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QTL analysis of pre-harvest sprouting resistance in SHA3/CBRD x Naxos spring wheat population under different environmental conditions

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Abstract

Pre harvest sprouting (PHS) in wheat refers to an untimely germination of mature kernels within the spikes before harvest while still in the mother plant. This is the single most important factor for quality-degradation in wheat around the world as the potential for baking quality is often destroyed by PHS. The major reason for PHS in the field is the lack of adequate seed dormancy when seeds are exposed to wet weather conditions. Due to the significant role of the environment in the PHS damage, wheat breeders are focusing on genetic resistance to control this trait. PHS is a heritable trait, which is maintained by the pleiotropic effect of multiple genes. The main objectives of this study were to identify quantitative trait loci (QTL) for two PHS traits and to investigate the relationship between PHS resistance QTL and QTL for agronomic traits when grown in different environmental conditions. To identify QTL for PHS under different weather conditions, one recombinant inbred line (RIL) population of SHA3/CBRD x Naxos spring wheat was grown at Vollebekk, Norway in 2016 and Chengdu, China in 2016 and 2015. Germination index (GI) and falling number (FN) were used to assess the dormancy of wheat grains. The result showed a weak, but significant negative correlation between GI and FN (R^2 = 3.6%). The best performing lines had low GI and high FN. A large variation was also observed in PHS values between most of the RILs, when grown in the different environments, indicating that they were more sensitive to environmental effects than others, and that the variation in PHS was not consistent across seasons and locations. A total of seven putative QTL for PHS traits were identified on chromosome arms 1BL, 2AL, 3BL, 4AL, 4BL, 5AS and 7BL. The largest proportions of the explained phenotypic variation were recorded for the FN QTL located on 1BL and GI QTL located on 3BL. Both of them were stable, expressed in two seasons and locations. A QTL controlling GI, days to maturity and plant height was identified on the long arm of chromosome 4A, and the LOD curve indicated also some effect of this locus on FN. Although, both parents contributed to PHS resistance, the German spring wheat cultivar Naxos was shown to be a good source of FN QTL. Further investigations of these and other genomic regions in different mapping population might help to detect important QTL for PHS resistance in spring wheat.

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List of abbreviations

°C…….....	Degree centigrade
°N…….....	Degree north
°S……	Degree south
ABA……	Abscisic acid
ANOVA ….....	Analysis of variance
BLAST ……	Basic Local Alignment Search Tool
BLASTn ……..	Nucleotide BLAST
CAPs ……..	Cleaved amplified polymorphic sequence
Cd15 ……...	Chengdu 2015
Cd16 ……...	Chengdu 2016
CIMMYT ……	International maize and wheat improvement center
cm ……..	Centimeter
cM …….	Centimorgan
DArT ……..	Diversity arrays technology
DF ……..	Degrees of freedom
DH …….	Days to heading
DI ……...	Dormancy index
dl ……...	Deciliter
DM …….	Days to maturity
FN ……..	Falling number
GA3 ……	Gibberelic acid 3
GI …….....	Germination index
g …….....	Gram
h …….....	Hour(s)
ha …….	Hectare(s)
IM ……..	Interval mapping
Kg …….	Kilogram
Km^2 ……..	Square kilometer
l …….....	Liter
LOD ……...	Likelihood ratio
m …….....	Meter
Max ……	Maximum
MAS ……...	Marker aided selection
MI ……..	Mist irrigated
mg …….	Milligram
Min …….	Minimum
ml ……..	Milliliter
mm …….	Millimeter
MQM ……..	Multiple-QTL Mapping
mRNA ……	Messenger ribonucleic acid
$mScm^{-1}$ ……	Milli siemens per centimeter
NMBU ……	Norwegian University of Life Sciences

NMI …….....	Non-mist irrigated
NPK …….....	Nitrogen, phosphorus, potassium
P …….....	Probability
PH …….....	Plant height
PHS …….....	Pre-harvest sprouting
PP …….....	Precipitation
QTL …….....	Quantitative trait locus/loci
RH …….....	Relative humidity
RIL …….....	Recombinant inbreed line
SAS ……	Statistical analysis system
SD …….....	Seed dormnacy
SEM …….....	Scanning electron microscopy
SHA3/CBRD..	Shanghai3/Catbird
SI …….....	Sprouting index
SKP …….....	Senter for klimaregulert planteforskning
SNP …….....	Single nucleotide polymorphism
Sq …….....	Square
SSR …….....	Simple sequence repeat
SxN …….....	Shanghai3/Catbird x Naxos
T …….....	Temperature
t …….	Ton(s)
v …….....	Version
VL16 …….....	Vollebekk 2016
YR …….....	Yellow ripeness
α-amylase …….	Alpha amylase
% …….....	Percentage
% Expl. ……	Phenotypic variation explained by the QTL

Chapter 1

Literature review

1.1. Wheat: importance on food security

Wheat, *Triticum aestivum* L., is one of the most important crops on earth, which provides 20% of the food calories ("Wheat facts," 2017) for more than 7.6 billion people throughout the world ("Worldometers," 2018). It also provides 20% of the world's protein consumption by the world's poorest population ("Wheat facts," 2017). Wheat is grown from temperate, irrigated to dry and high-rain-fall areas, and from warm, humid to dry, cold environments, i.e., from the 67°N to 45°S latitude (Trethowan, Hodson, Braun, Pfeiffer, & van Ginkel, 2005), and covers 17% area in the world. Obviously, this wide adaptation has been possible as the wheat genome is comparatively more complex in nature, which provides great plasticity to the crop. Wheat flour is used as a key ingredient to make varieties of foods such as bread, noodles, and tortillas. Studies showed that whole wheat is associated with reduced risk of chronic diseases like diabetes and cancer, reduced risk of obesity, and better weight control ("Wheat facts," 2017). Due to its high nutritional value, good storing and transporting ability, wheat is considered as an important food crop. According to the International Maize and Wheat Improvement Center (*CIMMYT*, 2018) report, the global population will increase to more than 9 billion people by 2050, which indicates that the successes and failures of wheat farmers will continue to have a crucial impact on food security. To meet the global demands, greater wheat production can be achieved by expanding the wheat growing areas, and improving the yield per unit area sown. Moreover, reducing pre- and post-harvest losses will make more wheat available for consumption (Curtis, 2002).

Wheat is a C3 plant, which belongs to family Poaceae (Gramineae). Studies showed that common wheat originated approximately 8,000 years ago from the natural hybridization of three wild diploid grasses such as *Triticum urartu, Aegilops speltoides,* and *Aegilops tauschii* (Trethowan, Hodson, Braun, Pfeiffer, & van Ginkel, 2005). Genetically, wheat is an allohexaploid plant which genome is composed of 21 pairs of chromosomes originated from the A, B, and D genomes (Mangelsdorf, 1953). According to the "Sequencing the wheat genome" (2012-2017) project, the total wheat genome size is 16,700 Mb per chromosome and around 94,000 to 96,000 genes is included in its genome.

1.1.1. Kernel morphology and germination

Plant seed acts as both reproductive structures as well as a vital nutritional resource for human consumption worldwide. Botanically, the seed of wheat is known as a caryopsis. The size and shape of the kernels may vary according to wheat genotype and location in the spike (Delcour & Hoseney, 2010). Generally, the wheat seed is divided into three major parts. They are the seed coat, the endosperm and the embryo (germ) (figure 1.1).

The seed coat is the outer layer of the grain, which is divided into testa and nucellar layer. The seed coat is composed of dead cells and acts for protection of the embryo from outer environment (Bewley & Black, 1994; Delcour & Hoseney, 2010). Endosperm is divided into the aleurone layer and the starchy endosperm. The aleurone is the outer layer of the endosperm, which contains proteins and nutritional components (Evers & Millar, 2002). The starchy endosperm contains starch granules and storage proteins, and serves as fuel for the growth of embryo. During seed maturation, the cells of the starchy endosperm undergo desiccation and become dead in a mature dry grain. The cells of the aleurone layer are not prone to desiccation and are still alive in a mature dry grain (Rigor, 2008). The embryo is the most important part of the seed, which develops into seedling through germination. All of the major parts of kernel are enclosed with a fruit coat which is known as pericarp. The pericarp is divided into outer and inner pericarp. Outer pericarp contains hairs of brush, epidermis and hypodermis layer, and inner pericarp contains mesocarp and endocarp layer. The major function of pericarp is protecting seeds from the adverse effects of the environment. In total, the wheat grain consist of approximately 84% endosperm, 6.5 % aleurone, 2.5 % seed coat, 4.5 % pericarp and 2.5 % embryo (King, 1989; Delcour & Hoseney, 2010).

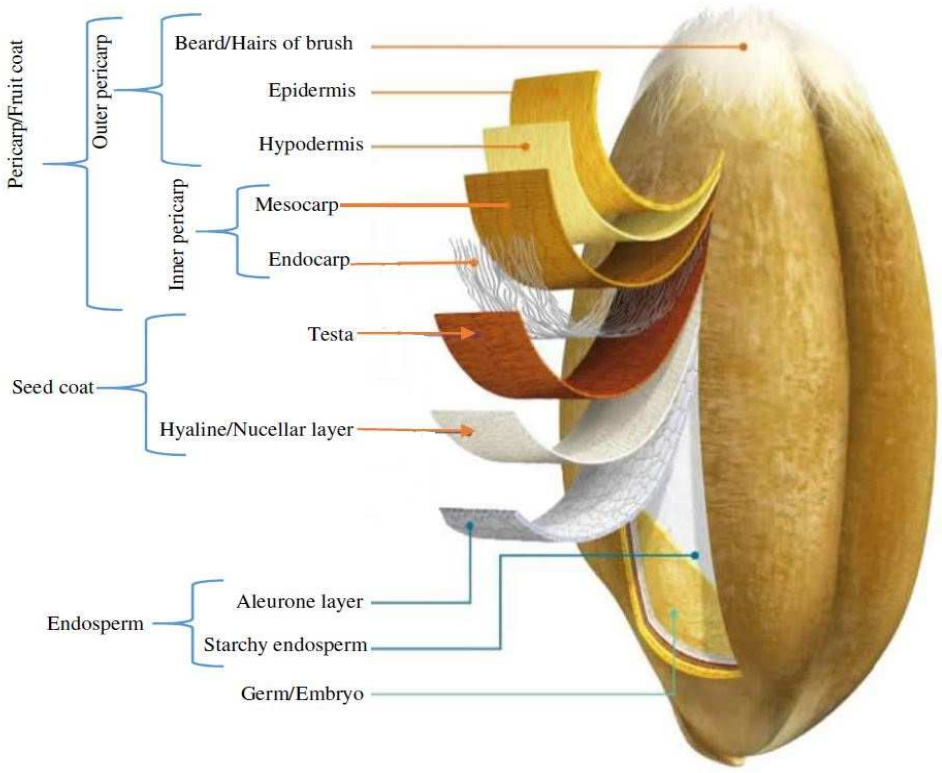


Figure 1.1. Different parts of wheat kernel. Adapted from Wheat Structure. In *nabim.* Retrieved May 13, 2018, from http://www.nabim.org.uk/wheat/wheat-structure/

Seed germination refers to the emergence of the shoot and root from the embryo of the seed. This is a physiological process, which usually begins with water uptake by the seed and ends with the emergence of the

embryonic axis through the structures surrounding it (Bewley & Black, 1994). When water enters into the seed coat, it starts softening the dry and hard tissue inside, and the grain swells up. Absorption of water within the kernel increases the hormonal activity, such as gibberellic acid (GA3), which causes the release of the hydrolytic enzyme, alpha (α)-amylases (De Laethauwer, De Riek, Stals, Reheul, & Haesaert, 2013). GA3 is transported to the aleurone layer where it turns on certain genes leading to their transcription into mRNA, which translated into α-amylase. The α-amylase diffuses into the starchy endosperm and hydrolyzes starch granules into sugar molecules. These sugar molecules are transported to the embryo which provide energy for growing embryo (figure 1.2), and seed germination occurs (Koning, 1994).

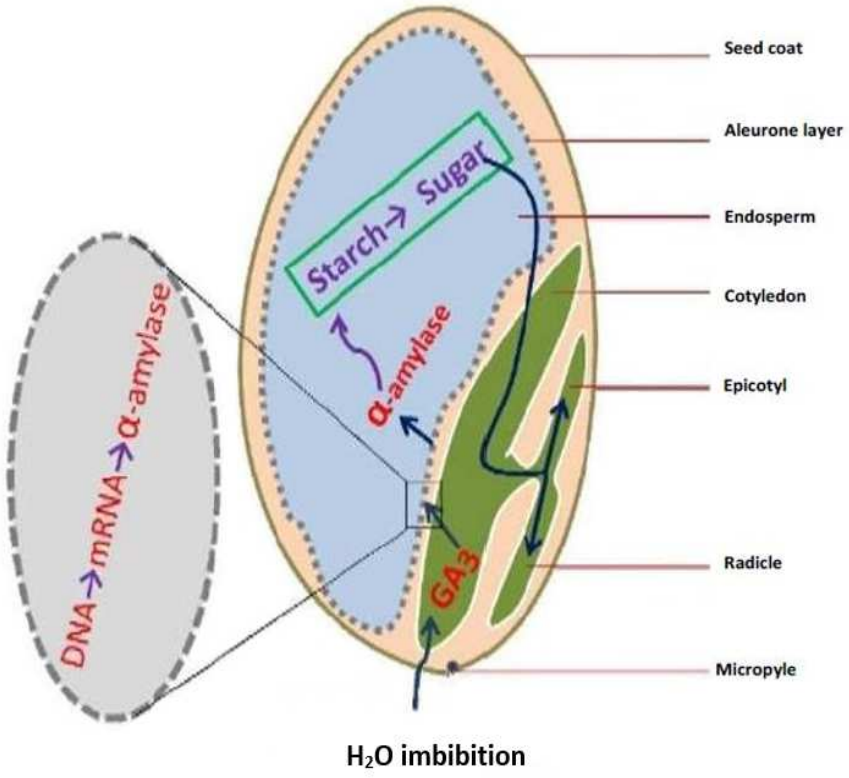


Figure 1.2. Mechanism of seed germination. Adapted from "Seed germination", In *Plant physiology information website,* by R. E. Koning, 1994, Retrieved from http://plantphys.info/

1.2. Pre-harvest sprouting in wheat: major cause of quality damage

Pre-harvest sprouting (PHS) in wheat refers to an "untimely germination of the mature kernels within the spikes" before harvest while still in the mother plant (figure 1.3) (Derera, 1989). PHS occurs when physiologically mature grain is exposed to frequent rain and high humidity along with higher temperature before harvest (Thomason, Hughes, Griffey, Parrish, & Barbeau, 2009). Generally, PHS may affect all the seed crops including wheat. PHS is considered as one of the most important factors for downgrading the bread-

making quality of the wheat in many regions around the world including Europe. It is also difficult to thresh sprouted grains, which results harvest losses.

Figure 1.3. Pre-harvest sprouting in wheat. Adapted from *Texas Row Crops Newsletter,* by C. B. Neely, 2016, Retrieved from https://agrilife.org/texasrowcrops/news/page/7/

During seed germination, a series of physiological, anatomical as well as biochemical changes occurs within the kernels to produce required energy and nutrients for the growth of emerging embryo. Sprouting involves with synthesis or activation of hydrolytic enzymes such as amylases, proteases, and lipases to breakdown starch, proteins and oils, respectively (Morad & Rubenthaler, 1983; Simsek et al., 2014a; Simsek et al., 2014b). This damages the quality of the wheat flour, and make it unusable for food processing.

Wheat kernel contains about 60 - 70% starch of the total dry weight which consists of 28 - 25% amylose and 72 - 75% amylopectin (Stone & Morell, 2009; Simsek et al., 2014a). Alpha-amylases are the enzymes responsible for hydrolyzing starch granules within the wheat kernel which accounts for about 30% of the total proteins synthesized during seed germination (Mohamed, Al-Malki, & Kumosani, 2009). The presence of alpha-amylases within wheat grain provides evidence for starch degradation and leads to poor end-use quality of baked goods made from flour of sprouted grains (Clarke, Ronald, De, & Christensen, 1984; Stone & Morell, 2009).

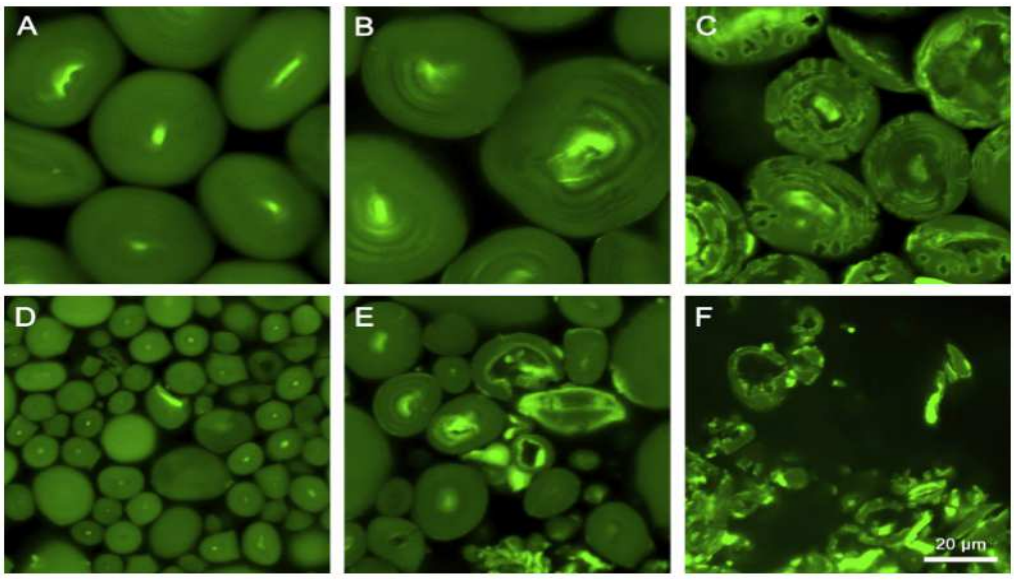


Figure 1.4. Structural changes of starch granules - honey-comb like structure (C and F). A - C: scanning electron micrographs of large starch granules of wheat, and D - F: scanning electron micrographs of small starch granules of wheat hydrolyzed by granular starch hydrolyzing enzyme at 55°C for 0 h (A and D), 1h (B and E) and at 30°C for 24h (C and F). Adapted from "*Amylolysis of large and small granules of native triticale, wheat and corn starches using a mixture of α-amylase and glucoamylase",* by Naguleswaran, Li, Vasanthan, Bressler, & Hoover, 2012, *Carbohydrate polymers, 88*(3), 864-874.

Studies by Naguleswaran et al. (2012) using scanning electron microscopy (SEM) of large and small starch granules of wheat grain hydrolyzed by starch hydrolyzing enzyme have suggested that the enzymatic cleavage of starch granules starts from the surface and generates pits towards the interior granule which produces a honey-comb like structure (figure 1.4). Thus, the higher activity of α-amylases during PHS causes structural changes of starch granules in PHS-damaged wheat grain, which results in reduced resistance of starch granules to swelling and lowered paste viscosity in dough made from flour of the sprouted grains (Simsek et al., 2014a).

Wheat proteins quantity and quality are the important factors in grading food wheat to access in industry. The protein content within kernel accounts for 10% - 18% of the total dry matter (Šramková, Gregová, & Šturdík, 2009). According to chemical nature, wheat proteins can be divided into two groups: the low molecular weight soluble protein, which consists of albumins, globulins, and peptides; and high molecular weight insoluble gluten, which consists of 80 - 85% of wheat storage proteins (Simsek et al., 2014b). Gluten proteins are important for better food processing and end product quality (Shewry, Halford, Belton, & Tatham, 2002) since the composition of gluten proteins is related with dough strength and baking quality (Simsek et al., 2014b). Gluten proteins are responsible to form cohesive viscoelastic dough during mixing flour with water. This viscoelastic property is responsible for the capacity of the dough to retain gas produced from fermentation (Veraverbeke & Delcour, 2002). PHS affects wheat grain quality by increasing proteolytic enzymes, which break high molecular weight storage proteins into smaller fractions. As a result, dough produced from flour of the sprouted grains results in decreased elasticity and strength with less gas holding capacity (Capocchi et al.,

2000). These studies suggest that the elevated protease activity causes degradation of proteins in sprouted wheat, which reduces the quality of wheat.

Economically, the main impact of PHS in wheat are the loss in yield and the decrease in quality (Edwards, Ross, Mares, Ellison, & Tomlinson, 1989). Bread baked from flour of sprout-damaged wheat possess low loaf volume, compact interior, and a dark crust. When sprout-damaged wheat flour is used for baking, the result may be a sticky dough with reduced water holding capacity. The extreme stickiness of dough creates handling problems which requires extra special handling (Paulsen & Auld, 2004). High levels of sprout damage also cause production problems of pasta and the resulting pasta products cannot withstand overcooking and become soft or mushy (Fu, Hatcher, & Schlichting, 2014). The discoloration of the noodles can be increased up to five times more than the normal kernel since sprouting raises alkaline activity within the kernels (Singh, 2008). This increased discoloration can make products unattractive to consumers (Hatcher & Symons, 2000). In most countries, sprouted wheat will be sorted out and down-graded to feed, and it will not appear in the lots used for milling. Thus, the farmers will also loose income because of the lower price for the low quality of the grain, and this is also a direct impact.

1.3. Factors affecting pre-harvest sprouting

There are some morphological, physiological and developmental traits of wheat kernel and spike, which can lead to PHS (Groos et al., 2002; King & Richards, 1984; Munkvold, Tanaka, Benscher, & Sorrells, 2009). The major factors affecting PHS include environmental conditions (Rasul et al., 2012), erectness of spikes and openness of florets (King & Richards, 1984; Tan, Sharp, Lu, & Howes, 2006), presence of the germination inhibiting compounds within the bracts (Derera & Bhatt, 1980), stage of maturity (Gale, Flintham, & Arthur, 1983), seed coat permeability, seed coat color, seed dormancy, hormonal activity, functional proteins (Gatford, Eastwood, & Halloran, 2002; King & Richards, 1984; Tan et al., 2006). However, resistance to PHS results from a combination of all the factors that affect water uptake of grains, both water uptake and drying rate of both ears and the individual grains, as well as factor affecting the mobilization of storage reserves to support germination. Thus, it is challenging to obtain resistance to PHS owing to the quantitative inheritance of the trait (Bailey et al., 1999; Flintham, 2000; Mares et al., 2005; Zanetti, Winzeler, Keller, Keller, & Messmer, 2000).

1.3.1. Role of phenology and ear morphology

The control of phenology is necessary to improve the resistance to PHS since the late heading results in the maturation of seeds in the rainy season which leads to PHS in the field (Derera, 1989; Mares & Mrva, 2014). Moreover, ear morphology, such as the presence of awn in the ear, and ear nodding angle are responsible for

more water absorption by the spike which promotes PHS (King, 1989). The awnless trait is preferable since it causes less water uptake of the spike and decreases the degree of PHS (King & Richards, 1984).

1.3.2. Role of seed coat permeability

The seed coat permeability acts as the first protecting wall to prevent PHS in wheat. When the external water, from rain or strong dewfall, enters into the seed coat, it turns on genes in aleuronic layer which release α-amylase. This α-amylase acts as precursor for starting the germination process (Koning, 1994).

1.3.3. Role of seed dormancy

1.3.3.1. Seed dormancy and germination

Among the factors affecting PHS, seed dormancy (SD) is the most important genetic factor (Zhang et al., 2017) which is considered as the primary inner factor leading to PHS resistance (Lan et al., 2005; Lin, Horsley, & Schwarz, 2008; Yang, Yu, Cheng, Tan, & Shen, 2011). SD can be defined as the temporary arrest of seed germination under favorable conditions for germination (Gubler, Millar, & Jacobsen, 2005). It is the type of adaptive strategy developed by some species to survive in adverse environmental conditions (Bewley, Black, & Halmer, 2006). There are some environmental factors like temperature, air, water and light conditions as well as biochemical factors such as hormonal activity play a crucial role for the process of seed germination. Some types of seeds may still fail to germinate even under optimal environmental conditions, these are known as dormant seeds, while a non-dormant seed can germinate under a widest range of environmental factors possible for the genotype (Baskin & Baskin, 2004).

1.3.3.2. Types of seed dormancy

According to the timing of development, there are two types of seed dormancy such as primary and secondary dormancy. Primary dormancy is build-up during seed maturation and maximum in the physiologically mature seeds. Both environmental and genetic factors may affect primary dormancy (Bewley, 1997). Secondary dormancy may initiate in mature seeds after losing primary dormancy. It can develop by prolonged inhibition of germination under unfavorable internal factors such as endogenous abscisic acid (ABA) and secondary metabolites or external factors such as unfavorable conditions for germination (Gubler et al., 2005; Hilhorst, 2007).

1.3.3.3. Mechanisms of seed dormancy

There are two main types of mechanisms for SD which can be defined as seed-coat imposed dormancy and embryo related dormancy. However, in some cases dormancy can also develop with the combination of the both seed-coat imposed and embryo related dormancy (Nikolaeva, 1977).

Seed-coat imposed dormancy is related to embryo surrounding structures such as seed coat, or endosperm. This type of dormancy can be found in cereals, conifers, and dicots seeds (Egley, 1989). Seed-coat also stores some germination inhibitors such as flavonoids (Debeaujon, Lepiniec, Pourcel, & Routaboul, 2007) which may delay the germination, and can lead to seed-coat imposed dormancy (Bewley & Black, 1994). Also, a relationship has been reported between PHS and wheat seed coat color, i.e., white-seeded wheat are much prone to PHS than red-seeded wheat (Derera, 1989; Flintham, 2000; Groos et al., 2002; Kottearachchi, Uchino, Kato, & Miura, 2006). The red color of the seed coat is determined by some flavonoids such as dihydroflavonols (DHQ, DHM), flavanol [(+)-catechin], and proanthocyanidins which accumulate in developing seeds of red but not white wheat (Kohyama et al., 2017).

Seed-coat usually prevents the absorption of water and gases by the seeds which is required for the germination. For instance, the pattern of epidermal cells arrangement of the seed coat has also an effect on SD as well as resistance to PHS, i.e., the loose arrangement of the epidermal cells in the seed coat easily allows the exchange of water and gases between seeds and outer environment which results in the germination of seeds and the susceptibility to PHS, whereas the tight arrangement ensures SD as well as the resistance to PHS (Cai & Chen, 2008).

Embryo related dormancy is intrinsic to the embryo and depends on the presence or absence of growth inhibitors or growth regulators, respectively. Embryo related dormancy releases when the seeds are stored for a long period with a certain level of the moisture content within the embryo (Bewley, 1997). King (1976) found that the isolated embryo was not able to germinate even under favorable conditions for the germination. This type of dormancy can be found in wild species of oat and Rosacea family (Kermode, 2005). In wheat, this is the main type of dormancy.

Primary dormancy is maximum in the physiologically mature seeds, and slowly released when seeds enter desiccation process. During the early stage of desiccation, seed can quickly pass the dormancy threshold, and PHS can occur if the environmental conditions are being favorable at this stage (Obroucheva & Antipova, 2000).

1.3.3.4. Importance of seed dormancy in agriculture

In agriculture, seed with low dormancy levels is very much prone to PHS, which damages the quality by affecting FN, an important quality parameter of wheat. Therefore, the maintenance of SD is essential to prevent PHS as well as to obtain uniform germination when seeds are sown in the field (Bewley & Black, 1994). PHS causes serious problem in wheat producing areas where the frequent rainfall is common during harvest season (Gale, 1989; Gerjets, Scholefield, Foulkes, Lenton, & Holdsworth, 2009). Therefore, wheat breeders focus on a high level of SD at harvesting period for preventing quality damage due to PHS and no SD at sowing to

obtain uniform germination. Thus, the identification and investigation of the mechanisms associated with PHS and subsequent germination of harvested seeds would be a better solution (Nakamura et al., 2011).

1.3.4. Roles of environmental factors: temperature and precipitation in seed dormancy

Temperature and precipitation are the most important environmental factors, which affect PHS in wheat during the late maturity stage (Hilhorst, 1995; Yanagisawa, Nishimura, Amano, Torada, & Shibata, 2005). Frequent rainfall after maturity (before harvest) is responsible for sprouting of the kernels while still attached to the mother plant (Groos et al., 2002). Although, low temperatures (10°C) and/or long photoperiods during seed development can induce higher seed dormancy (Benech-Arnold, 2004; Fenner, 1991; Gualano & Benech-Arnold, 2009; Nyachiro, Clarke, DePauw, Knox, & Armstrong, 2002), but sometimes low temperature with high humidity would break dormancy and promote sprouting (Argel & Humphreys, 1983; Ceccato, Bertero, & Batlla, 2011; Fonseca & Sa´nchez, 2000).

In wheat, temperature can affect SD at seed development as well as during seed germination (Black, Butler, & Hughes, 1987). Low temperatures during seed development induce higher SD (Black et al., 1987; Buraas & Skinnes, 1985; Reddy, Metzger, & Ching, 1985). For dormant seeds, however, low germination temperatures stimulate seed germination and intermediate germination temperature (20°C) allows different genotypes to express the degree of SD (Nyachiro et al., 2002). Whereas, a high germination temperature like 25-30°C can inhibit seed germination in wheat (George, 1967; Nyachiro et al., 2002; Walker-Simmons, 1988). Some seeds are not able to germinate at high temperature even after the dormancy is broken. This is considered as an adaptive strategy for plants, which allows seeds to germinate according to seasonal changes (Footitt, Douterelo-Soler, Clay, & Finch-Savage, 2011). Freshly harvested wheat grain is considered to have true SD (Corbineau & Côme, 2000). Generally, wheat cultivars exhibit maximum SD at physiological maturity, and then gradually lose dormancy through after-ripening (Gerjets et al., 2009; Paterson, Sorrells, & Obendorf, 1989). PHS tolerant wheat lines tend to lose dormancy during storage, or by cold stratification, imbibing water in the cold, more slowly than susceptible lines (reviewed in Baskin & Baskin, 2004; Bewley & Black, 1994; Bewley, Bradford, & Hilhorst, 2012; Gerjets et al., 2009; Tuttle et al., 2015). Selection for synchronous seedling emergence in the field has inadvertently led to low SD and PHS susceptibility in cereals including wheat (Gualano et al., 2007; Paterson & Sorrells, 1990; Ullrich et al., 2009).

1.3.5. Roles of endogenous hormones in seed dormancy

SD also depends on the absence or presence of plant hormones within the seeds. For example, ABA and GA are related with physiological dormancy of the plant embryo. They act antagonistically in maintaining seed dormancy (Iglesias & Babiano, 1997). A study by Pisipati (2008) has also revealed that ABA plays a role to induce dormancy during seed development, whereas GA promotes seed germination in non-dormant seeds.

ABA content increases within the seeds during seed development which directly maintenance SD by preventing the precocious hydrolysis of starch granules (Bewley, 1997; King, 1976). During early seed developmental stages, ABA level is higher in embryo than endosperm and it decreases with the maturation of seeds (Pisipati, 2008). The concentration of ABA within seeds is a critical factor for the successful germination of seeds and subsequent establishment of seedlings (Kermode, 2005). The α-amylase acts as main catalyst to breakdown starch during PHS (Walker-Simmons, 1987) which synthesizes by the GA3-enhanced biosynthesis pathway in aleuronic cells. ABA may suppress the synthesis of α-amylase by inhibiting the GA3-enhanced biosynthesis pathway (Ho & Varner, 1976; King, 1976). Study on the strongly dormant *A. thaliana* ecotype Cvi also showed that the dormant state is characterized by increased ABA biosynthesis and GA degradation (figure 1.5) (Cadman, Toorop, Hilhorst, & Finch-Savage, 2006).

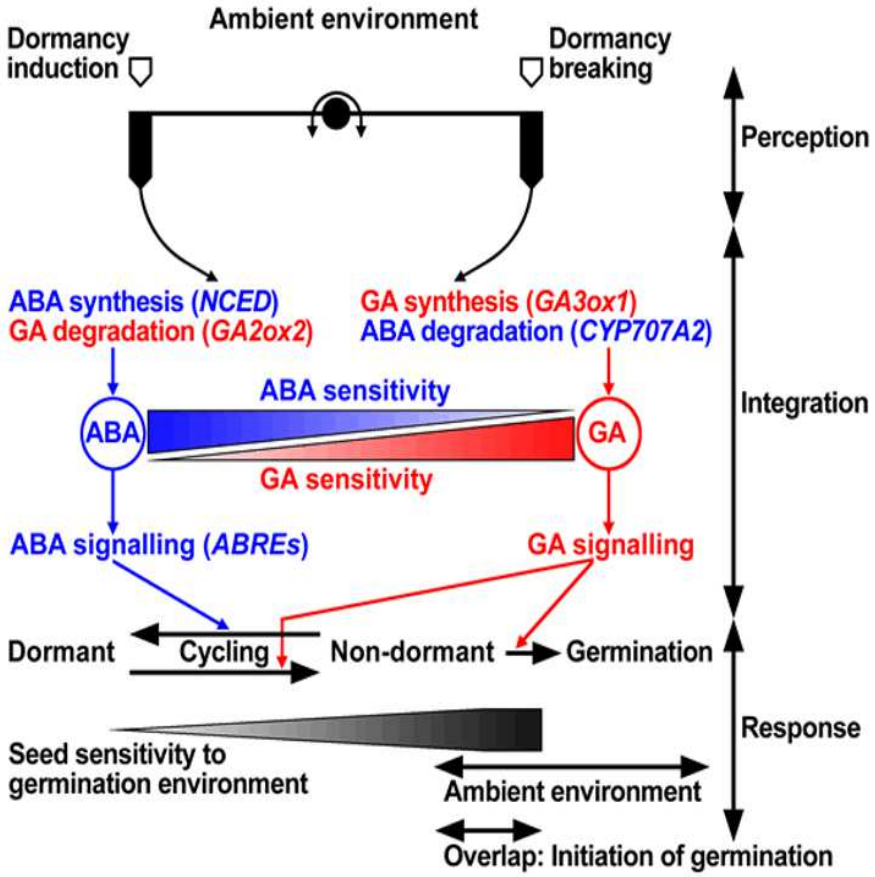


Figure 1.5. A schematic representation of the regulation of dormancy and germination by ABA and GA in response to the environment. Adapted from "Gene expression profiles of *Arabidopsis* Cvi seeds during cycling through dormant and non-dormant states indicate a common underlying dormancy control mechanism" by Cadman, Toorop, Hilhorst, & Finch-Savage, 2006, *The Plant Journal, 46*(5), 805-822.

1.3.6. Release of seed dormancy

Although GA is not associated with the maintenance of the SD, it acts as an important factor for seed germination and thus it plays a role for releasing SD. In developing embryo, the concentration of GA is usually high and most of the GA are deactivated with maturation (Ogawa et al., 2003). GA content increases again during germination just prior to radicle formation. During seed germination, GA increases embryo growth potential, reduces the mechanical barrier by weakening the tissues surrounding radicle, and prevents the inhibitory effect of ABA in germination (Bewley, 1997; Pisipati, 2008). When GA is released from the embryo, it triggers genes in aleurone cells to secrete α-amylases, which acts to supply energy for growing embryo (Koning, 1994).

1.4. Methods of dormancy assessment

There are several methods to assess dormancy in seeds. However, only two methods, Germination Index (GI) and Falling Number (FN), relevant to this research are being discussed. The GI test measures dormancy directly, while FN test measures it by measuring the activity of α-amylases.

1.4.1. Germination Index

GI is a weighted index that is measured by germinating seeds and counting number of germinated seeds each day for as long as the test is going (Reddy et al., 1985). The temperature for the germination test needs to be calibrated after material is being tested since dormancy is expressed differently with different temperatures. Nyachiro et al. (2002) reported that the greatest differences in seed germination tended to be at 15°C to 20°C, while GI decreased when temperature increased from 10°C to 30°C. These findings reveal that the level of SD depends on the genotype and germination temperature.

The GI values ranges from 0 to 1. GI is maximum if all the seeds germinate on the first day since it gives higher value to seeds that germinate early. The maximum GI indicates no SD. Whereas, lower GI values indicate the increasing level of SD when the seeds germinate later (Walker-Simmons, 1987). GI can predict the resistance of the genotypes to PHS since the ability of seed germination is inversely related to the degree of SD (Nyachiro et al., 2002). Therefore, measuring SD by GI is one of the main tools used for plant breeding to improve PHS tolerance in cereals (Biddulph, Plummer, Setter, & Mares, 2008). The GI is calculated with equation 1 according to Reddy et al. (1985).

$$GI = \frac{d\ X\ n_1 + (d-1) X\ n_2 + (d-2) X\ n_3 + \cdots + 1\ X\ n_L}{dN} \qquad equation\ 1$$

Where,

d = total number of days the seeds counted for

n_1, n_2, n_3 and n_L = number of germinated seeds on day 1, day 2, day 3 and day last

N = total number of germinated seeds

1.4.2. Falling Number

The FN is an analytical method, which is recognized as an established trading parameter for detecting quality damages caused by sprouting in flour of cereals including wheat (Perten, 2005). This is a standard and accepted method in cereal industry worldwide. FN is indicative of the amylase (specifically α-amylase) activity in a wheat flour dough. The α-amylase is the specific enzyme measured in the FN test relative to its ability to liquefy starch. The rate of conversion of starch into sugar is determined by the quantity of α-amylases contained in flour. Higher the α-amylase content results in high fermentation sugar in the dough, whereas lower the α-amylase content results in a dough with little gassing power. Severely sprouted wheat becomes suitable only for animal feed and cannot be used for flour production or any other application in the food industry (Simsek et al., 2014b). Therefore, the resistance to PHS is a highly desirable characteristic in bread wheat (Zanetti et al., 2000; Li et al., 2004).

The FN values ranges from 60 to above 400 seconds. The flour of sprout-damaged grains contains high α-amylase activity results in low FN values, whereas the flour of non-sprouted grains possesses low α-amylase activity which results in higher FN values (Hagberg, 1961; Best & Muller, 1991). The wheat flour is used as substrate for suspension. During FN test, starch degradation by α-amylase is measured by a quick gelatinization of flour suspension in a boiling water-bath under condition that is similar to those found during baking (Perten, 1964). The FN method has been tested and approved for application to both meal and flour of grains and cereals by the American Association of Cereal Chemists International (*AACCI*, 1999).

However, a certain amount of α-amylase is essential for baking since the amount of α-amylase present in the flour have a direct effect on the bread quality. The α-amylase in flour hydrolyzes starch to sugar molecules during mixing flour with water, which provide fuels for the fermentation process (Smith et al., 2006). The bread, produced from flour with optimal α-amylase activity, will be firm and with high volume and soft texture (figure 1.6; table 1.1). While, when a bread is produced from flour with excessive α-amylase activity results in low loaf volume with sticky crumb structure (figure 1.6; table 1.1). Moreover, a bread with small volume and dry crumb may result if the α-amylase activity is very low (figure 1.6; table 1.1) (Perten, 2005).

Table 1.1: Enzyme content and fermenting ability. The FN values are inversely proportional to the amylase activity. Adapted from Criteria for judging quality, In *The artisan*, Retrieved May 12, 2018, from http://www.theartisan.net/flour_criteria_judging.htm

FN	Comments
62 - 150	Strong amylase activity. Flour is nearly unusable for bread making without mixing with other flour with a higher FN.
150 - 220	Higher amylase activity to that which is normal. Flour requires a correction by being mixed with flours of a higher FN or using particular recipes during production.
220 - 280	Optimal amylase activity. Flour is suitable for bread making without any corrections.
>280 - 300	Weak amylase activity. Flour requires a correction by the baking recipe.

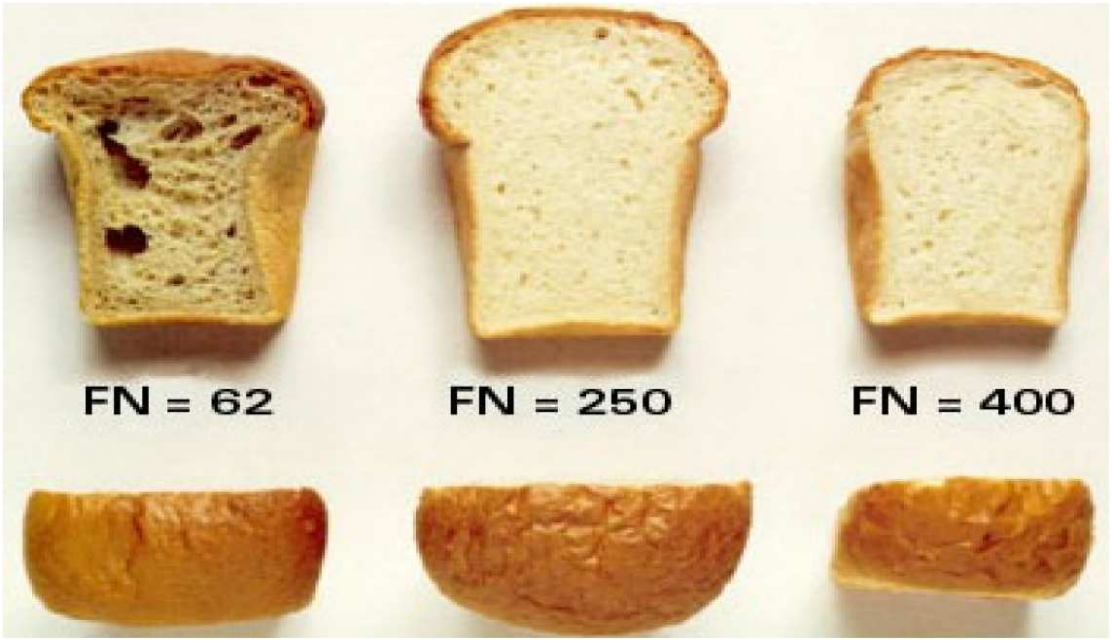


Figure 1.6. Bread loaves made from flour with different FN. Adapted from Falling number application and method, by H. Perten, 2005, Retrieved from https://www.perten.com/

1.5. Genetics of seed dormancy and pre harvest sprouting

1.5.1. Genes controlling seed dormancy

SD is a quantitative trait, determined by several genes with an additional strong impact of the environment (Finch-Savage & Leubner-Metzger, 2006). Several major QTL have been detected on chromosomes 2BS, 3AS, 3AL, 3BL, 3DL, and 4AL in different populations (Chen, Cai, & Bai, 2008; Fofana et al., 2009; Groos et al., 2002; Jaiswal, Mir, Mohan, Balyan, & Gupta, 2012; Kulwal et al., 2005; Kumar et al., 2015; Lin et al., 2015; Liu, Cai, Graybosch, Chen, & Bai, 2008; Liu, Bai, Cai, & Chen, 2011; Mori, Uchino, Chono, Kato, &

Miura, 2005; Ogbonnaya et al., 2008; Somyong et al., 2014). Candidate genes in these regions have been identified based on comparative genomics or transcriptomic analysis, such as *TaSdr-B1* on 2BS (Zhang, Miao, Xia, & He, 2014), *TaPHS1* (a *TaMFT*-like gene) on 3AS (Liu et al., 2013; Nakamura et al., 2011), *TaVp-1* and *Tamyb10* on group 3 chromosomes (Chang et al., 2010a, b, 2011; Himi, Maekawa, Miura, & Noda, 2011; Yang et al., 2007b), *PM19-A1* and *TaMKK3-A* on 4AL (Barrero et al., 2015; Torada et al., 2016).

TaSdr-B1, responsible for SD in wheat, identified on chromosome arm 2BS by Zhang et al. (2014). In addition, positional cloning of SD QTL has successfully found gene *Sdr4* (seed dormancy4) that affect SD in rice (*Oryza sativa)* (Sugimoto et al., 2010). *Sdr4* encodes proteins of unknown function and shows seed-specific expression, and highly expresses in dormant seeds.

Recently, a QTL, *QPhs.ocs-3A.1*, has been molecularly characterized on 3A chromosome, which has revealed that the mutation of the *MOTHER OF FT* and *TFL 1 (MFT)* gene affects SD in wheat (Nakamura et al., 2011). *MFT*-like genes belong to the PEBP (plant phosphatidyl ethanolamine binding protein) family. These genes are divided into three subfamilies such as *FLOWERING LOCUS T* (*FT*)-like, *TERMINAL FLOWER1* (*TFL1*)-like, and *MFT*-like (Chardon and Damerval, 2005). During seed germination in *Arabidopsis, MFT* acts as a negative regulator of ABA sensitivity (Xi, Liu, Hou, & Yu, 2010).

Leubner-Metzger (2003) reported that the wilty mutants of *Nicotiana plumbaginifolia* for *aba1* and *aba2* genes have reduced ABA contents and exhibit precocious germination with reduced primary dormancy. ABA-deficiency of the *aba2* mutant is for a mutation in the *ABA2* gene which encodes zeaxanthin epoxidase, a key step in ABA biosynthesis. In another study, *ENHANCED RESPONSE TO ABA8 (ERA8)* was isolated in a PHS susceptible cultivar Zak, a soft white spring wheat, based on increased sensitivity to ABA (Schramm, Nelson, Kidwell, & Steber, 2013).

High temperature germination sensitivity can vary according to the molecular mechanisms of different plant species (Lei et al., 2013). In *Arabidopsis*, high-temperature thermos-dormant seeds expressed high *FLC*, a flowering-time regulator, during germination at low temperature (Chiang, Barua, Kramer, Amasino, & Donohue, 2009; Xi et al., 2010). Thermos-dormancy is the failure of seed germination when seeds are imbibed at higher temperatures like 25-30°C or above. In lettuce, a QTL, *Htg6.1,* has identified for thermos-dormancy. *Htg6.1* is associated with *LsNCED4* gene, which plays a role in ABA biosynthesis (Argyris, Dahal, Hayashi, Still, & Bradford, 2008). Mutants with altered ethylene synthesis or sensitivity have also been found to exhibit the tolerance to seed germination at high temperatures (Lei et al., 2013). Hilhorst (2007) has reported that temperature signals can alter membrane fluidity, which results in a change in SD.

Although a number of genes have been identified involving SD process, our knowledge is still too limited to control SD in bread wheat (Bentsink & Koornneef, 2008; Finch-Savage & Leubner-Metzger, 2006; Finkelstein, Reeves, Ariizumi, & Steber, 2008; Holdsworth, Finch-Savage, Grappin, & Job, 2008).

1.5.2. Genes controlling pre-harvest sprouting in wheat

PHS is a complex trait which is controlled by several factors such as genotypes, environments as well as G*E interaction (Marzougui et al., 2012). In wheat, PHS is regulated by both embryonic and coat-imposed pathways, which are controlled by separate genetic systems (Himi, Mares, Yanagisawa, & Noda, 2002). Seed coat color is associated with both SD and PHS. Nilsson-Ehle (1914) first reported the relationship between the seed color genes and PHS. Several studies have revealed that red seeded wheat are comparatively more PHS-resistant than white seeded wheat (Rasul et al., 2012; Torada and Amano, 2002). It is widely known that the *R* genes such as *R-A1, R-B1, and RD1* are responsible for red grain color. They are located on 3AL, 3BL, and 3DL chromosomes arms of bread wheat respectively and responsible for the accumulation of the flavonoid pigments in the grain coat (Himi et al., 2011) and are heritable to the offspring. According to Himi and Noda (2005), *R* genes regulate the expressions of the *CHS, CHI, F3H* and *DFR* genes that encode enzymes in the early steps of flavonoid synthesis. Genes *TaDFR* and *Tamyb10* have been reported to regulate grain color, which are also located on homologous group 3 chromosomes. *Tamyb10* gene is located in the same regions as the *R* loci. Genes *R*, *TaDFR* and *Tamyb10* have been proven to be *Mybtype* transcription factors (Bi, Sun, Xiao, & Xia, 2014; Himi & Noda, 2004, 2005; Himi et al., 2011).

Since not all the red-grained wheat are resistant to PHS and also not all white-grained wheat are susceptible to PHS, which suggests that other genes might also be involved in regulation of PHS tolerance (Bassoi & Flintham, 2005; Zhang et al., 2017) and that *R* genes have only a minor effect in the determination of SD (Himi et al., 2002).

A study by McCarty et al. (1991) has revealed that *Vp-1* gene, *VIVIPAROUS-1*, is responsible for maintenance of SD in maize, and inactivation of this gene can disrupt the maturation of embryo and provoke germination while seeds are still attached in the cob. The *Vp-1* genes were also extensively investigated in wheat and proved to be associated with PHS tolerance (Bailey et al., 1999; Chang et al., 2010a, b, 2011; Hattori et al., 1992; McKibbin et al., 2002; Nakamura & Toyama, 2001; Xia et al., 2008; Yang et al., 2007a, b; Yang et al., 2014). Bailey et al. (1999) mapped the *TaVp1* genes on the long arms of group 3 chromosomes, and these were located within 30 cM distances from the *R* loci. This indicates that both *R* genes and *TaVp1* can influence on coat-imposed mechanisms of dormancy as well as on embryo dormancy (Groos et al., 2002).

Since many genes, with minor effect and interacting with each other, are contributing in determining the SD and the resistance to PHS in wheat, therefore, the understanding of the genetic mechanisms associated with PHS resistance will contribute in the breeding of new cultivars with resistance to PHS.

1.5.3. QTL controlling pre harvest sprouting

Previous studies on QTL analysis of SD and PHS in wheat indicates that SD and PHS are controlled by multiple QTL distributed through all 21 chromosomes (Mares & Mrva, 2014). Major QTL were reported on 2B (Chao, Xu, Elias, Faris, & Sorrells, 2010; Munkvold et al., 2009; Somyong et al., 2014), 3A (Liu et al., 2013) and 4A chromosomes in wheat genotype (Chen et al., 2008; Ogbonnya et al., 2008; Torada, Koike, Ikeguchi, & Tsutsui, 2008).

To date, a total of 22 QTL, derived from 9 mapping populations characterized worldwide, have been reported which are associated with the expression of PHS in wheat (Flintham, 2000). In a cross between AC Domain and White-RL4137, Fofana et al. (2007) identified seven PHS QTL on 3A, 3B, 3D and 5D chromosomes that overlapped with three seed coat color QTL on 3A, 3B, and 3D chromosomes and one PHS QTL on 5D chromosome. AC Domain is a Canadian red spring wheat, possesses high levels of dormancy, while White-RL4137 is moderately resistant to PHS and is more susceptible to PHS than AC Domain. In another mapping population also involving AC Domain, Rasul (2007) has reported 13 PHS QTL on 3A, 3D, 4A, 4B, and 7D chromosomes. Many of the QTL reported in the two Canadian doubled haploid (DH) mapping populations were identified at the similar chromosomal locations reported in other populations (Groos et al., 2002; Kulwal et al., 2005; Zanetti et al., 2000), suggesting that similar loci control PHS resistance in such populations. In several studies, major QTL free of interacting pleiotropic effects were found on chromosome 4A (Kato, Nakamura, Tabiki, Miura, & Sawada, 2001; Mares et al., 2005; Rasul, 2007), chromosome 5D (Anderson, Sorrells, & Tanksley, 1993; Fofana et al., 2007; Kulwal, Singh, Balyan, & Gupta, 2004), and group six chromosomes (Anderson et al., 1993; Roy et al., 1999; Zanetti et al., 2000).

Three QTL for SD, scored as the percentage of germinated seeds, have been detected on group four chromosomes of wheat by Kato et al. (2001) using 119 doubled haploid (DH) lines derived from a cross between AC Domain and Haruyutaka. Haruyutaka is a Japanese red-grained wheat, which possesses low levels of dormancy. AC Domain contributed on more than 80% of the total phenotypic variance in SD. Mori et al. (2005) mapped two putative QTL on 4A and 4B chromosomes, which are associated with dormancy. The QTL *QPhs.ocs-4A.1* is located on 4AL and *QPhs.ocs4B.1* is located on the centromeric region of 4BL chromosome arm. Mares et al. (2005) also reported that the resistance to PHS in bread wheat can be achieved through the introgression of putative SD genes or QTL located on 4A chromosome.

By evaluating the spike sprouting percentage, Groos et al. (2002) have reported that three marker loci, *Xfbb293, Xbcd131,* and *Xgwm3* located on 3AL, 3BL, and 3DL chromosomes arms, respectively, are associated with the resistance to PHS in wheat. These loci are co-localized with QTL for seed coat color where the *R* genes were previously mapped. Two QTL for SD designated as *QPhs.ocs-3A.1* and *QPhs.ocs-3A.2* evaluated by germination tests at 20°C were identified on the short and long arms of 3A, respectively (Osa et al., 2003). Roy et al. (1999) reported a significant association of 6BL chromosome arm with PHS tolerance. Zanetti et al. (2000) reported several QTL for PHS resistance by studying progenies derived from a cross between wheat x spelt that a QTL on 5A chromosome is associated with spelt ear morphology, and chromosome 3B, 7B from spelt as well as chromosome 6A from wheat is associated with FN and α-amylase activity.

However, it is obvious that a single major gene or QTL cannot fully explain the mechanisms associated with PHS resistance in wheat. In addition, SD is a complex trait, which is determined by the genetic factors along with environmental conditions (Cao et al., 2016; Jaiswal et al., 2012; Kulwal et al., 2004, 2012). Therefore, it is important to identify the genetic network among candidate QTL for SD as well as for other traits associated with PHS resistance in wheat. However, no QTL have been reported to date for PHS resistance in spring wheat adapted to Norwegian growing conditions.

1.6. Economic impact and research goals

1.6.1. Impact of PHS on wheat production in Norway

Norway is a western Scandinavian country stretching along the Atlantic from 58 degrees north to 71 degrees north (Arnoldussen, 1999). The total land covers about 323,787 km^2 with the total arable land only 2.6% of the total land area (Statistics Norway, 2017). In 2016, out of 832,099.1 ha of arable land only 220,973.6 ha were used for spring wheat production (table 1.2). The main wheat producing area is situated in the south-eastern part of the country (figure 1.7).

Table 1.2. Land area and use in Norway in 2017 (Statistics Norway, 2017).

Total land cover	323,787 km^2
Arable land	832,099.1 ha (2.6% of total area)
Spring wheat	220,973.6 ha

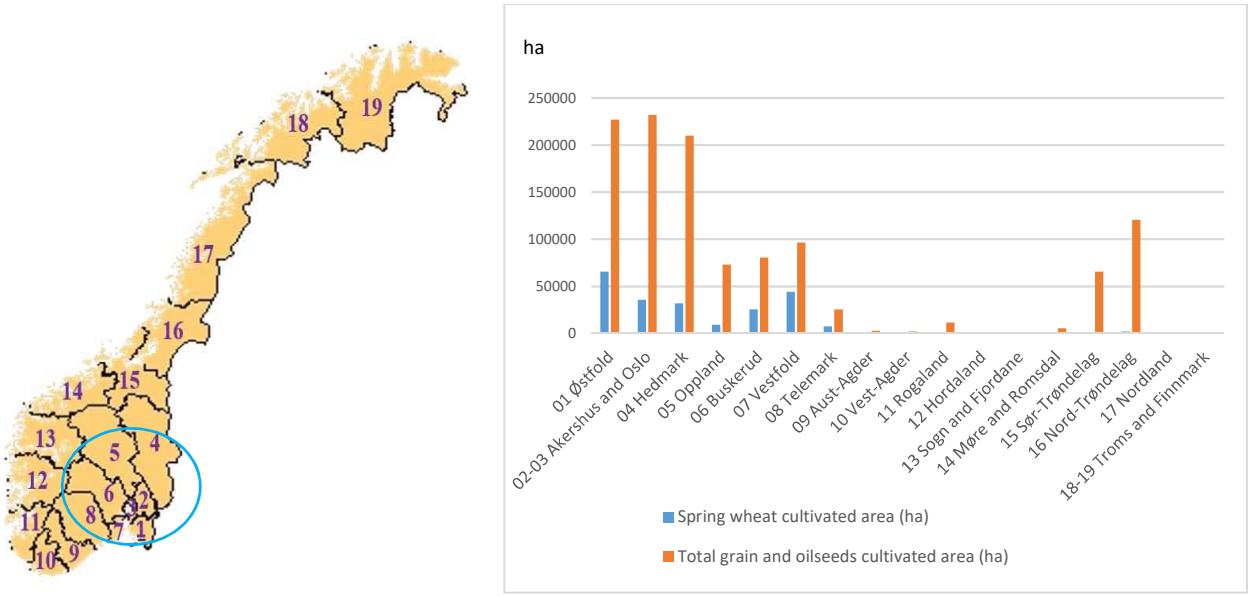


Figure 1.7. Distribution of wheat cultivation area in Norway in 2017 (figure in left). Main spring wheat producing areas are indicated by a circle. Right figure shows spring wheat cultivated area per county as well as the total area of grain and oilseeds in 2017 in Norway (Statistics Norway, 2017).

The first evidence for cultivation of cereals in Norway dates back to around 1800 BC during the Bronze Age. The main cereal grown in the beginning was naked barley (*Horedum vulgare nudum*), with more sporadic cultivation of einkorn (*T. monococcum*) and emmer wheat (*T. dicoccum*). Wheat cultivation declined during the Late Bronze Age, which also saw a transition to more cultivation of *T. spelta* and *T. aestivum*. In around 500 BC, naked barley was replaced by hulled barley (*H. vulgare*) and oats were introduced (Bakkevig, 1995; Myhre, 2004). During the Viking and Medieval times, wheat was considered as a luxury grain that was only grown in special fields at the big farms and used by the chieftain class (Mikkelsen, 1979). As the climate changed to cooler and wetter during period from around AD 1200 and onwards, domestic grain production became less reliable. However, barley and oats were the dominating cereals grown and used for foods until the twentieth century.

In around 1900, the country relied more on imported grains, mainly wheat from England and rye from the countries around the Baltic Sea (Lunden, 2004) and the vulnerability of not producing enough grain during the climatic misfortune often caused a widespread famine. However, the wheat producing area increased from about 10,000 ha in the 1920s to 41,000 ha in 1939 as the State paid good price to farmers (Lillemo & Dieseth, 2011).

However, the introduction of the combine harvester in around 1950s created new challenges for the wheat production since it is necessary that grain is dried enough in the field in order to be harvested by combine harvester. But frequent rainfall during harvesting period often created problems for harvesting on timely basis.

Since wheat cultivars were not resistant to PHS in the field, the quality of food wheat was poor. Because of this reason, farmers opted for barley instead of wheat, which nearly led to the extinction of wheat cultivation in Norway. As a result, a detailed investigation was started to improve the resistance of wheat cultivars to PHS, which led to the development of SD index (Strand, 1965).

During 1970s, the wheat production started to increase again in Norway because of releasing two landmark varieties, Runar and Reno, with resistance to powdery mildew, lodging, as well as PHS. The cultivation of those cultivars were dominated in Norway for more than two decades, which triggered the unprecedented increase in wheat production that has continued until today.

Due to the strong political will of increasing the domestic cereal production in Norway, the wheat production was increased remarkably during the last few decades, and the contribution of domestic wheat in the flour blend was also increased. This is also manifested by the Norwegian agricultural policy even after its integration with European Economic Area (EEA) as well as having an agreement with the World Trade Organization (WTO). However, the prolonged instability of the global wheat market with comparatively higher price also necessitates the increase in domestic wheat production for the self-sufficiency in the future (Lillemo & Dieseth, 2011).

Now it is obvious that the unstable autumn weather with frequent rainfall and high temperature during grain maturation and harvesting period is the main cause of quality damage in wheat (Archibald, 2013). Since a FN of 200 or above and protein content above 11.5% is used for grading wheat grain to industry for bread making, farmers are tend to harvest wheat grain with relatively higher moisture content to avoid price reduction due to the loss in quality from PHS.

In some recent years, PHS has caused a serious problem resulting in lower proportions of wheat that meet the requirements set to food quality. The total amount of wheat produced that holds food quality has been recorded approximately 180,000 tons during 2016/2017 with lowest production 50,000 tons during 2011/2012 and highest 250,000 tons during 2003/2004 (figure 1.8). In recent years, from 2008/2009 total food wheat production were not stable in amount and it is also predicted for 2017/2018 that the production will reduce again less than 100,000 tons which is almost half of the 2016/2017 but the consumption will still be at the same amount around 300,000 tons (figure 1.8). Although, just a few years ago from 2003/04 to 2007/08 seasons, Norwegian wheat comprised up to about 80% of Norwegian bread. During that most favorable wheat seasons in Norwegian history, less than 30% of the wheat for human consumption had to be imported (figure 1.8). Later, in the 2011/12 season, 90% of the wheat had to be imported due to challenging weather and problems with PHS (Archibald, 2013). In the years after 2007, low FN has been the most important factor to not achieve food grade. As a larger quantity of wheat with strong quality is likely to be imported during the seasons with weak quality, around 70% wheat might be imported during 2017/18 for human consumption

(figure 1.8). From this comparative statistical data of recent two decades of wheat production and consumption in Norway, it is obvious that Norwegian wheat production cannot meet the demand of national consumption and every year a lot of wheat is imported to mitigate this demand (figure 1.8). The major cause of the failure of producing required amount of wheat is PHS, resulting from excessive humidity during the maturation and harvesting period. Therefore, to achieve a more stable food wheat production, the resistance to PHS needs to be improved.

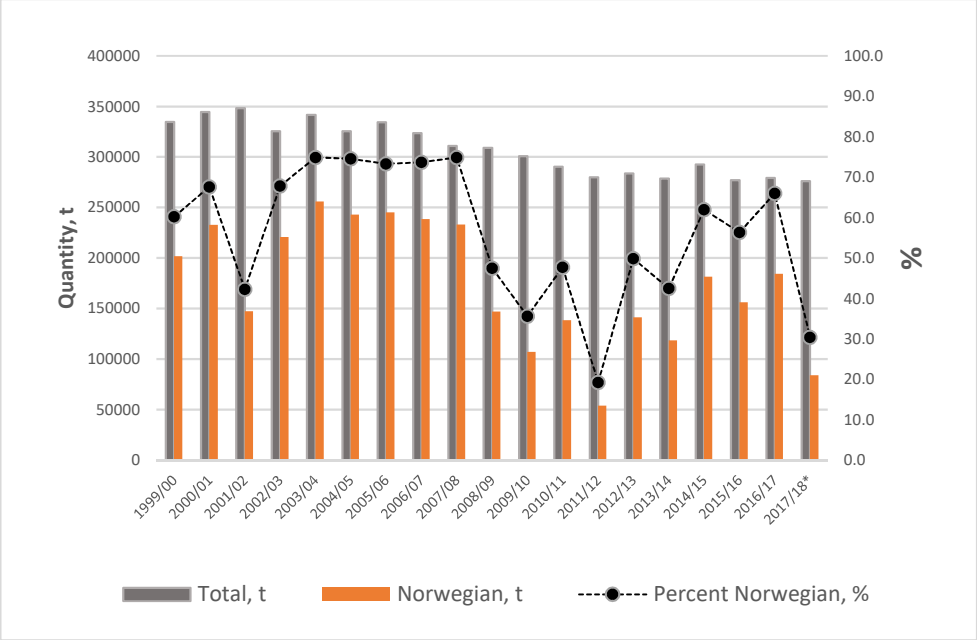


Figure 1.8. Total wheat consumption (ash bars) and total wheat production in country that holds food quality (dark brown bars) and the % contribution of domestic wheat in Norwegian wheat flour (Statistics Norway, 2017).

1.6.2. Research goals

In recent years, the yield reduction and quality deterioration of domestic wheat are the regular phenomenon in Norway, and this is likely to be more severe in the near future due to the effect of the climate change. Therefore, the improvement of the resistance power of Norwegian wheat cultivars to PHS is sought for reducing the losses due to reduced yield and quality damage. Although, enormous effort has already been paid in wheat breeding programs to integrate a moderate amount of SD in commercial varieties worldwide. However, little is known about the genetic basis for PHS resistance in the Norwegian wheat germplasm. As reviewed above, PHS occurs in susceptible cultivars and is to a large extent determined by the level of SD, a complex quantitatively inherited trait that is highly dependent on environmental conditions, genetic background and genotype-by-environment interactions. Thus, identification of the main genetic loci responsible for PHS resistance in spring wheat would be a great importance to develop PHS resistant wheat cultivar.

Therefore, the main objectives of this study were: (1) to detect QTL for two PHS traits: GI of seeds at yellow ripeness (YR) and FN of whole-meal flour; and (2) to investigate whether there are relationships between PHS QTL and QTL for agronomic traits: days to heading (DH), days to maturity (DM) and plant height (PH), on the integrated genetic map of the SHA3/CBRD x Naxos recombinant inbred lines (RILs) spring wheat population grown under different environmental conditions.

Chapter 2

Materials and Methods

2.1. Plant materials

A population of 168 RILs of Shanghai3/Catbird (SHA3/CBRD) x Naxos, crossed and propagated through F_1-F_6 by single seed descent method, was utilized in this study. The population was developed by Lu et al. (2012) at Norwegian University of Life Sciences (NMBU), mainly for mapping powdery mildew (Lu et al., 2012) and *Fusarium* head blight resistance (Lu et al., 2013), but later also found to be useful for other traits. Lu and Lillemo (2014) used this population to map the genetic factors related to *Parastagonospora nodorum* leaf blotch (SNB) resistance in the field. As the parents also segregated for PHS resistance, we used this population to identify responsible QTL for PHS. In the field trials in Norway and China, the CIMMYT line SHA3/CBRD was moderately susceptible while the German spring wheat Naxos was moderately resistant to PHS.

2.2. Field experiments

The wheat lines were sown at Vollebekk research farm of NMBU, Ås, Norway on 26th of April in 2016 and at Chengdu, China on 6th of November, 2014 for the 2015 season, and on 5th of November, 2015 for the 2016 season. The field trials at Vollebekk were laid out using Alpha Lattice Design with two replications. The trials were sown with plot size 0.75 m x 3 m with 15 cm of space between the rows. The amount of seeds was 80 g per plot. After emergence, 1 m alleys were sprayed out with glyphosate, giving a final harvested plot size of 0.75 m x 2 m. Fertilization was done with 12 kg N/daa in NPK fertilizer 22-3-10 (Yara). Herbicide treatment was done as normally practiced (Areane at Zadoks 13-15).

At Chengdu in 2015 and 2016, each line was planted in rows, one row with 1.5 m length and 0.5 m space between rows. The amount of seeds sown were 30 grains per row. Fertilization was done with 11.25 g pure N, 11.25 g K and 11.25 g P per square meter. Herbicide treatment was done with fluroxypyr and Clodinafop-propargyl, as normally practiced. The RILs were not randomized and no lines were replicated.

2.2.1. Recording days to heading, days to maturity and measurement of plant height

The dates of heading were recorded when 50% of the plants in the plot completed the emergence of spike (head) from the flag leaf sheath but had not yet started to flower. By using recorded dates of heading we calculated the number of days since planting to heading and referred this trait as days to heading (DH).

The days to maturity (DM) was determined on the basis of yellow ripeness (YR) of the plant. A plot was considered as in YR when 50% of the plants in that plot lost green color of the spike and the peduncle, and the moisture content of grains was 38%. For some plots, the grain moisture content was predicted to contain 38%

moisture content based on experience. At YR, kernel formation was completed and the kernel accumulated most of its dry weight since by the end of the YR stage the transport of nutrients from the leaves, stems, and spike to the developing seed is completed. The kernel is physiologically mature at this stage even though it still contains approximately 38% water. The dates of YR were recorded for all the plots. By using recorded dates of YR, the number of days were calculated since planting to YR, and this trait was referred to days to maturity (DM). At this stage, small samples of spikes were harvested for GI analyses.

Crop height is classified as the distance between the upper boundary of the main photosynthetic tissues on a plant and the ground level (Cornelissen et al., 2003; Perez-Harguindeguy et al., 2013). Anthesis, or flowering, is the point at which crop height is considered to be at maximum. In our study plant height (PH) scoring was done after completing anthesis by measuring the mean height of the plants grown in a plot. We took a handful of spikes and take the average height of them from the top of the soil surface to the tip of the spike. Measuring of the height was done by using scale in centimeter (cm) unit.

The DH, DM and PH of the plant were determined in both Vollebekk 2016 and Chengdu 2016 trial, but not in the Chengdu 2015 trial. As the sowing date were almost similar for Chengdu 2016 and 2015 trials, an approximate DM was estimated for the RILs grown in Chengdu 2015 by comparing planting date and DM for Chengdu 2016.

2.3. Assessment of PHS

GI, FN and weather parameters were used to assess the PHS of the SHA3/CBRD x Naxos RILs wheat population. Percentage of germination was also done as an additional test to evaluate the GI method for the most dormant lines. To determine the dormancy level and interaction with different temperatures, GI analyses were also done for the parents grown at the three different temperature regimes in the green house at SKP (Senter for klimaregulert planteforskning), NMBU.

2.3.1. GI analyses from field trials

SD was evaluated based on the GI method. For GI analyses of the SHA3/CBRD x Naxos RILs population from the trial at Vollebekk in 2016, 10 spikes were harvested at YR by cutting with scissors. Spikes were dried in a drying chamber with ventilation system at 25°C for 3 days (72 hours) to ensure the moisture content in grain to be below 15%. The dried spikes were then placed in a freezer at temperature below -20°C in order to avoid loss of dormancy until the GI analyses were performed. After completing the harvesting of all the samples, the ears were threshed by hand and the grain samples were placed back into the freezer at -20°C until all samples were being threshed.

A preliminary experiment was conducted at the three different temperatures of 15°C, 17°C and 19°C in order to find the optimal germination temperature for GI analyses. Four wheat cultivars (Bjarne, T7347, Polkka, and

Saar) expected to vary in the level of dormancy were chosen for the preliminary experiment. 25 well developed seeds from each line were placed in a petri-dish having 9 cm diameters with a single layer of 70 mm filter paper and 5 ml of distilled water. Two replications of each grain sample were prepared for the three different temperature with a total of 24 petri-dishes. Two petri-dishes from each cultivar was taken and kept in incubators (Termaks, type KBP2324) at 15°C, 17°C and 19°C under 16 h light and 8 h darkness with a total of 8 petri-dishes for each temperature. Germinated seeds were counted and thereafter removed every day for a period of 14 days, and at the same time each day. Based on this trial experiment, 19°C was chosen as an appropriate temperature for the final germination experiment as it showed the best differentiation in GI compared to 15°C and 17°C.

For the final germination experiment, 25 well developed grains of each grain sample were laid out in petri-dishes as described for the preliminary experiment. The petri-dishes were placed in plastic boxes with a cover and the plastic boxes were placed in an incubator for 14 days at 19°C as described before. Germinated seeds were counted daily and then removed for a period of 14 days. Two replications of each sample were performed. GI was calculated using equation (1) described by Reddy et al (1985).

For GI analyses of the SHA3/CBRD x Naxos RILs from Chengdu 2015 and 2016 trials, the dried spikes were harvested around 35-40 days after anthesis and threshed by machine. Then the grains were placed in a freezer below -20°C in order to keep the samples away from the effect of environment until all samples were being threshed. 50 well developed grains of each grain sample were laid out in the petri-dish as described before. The germination temperature was set at 20°C. Germinated seeds were counted daily as described before. Two replications of each sample were performed. GI was calculated according to equation (1).

2.3.1.1. Percentage of germination (%)

Germination tests at low temperature were performed to test if fresh and un-sprouted grains, counted as dormant in the GI test, were viable. This was done for selected samples from the Vollebekk trial in 2016 having very low GI. It was determined by using pre-chilling (cold stratification) treatment. 25 seeds from each most dormant lines, 9 lines with lower GI, were placed in the petri-dishes as described for the GI experiment. Two replications of each grain sample were performed. The petri-dishes were placed in a plastic box with a cover and the plastic box was kept at 4°C in a refrigerator for seven days under darkness. Once the stratification was complete, the plastic box was removed from refrigerator and seeds were allowed to germinate in an incubator as described before for 19 days at 19°C. Germinated seeds were counted daily and removed until 19 days. Germination percentage was calculated using following formula:

*Germination percentage = (total no of germinated seeds*100)/total no of viable seeds* *equation 2*

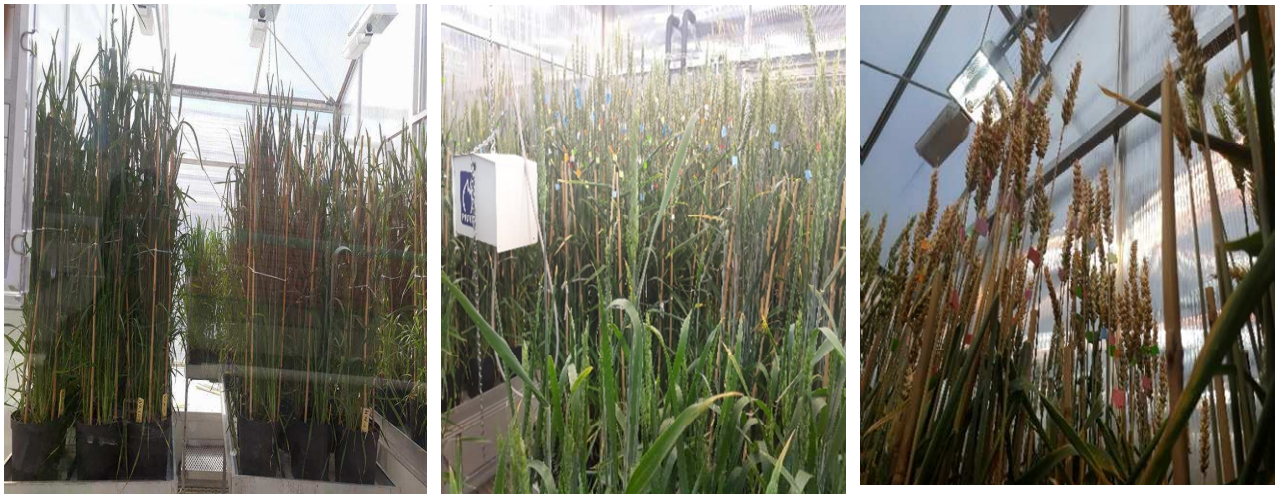
2.3.1.2. Experiment in greenhouse at three different temperature regimes

Plants were raised in three controlled climate chambers of the greenhouse at SKP, NMBU at optimal temperatures (15/10°C and 16 h day). 8 seeds for each cultivar were planted in a 3 L pot and thinned to 6 seedlings after emergence. Extra fertilization were given to each pot weekly from when the plants approached 15 cm. This was given as nutrient solution composed of 50% solution A and 50% solution B. Solution A was made of 25 parts calcium nitrate, one part ammonium nitrate, and 25 parts Kristalon (YARA, Inc) mixed with water to obtain ionic strength 1.5 m S cm^{-1}. Solution B was 25 parts Rød Superba, also mixed with water to 1.5 m S cm^{-1}. 2 - 3 dl were given per pot until the booting stage, and thereafter increased to approximately 5 dl. This was repeated until 2 weeks after anthesis (for the earliest varieties), which in total gave 10 applications. For one of the applications, 4 g of calcium nitrate was given per pot instead of the nutrient solution, as the plants at these stages had a bit light green color. Watering was done manually and optimized according to the plants requirements during their development. After anthesis was completed, the pots were re-organized and the three different temperatures were fixed for the three different climatic chambers. Two replications for each temperature regime were performed, and each replicate included 4 pots. Single ears were tagged at anthesis which made it possible to harvest ears at precise developmental stages by calculating developmental stage according to day-degrees after anthesis. The temperatures during day (16 h) and night (8 h) were as following:

Low temperature chamber with 15°C: day/night 17/12°C, mean temperature 15.33°C

High temperature chamber with 20°C: day/night 22/17°C, mean temperature 20.33°C

Very high temperature chamber with 25°C: day/night 27/22°C, mean temperature 25.33°C



a: growing parents in the greenhouse (Photo: Most Champa Begum)

b: anthesis stage (Photo: Most Champa Begum)

c: YR stage (Photo: Most Champa Begum)

Figure 2.1 (a-c): Different stages of growing parents (SHA3/CBRD and Naxos) in the greenhouse at SKP, NMBU in 2017.

2.3.1.3. GI analyses from greenhouse experiments for parents

For GI analyses of parents grown in the greenhouse at three different temperature regimes, 10 ears from each replicate were harvested at YR. The tagged ears were used to avoid tiller ears. The ear samples were placed into dryer with ventilation system at 25°C for 3 days (72 h) to ensure the moisture content in grain was below 15%. Then the dried ears were immediately placed in the freezer at -20°C to avoid loss of dormancy until the GI analyses were performed. After completing the harvesting of all the samples, the ears were threshed by hand, and 25 well filled grains of sound appearance were laid down for GI analyses as described before. Germination temperature was set at 20°C. Germinated seeds were counted daily and removed until 25 days. Two replications of each sample were performed. GI was calculated according to equation (1).

a: Hand threshing of wheat spikes (Photo: Most Champa Begum)

b: Germinated seeds in petri dish (Photo: Most Champa Begum)

c: Boxes in an incubator (Termaks, type KBP2324) (Photo: Most Champa Begum)

Figure 2.2 (a-c): Main steps of GI analyses

2.3.2. FN analyses

The field trial used for GI analyses was harvested with combiner and the samples were analyzed for FN. This field trial was conducted without mist-irrigation and is referred to as NMI. In addition, another and identical field trial was laid out, which were subjected to mist irrigation to provoke sprouting. This field trial is referred to as MI. For MI, the watering started on 11th of August, 108 days after sowing (42 days after completing heading for all the lines) and ended after about two weeks on 26th of August. When the watering was completed on 26th of August, all the lines had passed YR. However, there had been a lot of natural rain in the last few days before watering was stopped. The mist irrigation was programmed as follows: mist irrigation for 30 minutes at 16:30, 17:30, 19:00 and 20:30 on Fridays, Saturdays, Wednesdays and Thursdays. This irrigation gave about 7-11 mm (average 9 mm, but some variation in the field) rain during the day in the afternoons when the field was watered. The NMI trial was only subjected to natural weathering until harvest.

For FN analyses of the SHA3/CBRD x Naxos RILs population from Vollebekk 2016 trials, the grain samples were harvested with a combine harvester after YR on 30th and 31st of August from NMI trial and 14th of September from MI trial. Grains were dried in a drying chamber with ventilation system at room temperature to ensure the moisture content in grain to be below 15%. The grains were cleaned and milled by Perten 3100 FN mill (Perten Instruments AB, Huddinge, Sweden). Flour was kept at room temperature until FN analyses could be performed. For analyzing moisture content about 2 to 3 g of whole meal flour was placed in a moisture dish and heated in an oven (Termaks, type: T1056) at 105°C overnight. Afterwards, the samples were left to cool in a desiccator for 1 hour and the residue was weighed. The amount of weight loss due to heating is the moisture content of the sample, which was determined by the difference between the weight of the sample before and after heating, and expressed as percent moisture content. The moisture content of the flour was adjusted for 15% moisture content by using equation 3.

$$F = \frac{595}{100 - x} \qquad equation\ 3$$

Where,

F = flour, in g

x = moisture content of the flour

To analyze the FN, a suspension was made by mixing 7 g of whole meal flour with 25 ml distilled water and the FN values was measured by a quick gelatinization of flour suspension in a boiling water-bath under condition that is similar to those found during baking. FN analysis was performed according to AACC 56-81.03 (*AACC International*, 1999) by Perten 1700 FN instrument (Perten Instruments). Two replications per sample were run and the mean values were calculated for per sample. A sample that gave a difference of more than 20 seconds in a run was repeated again.

2.3.3. Weather data

Meteorological information about mean temperature, minimum (min) temperature, maximum (max) temperature, precipitation (PP) and relative humidity (RH) was collected for the period of 2 weeks before YR for the earlier genotypes to approximately 1 week after YR for the latest for Vollebekk 2016 and Chengdu 2016 trials. The similar weather data were collected for an approximate period of YR for the Chengdu 2015 trial. Mean, max and min temperature, accumulated PP and average RH were calculated for the periods of 0-7 days and 8-14 days before maturity by using Excel 2016.

2.3.4. Statistical analysis

Phenotypic data were analyzed by using SAS 9.4 (SAS Institute Inc., Cary, NC, USA). For GI of parents from the greenhouse experiment a two-way analysis of variance was performed using R v3.3.3 to determine effect of temperature and variety. Simple correlation coefficients were done using Minitab 18. The test was considered significant at P-value ≤ 0.05. Graphs were produced by Minitab 18 and Excel 2016.

2.3.5. Linkage Mapping

Linkage groups and genetic linkage map of 166 individuals from the SHA3/CBRD x Naxos RILs population were used according to Ruud, Windju, Belova, Friesen, and Lillemo (2017). Briefly, a total of 166 individuals from the SHA3/CBRD x Naxos RILs population were genotyped with the Illumina iSelect 90 K wheat SNP Chip (Wang et al., 2014). The genotype results were analyzed and scored manually using the Genome Studio Genotyping Module v1.0 software from Illumina. Linkage groups and genetic linkage maps were constructed by using markers scored as polymorphic. MST map was used to sort the markers into linkage groups (Wu, Bhat, Close, & Lonardi, 2008). The linkage groups were assigned to chromosomes based on the best BLASTn hit from a comparison of SNP flanking sequences with the Chinese Spring chromosome survey sequences (http://wheat-urgi.versailles.inra.fr/Seq-Repository). Previously developed SSR and DArT markers in the SHA3/CBRD x Naxos RILs population (Lu et al., 2012) were also added to the SNP marker data. Markers which are belonging to the linkage groups assigned to the same chromosomes were loaded into JoinMap v. 4.0 (Van Ooijen, 2006), and the linkage groups were refined using the maximum likelihood mapping algorithm. Recombination fractions were converted into map distances (centi-morgans) according to Kosambi mapping function to calculate genetic distances between markers with LOD significance threshold level 3.0 (Kosambi, 1943).

2.3.6. QTL analysis

To detect responsible QTL for PHS resistance in SHA3/CBRD x Naxos RILs population, interval mapping (IM) and multiple QTL model (MQM) mapping were performed by using the software MapQTL6 (Van Ooijen, 2011). At first, IM was conducted to detect possible major QTL for PHS resistance. Closely linked SNP markers to each QTL, observed in LOD profiles from IM, were used as cofactors in MQM mapping. The LOD significance threshold level was set to 3 after a permutation test with 1000 permutations. QTL with highest LOD score in the MQM mapping was reported when several peaks were observed above the threshold level for a trait in each chromosome. The additive effect and percentage (%) of phenotypic variance explained by each QTL were obtained from the MQM mapping. Genetic maps and LOD curves were drawn by using the software MapChart, v.2.3.2 (Voorrips, 2002).

Chapter 3

Results

3.1. Plant development and climatic conditions

In our study, grain filling and YR were followed from middle of July to end of August in 2016 for the NMI trial at Vollebekk, Ås, Norway. Figure 3.1 summarizes the temperature conditions and PP for the period of approximately two weeks before YR for the earliest genotypes to one week after YR of the latest genotypes. The date of YR varied from 2nd of August for the earliest genotypes (YRF) to 25th of August for the latest genotypes (YRL). A rainy period in the beginning of August was found in Ås with highest daily rainfall of 31.8 mm. Afterwards, the PP was quite low from middle of August to the end of YR on 25th of August (table 3.1 and figure 3.1). The total PP of the whole grain filling period (calculated from 20th of July to 3rd of September) was 160.8 mm (table 3.1) in 2016. Compared to the normal (1961 - 90, table 3.2) for Ås, the PP during grain filling in 2016 is close to the normal (total PP for July and August was 81 and 83 mm, respectively) (table 3.2). A wide fluctuation in daily temperature was seen, ranging from as low as below 5°C to above 25°C (figure 3.1 and table 3.1). Mean daily temperature was high for some days after 10th of July, thereafter it was quite stable around 15°C with two small drops around 9th and 27th of August (figure 3.1). However, the mean daily temperatures for this period are close to the normal (average 1961-90), which is 16.1°C in July and 14.9°C in August (table 3.2). RH varied from 58% to over 90% in the period of grain filling to YR (table 3.1 and figure 3.1).

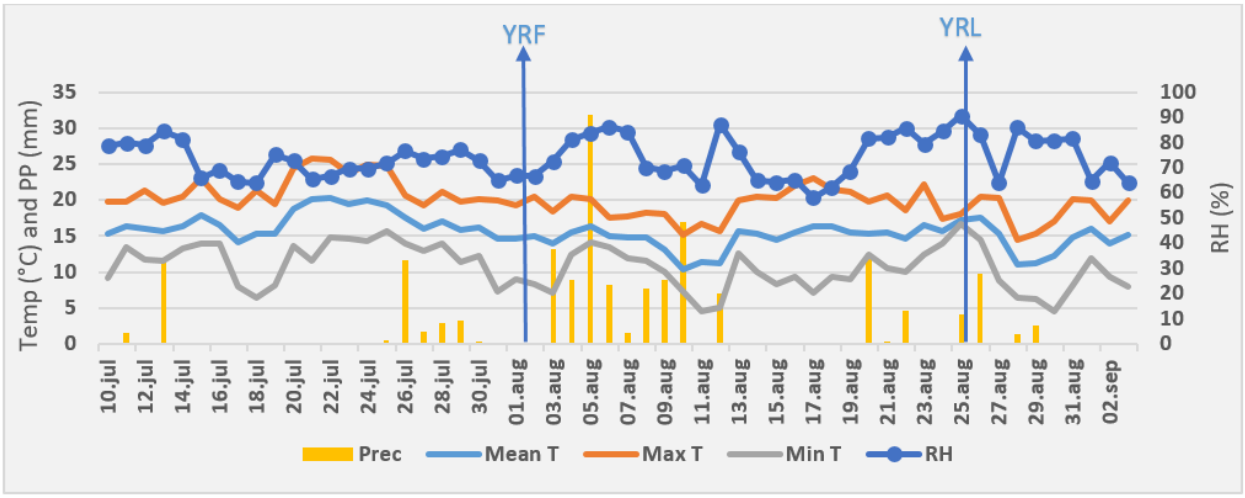


Figure 3.1: Weather conditions during grain filling period (two weeks before YR for the earlier genotypes to approximately one week after YR for the latest) in Ås in 2016 (Data source: Landbruks Meteorologisk Tjeneste (www.nibio.no)). Blue arrows show the date of YR for the earliest genotypes (YRF - yellow ripening first day, 2nd of August) and YR for the latest genotypes, (YRL - yellow ripening last day, 25th of August).

Average max temperature, min temperature, mean temperature, total PP and average percent RH were determined for 0-7 days (first week) and 8-14 days (second week) before YR for all genotypes in the experiment to get an overview of weather conditions during the time-periods associated with build-up of dormancy (table 3.1). There was a bit colder period with lowest mean temperature of 11.4°C during 8-14 days (second week) before YR than during 0-7 days (first week) before YR with lowest 13.3°C. However, there was not much variation in average max and min temperature, total PP and average percent RH during two weeks period before YR (table 3.1).

Table 3.1: Weather data recorded in Ås, Norway in growing period 2016 (Data source: Landbruks Meteorologisk Tjeneste (www.nibio.no)). Data in bold indicate the mean values and data in parenthesis indicate max and min values throughout the corresponding periods for all genotypes in the experiment.

Weather parameters	**Whole grain filling period 20th July – 3rd September**	**0-7 days before YR 26th July – 25th August**	**8-14 days before YR 19th July – 17th August**
Tmax (°C)	**19.5** (25.7, 14.5)	**16.2** (18.8, 13.9)	**16.3** (21.1, 14.9)
Tmin (°C)	**10.5** (15.6, 4.5)	**8.4** (10.5, 7.2)	**8.9** (12.1, 7.1)
Tmean (°C)	**15.2** (20.3, 10.3)	**13.9** (15.9, 13.3)	**12.6** (16.7, 11.4)
Total PP (mm)	**160.8** (31.8, 0)	**89.8** (9.8, 0.8)	**100.3** (10.6, 0.1)
RH (%)	**72.3** (90.6, 58.2)	**61.7** (61.1, 70.5)	**61.3** (68.3, 59.9)

Table 3.2: The monthly averages of mean daily temperature (°C) and total PP (mm) at Ås for 1961-1990 (Data source: Landbruks Meteorologisk Tjeneste (www.nibio.no)).

Months	**Mean daily temperature (°C)**	**Total PP (mm)**
April	4.1	39
May	10.3	60
June	14.8	68
July	16.1	81
August	14.9	83
September	10.6	90

For the field trial in Chengdu, China in 2016, the grain filling and YR was followed from middle of April to the end of May in 2016. The total PP of whole grain filling period (two weeks before YR for the earlier genotypes to YR for latest genotypes) was 100.5 mm (table 3.3) in 2016, with an highest daily rainfall of 44.3 mm on 6th of May (figure 3.2, table 3.3). Afterwards, there was almost no rain from middle of May to the end of YR (figure 3.2). However, the total PP was much lower in Chengdu 2016 compared to the Vollebekk 2016 (table 3.1 and 3.3). A wide fluctuation in daily temperature was seen, ranging from as low as 11.2°C to 33.4°C (figure 3.2 and table 3.3) which is much higher than Vollebekk 2016 (table 3.1). Mean daily temperature was quite stable around 20°C with two small fluctuations around 5th and 12th of May (figure 3.2). The difference of mean daily temperature was also obvious between the two locations, with approximately 5°C lower

temperatures at Vollebekk in 2016 than in Chengdu 2016 (table 3.1 and table 3.3). RH varied from 47% to 89% in the period of grain filling to YR (table 3.3 and figure 3.2), which is very similar to Vollebekk 2016 (table 3.1).

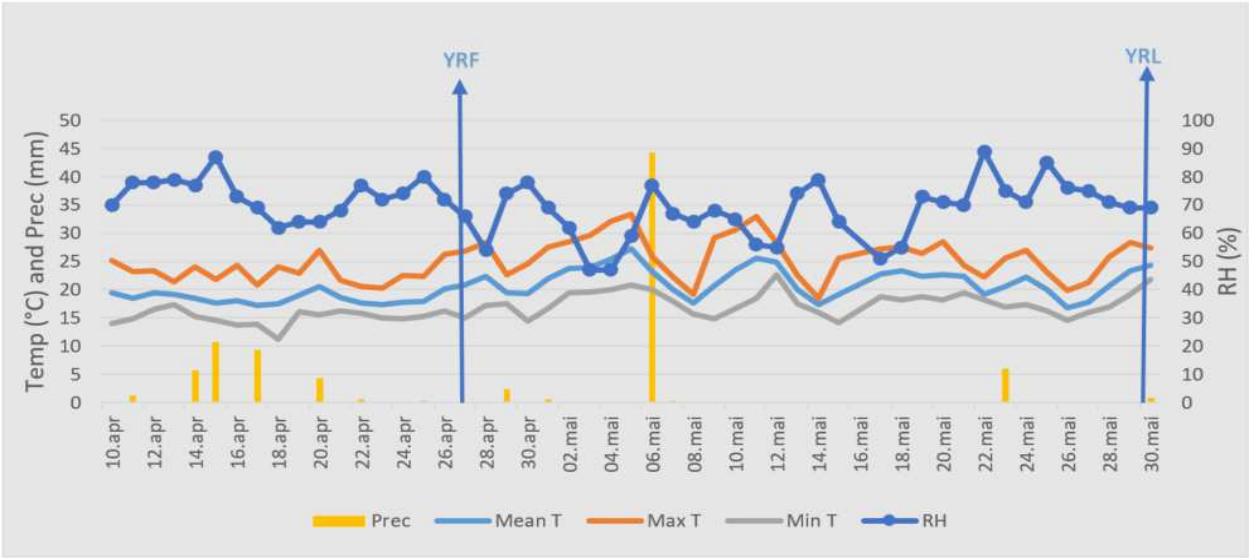


Figure 3.2: Weather conditions during grain filling period (two weeks before YR for the earlier genotypes to YR for latest genotypes) in Chengdu in 2016 (Data source: weather bureau in Xindu county). Blue arrows show the date of YR for the earliest genotypes (YRF - yellow ripening first day, 27th of April) and YR for the latest genotypes, (YRL - yellow ripening last day, 30th of May).

In addition, average max temperature, min temperature, mean temperature, total PP and average percent RH were determined for 0-7 days and 8-14 days before YR (table 3.3). There was a warmer period with lowest mean temperature around 18°C during both 8-14 days (second week) before YR and 0-7 days (first week) before YR compared to Vollebekk 2016 (table 3.3 and table 3.1). However, there was not much variation in max and min temperature, total PP and percent RH during two weeks period before YR in Chengdu 2016 (table 3.3).

Table 3.3: Weather data recorded in Chengdu, China in growing period 2016 (Data source: weather bureau in Xindu county). Data in bold indicate the mean values and data in parenthesis indicate max and min values throughout the corresponding periods for all lines in the experiment.

Weather parameters	**Whole grain filling period** **12th April – 31st May**	**0-7 days before YR** **20th April – 30th May**	**8-14 days before YR** **13th April – 22nd May**
Tmax (°C)	**25.2** (33.4, 18.5)	**24.5** (28.4, 23.4)	**23.8** (28.8, 22.5)
Tmin (°C)	**17.0** (22.6, 11.2)	**16.4** (18.6, 15.5)	**15.7** (18.7, 14.6)
Tmean (°C)	**20.7** (27.2, 16.8)	**20.1** (23.1, 18.9)	**19.4** (23.5, 18.1)
Total PP (mm)	**100.5** (44.3, 0)	**27.0** (5.7, 0.0)	**29.7** (6.4, 0.0)
RH (%)	**69.2** (89, 47)	**63.0** (76.5, 61.3)	**63.4** (73.0, 61.3)

For the field trial at Chengdu in 2015, the total PP of whole grain filling period was 108.5 mm (table 3.4) with a highest rainfall of 44 mm on 24th of May (figure 3.3, table 3.4). Overall, the whole grain filling period was quite dry similar to 2016 Chengdu field trial. Also, a wide fluctuation in daily temperature was seen ranging from as low as 10.9°C to 35.3°C (figure 3.3 and table 3.4). Mean daily temperature was quite stable around 22°C with a small drop around 18th to 24th of April (figure 3.3) which was much higher than both Vollebekk 2016 and Chengdu 2016 environment (table 3.4, 3.3 and 3.1). However, weather parameters were not calculated for 0-7 days and 8-14 days before YR as heading and YR dates were not recorded in Chengdu 2015 trial.

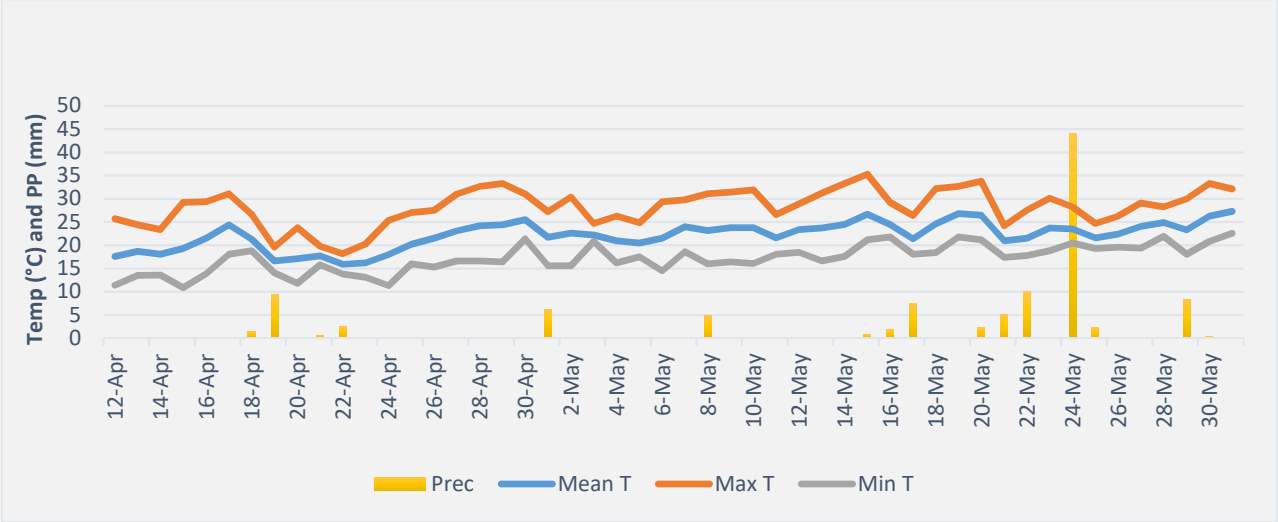


Figure 3.3: Weather conditions during approximate grain filling period in Chengdu in 2015 (Data source: weather bureau in Xindu county).

Table 3.4: Weather data recorded in Chengdu, China in growing period 2015 (Data source: weather bureau in Xindu county). Data in bold indicate the mean values and data in parenthesis indicate min and max values for all lines in the experiment.

Weather parameters	**Whole grain filling period** **12th April – 31st May**
Tmax (°C)	**28.2** (18.2, 35.3)
Tmin (°C)	**17.2** (10.9, 22.6)
Tmean (°C)	**22.2** (15.9, 27.3)
Total PP (mm)	**108.5** (0, 44)

3.2. Evaluation of PHS indices

3.2.1. Additional test to evaluate the GI method - percentage of germination results

Very low GI results can be obtained also if the seeds are not viable. To test whether this might be the case, standard germination tests for RILs from Vollebekk field trial in 2016 were performed at low temperature for

selected samples having very low GI. The results are shown in table 3.5, and showed that for the majority of the samples the germination percentage were high. A few samples, among them T7347, had germination percentage of 43 to 59%. In this test, no indications of dead seeds or fungal infestations was found. We interpret the results as the high dormancy of these samples caused the lower germination percentage, also at the low temperature used in these germination tests. The results do not show indications of low vitality of the seeds that could have given erroneous GI values.

Table 3.5. Percentage of germination of highly dormant SHA3/CBRD x Naxos RILs with GI

Rutes	Lines	Name	% Germination	GI
2124	SxN-159	SHA3/CBRD//NAXOS	83.67	0.28
2139	SxN-083	SHA3/CBRD//NAXOS	43.08	0.19
2229	1424	T7347	56.18	0.06
2312	SxN-018	SHA3/CBRD//NAXOS	94	0.31
2412	39	Naxos (x3)	95.83	0.24
2421	SxN-081	SHA3/CBRD//NAXOS	95.83	0.69
2429	SxN-025	SHA3/CBRD//NAXOS	98	0.24
2430	SxN-126	SHA3/CBRD//NAXOS	92.37	0.33
2626	1424	T7347	59.82	0.32

3.2.2. GI results

Analysis of variance (ANOVA) was conducted for the parents from the greenhouse experiment grown at three different temperature regimes, which shows a significant effects of both temperature, variety and G*E interaction on GI (table 3.6).

Table 3.6: ANOVA for GI of parents from greenhouse experiments in 2017 at SKP.

Source	DF	Sum Sq	F-value	Pr(>F)
Temperature	2	0.19	19.05	3.599e-05
Variety	1	0.07	13.19	0.0019051
Temperature:Variety	2	0.11	11.03	0.0007478
Residuals	18	0.09		

GI results showed higher dormancy levels at lower temperature (figure 3.4). Further, Naxos and SHA3/CBRD showed different responses to the temperature regimes: Naxos showed higher dormancy than SHA3/CBRD when temperature is low or moderate (17/12°C and 22/17°C) (figure 3.4). These results suggest that Naxos has higher dormancy than SHA3/CBRD when temperature is moderate to low during grain filling period. But, when the temperature is higher, the dormancy of Naxos is less expressed, which suggests that the dormancy level of Naxos is more dependent on temperature. In the field experiments, no differences in GI were found

between the parents neither at Vollebekk 2016 nor in Chengdu 2015, but again, we see that Naxos is more dormant in Chengdu 2016 than 2015.

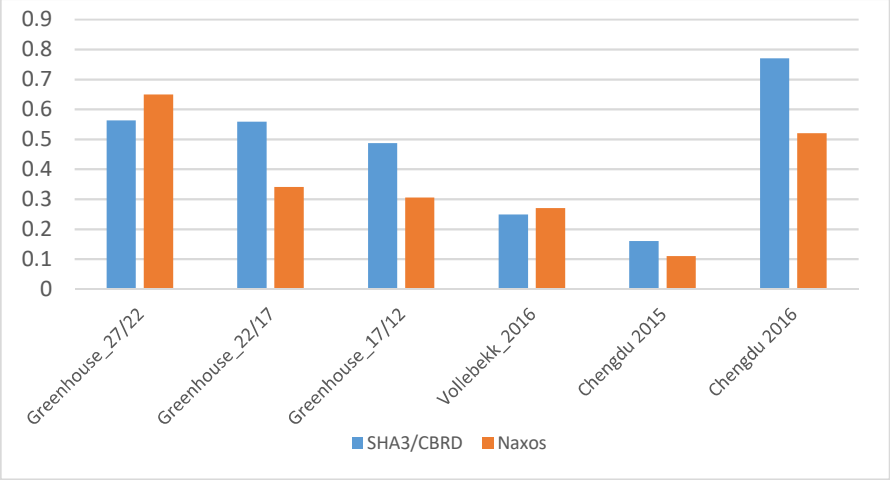


Figure 3.4. Comparison of mean GI of parents grown in different temperature regimes in 2017 in the greenhouse at SKP, NMBU and in different field with different environmental conditions: at Vollebekk research field in 2016, Norway, Xindu research field in 2015 and 2016, China. 27/22, 22/17 and 17/12 are the three different temperature regimes during grain filling period. The first one is the day temperature and the second one is the night temperature in °C.

Histograms for GI based on the individual RILs and parents are shown in figures 3.5, 3.6 and 3.7 for Vollebekk 2016, Chengdu 2016 and Chengdu 2015, respectively. The RILs grown at Chengdu in 2015 showed very high dormancy, and higher than RILs grown at Vollebekk in 2016. RILs grown at Chengdu in 2016 showed lower dormancy than the two other field trials. From the experiment at Vollebekk 2016, 11 progenies out of 169 (parents included), showed mean GI between 0 and 0.1 and only one progeny showed mean GI higher than 0.8 (0.85) (figure 3.5). The rest of the RILs were well differentiated between 0.1 and 0.6 (figure 3.5). In the Chengdu 2016 trial, three RILs had mean GI from 0.03 to 0.09 and 10 RILs had mean GI from 0.82 to 0.85 (figure 3.6a). Interestingly, even higher GI values were observed from Chengdu field trial in 2015. A total of 72 RILs showed mean GI from 0.0007 to 0.1 and only one RIL showed higher mean GI over 0.8 (0.84) (figure 3.7a).

Moreover, there were some RILs which had lower GI in at least two field trials, i.e., five RILs, out of 11 RILs with lowest GI from Vollebekk 2016 trial, had lower GI in both Vollebekk 2016 and Chengdu 2015. The line numbers of these RILs are SxN-066, SxN-108, SxN-141, SxN-169 and SxN-176 (table 3.7). In addition, three RILs with lower GI from Chengdu 2015 trial showed also lower GI in Chengdu 2016 trial. The number of those RILs are SxN-045, SxN-062 and SxN-072 (table 3.8). Among the RILs with higher GI above 0.8, only

one RIL, SxN-100, showed higher GI in both Chengdu 2015 and 2016 trials. The GI were 0.84 in 2015 and 0.80 in 2016 at Chengdu trials.

As the majority of RILs were not well differentiated for the field trials in Chengdu neither in 2016 nor 2015 (figure 3.6a and 3.7a), the GI results from these two trials were transformed into square ($Y=X^2$) and square root ($Y=\sqrt{x}$) (figure 3.6b and 3.7b) to get the transformed data better fitted to a normal distribution. By comparing transformed and original mean data for Chengdu 2016 and 2015, there were no change in ranking of lines in terms of lower and higher GI. In addition, there are 32 RILs which showed almost similar expression in at least two field trials. Among them, one RIL, SxN-005, showed about similar expression in all trials (table 3.9).

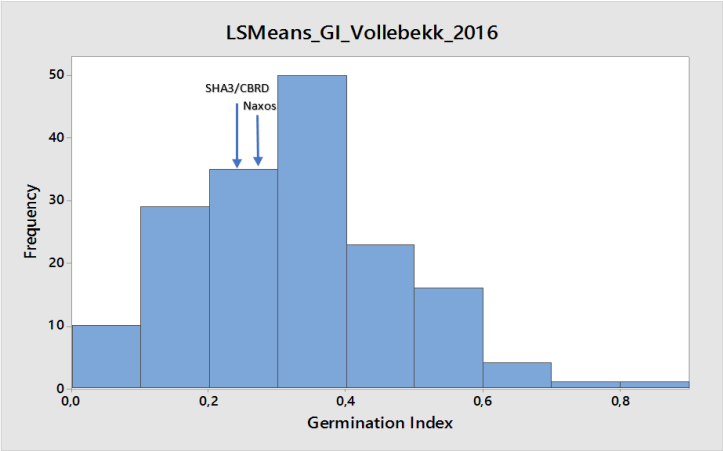
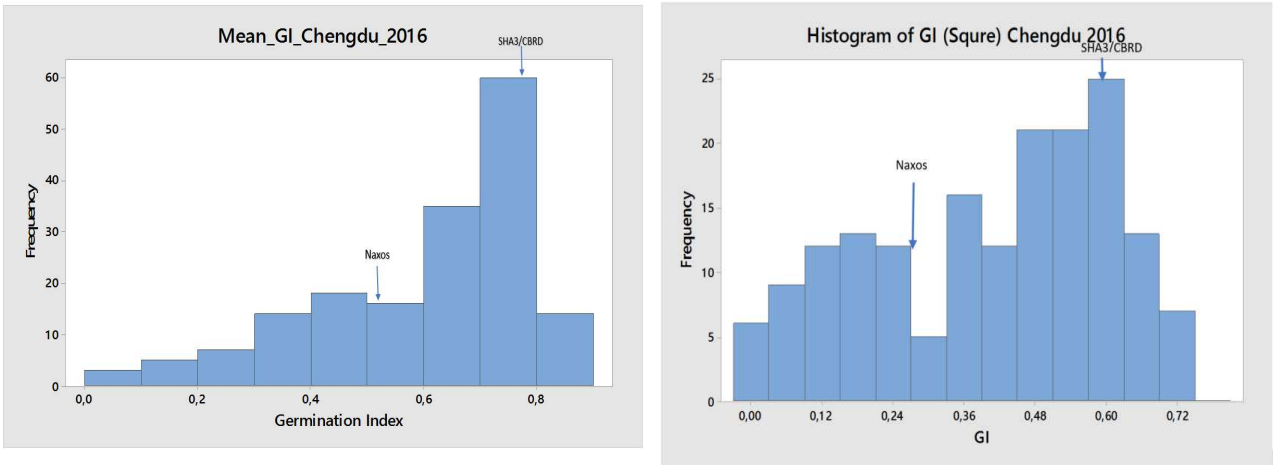
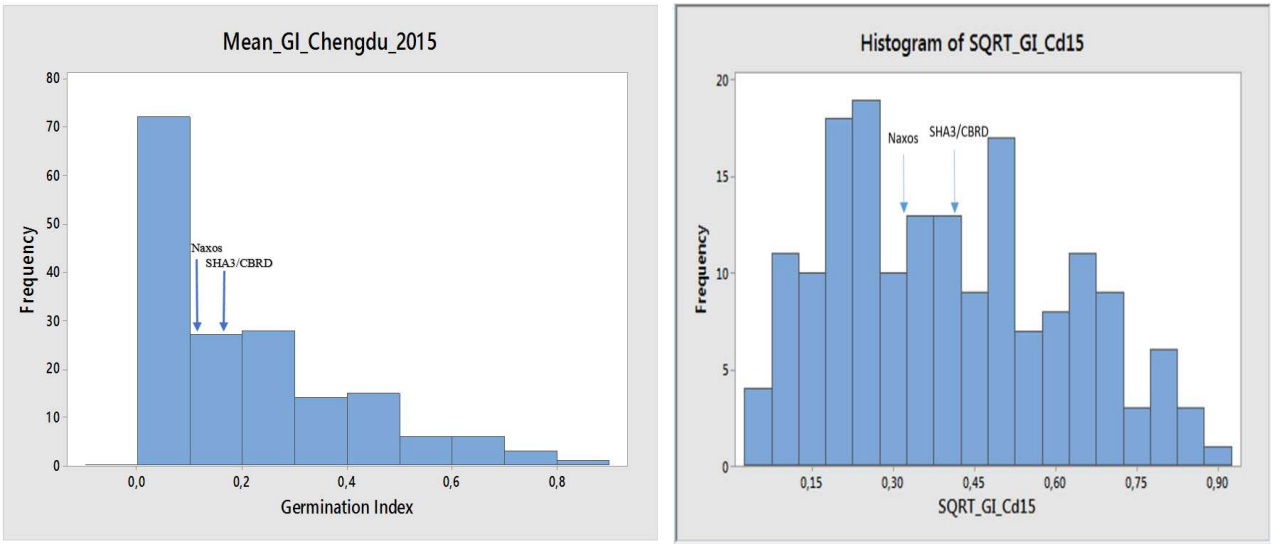


Figure 3.5. Histograms based on the LSMeans of GI of RILs from SHA3/CBRD x Naxos grown in 2016 at Vollebekk research farm, Ås, Norway. Arrows indicate the GI of parents: SHA3/CBRD and Naxos.



a b

Figure 3.6. Histograms based on the mean GI of RILs from SHA3/CBRD x Naxos grown in 2016 at Xindu research farm, Chengdu, China (a), and transformed data using square ($Y=X^2$) (b). Arrows indicate the GI of parents: SHA3/CBRD and Naxos.



a b

Figure 3.7. Histograms based on the mean GI of RILs from SHA3/CBRD x Naxos grown in 2015 at Xindu research farm, Chengdu, China (a), and transformed data using square root (Y=$\sqrt{x}$) (b). Arrows indicate the GI of parents: SHA3/CBRD and Naxos.

Table 3.7: RILs with lower GI from Vollebekk 2016 and Chengdu 2015 trials

Line	GI_Vollebekk_16	GI_Chengdu_2015	GI_Chengdu_SQRT_2015
SxN-066	0.02	0.04	0.20
SxN-108	0.06	0.06	0.25
SxN-141	0.00	0.04	0.20
SxN-169	0.04	0.00	0.04
SxN-176	0.09	0.06	0.24

Table 3.8: RILs with lower GI from Chengdu 2015 and 2016 trials

Line	GI_Chengdu 2016	GI_Chengdu_Square 2016	GI_Chengdu 2015	GI_Chengdu_SQRT 2015
SxN-045	0.09	0.01	0.01	0.10
SxN-062	0.08	0.01	0.00	0.03
SxN-072	0.03	0.00	0.06	0.25

Table 3.9: RILs with similar expression of GI in at least two trials

Lines	GI_Vl16	GI_Cd16	GI_Cd15	Lines	GI_Vl16	GI_Cd16	GI_Cd15
SxN-043	0.08	0.77	0.14	SxN-052	0.27	0.68	0.25
SxN-009	0.11	0.67	0.07	SxN-005	0.32	0.39	0.31
SxN-026	0.13	0.46	0.07	SxN-021	0.32	0.33	0.04
SxN-110	0.13	0.5	0.08	SxN-041	0.32	0.78	0.33
SxN-138	0.13	0.59	0.2	SxN-096	0.32	0.69	0.28
SxN-020	0.16	0.22	0.03	SxN-050	0.33	0.38	0.01
SxN-054	0.16	0.15	0.08	SxN-016	0.36	0.37	0.22
SxN-148	0.16	0.18	0.08	SxN-061	0.36	0.69	0.37
SxN-160	0.16	0.8	0.21	SxN-060	0.38	0.74	0.33
SxN-036	0.17	0.77	0.17	SxN-053	0.43	0.66	0.4
SxN-048	0.17	0.6	0.25	SxN-102	0.43	0.19	0.13
SxN-162	0.17	0.8	0.12	SxN-104	0.45	0.54	0.24
SxN-105	0.25	0.78	0.7	SxN-130	0.46	0.46	0.25
SxN-092	0.26	0.39	0.38	SxN-166	0.6	0.66	0.16
SxN-120	0.26	0.5	0.2	SxN-090	0.61	0.69	0.25
SxN-008	0.27	0.56	0.18	SxN-109	0.77	0.72	0.27

Non-significant differences in GI between parents were observed in Vollebekk 2016 (mean GI of SHA3/CBRD was 0.24 and Naxos was 0.27) (figure 3.4, 3.5 and table 3.10). Results from earlier experiments (unpublished, Dahal, 2012; unpublished, Sæbø, 2014) were collected to further explore differences in dormancy between the two parents, as non-consistent results were achieved in the greenhouse experiment and in the field. These parents were included in some earlier experiments to test dormancy by using the Dormancy Index (DI) method (Strand, 1965) in 2010, 2012, 2013 and 2014, and by using the GI method in 2010. All these experiments were done at Vollebekk and the results are summarized in table 3.10. High value of DI indicates more dormant genotype. From the previous DI results from 2010 to 2014, Naxos was the more dormant in 2013 and 2014, while SHA3/CBRD was the more dormant in 2010 and 2012. Thus, no consistent differences in DI could be found in these experiments (table 3.10). No differences were found in GI in 2010 between the parents either, and the results from the earlier field trials are thus consistent with the results obtained in 2016 at Vollebekk and in Chengdu 2015 (figure 3.7a). However, a significant difference was observed from the Chengdu 2016 field trial where Naxos had significantly lower mean GI than SHA3/CBRD, suggesting that Naxos was more dormant than SHA3/CBRD in this environment (figure 3.6a and b). This result is consistent with the greenhouse results when plants were grown in low (17/12°C) and moderate (22/17°C) temperatures (figure 3.4 and 3.6a, b).

Several check varieties were included in the field experiments at Vollebekk in 2016, and the GI results are compared to earlier testing of dormancy at Vollebekk (table 3.10). GI results of check varieties from Vollebekk

2016 field trial ranged from 0.19 to 0.46, and the ranking was consistent with previous results performed in 2015 at Vollebekk (table 3.10). These selected check varieties were known market varieties/lines to develop low, medium and high dormancy. T7347 showed very high dormancy for all the experiment years i.e., with the lowest GI in 2015 and the highest DI in 2013 and 2014 (table 3.10). Krabat showed medium to high DI. The ranking at Vollebekk experiment 2016 was according to these expectations.

Table 3.10. Comparisons of GI and DI for parents and check varieties from several years at Vollebekk field trial in Norway. GI data in 2010 are collected from Dahal's Master Thesis 2012 (unpublished). DI data from 2010 to 2014 and GI data for 2015 are collected from Sæbø's Master Thesis 2014 (unpublished).

Varieties	Contribution	GI_2016	GI_2015	GI_2010	DI_2014	DI_2013	DI_2012	DI_2010
Naxos	Parent 1	0.27	0.20	0.76	15.7	28.7	11.3	1.8
SHA3/CBRD	Parent 2	0.24	0.25	0.79	10.0	17.0	22.0	14.8
Saar	Check	0.44	0.41	0.86	12.0	0.0	5.3	2.5
Krabat	Check	0.36	0.35	0.84	12.0	30.6	21.9	17.8
Polkka	Check	0.46	-	-	1.5	15.8	12.5	-
T7347	Check	0.19	0.13	-	40.3	38.3	-	-

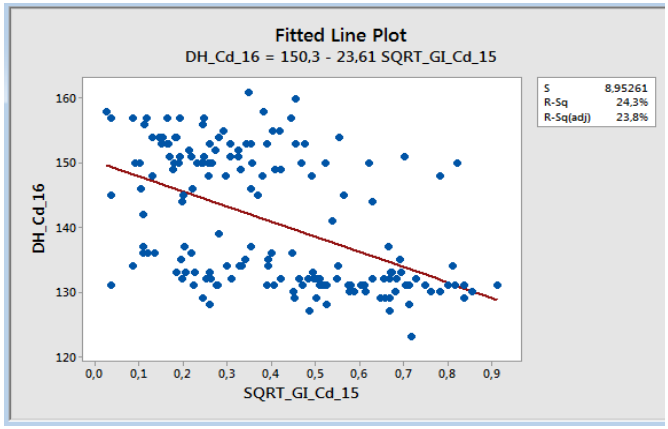
3.2.2.1. Relationship between GI and agronomic traits

The RILs, including parents varied in earliness, as verified by the date of YR, which occurred over a period of 25 days for the trial at Vollebekk in 2016 and at Chengdu in 2016. Variation in the weather, both prior to and after YR might affect both the build-up and the release of dormancy differently for progenies that vary in YR. This may have affected the results. To determine possible effects of weather parameters on PHS resistance on SHA3/CBRD x Naxos RILs, the weather data, the recorded DH, PH and DM from both locations and years, were analyzed by pairwise simple regression to find the possible relationship of GI and agronomic traits.

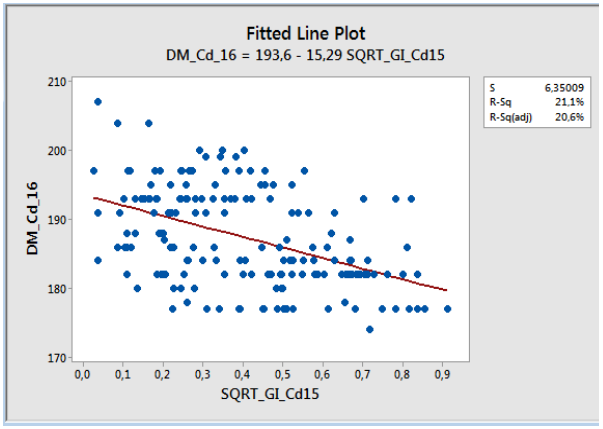
Results from these correlation analyses are shown in figure 3.8 and table 3.11. Statistically significant, but negative correlation was observed between DH from Chengdu field trial in 2016 and square root of mean GI from Chengdu field trial in 2015 (figure 3.8a and table 3.11) as well as DM from Chengdu field trial in 2016 and square root of mean GI from Chengdu field trial in 2015 (figure 3.8b and table 3.11). These two correlation results were also statistically significant when they were analyzed with mean GI. In addition, statistically significant, but positive correlation was observed between square root of mean GI from Chengdu 2015 and square of mean GI from Chengdu 2016 (figure 3.8 c and table 3.11). This correlation was also statistically significant when it was analyzed with mean GI from both fields (table 3.11). But, the transformed data gave a slight increased value of R^2. However, no statistically significant correlation was observed for GI from Vollebekk in 2016 with GI Chengdu from 2015 and 2016 (table 3.11).

Table 3.11: R^2 (%) values from regression analyses between GI and agronomic traits. Data in parentheses (green) and without parentheses (black) are negatively and positively correlated, respectively. Marked (*) data are statistically significant (P<0.05).

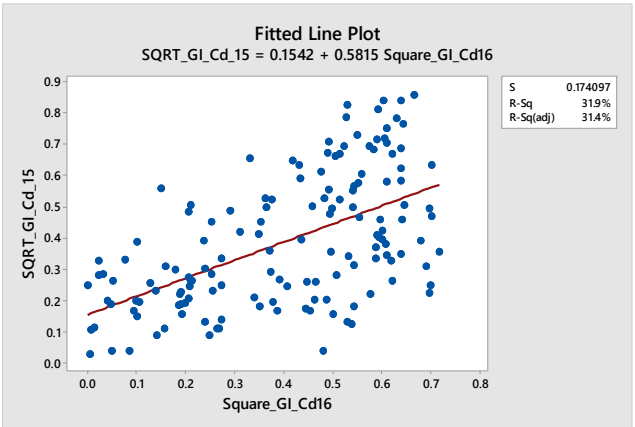
	GI_NMI_VL16	GI_Cd15	SQRT_GI_Cd15	GI_Cd16	Squre_GI_Cd16
DH_NMI_VL16	0.03	-	-	-	-
DM_NMI_VL16	1.01	0.1	-	0.3	-
PH_NMI_VL16	0.78	-	-	-	-
DH_Cd16	-	(22.8)*	(24.3)*	1.6	(1.21)
DM_Cd16	0.2	(20.3)*	(21.1)*	1.9	(1.51)
PH_Cd16	-	0.1	0.3	0.2	0.61
GI_NMI_VL16	-	0.08	0.02	0.00	0.00
GI_Cd15	-	-	-	25.56*	27.77*
SQRT_GI_Cd15	-	-	-	30.28*	31.92*



a



b



c

Figure 3.8: Regression plots of a) DH_Cd_16 (days to heading Chengdu 2016) vs SQRT_GI_Cd_15 (square root of mean GI Chengdu 2015); b) DM_Cd16 (days to maturity Chengdu 2016) vs SQRT_GI_Cd15 (square root of mean GI Chengdu 2015) and c) SQRT_GI_Cd15 (square root of mean GI Chengdu 2015) vs Square_GI_Cd16 (square of mean GI Chengdu 2016)

3.2.3. FN results

RILs from SHA3/CBRD x Naxos population, grown at Vollebekk in 2016 with MI and NMI to provoke sprouting, were analyzed for FN (figure 3.9 and 3.10). The average FN for all the RILs were 158 from MI trial and 254 from NMI trial. This suggests that the MI succeeded to provoke low FN. A total of 10 RILs, out of 147 RILs including parents, from the MI trial had FN higher than 250 (256-350) (figure 3.9), while eight RILs - out of 169 RILs including parents from the NMI trial had FN over 350 (356-397) (figure 3.10). On the other hand, 24 RILs from the MI trial showed FN less than 90, while only two RILs from the NMI trial had FN less than 90 (figure 3.9 and 3.10). Interestingly, 10 RILs, probably the less dormant, showed low FN also without mist-irrigation, and 14 RILs, probably the most dormant, showed higher FN also after mist-irrigation (table 3.12).

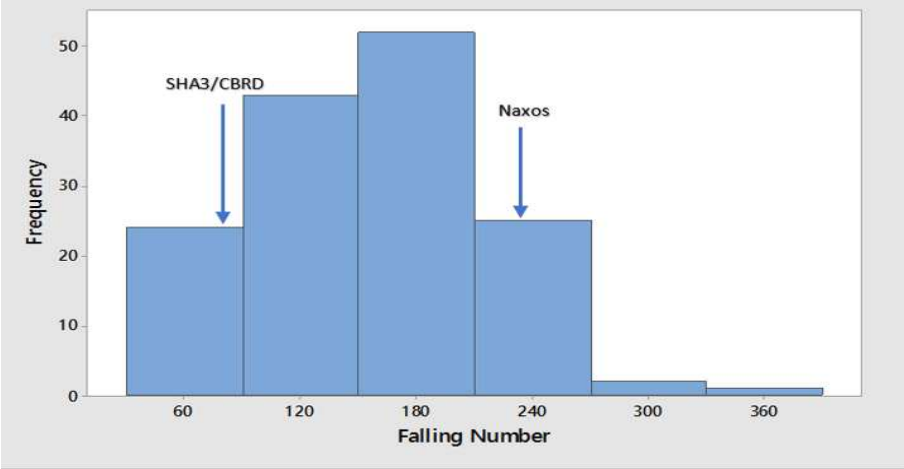


Figure 3.9. Histograms based on LSMeans of FN, from MI trial, of RILs from SHA3/CBRD x Naxos grown in 2016 at Vollebekk research farm, Ås, Norway. Arrows indicate the FN of parents: SHA3/CBRD and Naxos.

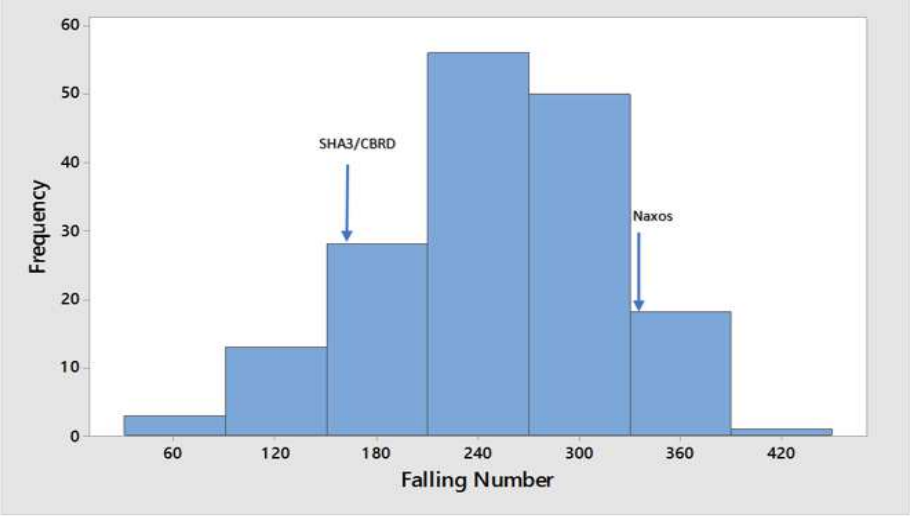


Figure 3.10. Histograms based on LSMeans of FN, from NMI trial, of RILs from SHA3/CBRD x Naxos grown in 2016 at Vollebekk research farm, Ås, Norway. Arrows indicate the FN of parents: SHA3/CBRD and Naxos.

Table 3.12: RILs with similar expression for FN in both NMI and MI trials at Vollebekk in 2016

Lines	FN_MI_Vl16	FN_NMI_Vl16	Lines	FN_MI_Vl16	FN_NMI_Vl16
SxN-095	61	63	SxN-117	206	248
SxN-164	66	66	SxN-056	225	259
SxN-035	86	94	SxN-175	227	234
SxN-126	93	110	SxN-147	231	283
SxN-129	104	134	SxN-123	232	253
SxN-048	148	166	SxN-066	234	262
SxN-020	148	128	SxN-122	242	256
SxN-171	157	187	SxN-039	250	220
SxN-131	160	161	SxN-157	256	269
SxN-120	175	193	SxN-099	267	299
SxN-110	201	247	SxN-017	300	297
SxN-080	203	265	SxN-159	350	341

In addition, significant differences of FN were observed between parents from both fields; for the MI trial, SHA3/CBRD had FN of 82 and Naxos had FN of 231 (figure 3.9 and table 3.13), and the corresponding FN for the NMI trial were 165 for SHA3/CBRD and 333 for Naxos (figure 3.10 and table 3.13). These results suggest that Naxos is more resistant to PHS compared to SHA3/CBRD. This is in agreement with previous FN tests which have been analyzed in 2015, 2010 and 2009 (table 3.13). Although well differentiation in FN of the RILs were obtained from both trials in 2016, the two cases gave different histograms, and as expected, the MI samples had lower FN.

FN results of check varieties from Vollebekk 2016 from the MI trial varied from 109 to 407, while for the NMI trial, the variation ranged from 223 to 365 (table 3.13). Higher FN were observed for T7347 for both 2016 and 2015. While, Saar, Krabat and Polkka showed lower FN than T7347 for 2016 and 2015 at Vollebekk and Staur (table 3.13).

Table 3.13. LSMeans of FN of parents and check varieties from both MI and NMI trial grown in several years at Vollebekk research farm, Ås, Norway. The data in 2010 are collected from Dahal's Master Thesis (unpublished, 2012) and the data in 2009 are collected from Tayor's Master Thesis (unpublished, 2010).

Varieties	Contribution	FN_2016_Vol_NMI	FN_2016_MI	FN_2015_Vol	FN_2010_Vol	FN_2009_Staur	FN_2009_Vol
Naxos	Parent 1	333	231	235	139	280	309
SHA3/CBRD	Parent 2	165	82	75	134	136	194
Saar	Check	223	109	62	145	118	272
Krabat	Check	354	222	275	193	382	391
Polkka	Check	359	122	157	-	-	-
T7347	Check	365	407	342	-	-	-

3.2.3.1. Relationship between FN and agronomic traits

As the samples for FN were harvested at the same time, early varieties might have received more rain between YR and harvest date, and could have been more subjected to PHS because of this. To determine possible effects of weather parameters on PHS resistance on SHA3/CBRD x Naxos RILs, pairwise correlation analyses were performed based on the weather data, the recorded DH, PH and DM from both NMI and MI fields at Vollebekk in 2016 to find the possible relationship of FN and agronomic traits.

Results from correlation analyses are shown in figure 3.11 and table 3.14. A statistically significant, but weak negative correlation was observed between DM and FN from NMI field at Vollebekk trial in 2016 (figure 3.11a and table 3.14). In addition, a weak statistically significant negative correlation was observed between GI from Chengdu field trial in 2015 and FN from MI field at Vollebekk in 2016 (figure 3.11b and table 3.14). This correlation was also significant when it was done with transformed (square root) data of mean GI from Chengdu 2015. But, the R^2 value was a bit higher for mean GI than transformed data. Moreover, a significant positive correlation was observed between FN from NMI and FN from MI trial at Vollebekk in 2016 (figure 3.11c and table 3.14). However, no statistically significant correlation was observed between GI from Vollebekk in 2016 with FN from both MI and NMI field at Vollebekk in 2016, as well as FN from NMI field at Vollebekk in 2016 with GI from Chengdu in 2016 and 2015 (table 3.14).

Table 3.14: R^2 (%) values from regression analysis between PHS traits. Data in parentheses (green) and without parentheses (black) are negatively and positively correlated, respectively. Marked (*) data are statistically significant (P<0.05).

	FN_NMI_VL16	FN_MI_VL16
DH_NMI_VL16	0.37	-
DM_NMI_VL16	(4.01)*	-
PH_NMI_VL16	0.41	-
DH_MI_VL16	-	2.42
PH_MI_VL16	-	0.80
GI_NMI_VL16	0.42	0.42
GI_Cd15	(0.32)	(3.58)*
SQRT_GI_Cd_15	0.21	(3.30)*
GI_Cd16	0.28	(0.37)
Squre_GI_Cd16	0.01	(0.91)
FN_NMI_VL16	-	39.36*

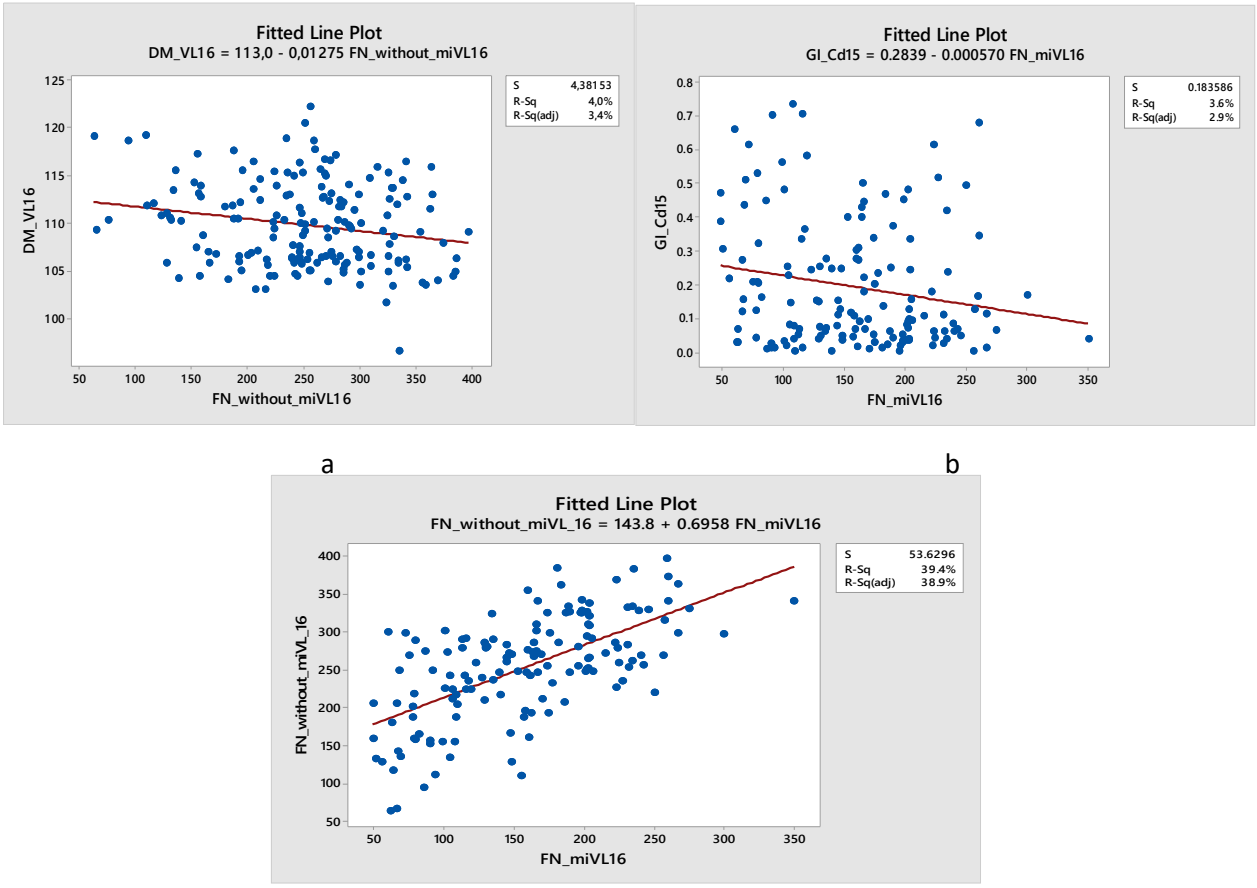


Figure 3.11: Regression plots of a) DM_VL16 (days to maturity NMI field Vollebekk 2016) vs FN_without_miVL16 (FN NMI field Vollebekk 2016), b) GI_Cd15 (mean GI Chengdu 2015) vs FN_miVL16 (FN MI field Vollebekk 2016), and c) FN_without_miVL16 (FN NMI field Vollebekk 2016) vs FN_miVL16 (FN MI field Vollebekk 2016)

3.3. QTL analysis results

Out of the 81,587 SNP chip from Illumina, 9230 SNP markers were scored as polymorphic which were sorted into 45 linkage groups. Among the scored polymorphic markers, 3512 SNPs were placed on the map after removing redundant markers. In addition, 224 SSR, DArT and gene-specific markers genotyped by Lu et al. (2012) were also placed on the map. The map spanned 3130 cM, covering all 21 chromosomes. QTL detected within 20 cM distances are considered as the same QTL.

In simple IM analysis, QTL for PHS resistance were detected on chromosome arms 1BL, 2AL, 3BL, 4AL, 4BL, 5AS, and 7BL in different environments (table 3.16). Closely linked SNP markers of detected QTL were chosen as cofactors for MQM analysis. After MQM analysis, major QTL, based on the percentage of explained phenotypic variance above 10, were detected on 1BL and 3BL and minor QTL, based on the percentage of explained phenotypic variance below 10, were detected on 2AL, 4AL, 4BL, 5AS and 7BL chromosome arms for PHS resistance (table 3.16). Whereas, for agronomic traits, major QTL were detected on 4BS, 5AL and 7DS as well as minor QTL on 1AL, 1BL, 2AL, 2DL, 2DS, 4AL and 7DS (table 3.15). QTL analyses were also done by using transformed data for Chengdu 2015 and 2016, but no new QTL were detected from these analyses.

3.3.1. QTL for agronomic traits

For agronomic traits, a total of 11 QTL were detected (table 3.15), of which three showed major effects: one QTL on 5AL for DH, explained from 11.7 to 21.1% of the phenotypic variance with LOD scores 6.9 and 11.3 and positions 180.3 and 178.9, was detected from the both NMI and MI field Vollebekk 2016, respectively. Among other two major QTL, one QTL for DM with position 25.2 on 7DS was detected from NMI field at Vollebekk in 2016 which explained 10.2% of the phenotypic variance with LOD score 6.6, which was also detected for DH from both NMI and MI field at Vollebekk in 2016. Another QTL with position 268.8 on 4BS for PH was detected from both NMI and MI field at Vollebekk in 2016 which explained 21.1% and 26.5% of the phenotypic variance with LOD scores 13.8 and 14.6, respectively. This QTL was also detected for the same trait from Chengdu 2016 trial in 278.6 position with LOD score 4.1 which explained 9.9% of the phenotypic variance (table 3.15).

Among minor QTL for agronomic traits, two QTL for DH were detected on 2AL, 2DS in Chengdu 2016 and one QTL on 4AL from MI trial in Vollebekk 2016. These QTL explained from 5.5 to 7% of the phenotypic variance, and none of them were common between the two locations. For DM trait, one QTL on 2DS was detected from Chengdu 2016, and three QTL on 1AL, 2DL and 4AL were detected from NMI trial at Vollebekk 2016. These QTL explained from 6.3 to 9.4% of the phenotypic variance, and none of them were common between the two locations. For PH trait, one QTL was detected on 4BS in Chengdu 2016, one QTL on 1AL

from MI trial in Vollebekk 2016, and two QTL on 1BL and 4AL from NMI trial in Vollebekk 2016. These QTL explained from 4 to 9.9% of the phenotypic variance, and one of them, located on 4BS, was common between the two locations at Vollebekk 2016 in both MI and NMI trial, and Chengdu 2016 which was detected as a major QTL from Vollebekk 2016 (table 3.15).

However, among all the QTL detected for agronomic traits five QTL were overlapping (located within 20 cM distances). One QTL on 2DS was detected for both DH and DM from Chengdu 2016 field trial which explained 5.8% and 9.4% of the phenotypic variance with LOD scores 2.5 and 4.1, respectively. QTL detected on 4AL for DM from NMI trial at Vollebekk 2016 was also detected for PH from the same field trial. A QTL on 4BS for PH was detected from both NMI and MI field in 2016 at Vollebekk trial as well as at Chengdu 2016 trial (table 3.15). QTL detected on 7DS for DH from both NMI and MI field Vollebekk 2016 which explained 5.2 to 9% of the phenotypic variance with LOD score 3.2 to 5.1. This minor QTL was also detected as a major QTL for DM trait from the NMI field.

Table 3.15. Results of QTL mapping (MQM mapping) in the RIL population of SHA3/CBRD x Naxos for agronomic traits.

Traits	Chromosome arm[a]	Marker(s)-Locus	Position (cM)	LOD[b]	%Expl.[c]	Additive
DH _Chengdu 2016	2AL	*wmc658*	274.9	2.4	5.5	-2.52
	2DS	*Ku_c42623_88*	64.1	2.5	5.8	-2.71
DH_NMI field - Vollebekk 2016	***5AL***	***Ex_c24587_139***	***180.3***	***6.9***	***11.7***	***0.76***
	7DS	*Exc_c22419_460*	25.2	3.2	5.2	0.47
DH_ MI field - Vollebekk 2016	4AL	*Td_con97425_92*	101.3	4.0	7	-0.52
	5AL	***Excrc111129_125***	***178.9***	***11.3***	***21.1***	***1.02***
	7DS	*D_GDS7LZN02FSYZC_227*	25.3	5.1	9	0.60
DM_ Chengdu 2016	2DS	*Ku_c42623_838*	64.1	4.1	9.4	-2.36
DM_ NMI field - Vollebekk 2016	1AL	*TA002362-0785*	190.9	4.2	6.3	1.24
	2DL	*wmc41*	124.2	4.3	6.3	-1.24
	4AL	*BS00022932_51*	35.7	5.8	8.8	-1.48
	7DS	***Exc_c22419_460***	***25.2***	***6.6***	***10.2***	***1.70***
PH_ Chengdu 2016	4BS	*Td_con64772_417*	278.6	4.1	9.9	-4.76
PH_NMI field - Vollebekk 2016	1BL	*K_c8592_935*	115.5	3.1	4	-2.49
	4AL	*wPt-0610*	31.2	3.5	4.7	2.44
	4BS	***Rht-B1b***	***268.8***	***13.8***	***21.1***	***-5.31***
PH_MI field - Vollebekk 2016	1AL	*Bwc6820_199*	225.9	3.9	5.9	2.84
	4BS	***Rht-B1b***	***268.8***	***14.6***	***26.5***	***-5.94***

Major QTL are highlighted in bold italics. Overlapping QTL are shaded with the same color (located within 20 cM distances).

a Based on Somers, Isaac, & Edwards (2004); S: short arm; L: long arm
b Maximum likelihood LOD score for the QTL
c Phenotypic variation explained by the QTL

3.3.2. QTL for PHS traits

A total of seven QTL were detected for PHS traits (table 3.16). Two QTL, on 3BL and 1BL showed major effects and were detected in more than one field trial. The QTL on 3BL for GI with resistance from SHA3/CBRD was detected from both Chengdu 2015 and NMI trial at Vollebekk 2016, and explained from 8.5 to 10.8% of the phenotypic variance with LOD scores from 4.0 to 5.4. The QTL responsible for GI on 3BL was detected on positions 21.3 and 36.0 for Chengdu 2015 and NMI trial at Vollebekk 2016, respectively.

The QTL on 1BL for FN with resistance from Naxos was detected from both NMI and MI field Vollebekk 2016, and explained from 11.6 to 12% of the phenotypic variance with LOD score 5.7. The positions of detected QTL for FN trait from both NMI and MI trial at Vollebekk in 2016 are 115.3 and 115.1 on 1BL chromosome arm, respectively (table 3.16).

In addition, five minor QTL were detected for PHS traits: for GI, two QTL on 2AL and 4BL were detected from Chengdu 2015 trial and one QTL on 4AL was detected form Chengdu 2016 trial. Moreover, two QTL for FN on 5AS and 7BL were detected from MI trial at Vollebekk 2016 (table 3.16). However, the source of dormancy was Naxos for all FN QTL, while for GI, the source of dormancy was SHA3/CBRD except one QTL detected on 4BL with Naxos as the source of dormancy.

Table 3.16. Results of QTL mapping (MQM mapping) in the RIL population of SHA3/CBRD x Naxos for PHS traits.

Traits	Chromosome arm[a]	Marker(s)-Locus	Position	LOD[b]	%Expl.[c]	Additive	Source of dormancy
Mean GI - Chengdu 2015	2AL	*wEx_c3808_6925015*	207.6	4.9	8.6	-0.07	SHA3/CBRD
	3BL	***Bwc3721_224***	***21.3***	***5.4***	***10.8***	***-0.07***	***SHA3/CBRD***
	4BL	*wEx_c4148_7495656*	44.4	4.5	8.8	0.06	Naxos
Square root of mean GI - Chengdu 2015	2AL	*wEx_c3808_6925015*	207.6	3.7	6	-0.06	SHA3/CBRD
	3BL	*Bwc3721_224*	21.3	5.3	9.6	-0.07	SHA3/CBRD
	4BL	*Exc_c57603_523*	42.9	4.0	7.2	0.06	Naxos
Mean GI - Chengdu 2016	4AL	*GENE-0689_30*	41.5	3.1	6.2	-0.05	SHA3/CBRD
Square of mean GI - Chengdu 2016	4AL	*GENE-0689_30*	41.5	2.7	5.3	-0.05	
GI_NMI - Vollebekk 2016	3BL	*Excrc102300_102*	36.0	4.0	8.5	-0.05	SHA3/CBRD
FN_NMI field - Vollebekk 2016	***1BL***	***Exc_c55363_265***	***115.3***	***5.7***	***12***	***-25.85***	***Naxos***
FN_MI field - Vollebekk 2016	***1BL***	***BS00089514_51***	***115.1***	***5.7***	***11.6***	***-25.06***	***Naxos***
	5AS	*K_c17126_1211*	13.2	3.1	5.4	-17.50	Naxos
	7BL	*BS00064368_51*	74.4	3.9	6.7	-18.05	Naxos

Major QTL are highlighted in bold italics. Overlapping QTL are shaded with the same color (located within 20 cM distances).

a Based on Somers et al. (2004); S: short arm; L: long arm
b Maximum likelihood LOD score for the QTL
c Phenotypic variation explained by the QTL

Interestingly, one QTL detected for FN on 1BL was also detected for PH from NMI trial at Vollebekk 2016. In addition, one QTL detected for GI on 4AL was also detected for DM and PH (located within 20 cM distances) from NMI trial at Vollebekk 2016. However, other QTL for FN and GI were not detected for any PHS and agronomic traits.

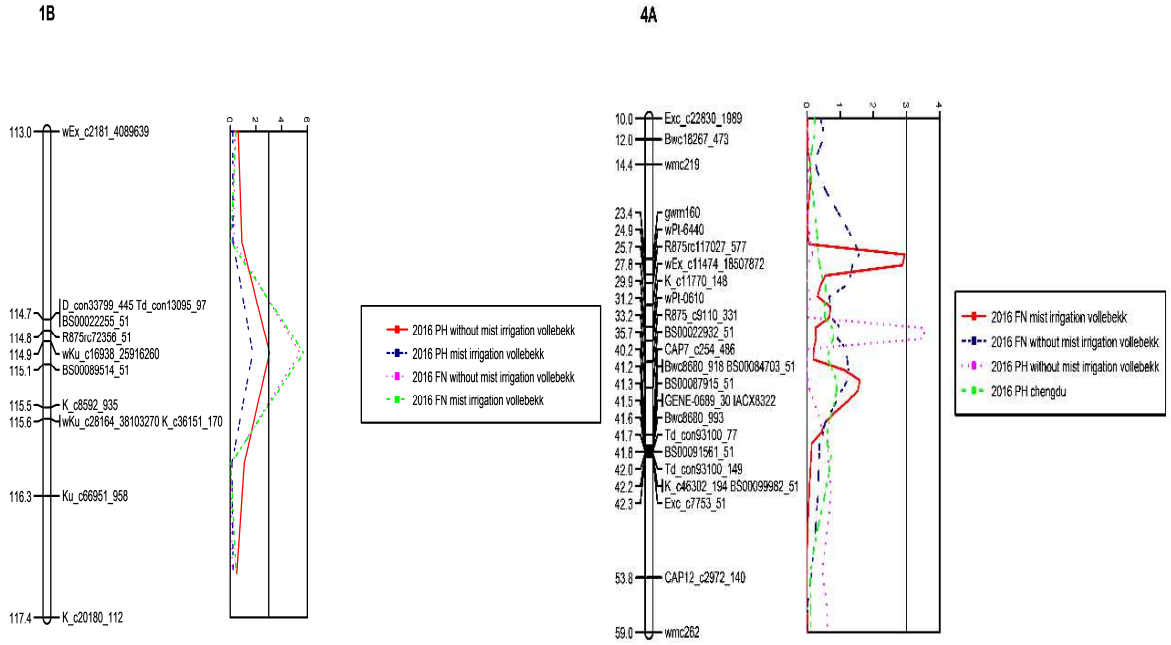
3.3.3. Genetic relationship between traits

LOD curves were drawn for detected QTL to find genetic relationship between agronomic and PHS traits as well as between PHS traits.

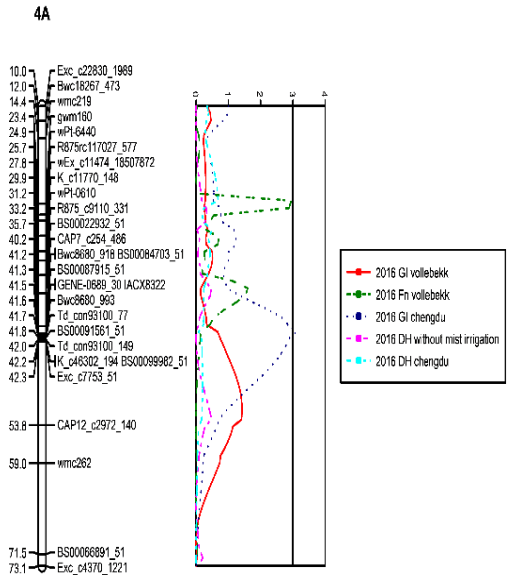
3.3.3.1. Genetic relationship between agronomic and PHS traits

On chromosome 1BL, one major QTL was detected for FN which is flanked by *wKu_c16938_25916260* and *K_c8592_935* markers. Interestingly, this flanking region also included QTL for PH which was detected from both NMI and MI trials at Vollebekk in 2016 (figure 3.12 a and table 3.15, 3.16).

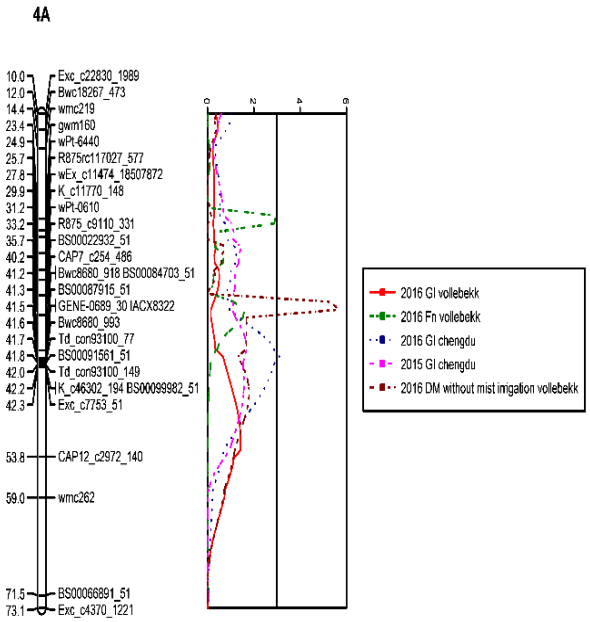
On chromosome arm 4AL, one QTL responsible for PH and DM was detected from NMI trial at Vollebekk in 2016. LOD curves of this region indicate that, another QTL could be responsible for the effect on FN on the



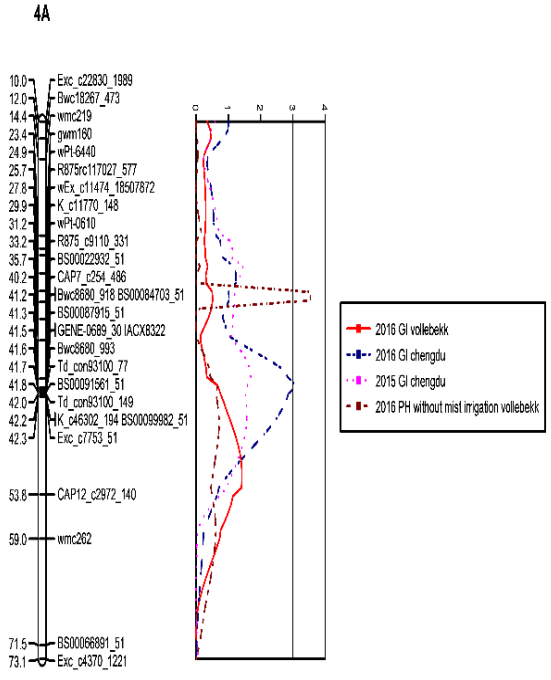
a b


4A
10.0 Exc_c22830_1989
12.0 Bwc18267_473
14.4 wmc219
23.4 gwm160
24.9 wPt-6440
25.7 R875rc117027_577
27.8 wEx_c11474_18507872
29.9 K_c11770_148
31.2 wPt-0610
33.2 R875_c9110_331
35.7 BS00022932_51
40.2 CAP7_c254_486
41.2 Bwc8680_918 BS00084703_51
41.3 BS00087915_51
41.5 GENE-0689_30 IACX8322
41.6 Bwc8680_993
41.7 Td_con93100_77
41.8 BS00091561_51
42.0 Td_con93100_149
42.2 K_c46302_194 BS00099982_51
42.3 Exc_c7753_51
53.8 CAP12_c2972_140
59.0 wmc262
71.5 BS00066891_51
73.1 Exc_c4370_1221
2016 GI vollebekk
2016 Fn vollebekk
2016 GI chengdu
2016 DH without mist irrigation
2016 DH chengdu

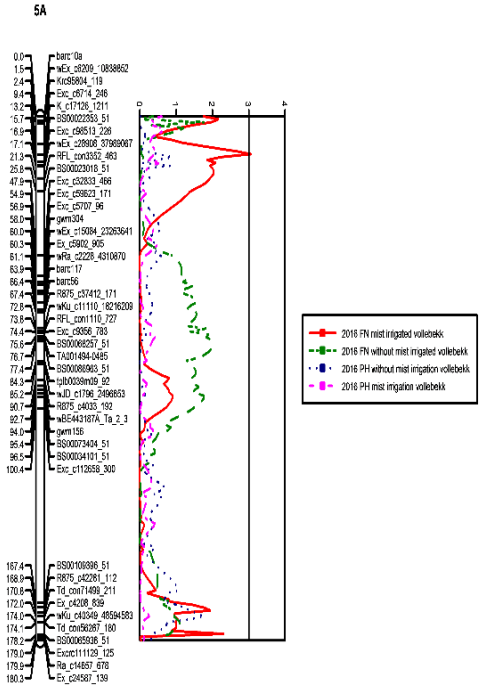
c


4A
10.0 Exc_c22830_1989
12.0 Bwc18267_473
14.4 wmc219
23.4 gwm160
24.9 wPt-6440
25.7 R875rc117027_577
27.8 wEx_c11474_18507872
29.9 K_c11770_148
31.2 wPt-0610
33.2 R875_c9110_331
35.7 BS00022932_51
40.2 CAP7_c254_486
41.2 Bwc8680_918 BS00084703_51
41.3 BS00087915_51
41.5 GENE-0689_30 IACX8322
41.6 Bwc8680_993
41.7 Td_con93100_77
41.8 BS00091561_51
42.0 Td_con93100_149
42.2 K_c46302_194 BS00099982_51
42.3 Exc_c7753_51
53.8 CAP12_c2972_140
59.0 wmc262
71.5 BS00066891_51
73.1 Exc_c4370_1221
2016 GI vollebekk
2016 Fn vollebekk
2016 GI chengdu
2015 GI chengdu
2016 DM without mist irrigation vollebekk

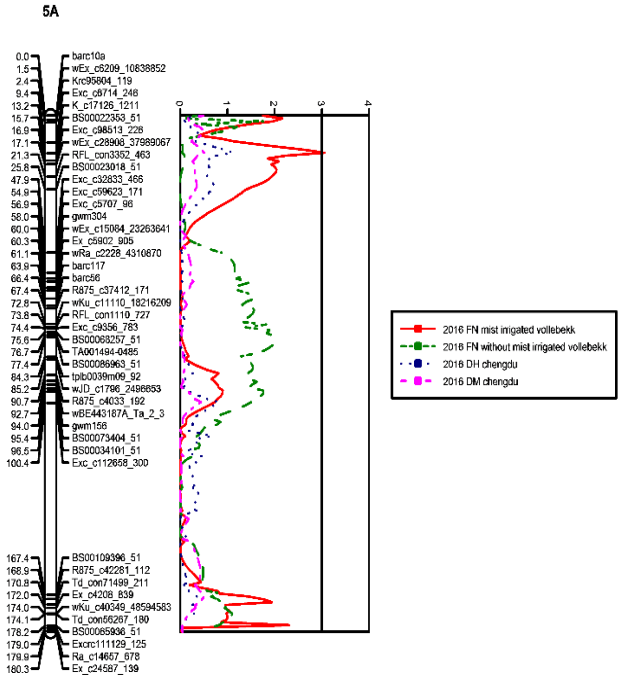
d


4A
10.0 Exc_c22830_1989
12.0 Bwc18267_473
14.4 wmc219
23.4 gwm160
24.9 wPt-6440
25.7 R875rc117027_577
27.8 wEx_c11474_18507872
29.9 K_c11770_148
31.2 wPt-0610
33.2 R875_c9110_331
35.7 BS00022932_51
40.2 CAP7_c254_486
41.2 Bwc8680_918 BS00084703_51
41.3 BS00087915_51
41.5 GENE-0689_30 IACX8322
41.6 Bwc8680_993
41.7 Td_con93100_77
41.8 BS00091561_51
42.0 Td_con93100_149
42.2 K_c46302_194 BS00099982_51
42.3 Exc_c7753_51
53.8 CAP12_c2972_140
59.0 wmc262
71.5 BS00066891_51
73.1 Exc_c4370_1221
2016 GI vollebekk
2016 GI chengdu
2015 GI chengdu
2016 PH without mist irrigation vollebekk

e


5A
2016 FN mist irrigated vollebekk
2016 FN without mist irrigated vollebekk
2016 PH without mist irrigation vollebekk
2016 PH mist irrigation vollebekk

f



g

Figure 3.12 (a-g): Segments of chromosomes with resistance QTL detected from SHA3/CBRD x Naxos RIL population. The LOD curves were obtained from MQM results. Genetic distances are shown in cM (left of the chromosomes). The threshold level is 3.

4AL chromosome arm (figure 3.12 b, d and table 3.15). Other indications of this region containing QTL for GI, FN, DM and PH can be seen from the LOD curves in figures 3.12 c, d and e, although LOD scores were below threshold level for all the PHS traits.

Moreover, LOD curves of chromosome 5A indicate that it contains several QTL for FN, PH, DH and DM (figure 3.12 f and g).

3.3.3.2. Genetic relationship between PHS traits

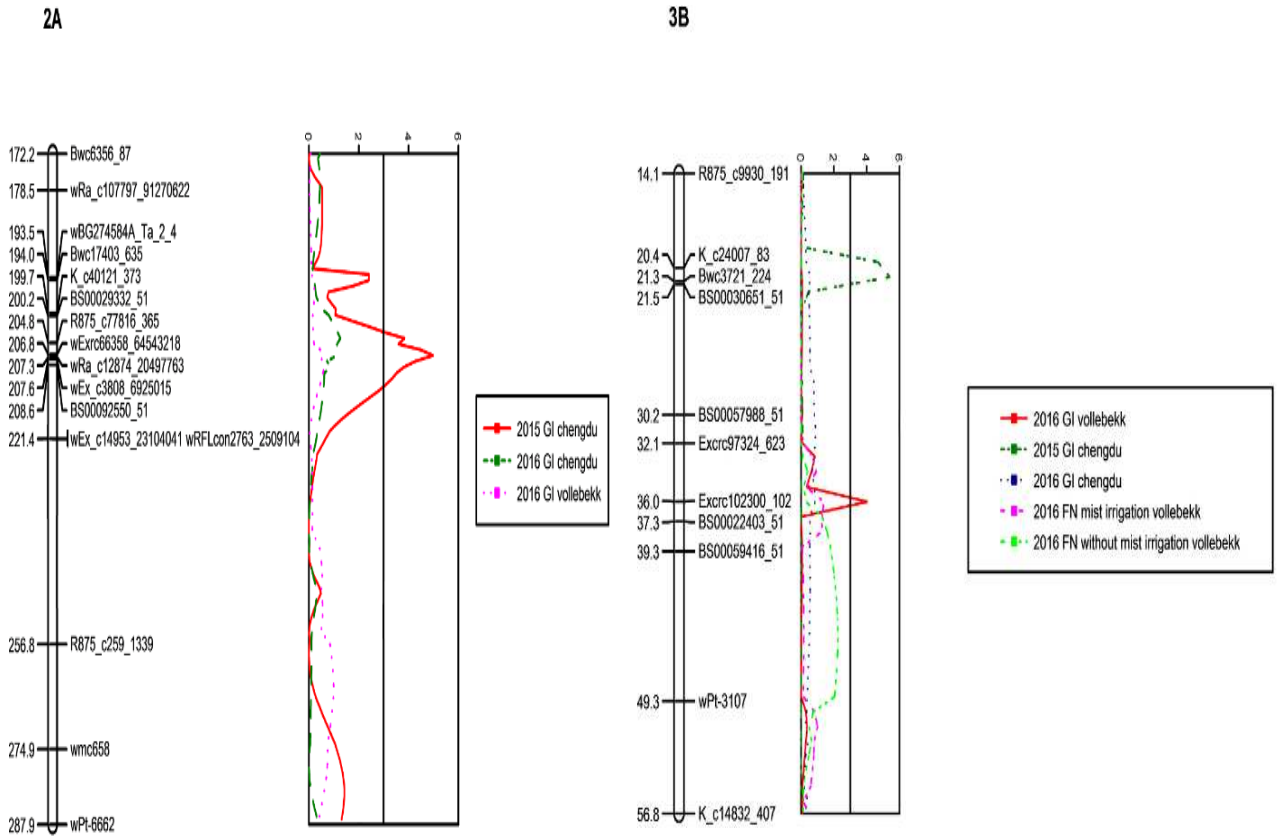
QTL on 2AL chromosome arm detected for GI from Chengdu trial 2015 is flanked by *wRa_c12874_20497763* and *BS00092550_51* markers which also showed some effect in Chengdu and Vollebekk in 2016 although the LOD score were below threshold level (figure 3.13a).

On chromosome arm 3BL, one QTL detected for GI from Chengdu field trial in 2015 was detected on position 21.3 and flanked by *K_c24007_83* and *BS00030651_51* markers. While, the position of the QTL detected from NMI trial at Vollebekk in 2016 is 36.0 and flanked by *Excrc97324_623* and *BS00022403_51* markers. From the LOD curves, it is confirmed that QTL for FN also could be responsible for PHS resistance from 36.0 position (figure 3.13b).

On chromosome arm 4BL, one QTL responsible for GI was detected on 42.9 and 44.4 positions which is flanked by *wPt-4243* and *Krc101259_159* markers. No QTL for PH was detected in this arm (figure 3.13c).

For FN one QTL was detected on 74.4 position of 7BL chromosome arm which is flanked by *Td_con5360_379 Td_5360_95* and *Td_con47317_100* markers (figure 3.13d).

On 4AL, one QTL for GI was detected from Chengdu 2016 trial and the position of this QTL is 41.5. However, no significant QTL was detected for FN on this chromosome arm (figure 3.13e).



a b

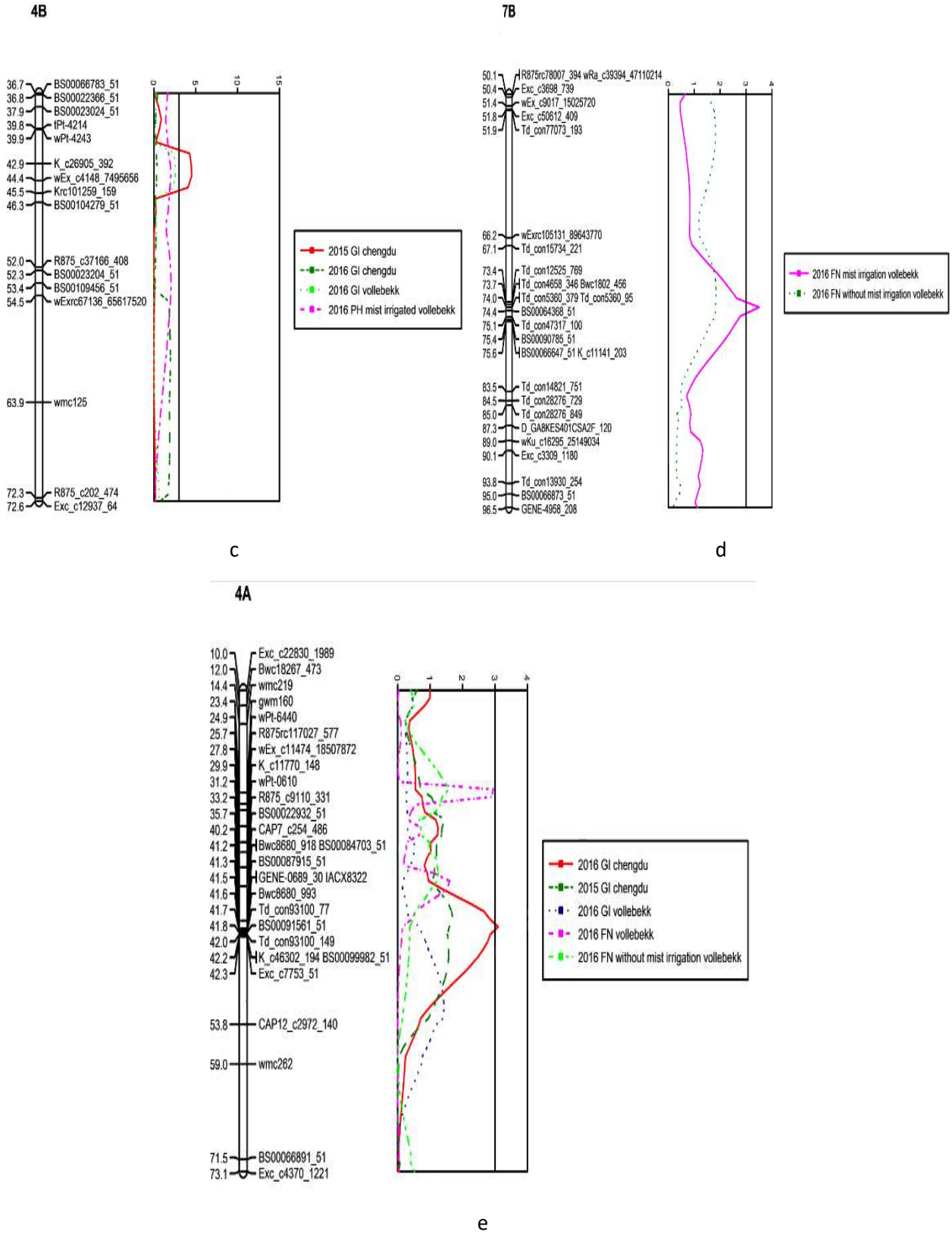


Figure 3.13 (a-e): Segments of chromosomes with resistance QTL detected from SHA3/CBRD x Naxos population. The LOD curves were obtained from MQM results. Genetic distances are shown in cM (left of the chromosomes). The threshold level is 3.

Chapter 4

Discussion

Although, a FN of 200 or above is considered acceptable for delivery of wheat grain to industry for bread making in Norway, recently many export markets require minimum tolerances of 300 to 350 seconds (German, 2006). Previous study reported that the lower the value of GI trait, the longer the SD period and, consequently, the better PHS resistance (Hagemann & Ciha, 1984). It is customary to refer lines whose GI does not exceed 0.1 - 0.2 to highly resistant to PHS (Biddulph et al., 2008). Interestingly, there were 41 RILs common including Naxos which achieved food grade (FN $\geq$200) from both NMI and MI trials. Among these 41 RILs, six RILs from Vollebekk, one RIL from Chengdu 2016 and 22 RILs from Chengdu 2015 showed lower GI which did not exceed 0.2. RIL with lower GI from Chengdu 2016, SxN-072, also expressed lower GI in Chengdu 2015 (did not exceed 0.2). In addition, three RILs from Vollebekk 2016, SxN-009, SxN-36 and SxN-108, which achieved food grade with lower GI values also expressed lower GI in Chengdu 2015 (did not exceed 0.2).

4.1. Assessment of PHS traits

In this experiment, dormancy was assessed according to the GI method, which has been successfully used in earlier studies for determination of dormancy in wheat breeding programs (unpublished, Dahal, 2012; Ellis, Biddulph, & Young, 2012; Krupnova & Svistunov, 2014). This trait is compared with the response to weathering in the field to provoke PHS, analyzed by FN. Some unpublished data from earlier studies of dormancy, analyzed by DI, are also included. In this study, GI and FN were used for the assessment of PHS resistance. GI analyses were conducted on grains harvested at YR. Thus, the GI measures primary dormancy of grains at YR, whereas FN, analyzed from threshed samples, measures degree of PHS and also involves the after-YR period as the grains for FN experiments were harvested after YR.

The variation in GI between lines was large compared to the experimental error. When inspecting results from individual lines, it could be found lines with very low variation between replicates, but for others the variation between replicates were larger. Variation due to the plot site in the field were corrected for by the experimental design and calculation of the LSmeans, but there may still be some field variation that is not accounted for. Other reasons for experimental error could be un-precise harvesting and drying of ears, and that a few grains subjected to the germination test were infested by fungi. These grains were however removed, and not included in the calculations of GI. The standard germination test performed at low temperature for selected lines with very low GI from the Vollebekk trial revealed that the fresh un-sprouted grain seeds were viable, and thus correctly scored in the GI test. It is concluded that the method was carried out satisfactory, and it was successful. However, the aim of GI analyses was to achieve a good differentiation between the RILs, and at

Vollebekk, we adjusted the temperature for the germination test to achieve this. But, adjustment of the temperature for the germination test at Chengdu was not done.

The average GI varied between seasons and locations, and revealed high dormancy at Vollebekk 2016, high dormancy in Chengdu 2015 and low dormancy in Chengdu 2016. The temperature before YR, during the period with build-up of dormancy, were higher in Chengdu in both seasons (20.7°C in Chengdu 2016 and 22.2°C in Chengdu 2015) compared to Vollebekk (15.2°C). It was expected that increasing temperature prior YR would give decreasing build-up of dormancy, but the variation in dormancy level between these sites and seasons did not reflect these differences in temperatures. Thus, the reasons for the absence of a strong temperature effect remain unclear in this study.

It is interesting to report that one RIL showed similar expression in all the environmental conditions. In addition, some other RILs showed similar expression of GI in two of the three locations, such as 11 RILs between Vollebekk and Chengdu 2016, 17 RILs between Vollebekk and Chengdu 2015 as well as only three RILs between Chengdu 2016 and 2015. Still, most of the RILs showed great difference in GI level between the environments (locations and seasons). On the basis of this study, it can be expected that the different RILs respond differently for GI trait with various environmental conditions. This is also supported by Rodríguez, Margineda, González-Martín, Insausti, and Benech-Arnold (2001) who showed that different genotypes may vary widely in their sprouting behavior due to different environments.

A good differentiation in GI between the lines was obtained at Vollebekk 2016, and the histograms follow a normal distribution. Unfortunately, not so good differentiation was recorded in Chengdu in any of the two seasons. But, the transformation of the data improved differentiation between RILs. Also, the results of correlation coefficients (R^2) were slightly increased when transformed data were used compared to original data. After transformation, the ranking of the lines within each location and season were consistent with the original data. The most dormant and the least dormant lines ranked more similar at the Chengdu location in 2015 and 2016, but Vollebekk ranked more differently.

The difference between parents in GI were not significant both at Vollebekk 2016 and Chengdu 2015, while in Chengdu 2016, Naxos were significantly more dormant than SHA3/CBRD. However, the greenhouse experiment showed that Naxos was more dormant at low and moderate temperatures, but there was no differences between the parents at high temperatures. Thus, the results from the field trial at Vollebekk were not consistent with the results from the greenhouse study, as we should have expected Naxos more dormant than SHA3/CBRD at the relatively lower temperatures at Vollebekk. From the earlier field trials performed at Vollebekk, the analyses of dormancy have shown variable results, and Naxos were more dormant than SHA3/CBRD in some seasons, but similar to SHA3/CBRD in others.

To evaluate the limitation of variation in weather conditions during the build up of dormancy at YR, DM were determined for Vollebekk and Chengdu trials. The RILs showed a wide variation in DM, which could imply that the early and late RILs were subjected to different weather conditions during the period with build-up of dormancy. Significant correlations between GI and DM could indicate changes in weather prior to YR and thus differences in build-up of dormancy. The GI was significantly and negatively related to DM only in Chengdu 2015. Non-significant correlations between GI and DM from Vollebekk and Chengdu 2016 indicate that the changes in weather conditions prior to YR is not an important factor influencing the build-up of dormancy.

FN was analyzed only on samples from Vollebekk 2016, and for both the NMI and MI trials. The aim of this experiment was to analyze FN after conditions that provoke PHS had occurred, and thus be able to differentiate the lines according to tolerance to PHS. As expected, the MI samples had lower FN than the NMI samples, although both trials obtained relatively good differentiation between the RILs. The difference between parents were significant in both fields, where Naxos had the higher FN and were thus more resistant to PHS compared to SHA3/CBRD.

Lines of higher dormancy, having low GI, would be expected to give less PHS and thus high FN. This has previously been reported by several authors (unpublished, Dahal, 2012; Rasul et al., 2012). In this study, however, any strong negative correlations between FN and GI was not found. The only weak but significant negative correlation was between GI from Chengdu 2015 and FN from the MI trial at Vollebekk. These findings are however consistent with other authors findings (Ellis et al., 2012; Reitan, 1989).

Since, GI represents primary dormancy, whereas FN reflect both primary dormancy as well as secondary dormancy, and grains for GI analyses were harvested a few weeks earlier than the FN samples, the lower GI may not reflect well to higher FN. Furthermore, the grains from early YR RILs had longer post-YR exposure to adverse weather that may have resulted in lower FN compared to the later RILs, as all of the lines were harvested at the same time in each trial. This was also discussed by Ellis et al. (2012). Therefore, the variation caused by the time differences from YR to a specific weather conditions and decay of dormancy over time are considered as a major limitation of the FN test to assess PHS (Biddulph et al., 2008). In addition, FN manifests the quality of the endosperm at the time of harvest (Hagemann & Ciha, 1984) and can vary hugely based on the degree of ripening and amount of precipitation before harvest (Mares, 1993). This might be the reason that some of the most dormant lines in this study having very low GI have a relatively low FN. This is in agreement to that found by Dahal (unpublished, 2012).

In contrary, several studies have revealed that the FN based method is more reliable for characterization of PHS tolerance than dormancy based methods (Fofana, Humphreys, Rasul, Cloutier, & Somers, 2008; Humphreys & Noll, 2002). FN is often considered as the more ultimate test for PHS, affected both by

dormancy levels of the grains, but also other factors as those related to wetting and drying up of the ear due to rain, and uptake of water in the grains. Certainly, one of the main advantages of FN method is that it measures the α-amylase activity in the grain samples, which is an important quality trait for milling and baking. This can help to differentiate the flour quality of the cultivars, while on the other hand provides a measurement of tolerance to PHS.

4.2. Environmental effects on PHS

The RILs in trials used in this study ripened in different environments. Sprouting tolerance, measured by the response of grain to germination at YR, varied considerably with rainfall during 15 days period prior to YR. Max and min temperatures and rainfall were averaged over the 15 days periods prior to harvest at YR, since these periods covered the major part of grain dehydration and reported changes in grain germinability and establishment of dormancy (Mitchell, Black, & Chapman, 1980). These weather data was used to determine the possible relationship with PHS indices.

The results showed that some lines approached to YR at higher temperature and some lines at lower temperature. Dahal (unpublished, 2012) reported that higher max temperatures and higher min temperatures during the later stages of YR resulted in higher GI values with lower PHS resistance. GI results for parents from greenhouse at 27/22°C also reveal the same findings. Similarly, higher max temperature and higher min temperature after YR resulted in lower FN. This result is in consistence to findings by Strand (1989) and Mares (1993) that higher temperature after YR decreased grain dormancy.

Mares (1993) found that lower temperature during YR produced more dormant grains than higher temperature. Similar result has been obtained by Mares, Rathjen, Mrva, and Cheong (2009) and Nyachiro et al. (2002). GI results for parents from greenhouse grown at 22/17°C and 17/12°C were also consistent with these findings. The wheat-growing areas of north-western Europe and Scandinavia are influenced by a maritime climate characterized by high humidity and relatively low temperatures. For instance, Strand (1989) referred to mean temperatures of 16°C (heading to YR) and 13.5°C (YR to YR+30 days) and mean RH in the range 60-66% for a 20-year period in Norway. Our findings are also consistent with this historical data. Strand (1989) found that low temperatures and high moisture are favorable for the development of dormancy. In addition, higher PP during grain filling period resulted in higher dormancy, which means increased PHS tolerance. In contrary, higher PP after YR lead to loss of dormancy, meaning decreased PHS tolerance of the grains. This indicates that the observed higher PP during grain filling together with higher level of RH and lower temperature might explain the higher PHS tolerance obtained at Vollebekk 2016 compared to Chengdu 2016.

On the other hand, Nielsen, McCrate, Heyne, and Paulsen (1984) found that higher temperatures on two weeks prior to YR resulted in decreased tolerance to sprouting. Moreover, significant positive correlation between

higher min temperatures and PHS during the grain filling periods was found by Barnard and Smith (2009). However, this result is in contrary to our results from Chengdu 2015 where tolerance to sprouting was higher although the temperature was higher than in Chengdu 2016. These findings of Nielsen et al. (1984), and Barnard and Smith (2009) partly may support our interpretation from this study. However, the reasons for the high dormancy in the Chengdu 2015 trial is difficult to explain based on previous studies. It may indicate that more complex responses to environmental factors may be present, and our available data from Chengdu 2015 is not sufficient to interpret the results precisely.

However, there can be some variation in the typical germination pattern of a cultivar due to fungal infections, black points and other weather associated defects. In the field trials of our study, some of the lines were severely affected by infection, but the greater attention was made to exclude infested ears as well as the affected grains laid out for germination tests. Although, results of germination percentage indicate that the lines having lower GI have the optimum ability to germinate in standard growing environment and were not affected by any infestation, it is also possible that invisible symptoms might be influencing the germination indices in this study.

4.3. QTL responsible for agronomic traits

Identification of QTL responsible for agronomic traits can be used to identify novel candidate genes involved in PHS tolerance, and potentially provide tools to predict agronomical important traits. In this study, we detected several QTL for agronomic traits from different locations and seasons. Among them, the stable QTL are being discussed in our thesis as they were expressed in more than one location or season.

We detected a QTL on 2D chromosome for both DH and DM, and a QTL on 2A chromosome for only DH from Chengdu 2016 trial, but not from Vollebekk trial. Previous studies reported some homologous series of genes for photoperiod response, *Ppd-A1* (formerly *Ppd3*), *Ppd-B1* (formerly *Ppd2*) and *Ppd-D1* (formerly *Ppd1*), located on chromosomes 2A, 2B and 2D respectively, in which dominant alleles determine early heading (Law, Sutka, & Worland, 1978; Scarth & Law, 1983; Snape, Butterworth, Whitechurch, & Worland, 2001). Under long day conditions, the photoperiod sensitivity in wheat is controlled by one or several recessive *ppd* loci (Law et al., 1978; Scarth & Law, 1983) and photoperiod insensitivity in wheat is controlled by dominant *Ppd* loci (Snape et al., 2001). The photoperiod insensitive alleles promote flowering in the absence of long days and is widespread where wheat is grown as a short day winter crop. According to our QTL results, Naxos carries the alleles that prolong the DH in Chengdu, but not in Vollebekk. When plants are grown under long days, as in Norway, the lines carrying the recessive alleles at *Ppd* loci will flower early, while under short days, as in Chengdu, the flowering will be delayed. This is actually also what was observed, and indicates that the 2A and 2D QTL could be caused by *Ppd-A1* and *Ppd-D1*.

In wheat, height reducing dwarfing (*Rht*) genes such as *Rht-B1b and Rht-D1b* on 4B and 4D chromosomes, respectively, are used for developing semi-dwarf high-yielding commercial wheat cultivars around the world (Maluszynski, Szarejko, & Maluszynska, 2001). Both of the dwarfing genes encode protein involved in GA signal transduction and share the same GA-insensitivity mechanism (Peng et al., 1999). These genes are derived from a Japanese dwarf variety 'Norin 10' (Allan, 1989). In our study, SHA3/CBRD x Naxos RILs population segregated for *Rht-B1b,* which was detected on 4BS chromosome arm from both MI and NMI trials in Vollebekk 2016. In addition, another PH QTL detected on 4BS in 9.4 cM distance of *Rht-B1b* from Chengdu 2016 trial which indicates this QTL is also a part of the dwarfing gene and studied SHA3/CBRD x Naxos RILs population strongly express this dwarfing gene.

In this study, we reported a QTL on 5AL for DH from both MI and NMI trials in Vollebekk, but not in any trial in Chengdu, which is likely to be responsible for vernalization. Vernalization refers to a period of low temperature treatment for the transition of vegetative stage to reproductive stage. Spring wheat does not require vernalization but winter wheat requires a long period of exposure to low temperatures for proper flowering. Earlier studies reported a dominant vernalization gene (*Vrn-1*) located in the middle of long arm of chromosome 5A (Worland, 1996). Worland (1996) also reported that most of the spring growth habits of plants have dominant vernalization genes (*Vrn-1*) on group five chromosomes. The presence of the *Vrn-1A* allele results in the complete elimination of vernalization requirement (Yan et al., 2003), whereas the presence of the *Vrn-B1* and *Vrn-D1* alleles requires partial vernalization (Stelmakh, 1992, 1997). Such variable response of *Vrn-1* genes vernalization requirement determines the geographical adaptation of certain wheat genotypes under a particular environment and growing season (Shrestha, 2014). By detecting QTL on 5AL from both MI and NMI trials in Vollebekk, it is confirmed that vernalization is not required for the studied SHA3/CBRD x Naxos RIL population and it is more adapted in Norway than China. This dominant *Vrn-1A* allele were also obtained during the study of modern cultivars of central Europe, United States, and Canada, as well as Russia (Potokina et al., 2012; Shcherban, Emtseva, & Efremova, 2012; Shcherban, Börner, & Salina, 2015).

In 2014, Cabral et al. mapped a maturity QTL on 7DS chromosome by using 90K SNP chip in a doubled haploid population of 183 progenies derived from a cross between Canadian spring wheats RL4452/AC Domain. This QTL was also coincided with a DH and a FN QTL. In this study, we detected a QTL for DH from both MI and NMI trial, as well as for DM from NMI trial at Vollebekk in 2016. But we did not find this QTL for DH and DM from Chengdu trial. In addition, this QTL was not detected for FN from both MI and NMI trial at Vollebekk. Cabral et al. (2014) reported the number of DM as a quantitative trait and an important determinant of a cultivar's suitability to a specific growing region. Since we could not detect this QTL in Chengdu for any DH or DM trait, this further confirmed that this studied RIL population is more adapted in Norwegian growing conditions than Chinese growing conditions. However, further investigation is needed to certify this QTL as the same described by Cabral et al. (2014).

In addition, we detected five more QTL for agronomic traits, but they were only detected in single location or season.

4.4. QTL responsible for PHS

The main objectives of our study were to identify candidate QTL for PHS resistance in the SHA3/CBRD x Naxos RILs wheat population. Our objectives were achieved using high-density marker maps with both SNP, SSR, DArT and gene-specific markers. In our research, two parameters FN and GI were used for evaluating PHS resistant of the SHA3/CBRD x Naxos RILs population. RILs were grown in several environments, and QTL analyses indicated that abundant genetic variations existed in these RILs. This may provide opportunities for the application of marker-assisted selection (MAS) to improve tolerance to PHS in wheat breeding programs.

In wheat, many QTL associated with PHS and SD were reported previously and they are distributed across all 21 chromosomes (Arif et al., 2012; Imtiaz et al. 2008; Jahoor, Eriksen, & Backes, 2006; Langridge et al., 2001; Mares et al., 2005; Ogbonnaya et al., 2008; Roy et al., 1999; Tuberosa & Salvi, 2004; Tan et al., 2006). A whole-genome molecular map constructed during the present study allowed detection of seven putative QTL for PHS on chromosome arms 1BL, 2AL, 3BL, 4AL, 4BL, 5AS and 7BL (table 3.16). Only two of the seven QTL located on chromosome arms 1BL and 3BL were stable (detected in two trials), the remaining five QTL on 2AL, 4AL, 4BL, 5AS and 7BL were detected in only one trial suggesting that these QTL were unstable. Nevertheless, identification of several QTL for PHS during the present study underlined the importance of genome wide QTL analysis.

Previous studies suggest that the chromosomes groups 2, 3, and 4 are of particular importance for PHS tolerance. Many major QTL responsible for PHS have been reported on these chromosomes groups in both white and red seeded wheat varieties (Jaiswal et al., 2012; Kulwal et al., 2004, 2005; Kulwal et al., 2012; Mohan et al., 2009; Munkvold et al., 2009). Other studies revealed that major SD QTL are mainly located on chromosomes 3A (Mori et al., 2005; Osa et al., 2003) and 4A (Kato et al., 2001; Mares et al., 2005; Noda, Matsuura, Maekawa, & Taketa, 2002).

Among the seven PHS QTL detected, the QTL on chromosome 1BL, with resistance from Naxos, had the highest LOD scores; explained the largest proportion of phenotypic variation; and were the most consistently detected QTL for FN trait in both MI and NMI trials. In the same position, one significant QTL for PH was also detected from NMI trials and the same, but non-significant QTL was detected from MI trial. Which give a clear indication of the influence of PH on FN. Jaiswal et al. (2012), Munkvold et al. (2009), and Singh et al. (2014) also reported a QTL for PHS on 1B chromosome.

A QTL for GI was reported by Kumar et al. (2015) on chromosome 2A by using SSR marker which was on different position from the previous reports (Jaiswal et al., 2012; Kumar et al., 2009; Kobayashi, Takumi, & Handa, 2010; Knox et al., 2012; Mohan et al., 2009). Although, we detected a GI QTL with resistance from SHA3/CBRD on 2AL chromosome arm, we were unable to compare whether this QTL is the same as reported previously since we used SNP markers while most of the published studies were based on SSR markers.

In previous studies, although several major QTL were detected on chromosome arm 3A for seed coat color, SD (Bailey et al., 1999; Mori et al., 2005; Nelson et al., 1995; Osa et al., 2003) and PHS tolerance (Kulwal et al., 2005). We did not find any QTL for PHS resistance on chromosome 3A in our population. Lin et al. (2015) were also not found QTL, *TaPHS1*, on 3A chromosome when they studied on 155 RILs derived from a cross between PHS-resistant Tutoumai A and PHS-susceptible Siyang 936 cultivars. Moreover, no QTL was recorded on 3D chromosome, although a bunch of QTL were recorded in previous studies responsible for PHS tolerance (Groos et al., 2002; Somers et al., 2004).

Bailey et al. (1999) reported that a viviparous gene (*Vp-1*) is located in chromosomes 3AL, 3BL and 3DL approximately 30 cM distal to the *R* loci. Rathjen, Mares, Mrva, Schultz, and Cheong (2008) also detected a QTL on chromosome 3BL close to locus *R-B1a*, which seems to be associated with higher expression of genes controlling key enzymes in the flavonoid pathway. In our study, one QTL was detected on chromosome 3B for GI. However, we were unable to compare whether this QTL is the same as reported previously. Moreover, Kumar et al. (2015) were not able to detect any QTL for GI on group three chromosomes. Yang et al. (2007) reported that the variability of these loci may be related to GI and hence to PHS.

The QTL for PHS traits on chromosome 4AL were found in several studies (Barrero et al., 2015; Chen et al., 2008; Hickey et al., 2010; Mares et al., 2005; Shorinola et al., 2016; Torada, Ikeguchi, & Koike, 2005; Torada et al., 2016) indicating that they are stable. In a Chinese landrace, Tutoumai, a major QTL for PHS tolerance was mapped on chromosome arm 4AL (Chen et al., 2008; Lin et al., 2015). In our study, a GI QTL was detected on 4AL chromosome arm. In addition, a statistically non-significant QTL for FN, a significant QTL for PH and DM were also detected within 20 cM on this 4AL chromosome arm. Others also have reported QTL on chromosome 4AL associated with SD measured by germination test (Flintham, Adlam, Bassoi, Holdsworth, & Gale, 2002; Mares & Mrva, 2001; Mares et al., 2005; Mori et al., 2005). Rasul et al. (2009) reported a sprouting index (SI) QTL, (*QSI.crc-4A.2*), on 4AL chromosome arm. SI measures SD on intact spikes of wheat. Kato et al. (2001) also reported a QTL for PHS resistance on 4AL chromosome arm associated with SD in red-grained wheat. Kato et al. (2001) reported that *QPhs.ocs-4A.1* was homologous to the barley gene *Sd4*. Lohwasser, Röder, & Börner (2005) identified a QTL on 4AL linked to the PHS by measuring sprouting of intact spikes. A QTL for RVA peak viscosity was also mapped on the 4AL chromosome arm (McCartney et al., 2006). The Rapid Visco Analyser (RVA) is a widely used and well known for assessing the

pasting properties of flour. RVA peak viscosity measures starch breakdown which indirectly related to FN. McCartney et al. (2005) also reported that a QTL associated responsible for time to maturity QTL (*QMat.crc-4A*) at the same locus as *QSI.crc-4A.2* and *QFN.crc-4A.2*. Taken together, the chromosome arm 4AL contribute a lot to the resistance to PHS. These results provide direct evidence that SD plays an important role to improve the PHS resistance in wheat and that the QTL on 4AL chromosome arm controls both PHS resistance and SD in wheat. Although, QTL analyses from our study also demonstrate the close relationship between PH, DM, FN, and GI, we were not able to compare this detected QTL with previous studies. Furthermore, although Barrero et al. (2015) used 90K SNP chip for QTL analyses, we did not find any common markers for this QTL area with those used in our study.

In this study, a significant QTL for GI was detected on 4BL with resistance from Naxos. The QTL findings of Kato et al. (2001) for SD on chromosome 4BL using doubled haploid lines from a cross AC Domain/Haruyutaka, and the AC Domain confirmed that the 4BL chromosome arm contributes to increase SD. Kato et al. (2001) also reported that no significant effects on SD linked to semi-dwarf *Rht-B1* gene. This was also detected in our study for PH on 4BS chromosome arm with no effects on SD. However, previous studies showed that the dwarfing genes located on the group four chromosomes have pleiotropic effects on PHS resistance in wheat (King, Gale, & Quarrie, 1983; Wu et al., 2011).

One QTL for FN was detected on 7BL chromosome arm with resistance from Naxos in our study when plants were grown in MI field. Cabral et al. (2014) reported that a QTL for SI is located on chromosome 7BL. In another study on a population derived from a cross between two Australian wheat lines, Cranbrook and Halberd, a QTL that is responsible for tolerance to late maturity α-amylase has also been mapped on 7BL chromosome arm (McNeil et al., 2009). QTL for PHS was also detected on 7BL chromosome arm in several other studies (Jaiswal et al., 2012; Kobayashi et al., 2010; Knox et al., 2012; Singh et al., 2014). These results from other studies suggest that PHS, FN and late maturity α-amylase QTL on chromosome 7B are likely the same.

Similarly, the QTL on 5AS was detected for only FN with resistance from Naxos. Groos et al. (2002), Lin et al. (2015), and Nakamura, Komatsuda, and Miura (2007) reported a QTL on 5AS chromosome arm for PHS resistance. However, these studies suggest that they could be the same QTL, but this needs further study.

4.5. Implications for breeding

QTL results from the SHA3/CBRD x Naxos RILs population linked with PHS suggest that lines with a high level of PHS resistance can be achieved when the desirable QTL alleles are combined from both parents. QTL alleles on group four chromosomes seem to be the most important in maintaining high levels of PHS resistance. These findings have also been reported from other studies (Mori et al., 2005; Torada et al., 2005). Candidate

markers on chromosome 4AL and 4BL may be useful for MAS for PHS resistance. In addition, understanding the major QTL for PHS resistance detected on chromosomes 1B and 3B observed through measuring α-amylase activity with FN and germination with GI may aid in MAS for improved PHS resistance in wheat. However, only one GI QTL on 3BL and one FN QTL on 1BL were detected in two environments, while other QTL were detected only in one environment. It may be that the number of QTL detected is associated with the degree of damage due to sprouting. These results again reveal the complex genetic control of SD and PHS which is consistent with earlier studies (Fofana et al., 2009; Imtiaz et al., 2008; Knox et al., 1998; Kulwal et al., 2004; Mohan et al., 2009).

Even though, we detected many QTL in the SHA3/CBRD x Naxos RILs population, several QTL reported in previous studies by others were not detected in our population. The possible reasons for that could be due to the lack of presence of responsible alleles in the parental genotypes, or the effective alleles were common between the two parents so no segregation occurred. In addition, the sampled environments also can affect to express candidate loci. This study also reveals that favorable alleles for improving sprouting resistance were present in both Naxos and SHA3/CBRD parents. Our results also support conclusions from other studies that QTL for PHS resistance are located throughout the whole wheat genome (Flintham et al., 2002).

Besides the PHS QTL, detected agronomic traits QTL also give a better understanding about the suitability of the growing areas for this studied SHA3/CBRD x Naxos RILs population. Pyramiding of these QTL will improve the level of resistance to PHS in wheat. The chromosome-based sequences and high-throughput genotyping will allow further fine mapping and better understanding of these QTL.

Chapter 5

Conclusion

With the help of high-density marker maps with SSR, DArT and SNP markers, seven QTL were detected in the SHA3/CBRD x Naxos RILs population for PHS traits, four of them with Naxos as the resistance source. They were located throughout the genome especially on 1BL, 3BL, and 4AL chromosomes arms. The largest proportions of the explained phenotypic variation were recorded for the FN QTL located on 1BL and GI QTL located on 3BL. Both of them were stable, expressed in two seasons and locations. A QTL controlling both FN and GI including DM and PH was identified on 4AL chromosome arm. These chromosomal regions, previously identified as being involved in the control of PHS in wheat, were also active in this studied population. All of these QTL are possibly reported previously by many authors. However, these findings need to be considered with caution for precise identification of closely linked SNP markers as it was difficult to compare QTL positions due to the use of different marker systems. Further investigations of these and other genomic regions might help to detect the location and effects of QTL for PHS resistance in spring wheat.

The results showed that the sources of resistance for PHS existed in both parents, Naxos and SHA3/CBRD, but the parent Naxos was the most important source of dormancy. However, a few RILs did score better than the parents, and further evaluation of the genotypes belonging to the top 10% group for FN and GI score in this population might help to identify new and improved spring wheat germplasm with acceptable levels of resistance to PHS in the field. In addition, this study also showed that different RILs responded differently in dormancy build-up regarding weather characteristics at different stages of grain development. The result showed a significant but weak negative correlation for GI and FN. This study also demonstrates that variations caused by weather factors may create difficulties to interpret PHS result precisely. As a lot of information becomes available regarding the location of these loci as well as their individual and combined effect on PHS resistance, breeders will be able to combine techniques of molecular genetics with conventional breeding methods through MAS in order to address the serious problem caused by PHS in wheat.

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Appendix A

Germination Index and Falling Number data from non-mist irrigated trial Vollebekk 2016

Line	Pedigree	DH	DM	PH	GI	FN
14	Saar	60.07	110.09	74.39	0.44	223
29	SHA3/CBRD	62.74	107.00	75.06	0.24	165
39	Naxos (x3)	59.34	111.95	70.41	0.27	333
45	Krabat	61.64	109.04	65.14	0.36	354
1419	Polkka	61.92	103.47	77.37	0.46	359
1424	T7347	62.99	113.01	82.75	0.19	365
SxN-001	SHA3/CBRD//NAXOS	62.19	107.82	75.36	0.38	327
SxN-002	SHA3/CBRD//NAXOS	59.95	110.41	67.23	0.14	192
SxN-003	SHA3/CBRD//NAXOS	62.46	115.28	73.06	0.43	326
SxN-004	SHA3/CBRD//NAXOS	61.41	105.10	86.46	0.36	286
SxN-005	SHA3/CBRD//NAXOS	58.98	104.62	87.68	0.32	243
SxN-006	SHA3/CBRD//NAXOS	61.74	118.65	68.45	0.42	259
SxN-007	SHA3/CBRD//NAXOS	61.35	109.41	78.17	0.61	271
SxN-008	SHA3/CBRD//NAXOS	61.70	113.06	57.25	0.27	274
SxN-009	SHA3/CBRD//NAXOS	62.44	112.77	71.44	0.11	342
SxN-010	SHA3/CBRD//NAXOS	58.51	106.84	64.91	0.22	204
SxN-011	SHA3/CBRD//NAXOS	58.26	106.55	65.14	0.41	269
SxN-012	SHA3/CBRD//NAXOS	61.60	115.26	75.82	0.20	249
SxN-013	SHA3/CBRD//NAXOS	60.84	109.96	53.93	0.19	247
SxN-014	SHA3/CBRD//NAXOS	60.22	105.96	69.66	0.53	193
SxN-015	SHA3/CBRD//NAXOS	63.73	116.28	84.60	0.20	246
SxN-016	SHA3/CBRD//NAXOS	60.29	107.69	73.33	0.36	240
SxN-017	SHA3/CBRD//NAXOS	61.20	107.24	74.81	0.40	297
SxN-018	SHA3/CBRD//NAXOS	63.06	112.09	66.04	0.36	286
SxN-019	SHA3/CBRD//NAXOS	63.74	112.22	77.24	0.22	273
SxN-020	SHA3/CBRD//NAXOS	61.62	110.98	89.04	0.16	128
SxN-021	SHA3/CBRD//NAXOS	61.80	105.54	79.65	0.32	286
SxN-023	SHA3/CBRD//NAXOS	62.26	115.52	74.40	0.57	196
SxN-024	SHA3/CBRD//NAXOS	57.69	103.35	70.86	0.25	329
SxN-025	SHA3/CBRD//NAXOS	58.81	104.90	85.84	0.23	385
SxN-026	SHA3/CBRD//NAXOS	60.17	105.89	82.84	0.13	279

Line	Pedigree	DH	DM	PH	GI	FN
SxN-027	SHA3/CBRD//NAXOS	57.97	103.89	70.52	0.61	271
SxN-028	SHA3/CBRD//NAXOS	58.82	106.75	82.18	0.19	172
SxN-029	SHA3/CBRD//NAXOS	60.54	114.92	62.66	0.27	270
SxN-030	SHA3/CBRD//NAXOS	60.58	106.97	75.35	0.35	274
SxN-031	SHA3/CBRD//NAXOS	62.89	106.14	72.81	0.31	342
SxN-032	SHA3/CBRD//NAXOS	58.72	103.95	59.24	0.39	369
SxN-033	SHA3/CBRD//NAXOS	58.24	106.40	82.30	0.01	240
SxN-034	SHA3/CBRD//NAXOS	59.03	106.93	76.69	0.36	246
SxN-035	SHA3/CBRD//NAXOS	64.35	118.58	44.12	0.27	94
SxN-036	SHA3/CBRD//NAXOS	58.81	109.01	68.10	0.17	397
SxN-037	SHA3/CBRD//NAXOS	60.10	105.84	75.09	0.85	241
SxN-038	SHA3/CBRD//NAXOS	60.95	106.97	79.02	0.25	299
SxN-039	SHA3/CBRD//NAXOS	60.54	104.40	73.46	0.10	220
SxN-040	SHA3/CBRD//NAXOS	61.19	110.27	83.77	0.36	233
SxN-041	SHA3/CBRD//NAXOS	60.73	112.56	85.42	0.32	267
SxN-042	SHA3/CBRD//NAXOS	59.03	104.96	62.91	0.40	194
SxN-043	SHA3/CBRD//NAXOS	62.16	104.81	84.52	0.08	286
SxN-044	SHA3/CBRD//NAXOS	60.99	112.93	62.73	0.41	299
SxN-045	SHA3/CBRD//NAXOS	59.79	109.58	63.80	0.27	291
SxN-046	SHA3/CBRD//NAXOS	61.59	106.45	67.14	0.45	282
SxN-047	SHA3/CBRD//NAXOS	61.77	115.86	68.95	0.33	316
SxN-048	SHA3/CBRD//NAXOS	61.77	105.81	58.27	0.17	166
SxN-050	SHA3/CBRD//NAXOS	61.77	114.55	76.45	0.33	212
SxN-051	SHA3/CBRD//NAXOS	59.76	114.04	66.06	0.45	290
SxN-052	SHA3/CBRD//NAXOS	58.59	105.86	64.69	0.27	335
SxN-053	SHA3/CBRD//NAXOS	61.00	112.70	91.38	0.43	268
SxN-054	SHA3/CBRD//NAXOS	63.46	117.55	80.32	0.16	188
SxN-055	SHA3/CBRD//NAXOS	62.27	113.03	79.81	0.16	156
SxN-056	SHA3/CBRD//NAXOS	61.88	110.02	85.77	0.24	259
SxN-057	SHA3/CBRD//NAXOS	60.81	113.63	79.38	0.58	328
SxN-058	SHA3/CBRD//NAXOS	59.28	112.84	60.63	0.49	235
SxN-059	SHA3/CBRD//NAXOS	58.70	110.57	73.60	0.13	131
SxN-060	SHA3/CBRD//NAXOS	58.01	106.02	84.58	0.38	243
SxN-061	SHA3/CBRD//NAXOS	61.26	112.50	69.54	0.36	326
SxN-062	SHA3/CBRD//NAXOS	59.95	111.65	87.86	0.30	281
SxN-063	SHA3/CBRD//NAXOS	59.76	115.49	57.47	0.40	136
SxN-064	SHA3/CBRD//NAXOS	62.83	110.79	85.64	0.37	226
SxN-065	SHA3/CBRD//NAXOS	59.20	110.34	64.50	0.59	132
SxN-066	SHA3/CBRD//NAXOS	60.28	105.82	77.62	0.02	262

Line	Pedigree	DH	DM	PH	GI	FN
SxN-067	SHA3/CBRD//NAXOS	62.10	111.29	73.63	0.31	295
SxN-068	SHA3/CBRD//NAXOS	58.36	101.66	75.84	0.23	324
SxN-069	SHA3/CBRD//NAXOS	61.28	106.43	78.67	0.37	246
SxN-070	SHA3/CBRD//NAXOS	60.31	111.65	64.30	0.19	180
SxN-071	SHA3/CBRD//NAXOS	62.63	111.98	81.84	0.43	116
SxN-072	SHA3/CBRD//NAXOS	62.42	116.69	65.93	0.33	269
SxN-073	SHA3/CBRD//NAXOS	65.11	119.20	65.41	0.42	109
SxN-074	SHA3/CBRD//NAXOS	60.67	114.17	68.48	0.33	152
SxN-075	SHA3/CBRD//NAXOS	62.03	117.19	75.01	0.33	155
SxN-076	SHA3/CBRD//NAXOS	59.90	106.28	55.37	0.18	239
SxN-078	SHA3/CBRD//NAXOS	63.13	116.36	65.75	0.67	205
SxN-079	SHA3/CBRD//NAXOS	60.94	105.77	53.37	0.10	289
SxN-080	SHA3/CBRD//NAXOS	64.71	115.64	59.00	0.21	265
SxN-081	SHA3/CBRD//NAXOS	60.47	106.11	62.67	0.54	217
SxN-082	SHA3/CBRD//NAXOS	61.98	114.44	58.19	0.41	338
SxN-083	SHA3/CBRD//NAXOS	64.26	116.55	58.89	0.20	273
SxN-084	SHA3/CBRD//NAXOS	62.38	104.87	90.66	0.30	301
SxN-085	SHA3/CBRD//NAXOS	62.31	106.24	85.38	0.41	386
SxN-086	SHA3/CBRD//NAXOS	59.50	106.04	82.14	0.31	334
SxN-087	SHA3/CBRD//NAXOS	61.16	106.52	62.62	0.37	325
SxN-088	SHA3/CBRD//NAXOS	64.79	112.67	67.11	0.25	158
SxN-089	SHA3/CBRD//NAXOS	62.18	110.71	83.15	0.29	325
SxN-090	SHA3/CBRD//NAXOS	58.54	104.14	58.53	0.61	183
SxN-091	SHA3/CBRD//NAXOS	58.21	107.43	77.31	0.40	154
SxN-092	SHA3/CBRD//NAXOS	58.94	96.63	71.98	0.26	336
SxN-093	SHA3/CBRD//NAXOS	61.96	107.88	76.13	0.12	374
SxN-094	SHA3/CBRD//NAXOS	63.43	114.73	69.54	0.55	309
SxN-095	SHA3/CBRD//NAXOS	62.19	119.03	65.83	0.20	63
SxN-096	SHA3/CBRD//NAXOS	59.46	103.73	70.44	0.32	356
SxN-097	SHA3/CBRD//NAXOS	61.92	115.88	64.99	0.34	364
SxN-098	SHA3/CBRD//NAXOS	62.08	117.63	36.59	0.43	260
SxN-099	SHA3/CBRD//NAXOS	60.07	106.40	67.31	0.31	299
SxN-100	SHA3/CBRD//NAXOS	59.96	113.73	78.97	0.39	266
SxN-101	SHA3/CBRD//NAXOS	60.19	108.69	77.51	0.39	250
SxN-102	SHA3/CBRD//NAXOS	60.05	104.25	83.20	0.43	139
SxN-103	SHA3/CBRD//NAXOS	61.08	111.02	75.30	0.34	249
SxN-104	SHA3/CBRD//NAXOS	59.42	104.39	74.65	0.45	383
SxN-105	SHA3/CBRD//NAXOS	58.24	104.48	82.22	0.25	224
SxN-106	SHA3/CBRD//NAXOS	60.40	109.33	77.35	0.15	224

Line	Pedigree	DH	DM	PH	GI	FN
SxN-107	SHA3/CBRD//NAXOS	62.78	120.45	53.56	0.56	251
SxN-108	SHA3/CBRD//NAXOS	61.47	108.60	83.62	0.06	331
SxN-109	SHA3/CBRD//NAXOS	64.54	122.12	52.31	0.77	256
SxN-110	SHA3/CBRD//NAXOS	59.78	105.87	80.90	0.13	247
SxN-111	SHA3/CBRD//NAXOS	59.35	110.13	66.03	0.32	286
SxN-112	SHA3/CBRD//NAXOS	60.81	105.36	83.95	0.52	342
SxN-113	SHA3/CBRD//NAXOS	62.55	109.14	65.13	0.29	251
SxN-114	SHA3/CBRD//NAXOS	59.08	105.50	65.28	0.30	310
SxN-115	SHA3/CBRD//NAXOS	60.65	106.38	75.68	0.44	266
SxN-116	SHA3/CBRD//NAXOS	60.53	109.88	70.67	0.15	252
SxN-117	SHA3/CBRD//NAXOS	60.69	105.69	79.37	0.35	248
SxN-118	SHA3/CBRD//NAXOS	60.32	104.84	81.72	0.30	325
SxN-119	SHA3/CBRD//NAXOS	60.62	106.60	83.60	0.06	310
SxN-120	SHA3/CBRD//NAXOS	60.55	106.47	69.58	0.26	193
SxN-121	SHA3/CBRD//NAXOS	60.63	110.36	63.07	0.19	280
SxN-122	SHA3/CBRD//NAXOS	60.29	105.00	82.90	0.18	256
SxN-123	SHA3/CBRD//NAXOS	59.28	106.15	72.08	0.33	253
SxN-124	SHA3/CBRD//NAXOS	60.19	108.48	82.90	0.52	272
SxN-125	SHA3/CBRD//NAXOS	64.73	115.41	47.39	0.29	223
SxN-126	SHA3/CBRD//NAXOS	60.99	111.85	73.42	0.32	110
SxN-127	SHA3/CBRD//NAXOS	62.88	106.52	88.41	0.11	276
SxN-128	SHA3/CBRD//NAXOS	61.12	103.54	80.78	0.39	300
SxN-129	SHA3/CBRD//NAXOS	62.19	113.44	78.98	0.11	134
SxN-130	SHA3/CBRD//NAXOS	63.02	114.90	78.86	0.46	242
SxN-131	SHA3/CBRD//NAXOS	60.38	108.72	70.68	0.39	161
SxN-132	SHA3/CBRD//NAXOS	61.91	112.10	86.53	0.25	194
SxN-133	SHA3/CBRD//NAXOS	61.38	107.03	88.56	0.46	209
SxN-134	SHA3/CBRD//NAXOS	60.02	113.88	81.03	0.21	158
SxN-135	SHA3/CBRD//NAXOS	60.76	109.41	63.81	0.41	292
SxN-136	SHA3/CBRD//NAXOS	58.94	111.44	87.34	0.39	362
SxN-137	SHA3/CBRD//NAXOS	60.12	111.63	82.80	0.33	283
SxN-138	SHA3/CBRD//NAXOS	57.95	104.45	69.35	0.13	158
SxN-139	SHA3/CBRD//NAXOS	62.99	110.80	80.74	0.47	124
SxN-140	SHA3/CBRD//NAXOS	59.57	113.88	77.01	0.20	226
SxN-141	SHA3/CBRD//NAXOS	59.77	106.61	58.92	-0.03	201
SxN-142	SHA3/CBRD//NAXOS	61.27	115.21	64.04	0.25	236
SxN-143	SHA3/CBRD//NAXOS	61.09	107.53	66.62	0.37	246
SxN-144	SHA3/CBRD//NAXOS	60.25	104.47	66.76	0.30	244
SxN-145	SHA3/CBRD//NAXOS	62.45	112.91	62.01	0.15	237

Line	Pedigree	DH	DM	PH	GI	FN
SxN-146	SHA3/CBRD//NAXOS	62.78	103.05	67.71	0.58	207
SxN-147	SHA3/CBRD//NAXOS	61.72	112.34	71.05	0.34	283
SxN-148	SHA3/CBRD//NAXOS	62.92	108.49	82.67	0.16	223
SxN-151	SHA3/CBRD//NAXOS	63.29	109.17	76.24	0.55	278
SxN-155	SHA3/CBRD//NAXOS	64.04	113.68	87.86	0.36	330
SxN-157	SHA3/CBRD//NAXOS	61.85	115.15	67.81	0.58	269
SxN-159	SHA3/CBRD//NAXOS	66.43	116.44	78.03	0.22	341
SxN-160	SHA3/CBRD//NAXOS	60.13	105.57	58.73	0.16	218
SxN-161	SHA3/CBRD//NAXOS	64.19	109.97	66.87	0.21	301
SxN-162	SHA3/CBRD//NAXOS	62.58	110.46	84.57	0.17	187
SxN-163	SHA3/CBRD//NAXOS	61.91	111.59	71.10	0.34	246
SxN-164	SHA3/CBRD//NAXOS	58.55	109.25	75.78	0.28	66
SxN-165	SHA3/CBRD//NAXOS	58.47	106.84	80.49	0.44	255
SxN-166	SHA3/CBRD//NAXOS	61.39	110.15	67.81	0.60	141
SxN-167	SHA3/CBRD//NAXOS	60.74	113.50	56.97	0.13	206
SxN-168	SHA3/CBRD//NAXOS	60.23	112.39	71.79	0.37	211
SxN-169	SHA3/CBRD//NAXOS	60.82	103.08	61.42	0.04	216
SxN-170	SHA3/CBRD//NAXOS	61.39	105.81	77.93	0.08	129
SxN-171	SHA3/CBRD//NAXOS	60.36	111.83	86.02	0.36	187
SxN-172	SHA3/CBRD//NAXOS	59.75	105.04	79.41	0.54	255
SxN-173	SHA3/CBRD//NAXOS	60.57	109.17	67.75	0.59	321
SxN-175	SHA3/CBRD//NAXOS	59.66	118.85	44.38	0.33	234
SxN-176	SHA3/CBRD//NAXOS	62.80	117.08	73.37	0.09	278
SxN-180	SHA3/CBRD//NAXOS	65.13	110.36	88.08	0.42	76
SxN-182	SHA3/CBRD//NAXOS	60.16	109.68	61.85	0.59	290

Appendix B

Germination Index data Chengdu 2016

Line	Pedigree	DH	DM	PH	GI	Square_GI
Parent 1	Naxos(x3)	149	195	115	0.52	0.27
Parent 2	SHA3/CBRD	136	182	98	0.77	0.59
SxN-001	SHA3/CBRD//NAXOS	148	193	132	0.42	0.18
SxN-002	SHA3/CBRD//NAXOS	137	182	114	0.70	0.48
SxN-003	SHA3/CBRD//NAXOS	133	180	117	0.51	0.26
SxN-004	SHA3/CBRD//NAXOS	132	178	98	0.58	0.33
SxN-005	SHA3/CBRD//NAXOS	154	197	102	0.39	0.15
SxN-006	SHA3/CBRD//NAXOS	133	182	106	0.71	0.50
SxN-007	SHA3/CBRD//NAXOS	153	199	125	0.40	0.16
SxN-008	SHA3/CBRD//NAXOS	155	197	113	0.56	0.31
SxN-009	SHA3/CBRD//NAXOS	148	195	110	0.67	0.45
SxN-010	SHA3/CBRD//NAXOS	145	191	94	0.29	0.09
SxN-011	SHA3/CBRD//NAXOS	153	195	110	0.77	0.60
SxN-012	SHA3/CBRD//NAXOS	129	182	115	0.71	0.50
SxN-013	SHA3/CBRD//NAXOS	144	191	101	0.84	0.70
SxN-014	SHA3/CBRD//NAXOS	150	195	127	0.61	0.37
SxN-015	SHA3/CBRD//NAXOS	129	182	98	0.72	0.51
SxN-016	SHA3/CBRD//NAXOS	131	182	111	0.37	0.14
SxN-017	SHA3/CBRD//NAXOS	149	193	118	0.59	0.35
SxN-018	SHA3/CBRD//NAXOS	149	193	115	0.77	0.60
SxN-019	SHA3/CBRD//NAXOS	134	184	116	0.49	0.24
SxN-020	SHA3/CBRD//NAXOS	133	182	109	0.22	0.05
SxN-021	SHA3/CBRD//NAXOS	151	197	109	0.33	0.11
SxN-022	SHA3/CBRD//NAXOS	132	187	122	0.66	0.44
SxN-023	SHA3/CBRD//NAXOS	151	193	141	0.15	0.02
SxN-024	SHA3/CBRD//NAXOS	157	204	120	0.67	0.45
SxN-025	SHA3/CBRD//NAXOS	142	188	125	0.52	0.27
SxN-026	SHA3/CBRD//NAXOS	128	178	82	0.46	0.21
SxN-027	SHA3/CBRD//NAXOS	146	191	142	0.85	0.72
SxN-028	SHA3/CBRD//NAXOS	133	182	119	0.70	0.48
SxN-029	SHA3/CBRD//NAXOS	146	191	143	0.84	0.70
SxN-030	SHA3/CBRD//NAXOS	134	186	97	0.50	0.25
SxN-031	SHA3/CBRD//NAXOS	150	193	108	0.73	0.53
SxN-032	SHA3/CBRD//NAXOS	136	180	103	0.52	0.27

Line	Pedigree	DH	DM	PH	GI	Square_GI
SxN-033	SHA3/CBRD//NAXOS	131	177	99	0.49	0.24
SxN-034	SHA3/CBRD//NAXOS	145	188	137	0.68	0.46
SxN-035	SHA3/CBRD//NAXOS	127	184	111	0.79	0.62
SxN-036	SHA3/CBRD//NAXOS	131	184	74	0.77	0.59
SxN-037	SHA3/CBRD//NAXOS	157	197	109	0.68	0.47
SxN-038	SHA3/CBRD//NAXOS	148	193	116	0.79	0.63
SxN-039	SHA3/CBRD//NAXOS	151	193	125	0.78	0.61
SxN-040	SHA3/CBRD//NAXOS	132	180	109	0.45	0.21
SxN-041	SHA3/CBRD//NAXOS	130	184	115	0.78	0.61
SxN-042	SHA3/CBRD//NAXOS	128	182	126	0.83	0.68
SxN-043	SHA3/CBRD//NAXOS	145	193	105	0.77	0.59
SxN-044	SHA3/CBRD//NAXOS	153	193	120	0.62	0.39
SxN-045	SHA3/CBRD//NAXOS	150	193	105	0.09	0.01
SxN-046	SHA3/CBRD//NAXOS	132	184	99	0.80	0.64
SxN-047	SHA3/CBRD//NAXOS	150	197	108	0.61	0.37
SxN-048	SHA3/CBRD//NAXOS	132	180	96	0.60	0.36
SxN-050	SHA3/CBRD//NAXOS	157	204	82	0.38	0.14
SxN-051	SHA3/CBRD//NAXOS	150	197	123	0.62	0.39
SxN-052	SHA3/CBRD//NAXOS	132	180	103	0.68	0.46
SxN-053	SHA3/CBRD//NAXOS	132	184	108	0.66	0.43
SxN-054	SHA3/CBRD//NAXOS	131	180	130	0.15	0.02
SxN-055	SHA3/CBRD//NAXOS	137	186	140	0.51	0.26
SxN-056	SHA3/CBRD//NAXOS	137	187	138	0.45	0.21
SxN-057	SHA3/CBRD//NAXOS	155	200	130	0.61	0.37
SxN-058	SHA3/CBRD//NAXOS	155	200	130	0.62	0.38
SxN-059	SHA3/CBRD//NAXOS	131	187	112	0.65	0.42
SxN-060	SHA3/CBRD//NAXOS	131	186	118	0.74	0.55
SxN-061	SHA3/CBRD//NAXOS	131	186	124	0.69	0.48
SxN-062	SHA3/CBRD//NAXOS	158	197	100	0.08	0.01
SxN-063	SHA3/CBRD//NAXOS	131	184	119	0.77	0.59
SxN-064	SHA3/CBRD//NAXOS	130	177	85	0.73	0.53
SxN-065	SHA3/CBRD//NAXOS	134	184	112	0.70	0.49
SxN-066	SHA3/CBRD//NAXOS	135	182	112	0.32	0.10
SxN-067	SHA3/CBRD//NAXOS	135	182	134	0.76	0.57
SxN-068	SHA3/CBRD//NAXOS	157	197	99	0.52	0.27
SxN-069	SHA3/CBRD//NAXOS	129	177	115	0.58	0.34
SxN-070	SHA3/CBRD//NAXOS	151	195	115	0.67	0.44
SxN-071	SHA3/CBRD//NAXOS	133	184	111	0.23	0.05
SxN-072	SHA3/CBRD//NAXOS	151	197	120	0.03	0.00

Line	Pedigree	DH	DM	PH	GI	Square_GI
SxN-073	SHA3/CBRD//NAXOS	135	177	100	0.73	0.53
SxN-074	SHA3/CBRD//NAXOS	153	193	85	0.71	0.50
SxN-075	SHA3/CBRD//NAXOS	131	177	100	0.78	0.61
SxN-076	SHA3/CBRD//NAXOS	141	191	100	0.63	0.40
SxN-077	SHA3/CBRD//NAXOS	150	191	98	0.66	0.44
SxN-078	SHA3/CBRD//NAXOS	150	191	96	0.72	0.51
SxN-079	SHA3/CBRD//NAXOS	145	191	101	0.74	0.54
SxN-080	SHA3/CBRD//NAXOS	150	188	95	0.45	0.20
SxN-081	SHA3/CBRD//NAXOS	132	182	97	0.20	0.04
SxN-082	SHA3/CBRD//NAXOS	132	177	100	0.74	0.54
SxN-083	SHA3/CBRD//NAXOS	154	193	83	0.49	0.24
SxN-084	SHA3/CBRD//NAXOS	131	182	128	0.70	0.49
SxN-085	SHA3/CBRD//NAXOS	131	182	118	0.73	0.53
SxN-086	SHA3/CBRD//NAXOS	129	182	113	0.65	0.42
SxN-087	SHA3/CBRD//NAXOS	149	191	94	0.59	0.35
SxN-088	SHA3/CBRD//NAXOS	132	182	98	0.74	0.55
SxN-089	SHA3/CBRD//NAXOS	133	184	109	0.70	0.49
SxN-090	SHA3/CBRD//NAXOS	129	177	96	0.69	0.47
SxN-091	SHA3/CBRD//NAXOS	130	177	109	0.82	0.66
SxN-092	SHA3/CBRD//NAXOS	130	177	94	0.39	0.15
SxN-093	SHA3/CBRD//NAXOS	130	182	99	0.66	0.43
SxN-094	SHA3/CBRD//NAXOS	137	182	90	0.70	0.50
SxN-095	SHA3/CBRD//NAXOS	153	193	111	0.31	0.10
SxN-096	SHA3/CBRD//NAXOS	128	177	99	0.69	0.48
SxN-097	SHA3/CBRD//NAXOS	136	182	96	0.40	0.16
SxN-098	SHA3/CBRD//NAXOS	131	186	67	0.71	0.51
SxN-099	SHA3/CBRD//NAXOS	134	184	115	0.77	0.59
SxN-100	SHA3/CBRD//NAXOS	131	177	121	0.80	0.64
SxN-101	SHA3/CBRD//NAXOS	131	177	116	0.80	0.64
SxN-102	SHA3/CBRD//NAXOS	153	193	119	0.19	0.04
SxN-103	SHA3/CBRD//NAXOS	131	182	114	0.80	0.64
SxN-104	SHA3/CBRD//NAXOS	127	177	105	0.54	0.29
SxN-105	SHA3/CBRD//NAXOS	129	177	112	0.78	0.60
SxN-106	SHA3/CBRD//NAXOS	130	182	119	0.80	0.64
SxN-107	SHA3/CBRD//NAXOS	131	184	90	0.75	0.57
SxN-108	SHA3/CBRD//NAXOS	132	182	117	0.36	0.13
SxN-109	SHA3/CBRD//NAXOS	131	184	85	0.72	0.51
SxN-110	SHA3/CBRD//NAXOS	154	193	118	0.50	0.25
SxN-111	SHA3/CBRD//NAXOS	152	191	130	0.78	0.61

Line	Pedigree	DH	DM	PH	GI	Square_GI
SxN-112	SHA3/CBRD//NAXOS	133	186	110	0.37	0.14
SxN-113	SHA3/CBRD//NAXOS	154	193	115	0.43	0.19
SxN-114	SHA3/CBRD//NAXOS	132	184	112	0.46	0.21
SxN-115	SHA3/CBRD//NAXOS	134	186	116	0.28	0.08
SxN-116	SHA3/CBRD//NAXOS	154	193	115	0.44	0.19
SxN-117	SHA3/CBRD//NAXOS	151	191	125	0.83	0.69
SxN-118	SHA3/CBRD//NAXOS	150	193	126	0.84	0.70
SxN-119	SHA3/CBRD//NAXOS	150	193	127	0.84	0.70
SxN-120	SHA3/CBRD//NAXOS	136	186	107	0.50	0.25
SxN-121	SHA3/CBRD//NAXOS	136	186	100	0.43	0.19
SxN-122	SHA3/CBRD//NAXOS	153	193	124	0.79	0.62
SxN-123	SHA3/CBRD//NAXOS	156	197	123	0.46	0.21
SxN-124	SHA3/CBRD//NAXOS	152	193	122	0.79	0.62
SxN-125	SHA3/CBRD//NAXOS	132	182	86	0.62	0.39
SxN-126	SHA3/CBRD//NAXOS	156	197	100	0.12	0.01
SxN-127	SHA3/CBRD//NAXOS	132	182	123	0.73	0.54
SxN-128	SHA3/CBRD//NAXOS	134	186	118	0.77	0.59
SxN-129	SHA3/CBRD//NAXOS	153	195	123	0.70	0.49
SxN-130	SHA3/CBRD//NAXOS	132	184	101	0.46	0.21
SxN-131	SHA3/CBRD//NAXOS	136	186	108	0.73	0.54
SxN-132	SHA3/CBRD//NAXOS	150	191	120	0.32	0.10
SxN-133	SHA3/CBRD//NAXOS	158	199	113	0.32	0.10
SxN-134	SHA3/CBRD//NAXOS	150	188	124	0.80	0.64
SxN-135	SHA3/CBRD//NAXOS	135	177	101	0.77	0.60
SxN-136	SHA3/CBRD//NAXOS	130	177	118	0.76	0.58
SxN-137	SHA3/CBRD//NAXOS	153	197	112	0.82	0.68
SxN-138	SHA3/CBRD//NAXOS	130	177	100	0.59	0.35
SxN-139	SHA3/CBRD//NAXOS	132	177	109	0.66	0.43
SxN-140	SHA3/CBRD//NAXOS	150	197	107	0.74	0.54
SxN-141	SHA3/CBRD//NAXOS	144	188	102	0.70	0.49
SxN-142	SHA3/CBRD//NAXOS	131	182	115	0.75	0.56
SxN-143	SHA3/CBRD//NAXOS	132	182	100	0.73	0.54
SxN-144	SHA3/CBRD//NAXOS	129	180	103	0.22	0.05
SxN-145	SHA3/CBRD//NAXOS	131	182	92	0.60	0.36
SxN-146	SHA3/CBRD//NAXOS	154	193	107	0.32	0.10
SxN-147	SHA3/CBRD//NAXOS	154	193	110	0.44	0.19
SxN-148	SHA3/CBRD//NAXOS	139	191	116	0.18	0.03
SxN-151	SHA3/CBRD//NAXOS	152	197	114	0.45	0.21

Line	Pedigree	DH	DM	PH	GI	Square_GI
SxN-155	SHA3/CBRD//NAXOS	151	195	121	0.76	0.58
SxN-157	SHA3/CBRD//NAXOS	157	207	87	0.69	0.48
SxN-158	SHA3/CBRD//NAXOS	146	186	88	0.76	0.58
SxN-159	SHA3/CBRD//NAXOS	157	197	88	0.62	0.38
SxN-160	SHA3/CBRD//NAXOS	160	197	143	0.80	0.64
SxN-161	SHA3/CBRD//NAXOS	133	182	100	0.72	0.52
SxN-162	SHA3/CBRD//NAXOS	161	200	141	0.80	0.64
SxN-163	SHA3/CBRD//NAXOS	150	193	109	0.68	0.47
SxN-164	SHA3/CBRD//NAXOS	153	199	112	0.78	0.61
SxN-165	SHA3/CBRD//NAXOS	131	182	114	0.80	0.64
SxN-166	SHA3/CBRD//NAXOS	134	184	107	0.66	0.43
SxN-167	SHA3/CBRD//NAXOS	132	182	90	0.80	0.64
SxN-168	SHA3/CBRD//NAXOS	148	193	112	0.78	0.61
SxN-169	SHA3/CBRD//NAXOS	131	184	106	0.22	0.05
SxN-170	SHA3/CBRD//NAXOS	132	182	118	0.74	0.55
SxN-171	SHA3/CBRD//NAXOS	133	182	109	0.58	0.34
SxN-172	SHA3/CBRD//NAXOS	148	188	111	0.73	0.53
SxN-173	SHA3/CBRD//NAXOS	148	186	101	0.83	0.70
SxN-175	SHA3/CBRD//NAXOS	123	174	76	0.78	0.61
SxN-176	SHA3/CBRD//NAXOS	150	193	91	0.64	0.41
SxN-180	SHA3/CBRD//NAXOS	157	195	115	0.74	0.55
SxN-182	SHA3/CBRD//NAXOS	131	177	99	0.44	0.19

Appendix C

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Line	Pedigree	GI	SQRT_GI
Parent 1	Naxos(x3)	0.11	0.33
Parent 2	SHA3/CBRD	0.16	0.40
SxN-001	SHA3/CBRD//NAXOS	0.09	0.30
SxN-002	SHA3/CBRD//NAXOS	0.44	0.66
SxN-003	SHA3/CBRD//NAXOS	0.05	0.23
SxN-004	SHA3/CBRD//NAXOS	0.43	0.65
SxN-005	SHA3/CBRD//NAXOS	0.31	0.56
SxN-006	SHA3/CBRD//NAXOS	0.24	0.49
SxN-007	SHA3/CBRD//NAXOS	0.10	0.31
SxN-008	SHA3/CBRD//NAXOS	0.18	0.42
SxN-009	SHA3/CBRD//NAXOS	0.07	0.26
SxN-010	SHA3/CBRD//NAXOS	0.00	0.04
SxN-011	SHA3/CBRD//NAXOS	0.21	0.46
SxN-012	SHA3/CBRD//NAXOS	0.44	0.66
SxN-013	SHA3/CBRD//NAXOS	0.40	0.63
SxN-014	SHA3/CBRD//NAXOS	0.27	0.52
SxN-015	SHA3/CBRD//NAXOS	0.45	0.67
SxN-016	SHA3/CBRD//NAXOS	0.22	0.47
SxN-017	SHA3/CBRD//NAXOS	0.17	0.41
SxN-018	SHA3/CBRD//NAXOS	0.18	0.42
SxN-019	SHA3/CBRD//NAXOS	0.09	0.30
SxN-020	SHA3/CBRD//NAXOS	0.03	0.19
SxN-021	SHA3/CBRD//NAXOS	0.04	0.19
SxN-022	SHA3/CBRD//NAXOS	0.45	0.67
SxN-023	SHA3/CBRD//NAXOS	0.11	0.33
SxN-024	SHA3/CBRD//NAXOS	0.03	0.16
SxN-025	SHA3/CBRD//NAXOS	0.01	0.11
SxN-026	SHA3/CBRD//NAXOS	0.07	0.26
SxN-027	SHA3/CBRD//NAXOS	0.13	0.36
SxN-028	SHA3/CBRD//NAXOS	0.45	0.67
SxN-029	SHA3/CBRD//NAXOS	0.05	0.22
SxN-030	SHA3/CBRD//NAXOS	0.01	0.09
SxN-031	SHA3/CBRD//NAXOS	0.68	0.82
SxN-032	SHA3/CBRD//NAXOS	0.02	0.14

Line	Pedigree	GI	SQRT_GI
SxN-033	SHA3/CBRD//NAXOS	0.15	0.39
SxN-034	SHA3/CBRD//NAXOS	0.04	0.20
SxN-035	SHA3/CBRD//NAXOS	0.45	0.67
SxN-036	SHA3/CBRD//NAXOS	0.17	0.41
SxN-037	SHA3/CBRD//NAXOS	0.01	0.12
SxN-038	SHA3/CBRD//NAXOS	0.61	0.78
SxN-039	SHA3/CBRD//NAXOS	0.49	0.70
SxN-040	SHA3/CBRD//NAXOS	0.23	0.48
SxN-041	SHA3/CBRD//NAXOS	0.33	0.58
SxN-042	SHA3/CBRD//NAXOS	0.51	0.71
SxN-043	SHA3/CBRD//NAXOS	0.14	0.37
SxN-044	SHA3/CBRD//NAXOS	0.03	0.16
SxN-045	SHA3/CBRD//NAXOS	0.01	0.10
SxN-046	SHA3/CBRD//NAXOS	0.25	0.50
SxN-047	SHA3/CBRD//NAXOS	0.13	0.36
SxN-048	SHA3/CBRD//NAXOS	0.25	0.50
SxN-050	SHA3/CBRD//NAXOS	0.01	0.09
SxN-051	SHA3/CBRD//NAXOS	0.07	0.27
SxN-052	SHA3/CBRD//NAXOS	0.25	0.50
SxN-053	SHA3/CBRD//NAXOS	0.40	0.63
SxN-054	SHA3/CBRD//NAXOS	0.08	0.28
SxN-055	SHA3/CBRD//NAXOS	0.01	0.11
SxN-056	SHA3/CBRD//NAXOS	0.04	0.21
SxN-057	SHA3/CBRD//NAXOS	0.09	0.29
SxN-058	SHA3/CBRD//NAXOS	0.16	0.40
SxN-059	SHA3/CBRD//NAXOS	0.26	0.51
SxN-060	SHA3/CBRD//NAXOS	0.33	0.58
SxN-061	SHA3/CBRD//NAXOS	0.37	0.61
SxN-062	SHA3/CBRD//NAXOS	0.00	0.03
SxN-063	SHA3/CBRD//NAXOS	0.51	0.71
SxN-064	SHA3/CBRD//NAXOS	0.61	0.78
SxN-065	SHA3/CBRD//NAXOS	0.31	0.55
SxN-066	SHA3/CBRD//NAXOS	0.04	0.20
SxN-067	SHA3/CBRD//NAXOS	0.48	0.69
SxN-068	SHA3/CBRD//NAXOS	0.06	0.25
SxN-069	SHA3/CBRD//NAXOS	0.21	0.45
SxN-070	SHA3/CBRD//NAXOS	0.03	0.17
SxN-071	SHA3/CBRD//NAXOS	0.07	0.26
SxN-072	SHA3/CBRD//NAXOS	0.06	0.25

Line	Pedigree	GI	SQRT_GI
SxN-073	SHA3/CBRD//NAXOS	0.12	0.34
SxN-074	SHA3/CBRD//NAXOS	0.02	0.15
SxN-075	SHA3/CBRD//NAXOS	0.56	0.75
SxN-076	SHA3/CBRD//NAXOS	0.29	0.54
SxN-077	SHA3/CBRD//NAXOS	0.01	0.09
SxN-078	SHA3/CBRD//NAXOS	0.27	0.52
SxN-079	SHA3/CBRD//NAXOS	0.32	0.57
SxN-080	SHA3/CBRD//NAXOS	0.04	0.19
SxN-081	SHA3/CBRD//NAXOS	0.04	0.20
SxN-082	SHA3/CBRD//NAXOS	0.10	0.31
SxN-083	SHA3/CBRD//NAXOS	0.02	0.13
SxN-084	SHA3/CBRD//NAXOS	0.50	0.71
SxN-085	SHA3/CBRD//NAXOS	0.64	0.80
SxN-086	SHA3/CBRD//NAXOS	0.42	0.65
SxN-087	SHA3/CBRD//NAXOS	0.03	0.18
SxN-088	SHA3/CBRD//NAXOS	0.53	0.73
SxN-089	SHA3/CBRD//NAXOS	0.45	0.67
SxN-090	SHA3/CBRD//NAXOS	0.25	0.50
SxN-091	SHA3/CBRD//NAXOS	0.73	0.86
SxN-092	SHA3/CBRD//NAXOS	0.38	0.61
SxN-093	SHA3/CBRD//NAXOS	0.35	0.59
SxN-094	SHA3/CBRD//NAXOS	0.13	0.36
SxN-095	SHA3/CBRD//NAXOS	0.03	0.17
SxN-096	SHA3/CBRD//NAXOS	0.28	0.53
SxN-097	SHA3/CBRD//NAXOS	0.01	0.11
SxN-098	SHA3/CBRD//NAXOS	0.08	0.28
SxN-099	SHA3/CBRD//NAXOS	0.11	0.33
SxN-100	SHA3/CBRD//NAXOS	0.84	0.91
SxN-101	SHA3/CBRD//NAXOS	0.67	0.82
SxN-102	SHA3/CBRD//NAXOS	0.13	0.35
SxN-103	SHA3/CBRD//NAXOS	0.70	0.84
SxN-104	SHA3/CBRD//NAXOS	0.24	0.49
SxN-105	SHA3/CBRD//NAXOS	0.70	0.84
SxN-106	SHA3/CBRD//NAXOS	0.58	0.76
SxN-107	SHA3/CBRD//NAXOS	0.28	0.53
SxN-108	SHA3/CBRD//NAXOS	0.06	0.25
SxN-109	SHA3/CBRD//NAXOS	0.27	0.52
SxN-110	SHA3/CBRD//NAXOS	0.08	0.28
SxN-111	SHA3/CBRD//NAXOS	0.05	0.22

Line	Pedigree	GI	SQRT_GI
SxN-112	SHA3/CBRD//NAXOS	0.05	0.23
SxN-113	SHA3/CBRD//NAXOS	0.03	0.18
SxN-114	SHA3/CBRD//NAXOS	0.07	0.26
SxN-115	SHA3/CBRD//NAXOS	0.11	0.33
SxN-116	SHA3/CBRD//NAXOS	0.03	0.19
SxN-117	SHA3/CBRD//NAXOS	0.09	0.31
SxN-118	SHA3/CBRD//NAXOS	0.06	0.25
SxN-119	SHA3/CBRD//NAXOS	0.22	0.47
SxN-120	SHA3/CBRD//NAXOS	0.20	0.45
SxN-121	SHA3/CBRD//NAXOS	0.05	0.22
SxN-122	SHA3/CBRD//NAXOS	0.07	0.26
SxN-123	SHA3/CBRD//NAXOS	0.06	0.24
SxN-124	SHA3/CBRD//NAXOS	0.11	0.33
SxN-125	SHA3/CBRD//NAXOS	0.18	0.42
SxN-126	SHA3/CBRD//NAXOS	0.01	0.11
SxN-127	SHA3/CBRD//NAXOS	0.30	0.55
SxN-128	SHA3/CBRD//NAXOS	0.66	0.81
SxN-129	SHA3/CBRD//NAXOS	0.23	0.48
SxN-130	SHA3/CBRD//NAXOS	0.25	0.50
SxN-131	SHA3/CBRD//NAXOS	0.02	0.12
SxN-132	SHA3/CBRD//NAXOS	0.05	0.23
SxN-133	SHA3/CBRD//NAXOS	0.15	0.38
SxN-134	SHA3/CBRD//NAXOS	0.39	0.62
SxN-135	SHA3/CBRD//NAXOS	0.16	0.39
SxN-136	SHA3/CBRD//NAXOS	0.47	0.68
SxN-137	SHA3/CBRD//NAXOS	0.15	0.39
SxN-138	SHA3/CBRD//NAXOS	0.20	0.45
SxN-139	SHA3/CBRD//NAXOS	0.26	0.51
SxN-140	SHA3/CBRD//NAXOS	0.03	0.18
SxN-141	SHA3/CBRD//NAXOS	0.04	0.20
SxN-142	SHA3/CBRD//NAXOS	0.36	0.60
SxN-143	SHA3/CBRD//NAXOS	0.25	0.50
SxN-144	SHA3/CBRD//NAXOS	0.06	0.25
SxN-145	SHA3/CBRD//NAXOS	0.28	0.52
SxN-146	SHA3/CBRD//NAXOS	0.02	0.15
SxN-147	SHA3/CBRD//NAXOS	0.02	0.16
SxN-148	SHA3/CBRD//NAXOS	0.08	0.28
SxN-151	SHA3/CBRD//NAXOS	0.08	0.27
SxN-155	SHA3/CBRD//NAXOS	0.05	0.22

Line	Pedigree	GI	SQRT_GI
SxN-157	SHA3/CBRD//NAXOS	0.00	0.04
SxN-158	SHA3/CBRD//NAXOS	0.01	0.11
SxN-159	SHA3/CBRD//NAXOS	0.04	0.19
SxN-160	SHA3/CBRD//NAXOS	0.21	0.46
SxN-161	SHA3/CBRD//NAXOS	0.48	0.69
SxN-162	SHA3/CBRD//NAXOS	0.12	0.35
SxN-163	SHA3/CBRD//NAXOS	0.07	0.26
SxN-164	SHA3/CBRD//NAXOS	0.12	0.34
SxN-165	SHA3/CBRD//NAXOS	0.34	0.58
SxN-166	SHA3/CBRD//NAXOS	0.16	0.39
SxN-167	SHA3/CBRD//NAXOS	0.47	0.69
SxN-168	SHA3/CBRD//NAXOS	0.15	0.38
SxN-169	SHA3/CBRD//NAXOS	0.00	0.04
SxN-170	SHA3/CBRD//NAXOS	0.22	0.46
SxN-171	SHA3/CBRD//NAXOS	0.04	0.21
SxN-172	SHA3/CBRD//NAXOS	0.02	0.13
SxN-173	SHA3/CBRD//NAXOS	0.24	0.49
SxN-175	SHA3/CBRD//NAXOS	0.52	0.72
SxN-176	SHA3/CBRD//NAXOS	0.06	0.24
SxN-180	SHA3/CBRD//NAXOS	0.20	0.45
SxN-182	SHA3/CBRD//NAXOS	0.05	0.23

Appendix D

Percentage of germination of most dormant lines from Vollebekk 2016 trial

Rutes	Lines	Name	% Germination
2124	SxN-159	SHA3/CBRD//NAXOS	83.67
2139	SxN-083	SHA3/CBRD//NAXOS	43.08
2229	1424	T7347	56.18
2312	SxN-018	SHA3/CBRD//NAXOS	94
2412	39	Naxos (x3)	95.83
2421	SxN-081	SHA3/CBRD//NAXOS	95.83
2429	SxN-025	SHA3/CBRD//NAXOS	98
2430	SxN-126	SHA3/CBRD//NAXOS	92.37
2626	1424	T7347	59.82

Appendix E

Germination Index data from greenhouse experiments for parents in 2017

Name	Rep	GI_27/22°C	GI_22/17°C	GI_17/12°C
Naxos	IA	0.61	0.47	0.28
Naxos	IB	0.65	0.32	0.38
Naxos	IIA	0.70	0.30	0.26
Naxos	IIB	0.63	0.28	0.30
SHA3/CBRD	IA	0.63	0.62	0.52
SHA3/CBRD	IB	0.57	0.41	0.53
SHA3/CBRD	IIA	0.46	0.61	0.41
SHA3/CBRD	IIB	0.59	0.60	0.50

Appendix F

Falling Number data from mist-irrigated trial Vollebekk 2016

Line	Name/Pedigree	DH	PH	FN
14	Saar	60.50	72.90	109
29	SHA3/CBRD	61.75	75.22	82
39	Naxos (x3)	59.00	69.12	231
45	GN03509 (Krabat)	61.00	65.23	222
1419	Polkka	61.50	77.99	122
1424	T7347	63.00	83.96	407
SxN-001	SHA3/CBRD//NAXOS	64.00	77.78	202
SxN-003	SHA3/CBRD//NAXOS	62.50	66.89	174
SxN-004	SHA3/CBRD//NAXOS	62.00	77.06	164
SxN-005	SHA3/CBRD//NAXOS	59.00	87.46	161
SxN-006	SHA3/CBRD//NAXOS	61.00	68.76	123
SxN-007	SHA3/CBRD//NAXOS	61.50	75.01	170
SxN-008	SHA3/CBRD//NAXOS	60.00	63.69	166
SxN-009	SHA3/CBRD//NAXOS	60.00	73.69	167
SxN-010	SHA3/CBRD//NAXOS	57.00	72.57	109
SxN-011	SHA3/CBRD//NAXOS	57.00	72.67	75
SxN-012	SHA3/CBRD//NAXOS	60.00	88.74	68
SxN-013	SHA3/CBRD//NAXOS	60.00	58.76	153
SxN-014	SHA3/CBRD//NAXOS	59.00	69.76	162
SxN-015	SHA3/CBRD//NAXOS	64.00	78.98	166
SxN-017	SHA3/CBRD//NAXOS	61.00	77.67	300
SxN-018	SHA3/CBRD//NAXOS	63.00	72.78	222
SxN-019	SHA3/CBRD//NAXOS	64.00	74.67	163
SxN-020	SHA3/CBRD//NAXOS	61.00	89.31	148
SxN-021	SHA3/CBRD//NAXOS	62.00	79.67	129
SxN-023	SHA3/CBRD//NAXOS	63.50	80.35	158
SxN-024	SHA3/CBRD//NAXOS	57.00	68.69	198
SxN-025	SHA3/CBRD//NAXOS	61.00	53.05	181
SxN-026	SHA3/CBRD//NAXOS	57.00	74.05	113
SxN-027	SHA3/CBRD//NAXOS	57.00	75.12	147
SxN-029	SHA3/CBRD//NAXOS	62.00	65.89	148
SxN-030	SHA3/CBRD//NAXOS	58.50	70.48	87
SxN-031	SHA3/CBRD//NAXOS	62.00	67.06	260
SxN-032	SHA3/CBRD//NAXOS	58.00	64.78	223

Line	Name/Pedigree	DH	PH	FN
SxN-033	SHA3/CBRD//NAXOS	59.00	87.78	127
SxN-034	SHA3/CBRD//NAXOS	58.50	76.15	190
SxN-035	SHA3/CBRD//NAXOS	63.00	45.04	86
SxN-036	SHA3/CBRD//NAXOS	58.50	66.28	259
SxN-038	SHA3/CBRD//NAXOS	61.50	72.91	72
SxN-039	SHA3/CBRD//NAXOS	61.00	71.74	250
SxN-040	SHA3/CBRD//NAXOS	60.00	86.67	177
SxN-041	SHA3/CBRD//NAXOS	61.00	88.04	204
SxN-043	SHA3/CBRD//NAXOS	61.00	86.04	182
SxN-044	SHA3/CBRD//NAXOS	62.00	65.57	175
SxN-045	SHA3/CBRD//NAXOS	61.00	66.50	116
SxN-046	SHA3/CBRD//NAXOS	61.00	69.12	130
SxN-047	SHA3/CBRD//NAXOS	61.00	63.25	257
SxN-048	SHA3/CBRD//NAXOS	62.00	64.03	148
SxN-050	SHA3/CBRD//NAXOS	62.00	79.68	170
SxN-051	SHA3/CBRD//NAXOS	58.00	62.50	135
SxN-052	SHA3/CBRD//NAXOS	57.00	63.67	188
SxN-053	SHA3/CBRD//NAXOS	61.00	97.68	164
SxN-054	SHA3/CBRD//NAXOS	63.00	89.31	108
SxN-055	SHA3/CBRD//NAXOS	63.00	83.00	90
SxN-056	SHA3/CBRD//NAXOS	61.00	78.76	225
SxN-057	SHA3/CBRD//NAXOS	60.00	78.00	239
SxN-060	SHA3/CBRD//NAXOS	59.00	76.50	115
SxN-061	SHA3/CBRD//NAXOS	61.00	68.20	190
SxN-062	SHA3/CBRD//NAXOS	59.00	91.67	196
SxN-063	SHA3/CBRD//NAXOS	59.00	59.76	69
SxN-064	SHA3/CBRD//NAXOS	60.50	79.94	223
SxN-065	SHA3/CBRD//NAXOS	58.00	66.67	62
SxN-066	SHA3/CBRD//NAXOS	60.00	85.25	234
SxN-067	SHA3/CBRD//NAXOS	63.00	73.68	202
SxN-068	SHA3/CBRD//NAXOS	58.00	75.57	134
SxN-070	SHA3/CBRD//NAXOS	61.00	65.78	63
SxN-071	SHA3/CBRD//NAXOS	63.00	86.68	63
SxN-072	SHA3/CBRD//NAXOS	63.00	67.31	240
SxN-073	SHA3/CBRD//NAXOS	64.50	66.04	155
SxN-074	SHA3/CBRD//NAXOS	60.00	71.57	90
SxN-075	SHA3/CBRD//NAXOS	62.00	70.17	99
SxN-078	SHA3/CBRD//NAXOS	63.00	63.69	66
SxN-079	SHA3/CBRD//NAXOS	60.00	55.76	80

Line	Name/Pedigree	DH	PH	FN
SxN-080	SHA3/CBRD//NAXOS	63.50	62.05	203
SxN-081	SHA3/CBRD//NAXOS	61.00	69.31	108
SxN-082	SHA3/CBRD//NAXOS	61.50	58.72	203
SxN-083	SHA3/CBRD//NAXOS	63.00	58.76	103
SxN-084	SHA3/CBRD//NAXOS	62.00	89.67	166
SxN-086	SHA3/CBRD//NAXOS	59.00	84.74	234
SxN-087	SHA3/CBRD//NAXOS	60.00	58.67	197
SxN-088	SHA3/CBRD//NAXOS	63.00	63.20	79
SxN-089	SHA3/CBRD//NAXOS	62.00	82.09	199
SxN-091	SHA3/CBRD//NAXOS	58.00	80.71	107
SxN-093	SHA3/CBRD//NAXOS	61.00	81.95	260
SxN-094	SHA3/CBRD//NAXOS	61.00	66.12	204
SxN-095	SHA3/CBRD//NAXOS	62.00	77.48	61
SxN-096	SHA3/CBRD//NAXOS	58.00	68.20	160
SxN-097	SHA3/CBRD//NAXOS	62.00	68.12	267
SxN-098	SHA3/CBRD//NAXOS	62.00	42.57	144
SxN-099	SHA3/CBRD//NAXOS	60.00	79.76	267
SxN-103	SHA3/CBRD//NAXOS	61.00	80.25	91
SxN-104	SHA3/CBRD//NAXOS	60.00	76.18	235
SxN-105	SHA3/CBRD//NAXOS	57.00	75.20	116
SxN-106	SHA3/CBRD//NAXOS	58.00	83.68	119
SxN-108	SHA3/CBRD//NAXOS	62.00	83.08	275
SxN-110	SHA3/CBRD//NAXOS	60.00	80.72	201
SxN-112	SHA3/CBRD//NAXOS	59.50	73.78	198
SxN-113	SHA3/CBRD//NAXOS	61.00	72.12	202
SxN-114	SHA3/CBRD//NAXOS	58.00	61.76	203
SxN-115	SHA3/CBRD//NAXOS	60.00	78.67	145
SxN-116	SHA3/CBRD//NAXOS	60.00	66.50	203
SxN-117	SHA3/CBRD//NAXOS	59.50	78.41	206
SxN-118	SHA3/CBRD//NAXOS	60.50	78.74	187
SxN-119	SHA3/CBRD//NAXOS	60.00	82.06	166
SxN-120	SHA3/CBRD//NAXOS	60.50	66.94	175
SxN-121	SHA3/CBRD//NAXOS	59.00	61.67	130
SxN-122	SHA3/CBRD//NAXOS	59.00	73.67	242
SxN-123	SHA3/CBRD//NAXOS	60.00	63.68	232
SxN-124	SHA3/CBRD//NAXOS	60.00	75.90	215
SxN-126	SHA3/CBRD//NAXOS	62.00	73.90	93
SxN-127	SHA3/CBRD//NAXOS	63.00	87.48	159
SxN-128	SHA3/CBRD//NAXOS	60.00	77.76	62

Line	Name/Pedigree	DH	PH	FN
SxN-129	SHA3/CBRD//NAXOS	63.50	83.94	104
SxN-130	SHA3/CBRD//NAXOS	61.00	66.20	104
SxN-131	SHA3/CBRD//NAXOS	59.00	77.48	160
SxN-133	SHA3/CBRD//NAXOS	61.00	86.50	129
SxN-134	SHA3/CBRD//NAXOS	60.00	77.06	49
SxN-135	SHA3/CBRD//NAXOS	60.00	60.86	205
SxN-136	SHA3/CBRD//NAXOS	58.00	84.67	184
SxN-137	SHA3/CBRD//NAXOS	60.00	76.74	145
SxN-138	SHA3/CBRD//NAXOS	58.00	65.25	79
SxN-140	SHA3/CBRD//NAXOS	59.00	78.69	101
SxN-141	SHA3/CBRD//NAXOS	58.00	65.04	78
SxN-142	SHA3/CBRD//NAXOS	58.00	65.05	117
SxN-143	SHA3/CBRD//NAXOS	61.00	60.12	140
SxN-145	SHA3/CBRD//NAXOS	63.00	69.31	135
SxN-146	SHA3/CBRD//NAXOS	63.00	71.04	186
SxN-147	SHA3/CBRD//NAXOS	61.00	67.74	231
SxN-148	SHA3/CBRD//NAXOS	61.00	85.48	105
SxN-151	SHA3/CBRD//NAXOS	62.00	72.50	130
SxN-155	SHA3/CBRD//NAXOS	63.00	87.63	246
SxN-157	SHA3/CBRD//NAXOS	61.00	68.00	256
SxN-158	SHA3/CBRD//NAXOS	61.00	50.05	113
SxN-159	SHA3/CBRD//NAXOS	66.00	76.05	350
SxN-160	SHA3/CBRD//NAXOS	58.00	70.25	78
SxN-161	SHA3/CBRD//NAXOS	62.00	67.48	100
SxN-162	SHA3/CBRD//NAXOS	63.00	86.67	78
SxN-163	SHA3/CBRD//NAXOS	61.00	64.76	159
SxN-164	SHA3/CBRD//NAXOS	58.00	67.89	66
SxN-165	SHA3/CBRD//NAXOS	59.50	75.68	174
SxN-166	SHA3/CBRD//NAXOS	61.00	71.00	67
SxN-167	SHA3/CBRD//NAXOS	61.00	61.06	62
SxN-168	SHA3/CBRD//NAXOS	59.00	83.20	106
SxN-169	SHA3/CBRD//NAXOS	59.00	66.67	140
SxN-170	SHA3/CBRD//NAXOS	60.00	73.67	62
SxN-171	SHA3/CBRD//NAXOS	61.00	84.89	157
SxN-172	SHA3/CBRD//NAXOS	59.00	82.76	196
SxN-173	SHA3/CBRD//NAXOS	61.00	66.67	204
SxN-175	SHA3/CBRD//NAXOS	59.50	49.08	227
SxN-176	SHA3/CBRD//NAXOS	63.00	76.25	223
SxN-182	SHA3/CBRD//NAXOS	59.00	62.04	113

Appendix G

Weather data Vollebekk 2016

Time	Mean T (°C)	Max T (°C)	Min T (°C)	Prec (mm)	RH (%)
10.jul	15.4	19.8	9.1	0.0	78.6
11.jul	16.3	19.8	13.4	1.6	79.8
12.jul	16.1	21.3	11.7	0.2	78.9
13.jul	15.6	19.7	11.5	11.4	84.5
14.jul	16.3	20.4	13.2	0.0	81.4
15.jul	17.9	23.1	14.0	0.2	65.8
16.jul	16.6	20.1	13.9	0.0	69.2
17.jul	14.1	18.9	8.0	0.2	64.5
18.jul	15.3	21.3	6.4	0.0	64.0
19.jul	15.4	19.5	8.1	0.0	75.1
20.jul	18.7	24.4	13.6	0.0	72.7
21.jul	20.1	25.7	11.5	0.0	65.4
22.jul	20.3	25.6	14.8	0.2	66.6
23.jul	19.5	23.7	14.7	0.0	69.7
24.jul	19.9	25.0	14.3	0.0	69.5
25.jul	19.3	24.7	15.6	0.6	71.8
26.jul	17.6	20.7	14.0	11.6	76.8
27.jul	16.0	19.3	12.9	1.8	73.4
28.jul	17.1	21.2	13.9	3.0	74.4
29.jul	15.8	19.8	11.4	3.2	77.1
30.jul	16.2	20.1	12.3	0.4	72.8
31.jul	14.7	20.0	7.3	0.0	64.9
01.aug	14.7	19.3	9.0	0.0	67.0
02.aug	15.0	20.5	8.3	0.0	66.6
03.aug	14.0	18.5	7.1	13.2	72.6
04.aug	15.5	20.4	12.5	9.0	81.1
05.aug	16.4	20.2	14.1	31.8	83.8
06.aug	15.0	17.6	13.5	8.2	86.2
07.aug	14.8	17.8	11.9	1.6	84.3
08.aug	14.9	18.2	11.5	7.8	70.0
09.aug	13.1	18.0	10.1	9.0	68.7
10.aug	10.3	15.1	7.3	17.0	70.7

Time	Mean T (°C)	Max T (°C)	Min T (°C)	Prec (mm)	RH (%)
11.aug	11.4	16.7	4.6	0.0	63.0
12.aug	11.3	15.6	5.0	7.0	87.3
13.aug	15.6	20.0	12.6	0.0	76.4
14.aug	15.3	20.5	10.0	0.0	64.9
15.aug	14.4	20.3	8.4	0.0	64.3
16.aug	15.5	22.0	9.3	0.0	65.1
17.aug	16.4	23.0	7.2	0.0	58.2
18.aug	16.3	21.5	9.4	0.0	62.3
19.aug	15.5	21.2	9.0	0.0	68.7
20.aug	15.3	19.8	12.4	12.2	81.7
21.aug	15.5	20.6	10.6	0.4	82.1
22.aug	14.6	18.6	10.0	4.6	85.8
23.aug	16.5	22.2	12.5	0.0	79.3
24.aug	15.6	17.4	13.9	0.0	84.4
25.aug	17.2	18.1	16.7	4.2	90.6
26.aug	17.6	20.5	14.5	9.8	83.1
27.aug	15.3	20.3	8.8	0.0	64.0
28.aug	11.0	14.5	6.5	1.4	86.0
29.aug	11.3	15.3	6.3	2.6	80.6
30.aug	12.2	17.0	4.5	0.2	80.9
31.aug	14.9	20.1	8.1	0.0	81.6
01.sep	16.0	20.0	12.0	0.0	64.6
02.sep	13.9	17.0	9.3	0.0	72.1

Weather data Chengdu

2015					2016				
Time	Mean T (°C)	Max T (°C)	Min T (°C)	PP (mm)	Max T (°C)	Min T (°C)	Mean T (°C)	RH (%)	PP (mm)
10-Apr	15.9	20.5	11.5	0.0	25.2	14.0	19.5	70.0	0.0
11-Apr	16.5	21.3	13.0	0.0	23.2	14.8	18.5	78.0	1.2
12-Apr	17.6	25.7	11.4	0.0	23.4	16.5	19.4	78.0	0.0
13-Apr	18.7	24.4	13.5	0.0	21.4	17.3	19.1	79.0	0.0
14-Apr	18.1	23.4	13.6	0.2	24.1	15.3	18.4	77.0	5.7
15-Apr	19.3	29.2	10.9	0.0	21.8	14.6	17.6	87.0	10.7
16-Apr	21.5	29.4	13.9	0.0	24.3	13.7	18.1	73.0	0.0
17-Apr	24.4	31.1	18.1	0.0	20.8	13.9	17.2	69.0	9.4
18-Apr	21.3	26.7	18.8	1.5	24.1	11.2	17.5	62.0	0.0
19-Apr	16.6	19.6	14.1	9.5	22.9	16.1	19.0	64.0	0.0
20-Apr	17.1	23.8	11.8	0.0	27.0	15.5	20.6	64.0	4.3
21-Apr	17.7	19.8	15.8	0.6	21.7	16.2	18.6	68.0	0.0
22-Apr	15.9	18.2	13.8	2.6	20.5	15.8	17.6	77.0	0.5
23-Apr	16.2	20.3	13.1	0.0	20.3	15.0	17.4	72.0	0.2
24-Apr	18.0	25.4	11.3	0.0	22.5	14.8	17.7	74.0	0.0
25-Apr	20.2	27.0	16.0	0.0	22.4	15.2	17.9	80.0	0.3
26-Apr	21.5	27.5	15.3	0.0	26.2	16.2	20.2	72.0	0.0
27-Apr	23.1	31.0	16.6	0.0	26.8	15.0	20.9	66.0	0.0
28-Apr	24.2	32.7	16.6	0.0	28.4	17.2	22.4	54.0	0.0
29-Apr	24.4	33.3	16.4	0.0	22.7	17.5	19.5	74.0	2.4
30-Apr	25.5	31.0	21.4	0.0	24.6	14.4	19.3	78.0	0.1
1-May	21.7	27.2	15.6	6.2	27.5	16.7	22.0	69.0	0.6
2-May	22.6	30.4	15.6	0.0	28.5	19.5	23.7	62.0	0.0
3-May	22.2	24.7	20.8	0.0	29.6	19.6	23.9	47.0	0.0
4-May	21.0	26.3	16.2	0.0	32.1	20.0	25.5	47.0	0.0
5-May	20.5	24.8	17.5	0.0	33.4	20.8	27.2	59.0	0.0
6-May	21.5	29.4	14.5	0.0	25.9	20.0	23.2	77.0	44.3
7-May	24.0	29.8	18.6	0.0	22.2	17.9	20.0	67.0	0.3
8-May	23.2	31.1	16.0	4.9	19.1	15.7	17.6	64.0	0.0
9-May	23.8	31.4	16.4	0.0	29.2	14.9	20.7	68.0	0.0
10-May	23.8	31.9	16.1	0.0	30.6	16.7	23.5	65.0	0.0
11-May	21.6	26.6	18.1	0.0	33.0	18.5	25.6	56.0	0.0
12-May	23.4	28.9	18.5	0.0	28.4	22.6	24.9	55.0	0.0
13-May	23.7	31.2	16.6	0.0	22.5	17.5	20.0	74.0	0.0
14-May	24.5	33.3	17.6	0.0	18.5	15.9	17.4	79.0	0.0

	2015				2016				
Time	**Mean T (°C)**	**Max T (°C)**	**Min T (°C)**	**PP (mm)**	**Max T (°C)**	**Min T (°C)**	**Mean T (°C)**	**RH (%)**	**PP (mm)**
15-May	26.7	35.3	21.2	0.8	25.6	14.2	19.1	64.0	0.0
16-May	24.5	29.2	21.8	1.8	missing	missing	missing	missing	missing
17-May	21.4	26.4	18.1	7.6	27.3	18.7	22.8	51.0	0.0
18-May	24.6	32.2	18.4	0.0	27.5	18.2	23.3	55.0	0.0
19-May	26.8	32.7	21.8	0.0	26.4	18.7	22.4	73.0	0.0
20-May	26.5	33.8	21.2	2.4	28.5	18.2	22.6	71.0	0.0
21-May	21.0	24.2	17.4	5.1	24.3	19.5	22.3	70.0	0.0
22-May	21.5	27.6	17.8	10.0	22.2	18.2	19.2	89.0	0.0
23-May	23.7	30.1	18.8	0.0	25.6	16.9	20.5	75.0	6.0
24-May	23.5	28.3	20.5	44.0	27.0	17.3	22.2	71.0	0.0
25-May	21.6	24.7	19.3	2.4	23.1	16.2	20.0	85.0	0.0
26-May	22.4	26.3	19.6	0.0	19.9	14.6	16.8	76.0	0.0
27-May	24.1	29.1	19.4	0.0	21.2	15.9	17.8	75.0	0.0
28-May	24.9	28.3	21.9	0.2	25.9	17.0	20.7	71.0	0.0
29-May	23.3	30.0	18.1	8.3	28.4	19.2	23.4	69.0	0.0
30-May	26.3	33.3	20.8	0.4	27.4	21.8	24.3	69.0	0.9
31-May	27.3	32.1	22.6	0.0	30.4	21.8	25.3	68.0	0.0

Appendix H

Parent's description

Variety name	Pedigree	Origin	Habitat
SHA3/CBRD	Shanghai3/Catbird	CIMMYT	Spring
Naxos (x3)	Tordo/St.Mir808-Bastion//Minaret	Germany	Spring

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