factors (Flintham, 2000; Mares *et al*., 2005). Wheat (*Triticum aestivum* L.), one of the world's largest food crops, is mainly planted in the north latitude 67 degrees to the south latitude 45 degrees. Generally, the seeds tend to be dormant in low temperature and long photoperiod, but sometimes the low temperature and high moisture would break dormancy and promote the seed sprouting (Argel *et al*., 1983; Ceccato *et al*., 2011). Temperature and moisture are the main environmental factors those affect PHS especially during the late maturity stage of wheat (Hilhorst, 1995; Yanagisawa *et al*., 2005; Gao *et al*., 2006). However, the influence of environmental conditions to PHS is very small usually less than 6% (Biddulph *et al*., 2008).

The major factors beside environment conditions affecting the tolerance to PHS are seed dormancy, seed coat permeability and color, α-amylase activities, endogenous hormones levels, genes and QTLs. Dormancy was regarded as the primary inner factor which leaded to the wheat resistance to PHS (Lan *et al*., 2005; Lin *et al*., 2008; Yang *et al*., 2011). The seed coat permeability is the first protecting wall which could increase the wheat PHS tolerance. The seed coat color also plays an important role in PHS. Generally, white wheat varieties have higher germination rates than the red ones (He *et al*., 2000). The α-amylase is also regarded as one of the factors that affect wheat germination rate, cold tolerance and production. Some other endogenous factors like gibberellic acid (GA), abscisic acid (ABA) and indole acetic acid (IAA) could also affect PHS through all kinds of ways.

PHS is a quantitative trait controlled by multiple genes. Viviparous-1(*Vp-1*) has been identified as the main gene that regulated seed germination and dormancy. Some other genes were also regarded to participate in embryos maturing, seed dormancy and germination through network regulation with *Vp-1* to control PHS. QTLs for dormancy and PHS were found in different materials through molecular markers. They were located on almost each chromosome (1A, 1B, 2A, 2B, 2D, 3A, 3B, 3D, 4A, 4B, 5B, 5D, 6A, 6B, 6D, 7A, 7B and 7D) in wheat (Flintham *et al*., 2000; Kato *et al*., 2001; Flintham *et al*., 2002; Groos *et al*., 2002; Osa *et al*., 2003; Kulwal *et al*., 2005; Lohwasser *et al*., 2005; Mori *et al*., 2005; Kottearachchi *et al*., 2006; Ogbonnaya *et al*., 2008; Ren *et al*., 2008; Kumar *et al*., 2009; Munkvold *et al*., 2009; Fofana *et al*., 2009; Mohan *et al*., 2009; Mares *et al*., 2009; Zhu *et al*., 2010; Zhang *et al*., 2011;Knox *et al*., 2012).

To avoid the grain sprouting on the ear and increase wheat production especially in wet harvest period, many studies were focused on the factors affecting PHS in the last decade. Here, we reviewed these new advances as influencing factors to wheat PHS, including seed dormancy, seed coat permeability and color, α-amylase activities, endogenous hormones levels, genes and QTLs.

Seed dormancy: Different wheat varieties have various period of dormancy. The varieties having shorter period of dormancy would easily germinate before harvest under

[1] These authors contributed equally to this work.

continuous raining conditions after seed maturation. For seed development, the dormancy capacity could be reduced with the mature of seed, but the ability to germinate could be formed at the fifth day after flowering and reach the peak after the seed physiological maturity (Gao *et al*., 2006). Different dormant levels could be determined by varied seed endogenous inhibitors, which usually affect seed dormancy by complicatedly interacting with each other (McCrate *et al*., 1982). The dormancy was also partially caused by high level of phenolic compounds in seeds through inhibiting the cell division. Most of the phenolic compounds existed as soluble ester, such as caffeic, p-coumaric, ferulic, and sinapic acids (Weidner *et al*., 1999). For seed development, the free phenolic compounds usually exist mainly at the early stage of seed development when the germination rate is low. At this point, the concentration of phenolic compounds significantly varied in different kinds of PHS resistant cultivars. The quantity of phenolic compounds was identified to present a negative relationship with the PHS tolerance (Weidner *et al*., 2002). Therefore, the relationship between seed dormancy and germination was revealed as varieties with stronger seed dormancy not prone to germinate before harvest and with stronger resistant to PHS.

Seed coat: Seed coat is the first protecting wall which could prevent water absorbed into seed to increase the PHS tolerance. The external water especially the rains once imbibed into the epidermic cells of seed, the α-amylase will be activated in the aleuronic layer and then the seeds will be germinated. Therefore, the seed coat especially the epidermic cells are very important for the tolerance to PHS. Once the epidermic cells of the seed coat arranged very loosely, the varieties will be very susceptible to PHS. On the contrary, the seeds of the PHS resistant varieties always have tightly arranged epidermal cells which could form a wall against absorbing water. (He *et al*., 2000; Cai and Chen, 2008; Wang *et al*., 2008). Beside the epidermal cells, an enzymatic oxidation of phenolic compounds was predicted to decrease the seed coat permeable against water and increase the PHS tolerance (Debeaujon *et al*., 2000).Generally, the PHS resistant varieties usually have voidless glumes and high level of lixivium of seed coat, which could prevent the embryos from germination in case of continuous raining after seed maturation before harvest.

The influences of seed color on dormancy and germination were indentified though creating recessive mutant Arabidopsis white seeds. The dormancy could be broken by cold treatment with most examined white mutants' seeds' dormancy reduced and germination easily (Debeaujon *et al*., 2000; Toradal and Amano, 2002). The color of seed coat has also been found as another influential factor to the tolerance of PHS in wheat. Generally, the white wheat varieties have a higher germination rate than the red ones (He *et al*., 2000).This may be caused by different anthocyanin amounts of seed color between two wheat varieties. The deficiency of oligo proanthocyanidin in seed coat especially of the white wheat was found to absorb water fast and caused high germination rate of seeds (Wu *et al*., 1996). The red wheat varieties were tolerance to PHS, but the white ones with higher quality were planted on larger areas than the red ones, which may be better valued and have great demand especially in Asia. Fortunately, the color of seed coat is determined by *R* gene and could transfer to the offspring. Therefore, the tolerance of PHS could be improved in white wheat through crossed with the red wheat varieties. Their offspring resistance to PHS of the self-crossed hybrids after 4-5 generations could be detected as strong as the red seed donor parent (DE PAUW and McCAIG, 1983; Groos *et al*., 2002). The white wheat varieties with high quality and production could be improved the PHS tolerance and planted in the wet harvest period.

α-Amylase activity: The α-amylase widely exists and participates in many physiology processes in plants, which could hydrolyze with α-1.4-glycosidic bond in the saccharides. The expression of α-amylase was involved in plant metabolism and could affect the germination rate, cold tolerance and production of seed (Khursheed and Rogers, 1988; Gubler and Jacobsen, 1992; Sogaard *et al*., 1993; Autio *et al*., 2001; Masojć and Milczarski, 2009). The relationship between α-amylase activity and PHS resistance was deemed to be very remarkable (Wu *et al*., 2002). This may be due to activity of α-amylase that would increase quickly once absorbed enough water and then promoted the seed sprouting (Wang *et al*., 2008). The activity of α-amylase was also found to have a significant difference between the resistant and sensitive varieties to PHS in wheat (Wang *et al*., 2008). Three isozymes of α-amylase in wheat have been identified affecting PHS, namely malt-α-amylase (*α-amylase*-1) located on homologous chromosomes 6, green-α-amylase (*α-amylase*-2) located on homologous chromosomes 7 and *α-amylase*-3 (Gale and Ainsworth, 1984). The expression level of *α-amylase*-1 and *α-amylase*-2 could be regulated by GA3 (Marchylo *et al*., 1983). The activity of *α-amylase*-1 was deemed to correlate with the degree of seed dormancy, which accounted for 84% of seed germination (Gale and Ainsworth, 1984). Besides the variation of α-amylase, the α-amylase/subtilisin inhibitors (ASI) in wheat, barley, rice and rye were indentified via restraining the activity of α-amylase to restrain the seeds germination (Mundy *et al*., 1984; Henry *et al*., 1992). Ten ASI isomerides were found through isoelectric focusing electrophoresis and monoclonal antibody immune imprinting (Macgregor *et al*., 1988; Masojć *et al*., 1993). The activity of α-amylase could be reduced by the combing complex of ASI and α-amylase-1 to increase the

variety's tolerance to PHS (Yuan *et al*., 2005). However, the mechanism of α-amylase regulating varieties tolerance to PHS still needs to be discussed, since the activity and quantity of α-amylase certainly increased after seed sprouting and very low in the dormant seed.

Growth hormones: The growth hormones, such as GA, ABA and IAA, affected wheat varieties tolerance to PHS through inducing or delaying seed dormancy and germination (Wickham *et al*., 1984; Weidner *et al*., 2002; Cai and Chen, 2008). Many studies have been focused to explain the relationship between them and indentified the genes involved them. The GA has been regarded to take part in promoting the seed sprouting. The ABA is one of the most important hormones regulating the development of plants and promoting the seed sprouting through promoting the ecclasis of separation layer and the maturation of embryo (Chen *et al*., 1999; Xia *et al*., 2000). It has been confirmed that there was a completely opposite relationship between the expression of GA and ABA.

Various mutants have been used to analyze the regulation process of the GA, ABA and IAA to seed dormancy and germination. The GA mutants failed to germinate and formed physically abnormal seed (Mitsunaga *et al*., 1994; Steber *et al*., 1998). The level of GA has been identified as a key determinant of seed germination, and could soften the tissue around the embryo to promote the embryo development by breaking the limitation of glume tenacity (McCrate *et al*., 1982). The GA could break seed dormancy by counterbalancing the primal endogenous inhibitors and promote seed germination (Wickham *et al*., 1984; Gashi *et al*., 2012). It also could induce the α-amylase hydrolyzing starch in endosperm and seed germination through regulating the expression of α-amylase synthetic related genes. The level of ABA could increase quickly to be 2.5-fold in dormant seeds but with no changes in the non-dormant ones (Ried and Walker-Simmons, 1990). The expression of ABA-response genes represented a long period delayed in the hydrated dormant seeds (Morris *et al*., 1991).

Lots of genes have been indentified in regulating the expression of GA, ABA and IAA, which have been found to participate in regulating seed dormancy and germination. The *RGL*2 and *ABI*5 could regulate the expression of ABA and GA (Piskurewicz *et al*., 2008). Some dwarf genes participated in regulating seed dormancy and germination in GA insensitive materials (Mitsunaga *et al*., 1994). One of the *Rht* alleles, named *Rht*3, was found to increase the varieties tolerance to PHS tolerance through reducing the amount of late maturity α-amylase (Flintham and Gale, 1982; Mrva and Mares, 1996; Wan *et al*., 2001). The activity of GA was also could be up-regulated by *fusca3* (*fus*3) and leafy cotyledon 2 (*lec*2) which led to germinate before maturity of Arabidopsis seed (Curaba *et al*., 2004; Lu *et al*., 2010). Transcription factors interacted with ABA mainly contained B3 domain or ring finger domain such as *AFL*, *VAL*, *DESPIERTO*, *ATHB*20 and ABA insensitive protein 2 (*AIP*2), which directly regulated seed dormancy. The sensitivity to ABA and the expression of *ABI*3 could be down-regulated by *DESPIERTO* (the mutant completely lost the ability of dormancy) and up-regulated by *ATHB*20 to promote seed dormancy of plant (Barrero *et al*., 2010). The development and maturation of seeds could be modulated by *ABI*3 through binding *AtGA3ox*2 promoter with the B1 and B3 domain of *ABI*3 (Mönke *et al*., 2004). The *FUS*3, *LEC*2, and *ABI*3 were also indentified to cooperate with each other and regulated the sensitivity to ABA during seed development (Nambara *et al*., 2000). The *FUS*3 and *LEC*2 could participate in regulating the abundance of ABA at the early stage of seed maturation (Nambara *et al*., 2000). The ABA was mainly regarded to regulate the germination pathways but not responsible for the loss of seed dormancy (Jacobsen *et al*., 2002). However, the level of ABA was significantly different in PHS resistant varieties compared with in PHS susceptible strains. Therefore, the tolerance to PHS would be improved in the future through spraying growth hormones with more and more genes identified to regulate the expression of growth hormones especially for the GA, ABA and IAA.

Controlled genes of PHS resistant: The dormancy of seeds and resistant to PHS were controlled by genotypes, environments and the interaction between these factors (Marzougui *et al*., 2012). During kernel development, the *Vp-1* gene expressed in cytoplasm after flowering regulated seed dormancy at the transcriptional level, promoted the seed maturation and repressed the expression of germination related genes (Hoecker *et al*., 1995; Paek *et al*., 1998; Wilkinson *et al*., 2002). There were multiple allelic variation of *Vp-1* gene in different cereal crops, but the predicted protein of *Vp-1* was conserved with four DNA binding regions A1, B1, B2, and B3 (Nakamura and Toyama, 2001). Three alleles *Vp-1A*, *Vp-1B*, *Vp-1D* of *Vp-1*, located on 3A, 3B and 3D homologous chromosomes in wheat, respectively, have been indentified (Yang *et al*., 2007; Chang *et al*., 2010).

Many studies also focused on the allele's variation of *Vp-1* to explain how *Vp-1* regulated the tolerance to PHS. Six alleles of *Vp-1A*, namely*Vp-1Aa*, *Vp-1Ab*, *Vp-1Ac*, *Vp-1Ad*, *Vp-1Ae* and *Vp-1Af*, were discovered in 81 wheat cultivars and advanced lines (Chang *et al*., 2010). Six alleles of *Vp-1B* termed *Vp-1Ba*, *Vp-1Bb*, *Vp-1Bc*, *Vp-1Bd*, *Vp-1Be* and *Vp-1Bf* were also found in wheat (Yang *et al*., 2007; Chang *et al*., 2010; Divashuk *et al*., 2012). However, no alleles of *Vp-1D* were found in wheat. The wheat variations with alleles of *Vp-1Ab* and *Vp-1Ad* were regarded to have low germination index (GI) and strong PHS tolerance (Chang

et al., 2010). However, the wheat variations with the allele *Vp-1Ba* have higher GI and more sensitive to PHS than the other five ones, which even positively affected on the reduction of germination rate (Yang *et al*., 2007; Chang *et al*., 2010; Divashuk *et al*., 2012).

Table 1. QTLs for dormancy and PHS indentified in wheat

Trait	Chromosome	Marker	Material	Reference
Dorf	4AL	Xcdo795 - Xpsr115	DHLs	Kato *et al*., 2001
	4BL	Xbcd1431.1/Xbcd1431.2		
	4DL	Xbcd1431.1/Xbcd1431.2		
PHS	3AL	Xfbb293	RILs	Groos *et al*., 2002
	3BL	Xgwm403, Xbcd131		
	3DL	Xgwm3		
	5AS	Xbcd1871		
PHS	1BS	Xpsp3000	RILs	Flintham *et al*., 2002
	4BL	Xpsp3030-Xpsp3078		
	7AS	Xpsp3050		
PHS and Dor	4AL	Xbarc 170-Xgwm 397	RILS	Cai and Chen, 2008
	2BS	barc328.217, stm773.183		
	2BL	wPt-666931		
	4AL	gwm610.162, gwm610.172		
	5B/7B	gwm397.191		
	2BL	wPt-3873		
	7BS	wPt-0697, wPt-666931	DHLs	
Dor	4A	Xgwm397- Xgwm269		Mares *et al*., 2005
Dor	3BL	wmc527-gwm77	DHLs	Mares *et al*., 2009
PHS and Dor	4AS	Xwmc48-Xgwm397	RILS	Ogbonnaya *et al*., 2008
	4AL	Xgwm0637-Xgwm937	RILs	Mohan *et al*., 2009
PHS	1AS	Xwmc24-Xbarc119		
	2AL	Xwmc170d-Xcfd168		
		Xgwm1045-Xgwm296		
	2B	XE36M605 – XE36M607		
	3AL	Xwmc153 – Xgwm155		
PHS and Dor	2A	521-2A	Single chromosome substitution lines	Chao *et al*., 2010
	2B	521-2B		
	3A	521-3A	RILs	
	4A	521-4A		
	7B	521-7B	RILs	
PHS	3AS	BARC310	RILs	Kottearachchi *et al*., 2006
	4AL	BARC170		Kulwal *et al*., 2005
PHS	3A	Xgwm155		Mori *et al*., 2005
PHS	3AS	Xbarc310- Xbcd907		
	4AL	Xcdo189-Xcdo795/Xcdo 808		
	4BL	Xgwm495 - Xgwm375	F_2 and F_6 populations	
PHS	2DS	Xgwm261-Xgwm484	RILs DHLs	Ren *et al*., 2008
Dor	3AS	Xgwm5-Xpsr394		Osa *et al*., 2003
PHS	3A	Xcfa2193-Xwmc594		Fofana *et al*., 2009
	3B	Xbarc77-Xwmc307		
	3D	Xwmc552-Xwmc533		
	5D	Xgwm469-Xcfd10		
PHS	2B	BARC55 - WMC474	DH	Munkvold *et al*., 2009
	2D	XWMC111-XwPt-9997		
	3D	XBARC1161-Xgpw4152		
	6D	XCFD37-XBARC196		

PHS	4AL	Xksuf8a-Xbcd402b	RILs	Lohwasser *et al*., 2005
Non-Dor	3AL	Xpsr903b-XATPased		
PHS	1B	XWMC766-XSWES158	DH	Zhu *et al*., 2010
	2B	XCWEM55-XBARC129.1		
	4A	XBARC373-XBARC1114 XWMC313-XWMC494		
	5D	XCFD40-XBARC1097		
PHS	1A	Xwmc611-Xwmc333	RILs	Knox *et al*., 2012
	2A	Xgwm515-Xgwm425		
	7B	Xgwm297-Xwmc532		

Note: PHS, pre-harvest sprouting DOR, dormancy
DHLs, doubled haploid lines RILs, recombinated inbred lines

And many researches also studied the expression level of *Vp-1*, which increased with the growth of caryopsis and reached highest at 40 days after flowering (McKibbin *et al*., 2002; Wilkinson *et al*., 2005; Yang *et al*., 2007). The allele of *Vp-1B* was more highly expressed than *Vp-1A* and *Vp-1D* during the seed development period in wheat (McKibbin *et al*., 2002; Wilkinson *et al*., 2005; Yang *et al*., 2007). The alternative splicing resulted in different tolerance and sensitivity to PHS, indentified at 35 days after flowering (McKibbin *et al*., 2002; Wilkinson *et al*., 2005; Yang *et al*., 2007).Only the correctly spliced *Vp-1* gene could encode the complete Vp-1 protein.

The *Vp-1*gene was also found to be regulated by some other genes participated in embryos maturing, seed dormancy and germination. They controlled PHS through network regulation. For example, the *LEC*1, *ABI*3 and *FUS*3 belonged to the members of B3 transcription factors family were involved into embryos maturing and seed germination (Meinke *et al*., 1994; Suzuki *et al*., 2007; Angeles-Núñez and Tiessen, 2011). The ABRE (ABA responding element), DREB (dehydration responsive element binding protein), MYB (v-myb avian myeloblastosis viral oncogene homolog) and GARE (GA responding element), four elements predicted in the *Vp-1B* promoter sequence, were involved in regulating seed dormancy and germination (Sun *et al*., 2011). The genetic mechanisms of the tolerance to PHS of wheat varieties would be revealed though more and more genes related to PHS indentified in further studies.

QTLS related to PHS resistant: The genetics of PHS resistant controlled by both additive and epistatic effects were easily affected by environment. However, the varieties resistant to PHS were regarded not significantly associated with environment by analyzing the interaction and contribution of main effect QTL and environmental effect QTL to PHS (Mohan *et al*., 2009). QTLs controlled the PHS were indentified to locate on almost each chromosome of wheat (1A, 1B, 2A, 2B, 2D, 3A, 3B, 3D, 4A, 4B, 5B, 5D, 6A, 6B, 6D, 7A, 7B and 7D) (Table 1) (Flintham *et al*., 2000; Kato *et al*., 2001; Flintham *et al*., 2002; Groos *et al*., 2002; Osa *et al*., 2003; Kulwal *et al*., 2005; Lohwasser *et al*., 2005; Mori *et al*., 2005; Kottearachchi *et al*., 2006; Ogbonnaya *et al*., 2008; Ren *et al*., 2008; Kumar *et al*., 2009; Munkvold *et al*., 2009; Fofana *et al*., 2009; Mohan *et al*., 2009; Mares *et al*., 2009; Chao *et al*., 2010; Zhu *et al*., 2010; Zhang *et al*., 2011; Knox *et al*., 2011). Some QTLs were associated with the activity of α-amylaselase in rye and barley (Masojc´ and Milczarski, 2009).

The explanation and interacting genes of different QTLs controlled the PHS resistant were different. QTLs located on chromosome 4A could interact with red seed (*R*) gene to affect the tolerance of PHS (Fofana *et al*., 2009). However, a QTL located on chromosome 5D independently from *R* gene has also been identified for the tolerance of PHS in wheat (Fofana *et al*., 2009). Although the PHS is quantitative trait controlled by multiple genes and QTLs, a major QTL controlled over 40% of phenotypic variation of PHS resistant in wheat has been indentified on chromosome 4A (Mares *et al*., 2005; Torada *et al*., 2005; Ogbonnaya *et al*., 2008). Different QTLs on the same chromosome were identified through multiple materials with molecular markers in wheat. Although several QTLs have already been identified, it will take a long time to apply them to molecular marker assisted selection (MAS) for wheat breeding.

Molecular makers for PHS resistant: Nowadays, MAS, the use of genetic markers, could facilitate the identification of favorable (or deleterious) alleles in a collection of diverse genotypes (Lazo *et al*., 2004). MAS could be used in the indirect selection of the objective trait, with labor-and time-saving (Anjali *et al*., 2007). The genetic markers were developed mainly on morphology, cytology, protein and DNA composition. Among them, the first three markers, mainly based on the consequence of gene expression, are the indirect reflection of gene. But DNA markers could directly reflect the information of genetic variation at molecular level. The *Vp-1* gene-specific STS marker of *Vp1B3* and *MST101*, SSR markers of *Vp1-b2*, *Xgwm937*, *Xgwm894* and *Xgwm15*, STMS markers of *Xwmc468*, *Xgwm397* and *wmc104*have been developed and used to identify PHS resistant in

different cultivars, and analyze the allelic variations of *Vp-1* (Yang *et al*., 2007; Yang *et al*., 2008; Ogbonnaya *et al*., 2008; Xia *et al*., 2009; Guo *et al*., 2009; Miao *et al*., 2011; Zhang *et al*., 2010; Zhao *et al*., 2010; Yang *et al*., 2011). The makers of *Xgwm937* and *Xgwm894* were indentified to be significantly associated with PHS resistance and could be utilized in wheat molecular breeding to improve the PHS resistance (Ogbonnaya *et al*., 2008). With more genes related to the tolerance of PHS identified, more and more markers especially the functional markers associated with PHS resistance would be developed and utilized in wheat molecular breeding to improve the varieties PHS resistance.

Prospect and conclusion: PHS-resistant cultivars are highly desirable in wheat growing areas where long periods of wet weather occur frequently during harvest. However, only a small number of PHS-resistant cultivars have been used in the field, and the grain quality of these cultivars remained to be improved. Studying the effect of factors to PHS is essential to improve the PHS-resistance in wheat. Besides, selecting and mining new materials also could help breeding PHS-resistant varieties. For example, RSP (*Triticum turgidum-Aegilops tauschii*) with extreme PHS resistance is the artificial synthetic hexaploid wheat crossed between *Aegilops tauschii Cosson* and tetraploid wheat (*T. turgidum* L.) (Lan *et al*., 2005). With the deepening research on PHS, varieties with PHS resistance, high quality and yield, and high good comprehensive traits will be bred and used in the future crop breeding program.

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