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Evaluation of Fusarium Head Blight susceptibility by genotyping NILs for natural and transgenic *Rht1* dwarfing genes and phenotyping of wheat mutant lines treated with the *Fusarium* mycotoxin deoxynivalenol

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Tulln, März 2019

Declaration of authorship

I hereby declare that this thesis submitted has been written to the best of my knowledge and belief. All sources of information, direct or indirect sources, including graphs, tables and figures, used are acknowledged as references.

This work was not previously submitted to another educational institution for the purpose of receiving an academic degree.

Tulln, _____ _____

Date Signature

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Abstract

Fusarium Head Blight (FHB) caused by *Fusarium spp*. is a devastating fungal disease all over the world. By producing mycotoxins, the fungal disease is not only harmful for human and animal health, but also accounts for big losses in yield and grain quality. The most promising tool for FHB control is the use of FHB resistant cultivars. However, breeding for resistance is a challenge as FHB resistance in small grain cereals is a quantitative trait, influenced by multiple genes and by the environment.

It is well documented that the semi-dwarfing alleles *Rht-D1b* and *Rht-B1b* are strongly associated with FHB susceptibility. The question is whether this is due to a pleiotropic effect of the semi-dwarfing alleles or due to close linkage with a susceptibility factor. Therefore, the *Rht-D1b* allele was transferred through particle bombardment into four tall wheat cultivars with different genetic backgrounds to break the assumed linkage between short plants and increased FHB severity. In the obtained transgenic plants, the *Rht-D1b* allele is linked to green fluorescent protein (GFP). The fluorescent signal of GFP can be used for visual screening to identify plants being homozygous for transformed *Rht-D1b* allele. The transgenic plants were screened for the presence/absence of the transgenic *Rht-D1b* allele using molecular marker technology and fluorescence microscopy. The positively transformed plants, together with plants of the same genetic background but with natural semi-dwarfing allele will be phenotyped for FHB severity to allow a direct comparison of transgenic and natural *Rht-D1b* allele carriers.

In the second part of this thesis a mutant population carrying three promising candidate genes, which could provide resistance to FHB and the major mycotoxin deoxynivalenol (DON), has been evaluated. Targeting Induced Local Lesions in Genomes (TILLING) has been used as a reverse genetics approach to identify lines with deleterious mutations in the candidate genes. Selected lines were phenotyped in the greenhouse by treatment of spikelets, either with a spore suspension of *Fusarium graminearum* or DON. After evaluation of symptom severity, the results showed that none of the three candidate genes could be identified to contribute to FHB or DON resistance.

Keywords: Fusarium head blight, semi-dwarfing alleles, linkage, pleiotropy, *Rht-D1b*, resistance breeding, TILLING

Kurzfassung

Fusarium Head Blight (FHB) verursacht durch *Fusarium spp.* ist eine weltweit bedeutende Pilzkrankheit. Durch die Produktion von Mykotoxinen ist FHB nicht nur für die Gesundheit von Menschen und Tieren gefährlich, sondern verursacht auch hohe Ertrags- und Qualitätsverluste. Die Verwendung von resistenten Sorten ist der vielversprechendste Ansatz zur Bekämpfung von FHB. Die Resistenzzüchtung ist jedoch eine Herausforderung, da die Krankheitsresistenz ein quantitatives Merkmal ist, welches von mehreren Genen und der Umwelt beeinflusst wird.

Im ersten Teil der Masterarbeit wurde ein transgener Ansatz gewählt, um das Kurzstroh-Gen *Rht-D1b* in verschiedene langstrohige Weizensorten einzuschleusen. Dies wurde gemacht, um die Ursache des Zusammenhangs zwischen kurzstrohigen Sorten und erhöhter FHB Anfälligkeit zu untersuchen. In der Literatur ist bereits gut dokumentiert, dass die Kurzstroh-Gene *Rht-D1b* und *Rht-B1b* stark mit erhöhter FHB Anfälligkeit assoziiert sind. Die Frage ist, ob dies auf einen pleiotropen Effekt des Kurzstroh-Gens selbst oder auf eine enge genetische Kopplung mit einem Anfälligkeitsfaktor zurückzuführen ist. In den transgenen Pflanzen ist das *Rht-D1b* Allel an das grün fluoreszierende Protein (GFP) gebunden. Dieses Fluoreszenzsignal ermöglicht es, durch Mikroskopieren des Pollens homozygot transgene Pflanzen zu identifizieren. Die positiv transformierten Pflanzen wurden selektiert und dieses Pflanzenmaterial wird in weiterer Folge auf FHB Resistenz untersucht.

Im zweiten Teil der Masterarbeit wurden drei vielversprechende Kandidatengene getestet, die verantwortlich für die Resistenz gegenüber FHB und das Mykotoxin Deoxynivalenol (DON) sein könnten. Eine Methode der reversen Genetik ist Targeting Induced Local Lesions in Genomes (TILLING). Diese Methode wird zur zielgerichteten Suche nach Mutanten verwendet, die induzierte Polymorphismen innerhalb der Kandidatengene tragen. Die selektierten Linien wurden im Gewächshaus durch Inokulation der Ährchen mit einer Sporensuspension von *Fusarium graminearum* oder DON getestet. Nach Auswertung der Symptome zeigten die Ergebnisse, dass keines der drei Kandidatengene zur FHB oder DON Resistenz beiträgt.

Schlagwörter: Fusarium Head Blight, Verzwergungsgene, Linkage, Pleiotropie, *Rht-D1b*, Resistenzzüchtung, TILLING

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Formula 2: Formula for unpaired t-test. $x1, x2$ … sample means; s^2 … pooled sample variance; $n1, n2$ … sample size 75

List of abbreviations

A.....Arginine
AGES.....Austrian Agency for Health and Food Safety
BAES ..... The Federal Office for Food Safety
BC_nF_n ..... Backcross n, Filial Generation n
BF ..... Brightfield
BME ..... β-Mercaptoethanol
CIMMYT..... International Maize and Wheat Improvement Center
CTAB . Mixed Alykltrimethyl-Ammonium Bromide (Sigma M-7635)
D3G..... DON-3-O-glycoside
dai .....days after inoculation
DELLA..... named after conserved N-terminal amino acid sequence: aspartate, glutamate, leucine, leucine, alanine
DHPLC..... Denaturing High-Performance Liquid Chromatography
DNA ..... Deoxyribonucleic Acid
dNTP..... Deoxynucleoside Triphosphate
DON .....Deoxynivalenol
EDTA .....Ethylene-Diamine-Tetra-Acetic Acid
EMS .....Ethyl-methane-sulfonate
FHB..... Fusarium Head Blight
G .....Glutamine
GFP.....Green Fluorescent Protein
GOTAQ..... Green Mastermix for PCR
GS.....Genomic Selection
GTF..... Glycosyltransferases
HGA ..... Homogalacturonan
IFA .....Inter-University Department of Agrobiotechnology
IPM..... Integrated Pest Management
LfL..... Bavarian State Research Institute for Agriculture
MAS.....Marker-assisted Selection
NDAES .....North Dakota Agricultural Experiment Station
NIL ..... Near Isogenic Line
NIV..... Nivalenol
PAGE..... Polyacrylamide Gel Electrophoresis, Polyacrylamide Gel Electrophoresis
PCR ..... Polymerase Chain eaction
PFT .....pore-forming toxin-like
QTL..... Quantitative Trait Locus
rcf..... Relative Centrifugal Force
REP ..... Repeatability
RFLP .....Restriction Fragment Length Polymorphism
RH.....Radiation Hybrid
Rht .....Reduced height
RNA .....Ribonucleic Acid
rpm ..... Revolutions per Minute
SNP ..... Single Nucleotide Polymorphism
spp. .....species pluralis
SSD .....Single Seed Descent
SSR ..... Simple Sequence Repeat
T..... Tryptophan
TILLING..... Targeting Induced Local Lesions in Genomes
T_m..... melting temperature of primers
UGT .....UDP-Glycosyltransferase
UPS .....Ubiquitin/proteasome system
UV..... Ultraviolet
ZON .....Zearalenone

1. Introduction

1.1. The importance of wheat (*Triticum aestivum*) as a worldwide food crop

Wheat is widely grown all around the world and in many different environments. It is not only used for human nutrition, but also an important source of animal feed, or used in industry (Curtis, 2007). The global wheat production increased from about 580 million tons in 2000 to about 754 million tons in 2016/17 (IGC, 2018; Statista, 2018).

According to market information of AMA the acreage of wheat (*T. aestivum, T. durum*) in Austria amounted to about 295,000 hectares in 2018, with a grain harvest of about 1.4 million tons (AMA, 2018). The main production area of grains in Austria is Lower Austria, Upper Austria and Styria (BMLFUW, 2017).

The increase in wheat production can be explained by successful plant breeding combined with improved cultivation methods to provide better conditions for the crop to grow, rather than by an increase in agricultural area. For example, dwarf or semi-dwarf wheat cultivars allow a higher nitrogen fertilizer application without having a strong tendency to lodge. Also, improved chemical control of diseases and pests, as well as modern technologies in sowing and mechanical weed control contribute to this yield increase (Brown et al., 2014).

As the world population is growing, agricultural production should increase too, in order to cover the worldwide wheat demand for estimated 9.8 billion people in 2050 (UN DESA, 2017). Further expansion of agricultural land is a challenge, because land is also used for commercial and residential purposes of this growing population. As land is scarce, more food will have to be produced on less land. Therefore, plant breeding is one of the most important tools, not only to breed higher yielding crops, but also crops that are resistant to diseases and abiotic stress (Acquaah, 2012).

1.2. The relevance of Fusarium Head Blight as a devastating disease in wheat

Fusarium Head Blight (FHB) has been studied for far more than a century now. FHB was first described in England by W.G. Smith as “wheat scab” in 1884 (Arthur, 1891). In 1890 Kirchner (1906) reported the disease in Europe, Germany. Until now FHB is a major threat to global food and feed safety. Today, the genus *Fusarium* includes at least 300 phylogenetically distinct species, 20 species complexes and nine monotypic lineages. Most of the identified *Fusarium* pathogens belong to the *F. solani* complex, *F. sambucinum* complex, *F. fujikuroi* complex and *F. oxysporum* complex (O’Donnell et al., 2013, 2015).

Fusarium spp. cause diseases in a wide range of hosts. The fungus not only affects wheat, rye, barley or oats, but also melon, pepper and tomato. In small-grain cereals *Fusarium spp.* may infest roots, stems, leaves and heads, whereby the most important species are *F. avenaceum*, *F. culmorum*, *F. graminearum*, *F. poae*. The symptoms are ear blight, root and stem rot, and wilting. FHB not only decreases grain quality and yield, but the major concern of mycotoxin contamination of grains is human and animal health (Early, 2009; Schmale III and Bergstrom, 2003).

The occurrence of *Fusarium spp.* varies from year to year for several reasons, such as the weather conditions, previous crop or cultural practices. Hence, there is also a yearly variation in mycotoxin levels. In the US, Canada and Europe *F. graminearum* is the major cause of FHB. This *Fusarium* species is most favored by warm temperatures and high moisture during anthesis (MacInnes and Fogelman, 1923; McMullen et al., 1997). *F. culmorum* is the dominant species in cooler wheat growing areas, such as northern, central, western Europe (Miller, 1994; Parry et al., 1995; Wagacha and Muthomi, 2007).

1.2.1. Lifecycle of the pathogen

The genus *Fusarium* is part of the phylum *Ascomycota*, class *Ascomycetes*, order *Hypocreales*. *Fusarium* is a saprophytic and facultative parasite, able to colonize living host tissue. This fungus is common in soil and organic substrates, and it can attack every part of the plant (Lindell, 2003).

F. graminearum is the asexual stage (anamorph) of the fungus, and *Gibberella zeae* is the sexual stage (teleomorph). The sexual spores (ascospores) are formed in asci which are inside the perithecium. A perithecium is a spherical or pear-shaped blueish-black structure, and it grows on the surface of affected spikelets. The asexual spores (macroconidia) of *F. graminearum* are formed on infected spikelets under warm and moist environmental conditions and appear as a pink colored sporodochium, which consists of many phialides clustered together. Phialides are the conidium-producing cells (Schmale III and Bergstrom, 2003).

In Figure 1 the disease cycle of *F. graminearum* in wheat is illustrated, which was published by Trail (2009). The overwintering structures are perithecia formed on crop residues (corn stalk, wheat straw, etc.) that remain on the field after harvest. The ascospores are forcibly discharged of the perithecium and dispersed by the wind, where they travel long distances as a main inoculum source of the disease. During anthesis wheat is most susceptible to these airborne spores (ascospores, or macroconidia), which land on wheat heads, germinate and enter the plants through natural openings. The grain grows, and the fungus expresses genes for mycotoxin synthesis to disable the plants defense mechanism. The main mycotoxin produced by *F. graminearum* is deoxynivalenol (DON).

When the weather is warm and humid sporodochia are formed on the surface of infected plants or crop debris. The macroconidia, which are formed in pink-colored sporodochia, are mainly short-distance dispersed from plant to plant by rain splash (Schmale III and Bergstrom, 2003; Shaner, 2003; Trail, 2009).

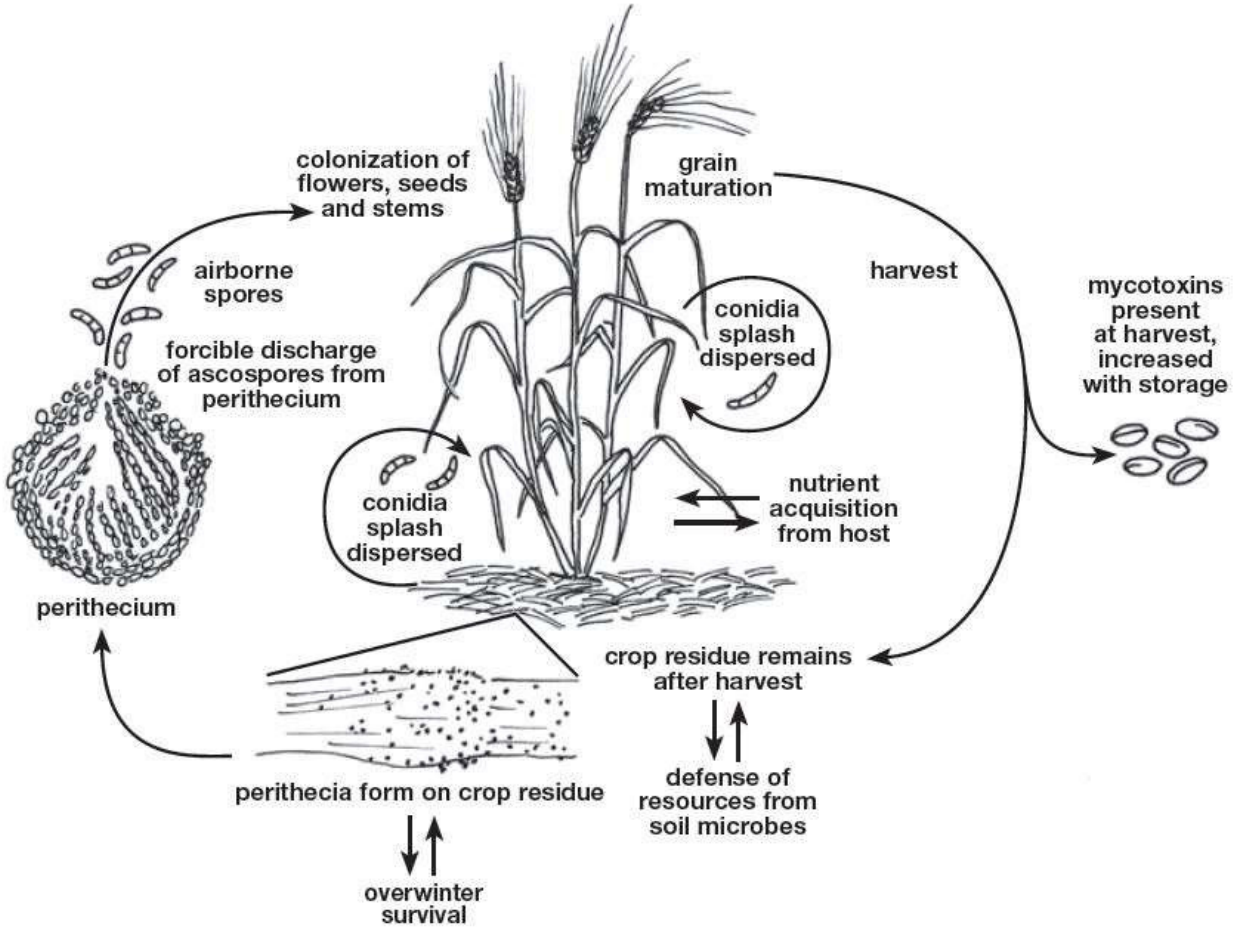

Figure 1: The life cycle of *F. graminearum* (Trail, 2009). Perithecia are formed on crop residues. Ascospores are forcibly discharged from the perithecium and dispersed by the wind. Colonization of flowers, seeds and stems. The grain matures and the fungus grows, and mycotoxins are present at harvest. Sporodochia can also be formed and conidia are splash dispersed by rain. After harvest crop residue remains on the field, where perithecia are formed and overwinter.

1.2.2. Symptoms of Fusarium Head Blight infection

The infestation with FHB can be easily recognized in the field, as the disease produces very distinctive symptoms on wheat heads, which can be seen in Figure 2.

The first symptoms of infection with *Fusarium spp.* occur shortly after anthesis. Infected spikelets are bleached where the fungus grows, and bleaching may spread across the entire spike. However, other diseases can also cause bleached heads, e.g. the stem maggot or *Stagonospora nodorum*. Under favorable conditions, pink colored spores (sporodochia) form on rachis and glumes of infected spikelets. Later in the season, blueish-black perithecia are formed on affected spikelets (Schmale III and Bergstrom, 2003; Shaner, 2003).

Infected florets will produce diseased kernels, which are shriveled and wrinkled (Buerstmayr et al., 2012; Bushnell et al., 2003). Infected kernels can be seen in Figure 3, published by the Government of Canada (2009). Even if kernels appear healthy, they could be contaminated with mycotoxins. These mycotoxins are a great danger to humans and animals. In chapter 1.2.3 the effects of mycotoxins are discussed in more detail.

Figure 2: Symptoms of FHB infection on wheat heads. (A) Bleached spikelets on infected wheat head. (B) Pink-colored spores (sporodochia) are formed on glumes.

Figure 3: Comparison of winter wheat kernels (Government of Canada, 2009). (A) Healthy winter wheat kernel. (B) Moderate symptoms of infection with *F. graminearum*. Mycelial growth is visible, kernel appears whitish and wrinkled. (C) Severe symptoms of infected kernels. Kernel is wrinkled, appears whitish and pink.

1.2.3. Mycotoxins produced by *Fusarium spp.*

Mycotoxins are secondary metabolites produced by fungi and are used in nature to disable plant defense mechanisms or protect the fungus against other microorganisms (Schmale III and Bergstrom, 2003). Many *Fusarium* species have the potential to produce mycotoxins. One of the most prevalent is DON, which is a type B trichothecenes (EFSA, 2004). Although, DON is one of the least acutely toxic members of the trichothecene group, it is a frequent contaminant of grains (Rotter et al., 1996; Streit et al., 2012).

T-2 toxin and HT-2 toxin are type A trichothecenes and the most toxic members of the trichothecene group (Fels-Klerx, 2010; McCormick et al., 2011). T-2 and HT-2 can be produced by e.g. *F. poae* or *F. sporotrichioides* (Logrieco et al., 2003; Torp and Nirenberg, 2004). In Table 1 a summary of the major mycotoxins in food, the fungal species, the most susceptible crops, and effects on human and animal health are given.

Table 1: Summary of major mycotoxins produced by different *Fusarium* species (according to Patriarca and Fernández Pinto, 2017). The major produced mycotoxin, the *Fusarium* species and the most susceptible crops are given. Also, the main diseases and symptoms shown in humans and animals.

Mycotoxin	Producing *Fusarium* species	Susceptible crops	Main disease/symptoms
Zearalenone	*F. culmorum, F. equiseti, F. verticillioides, F. graminearum*	Maize, Barley, Wheat, Rye	Estrogenic effects, cervical cancer
Fumonisins	*F. proliferatum, F. verticillioides*	Maize, Sorghum	Esophageal carcinoma, equine encephalomalacia pulmonary edema
T-2, HT-2	*F. langsethiae, F. poae, F. sporotrichioides*	Wheat, Maize	Alimentary toxic aleukia
Deoxynivalenol	*F. graminearum, F. poae, F. culmorum, F. crookwellense, F. sporotrichioides, F. tricinctum, F. acuminatum*	Wheat, Maize, Barley, Oat, Rye	Nausea, vomiting, diarrhea, abdominal pain, feed refusal
Nivalenol	*F. crookwellense, F. poae, F. nivale, F. culmorum, F. graminearum*	Wheat, Maize, Barley, Oat, Rye	Erythopenia, Leucopenia, Hematotoxicity

However, the most important mycotoxin produced by *F. graminearum* on small-grain cereals is DON (Rotter et al., 1996). The main effects of trichothecenes is the strong inhibitory effect on protein synthesis by binding to ribosomes, the inhibitory effect on RNA and DNA synthesis and a toxic effect on cell membranes (Scientific Committee on Food, 2000a, 2002). Acute DON toxicosis in animals and humans causes temporary nausea, vomiting, diarrhea, headache, dizziness, and fever. One of the main physicochemical properties and a big concern is the heat-stability of DON. Hence, DON remains active in processed food and feed (Desjardins, 2006; Sobrova et al., 2010).

Another important mycotoxin is Zearalenone (ZON), which is a nonsteroidal estrogenic mycotoxin and produced by several *Fusarium* species. ZON causes reproductive disorders, and especially pigs are very sensitive to this mycotoxin (Scientific Committee on Food, 2000b)

Karlovsky et al. (2016) and Peng et al. (2018) show strategies to prevent and reduce mycotoxin contamination in feed. These strategies should start from crop cultivation up to processing of feed and food. The most effective strategies are prevention methods, such as pre-harvest field management and post-harvest management, e.g. drying of grains before storage. Further mycotoxin reducing methods are available, including physical methods, thermal methods, chemical methods and mycotoxin-controlling feed additives. Physical removal of mycotoxins can be done by cleaning out external materials and sorting out of inferior kernels. Also, dehulling and milling can reduce mycotoxin contamination. DON is distributed throughout the whole kernel, with higher concentration in rachis and glumes (Cowger and Arellano, 2013). Therefore, to reduce DON concentration the rachis and glumes can be removed prior to milling. Also, DON is water-soluble and by cooking cereal products, such as noodles, the mycotoxin leaches into the cooking water to some extent (Kushiro, 2008; Sobrova et al., 2010).

Thermal processes, such as dry heating may not be ideal for mycotoxin reduction in feed, because mycotoxins are very heat-stable. For example, DON has a decomposing temperature of 151-153°C (Kabak, 2009). Due to concerns regarding consumers' health chemical treatments for mycotoxin reduction have been banned in food and feed processing by The European Commission (2006).

As mycotoxins are ubiquitously present in food and feed, the Austrian Agency for Health and Food Safety (AGES) has established a maximum level of contaminants in food and feed. For DON the maximum level in unprocessed grain, except durum wheat, oats and maize, is 1,250 µg/kg, in pasta products 750 µg/kg, and in bread the maximum level is 500 µg/kg. For processed cereal-based foods and baby foods for infants and young children the limit is 200 µg/kg.

For ZON the maximum level is generally lower due to its estrogenic effect, e.g. for unprocessed grain, except maize, the limit is 100 µg/kg and in processed cereal-based foods and baby foods for infants and young children the limit is 20 µg/kg (Öhlinger, 2017).

1.2.4. Disease management

There is always *Fusarium* inoculum present in the environment and FHB severity is heavily influenced by seasonal weather conditions and humidity. Currently, there is no single way to fully control FHB infection. However, there are several measures which can be combined altogether in an integrated pest management (IPM) plan to decrease the risk of FHB infection. An IPM plan includes the choice of the pre-crop type, cultural practices, fungicidal treatments or the cultivar selection. IPM not only helps to prevent *Fusarium* infection, but can also be effectively used against many other pests and diseases (Wenda-Piesik et al., 2017).

On the following pages several methods to control FHB are described.

Crop rotation

As the fungus survives on crop residues and in soil, a broad and diversified crop rotation can minimize the amount of inoculum and reduce the potential for infection with FHB. Clearly, a diverse crop rotation is recommended for several reasons and is also effective against weeds and other diseases. The findings of Wenda-Piesik et al. (2017) show, that wheat has a higher infection rate when grown after wheat or corn, compared to sugar beet. Also, mycotoxin content increased by a close crop rotation (Bernhoft et al., 2012).

Cultural practices

No-till or minimum tillage is practiced worldwide as a method of conserving soil moisture and structure, as well as improving soil properties. Crop residues remain on the soil surface and take a long time to degrade, which leads to a massive inoculum source. To reduce *Fusarium* inoculum in the soil a minimum tillage is recommended, at least in areas with high *Fusarium* pressure. In areas where tilling is no option, because of conserving soil moisture and structure, methods that accelerate the rotting process are appropriate. For example, crop residues can be chopped into small pieces, which speeds up the degradation process (Köpke et al., 2007).

Fungicidal treatment

To obtain the full efficiency of a fungicidal treatment many factors must be considered, as the right application time, weather or previous treatments. Forecasting models can help to predict sporulation, spore dispersal and infection of plant diseases.

In Bavaria the Institute for Plant Protection, Bavarian State Research Institute for Agriculture (LfL) informs farmers about modern strategies to protect crop plants from diseases and pests. Further, they ensure an economic and sustainable crop production and the production of high quality food and feed (Bayerische Landesanstalt für Landwirtschaft, 2018).

In Austria there is the 'Ikwarndienst' which provides information for farmers about the occurrence of diseases and pests on basis of national and regional forecasting systems. This information helps to optimize plant protection measures, reduce costs, increase efficiency in crop production and also to reduce environmental impacts (LK Österreich, 2018).

There are several plant protection products available, which are currently approved in Austria. The products can be found in the 'plant protection products register' of the Federal Office for Food Safety (BAES).

The findings of Hysing and Wiik (2014) show that treatment of seeds with fungicides mainly increased plant emergence in seed lots with low to moderate *Fusarium/Microdochium spp.* infection, but had little or no effects on other agronomic characters.

The application of fungicides at heading time has shown limited results to reduce the risk of FHB infection. However, there are recommendations for an optimal treatment, including the knowledge of susceptibility of the variety or regular infestation controls. A fungicidal treatment should be carried out during anthesis and shortly after rain. The uneven flowering of wheat and unfavorable weather conditions, as well as the fast development and spreading of the fungus, makes the treatment rather challenging. Moreover, the fungicide costs are a limiting factor, besides in many cases a treatment is not very effective (Bernhoft et al., 2012; McMullen et al., 1997; Schmale III and Bergstrom, 2003).

A single fungicidal treatment during flag-leaf stage and at the end of ear emergence is sufficient in most cases. When mulch seeding is done, and the pre-crop was maize, the risk of FHB infection for wheat is much higher. A double treatment at flag-leaf stage and during wheat flowering is recommended (Schmiedl, 2018).

The Austrian Chamber of Agriculture provides information for farmers on which products to use, or when the treatment should be done. In more humid areas an earlier fungicidal treatment can be necessary due to a higher disease pressure.

Cultivar selection

Although, there are several crop protection solutions to reduce *Fusarium spp.* pressure, the breeding for resistant cultivars seems to be the most effective method to combat FHB. Further, the use of healthy seeds and a homogenous population with rapid flowering makes it harder for the fungus to infest the wheat plants.

In the following chapters the recent approaches of resistance breeding in wheat are summarized.

1.3. Breeding for resistance to Fusarium Head Blight

The breeding for FHB resistant cultivars is an important target in many breeding programs around the world. It not only is the most effective way of controlling the disease and mycotoxin content, but also the most cost-efficient and environmentally-save way.

The resistance to *Fusarium spp.* is very complex. FHB resistance is a quantitative trait, which means it is influenced by multiple genes of the host (resistance factors in the plant) and by the pathogen (aggressiveness of the fungus). Also, the environment has an influence on disease establishment (Campbell and Lipps, 1998; Mesterházy, 1995). Further, resistance to FHB is species and race non-specific. This means, the resistance does not depend on the species of *Fusarium* and therefore, resistant lines from all over the world could provide general resistance to *Fusarium spp.* (Buerstmayr et al., 2009).

However, the difficulty in resistance breeding is creating regionally adapted cultivars that are not only high and stable yielding with good grain quality, but also with resistance against several different pests and diseases, e.g. powdery mildew, brown rust, yellow rust, *Septoria nodorum*, and *Fusarium spp* (Mesterházy, 2003).

The most resistant cultivars origin from China, Japan, and Brazil and are mostly spring wheat genotypes, such as Sumai-3, Nobeoka Bozu, Ning-8343, Ning-7840, Frontana. They carry FHB resistance genes, but are agronomically poor and regionally unadapted genotypes. Therefore, combining FHB resistance and other agronomically important traits is another main goal (Bai et al., 2003; Buerstmayr et al., 2009; Mesterházy, 2003).

1.3.1. Types of resistance

Several active and passive resistance mechanisms have been described in literature. The following classification of types of resistance for FHB of wheat and barley is largely based on Mesterházy (1995), Mesterházy (2003) and Leonard and Bushnell (2003).

Mesterházy (1995) also differentiates between active and passive resistance mechanisms. Active resistance factors depend on the physiological defense response of the host plant. Passive resistance is dependent on the morphological features of the plant, but independent of the physiological status. Passive factors may influence the likelihood of getting infected as well as alter conditions for primary infection and fungal growth development.

In Table 2 the active resistance mechanisms are listed. To evaluate type I resistance either artificial spray inoculation with *Fusarium* inoculum or alternatively the grain-spawn method can be used. For optimal infection conditions a mist irrigation system can be used. Disease severity is measured by visual scoring of disease symptoms on heads, or on harvested samples (Buerstmayr et al., 2009; Mesterházy, 2003). Other morphological and developmental traits, which act as passive resistance mechanisms, have been found to have an impact especially on type I resistance, e.g. plant height, presence of awns. Further, type II resistance can be assessed by point inoculation of single-florets or single-spikelets. Disease severity is measured as amount or speed of symptom spreading from the inoculation site along the head (Buerstmayr et al., 2009)

Table 2: Types of active resistance mechanisms.

Active resistance
I. Resistance against initial infection (Schroeder and Christensen, 1963)
II. Resistance to pathogen spreading in infected tissue (Schroeder and Christensen, 1963)
III. Resistance to kernel infection (Mesterházy, 1995)
IV. Tolerance against FHB. Tolerance means that yield is maintained despite the presence of disease (Mesterházy, 1995)
V. Resistance to toxins. This can be the result of toxin decomposition by the plant, or of plant tolerance to the toxin, or of limitation in DON concentration accumulating in wheat heads (Mesterházy, 2003)

In Table 3 the passive resistance mechanisms are listed. The role of plant height as a passive resistance mechanism was first described by Mesterházy (1995). Also, many other authors have discussed the relationship between plant height and severity of FHB (Buerstmayr et al., 2000, 2009; Gervais et al., 2003; Hilton et al., 2002). It is therefore well validated, that tall cultivars show increased FHB resistance. This relationship could be explained either by linkage between one or more genes controlling resistance and genes controlling straw height, or by pleiotropy, where genes that promote shorter straw also promote susceptibility (Hilton et al., 2002).

Also, the shorter distance of wheat heads to the natural inoculum source, the crop debris on soil promotes FHB infection in shorter plants (Mesterházy, 1995). Raindrops rebound from the ground and take spores up to flowering wheat heads (Rossi et al., 2002). Further, soil moisture has a stronger effect on smaller genotypes, because they preserve humidity better and the wind dries up taller genotypes more easily. Although, taller plants are generally more resistant to FHB, they are not desired by breeders for agronomic reasons (Parry et al., 1995).

Table 3: Types of passive resistance mechanisms.

Passive resistance
I. Plant height (Buerstmayr et al., 2000, 2009; Gervais et al., 2003; Hilton et al., 2002; Mesterházy, 1995)
II. Presence of awns (Mesterházy, 1995)
III. Spikelet density within the head (Mesterházy, 1995; Steiner et al., 2004)
IV. Flowering date (Gervais et al., 2003; Holzapfel et al., 2008; Schmolke et al., 2005; Steiner et al., 2004)
V. Flower opening and flower duration (Gilsinger et al., 2005)
VI. Extent of anther extrusion (Skinnes et al., 2010)

There are conflicting results on whether the presence of awns increases (Mesterházy, 1995), or decreases disease severity (Buerstmayr et al., 2000).

Also, spikelet density within the head is reported as a passive resistance mechanism. Loose ears show a lower natural infection than dense ones (Mesterházy, 1995; Steiner et al., 2004).

In several studies it was confirmed that wheat heads are most susceptible for FHB infection during anthesis (Gervais et al., 2003; Holzapfel et al., 2008; Schmolke et al., 2005; Steiner et al., 2004). Siou et al. (2014) showed in a greenhouse experiment that inoculation around anthesis caused the highest disease and toxin levels. Also, early or late infections with aggressive isolates led to detectable levels of the fungus and toxin.

Gilsinger et al. (2005) hypothesized that wheat lines with narrow flower opening and a short duration of flower opening will be affected to a lesser extent by FHB. This could be explained by a reduced area and time for the *Fusarium* spores to enter the florets. However, these traits are influenced by environmental conditions during anthesis (Gilsinger et al., 2005).

Several authors have already assessed the correlation of anther extrusion and FHB severity in different field trials. The results have shown a negative correlation between anther extrusion and type I FHB susceptibility. Partially extruded anthers are considered to be a source of FHB infection, whereas closed-flowering genotypes and genotypes that rapidly and completely extrude their anthers show improved resistance to FHB infection (Buerstmayr and Buerstmayr, 2015; Graham and Browne, 2009; He et al., 2014; Kubo et al., 2013; Lu et al., 2013; Skinnes et al., 2008, 2010).

1.3.2. Resistance to Fusarium Head Blight

The resistance to FHB is quantitatively inherited. To investigate FHB resistance a Quantitative Trait Locus (QTL) mapping study is the preferred method. However, upon FHB infection the difficulty is to distinguish pleiotropic effects of genes involved in morphological or developmental traits from true resistance genes, which may only be linked to these morphological or developmental genes. Many articles have been published about QTL mapping in the past several years. Buerstmayr et al. (2009, 2012) summarized 52 studies, which report more than 100 QTL for FHB resistance in wheat. These QTL are on chromosomes 1B, 1D, 2A, 2B, 2D, 3A, 3B, 3D, 4B, 4D, 5A, 5B, 6A, 6B, 7A and 7B. However, most of these QTL are only minor contributors to overall *Fusarium* resistance.

Many resistant cultivars in hexaploid wheat originate from Asia, such as Sumai-3, Ning-7840, Ning-8331, W14, Wangshuibai, and other lines. The resistance genes of these genotypes are used in adapted lines in breeding programs around the world. The Brazilian cultivar Frontana was first described by Schroeder and Christensen (1963) as a source of resistance, but until today no large-effect QTL has been detected in any populations derived from Frontana (Buerstmayr et al., 2009).

Important resistance QTL are *Qfhs.ndsu-3BS* (*Fhb1*) on chromosome 3BS (Waldron et al., 1999), *Qfhs.ifa-5A* on chromosome 5A, and *Qfhs.nau-6B*, *Qfhs.lfl-6BS* (*Fhb2*) on chromosome 6BS. All carrying the resistance alleles from Sumai-3 and related lines (Buerstmayr et al., 2009, 2012).

The QTL *Fhb1* shows resistance to fungal spread (type II resistance), whereas *Qfhs.ifa-5A* contributes to resistance against fungal penetration (type I resistance) (Buerstmayr et al., 2002, 2003). The resistance QTL *Fhb1* is found in about 50% of wheat varieties, and in 2/3 of advanced breeding lines of spring wheat breeding program at University of Minnesota. To a lower extent *Qfhs.ifa-5A* is present in these lines. In Europe only one registered cultivar, namely the French cultivar Jaceo (Syngenta Seeds), is known to carry *Fhb1.* The challenge of using Chinese resistance QTL in modern cultivars is linkage drag with unfavourably agronomic traits, such as increased lodging or decreased grain yield (Steiner et al., 2017).

Lemmens et al. (2005) found out that wheat lines carrying *Fhb1* can convert the mycotoxin DON into the less toxic DON-3-O-glycoside (D3G) as a detoxification product. He hypothesized that *Fhb1* either encodes for a DON-glucosyltransferase or modulates the expression of such an enzyme. Further, the D3G of DON-resistant wheat cultivars could be a ‘masked mycotoxin’. Some plants protect themselves from mycotoxins by transforming them into more polar metabolites, which can then, for example, be transported into vacuoles for further storage. This means that not only unaltered mycotoxins are a threat to food and feed safety, but also masked mycotoxins, which could regain biological activity in the intestinal tract of humans and animals. In contrast to DON, D3G is not detectable with routine analyses (Berthiller et al., 2015; Lemmens et al., 2005).

1.3.3. Methods of resistance breeding

The difficulty in resistance breeding is to combine high and stable yield with good product quality and resistance to variable environmental conditions, such as abiotic and biotic stresses. Therefore, FHB breeding programs are conducted over several years in different environments (Buerstmayr et al., 2014).

Conventional (phenotypic) selection

In resistance breeding the conventional or phenotypic selection is used for small and large effect QTL, usually in more advanced generations (e.g. from F_4 upward) and in separate resistance testing nurseries. This method is time and cost intensive, but until recently the phenotypic selection was the only option and has resulted in good cultivars (Buerstmayr et al., 2014; Steiner et al., 2017).

For this breeding method potential crossing partners are evaluated according to their resistance against FHB. Next, a new population is generated by crossing at least one parent with good FHB resistance or two moderately resistant parents. Among the progeny, the best lines with best trait combinations are selected (Buerstmayr et al., 2014).

Besides phenotyping of symptoms in the field, there is also the alternative method of pre-screening for FHB resistance at seedling stage. This screening method could be a simple, rapid and reliable screening method to evaluate FHB resistance (Shin et al., 2014). Further, post-harvest analysis of kernels by visual scoring can be done. Another alternative method, that is less labour-intensive and time-consuming is digital image analysis for estimation of *Fusarium*-damaged kernels (Maloney et al., 2014). Other methods are to measure yield and yield reduction relative to non-inoculated controls (Dill-Macky, 2003).

Marker-assisted selection

The marker-assisted selection (MAS) is a relatively recent tool in resistance breeding. It can be applied for traits that are difficult and cost intensive to assess. It is used for introgression of medium to large effect QTL in breeding populations (including early generations, e.g. F_2, F_3) and is based on molecular markers (Anderson, 2007; Buerstmayr et al., 2014). Many QTL mapping studies have been published and summarized by Buerstmayr et al. (2009) and thus identified QTL can be used for MAS.

Molecular markers associated with QTL for FHB resistance and the use of those markers for MAS have been summarized by Anderson (2007) and Kolb et al. (2001). The concept is that differences in the DNA sequence of wheat cultivars are genetically linked to genes that confer resistance to FHB. The differences in DNA sequences could be due to a different number of repeat units of a sequence (e.g. microsatellite or simple sequence repeat, SSR), or due to an insertion or deletion of a DNA segment, or due to a single base pair difference (single nucleotide polymorphism, SNP). Today, mostly PCR-based SSRs or KASP marker are used as markers (Anderson, 2007). A main advantage of SSR markers is that they are very robust and reliable. However, these markers need to be tightly linked to the QTL. Tight linkage ensures that the marker is a good substitute for the actual QTL (Collard et al., 2005).

For the successful implementation of MAS for a QTL, it must be a relatively large effect and stable QTL, also there should be tightly linked markers available. Until today, the major QTL *Fhb1*, *Qfhs.ifa-5A, Fhb2, Fhb4* have been repeatedly identified by QTL mapping studies (Buerstmayr et al., 2012; Liu et al., 2009).

Genomic selection

The genomic selection (GS) is already used in animal breeding and is now becoming an important tool in plant breeding. GS using genome wide prediction models is a very promising tool for small effect QTL, which are the rule in resistance breeding for FHB. With rapidly decreasing marker costs and increasing genome coverage, GS becomes more important for breeders (Buerstmayr et al., 2014; Heffner et al., 2010). Large numbers of individuals and several traits can be screened at once, which is more cost efficient than phenotypic evaluation of populations over years and in different environments. GS shortens the breeding cycle and increases selection gain per unit time for difficult phenotypic traits, such as mycotoxin content (Steiner et al., 2017).

1.3.4. Semi-dwarfing *Rht1* genes

During the green revolution from 1940-1970 new technologies, chemical fertilizers and crop genetic improvement led to high yields (Pingali, 2012). Until today, nitrogen fertilizers are used to achieve high grain yield and good grain quality. This entails taller plants with heavier ears, which are more vulnerable to lodging than smaller cultivars. Lodging wheat crops show a higher infestation rate with *Fusarium spp.,* because of the close contact of wheat ears and soil as a natural inoculum source. Also, lodging wheat plants make drying up harder after a rainfall (Bernhoft et al., 2012; Zhang et al., 2017). In contemporary agriculture reducing nitrogen fertilization is no practical tool in managing *Fusarium* infestation (Lemmens et al., 2004). However, with the introduction of wheat semi-dwarfing genes plants had shorter and stronger stems. Such plants were more resistant to lodging and have an improved harvest index (Allan, 1986; Zhang et al., 2017).

These semi-dwarfing genes originate from the Japanese genotype 'Norin 10'. This cultivar is the work of Japanese scientist Gonjiro Inazuka, who crossed a semi-dwarf Japanese wheat landrace with two American varieties, which resulted in an improved semi-dwarf variety. Norin 10 was registered in 1935 (Lumpkin, 2015).

In 1953 Norman Borlaug at CIMMYT (International Maize and Wheat Improvement Center) in Mexico received Norin 10 derived varieties and began to incorporate the semi-dwarfing genes into regionally adapted varieties. The result was a short and stiff-strawed high-output variety. The progeny of this variety was soon adapted all over Mexico, and in other developing countries, such as India or Pakistan, where it increased crop productivity and was crucial for the green revolution. Norman Borlaug was awarded with the Nobel peace prize in 1970 for his contribution to world peace by increasing food supply. With his contribution over a billion people worldwide could be saved from starvation (Hedden, 2003; Lumpkin, 2015; Voß, 2011).

The most widely used reduced height (*Rht*) genes are *Rht-B1b* (formerly *Rht1*), located on chromosome 4B, and *Rht-D1b* (formerly *Rht2*), located on chromosome 4D (Steiner et al., 2017; Thomas, 2017).

The *Rht1* genes (*Rht-B1b, Rht-D1b*) are now ubiquitously used in crop breeding programmes around the world to reduce lodging and increase grain yield. Though, lines carrying *Rht1* genes are more susceptible to FHB (Voss et al., 2008).

Distribution of the semi-dwarfing alleles (*Rht-D1b, Rht-B1b*)

Voß (2011) provides a comprehensive table of the most important *Rht* genes and its sources. Würschum et al. (2015) performed a genome-wide association study and identified two additional QTL affecting plant height within his material. These additional medium-effect QTL are located on chromosomes 6A and 5B. Since the 1970s the most widely used semi-dwarfing allele is *Rht-D1b* and the major QTL on chromosome 6A. The second major QTL on chromosome 5B has remained constantly high in the last decades, whereas *Rht-B1b* is only rarely used in the European winter wheat varieties. The findings of Würschum et al. (2015) also show the geographic range of the semi-dwarfing genes. It is revealed that *Rht-B1b* occurs in more than half of Eastern European varieties and in all Turkish varieties. *Rht-D1b* is more frequent in varieties from Central Europe, Denmark, the UK and France.

Association of the semi-dwarfing alleles (*Rht-D1b, Rht-B1b*) and FHB resistance

Reduced plant height and a high number of retained anthers are both traits associated with increased FHB severity and should be considered when breeding for FHB resistance (Buerstmayr et al., 2012). In several studies the negative effect of semi-dwarfing alleles *Rht-B1b* and *Rht-D1b* on FHB resistance was shown (Draeger et al., 2007; Holzapfel et al., 2008; Srinivasachary et al., 2008, 2009; Steiner et al., 2017; Voss et al., 2008). FHB resistance and anther retention are both quantitative traits, influenced by the environment and genetic background (Buerstmayr and Buerstmayr, 2015).

Both semi-dwarfing alleles (*Rht-D1b, Rht-B1b*) have similar effects on plant height and act additively when combined but have a different effect on FHB severity. Differences in disease severity of *Rht-D1b* and *Rht-B1b* alleles can partly be explained by different effects on the extent of anther retention. *Rht-D1b* allele has a greater impact on anther retention and FHB severity than *Rht-B1b* allele (Miedaner and Voss, 2008; Srinivasachary et al., 2009). The negative effect of retained anthers could be explained, because *Fusarium* infection establishes inside the floral cavity, forming hyphal networks on inner surface of palea, lemma and glume. Retained anthers within the florets could potentially support colonization of the fungus and hyphal growth, which leads to higher disease levels in these genotypes (Pugh et al., 1933).

As both semi-dwarfing genes (*Rht-D1b, Rht-B1b*) lead to approximately the same reduction on plant height, other factors than plant height must induce this gene-specific difference in FHB severity. Possibly the different proportion on florets with retained anthers are the cause of different FHB severity between genotypes with *Rht-D1b* or *Rht-B1b*. However, the question is, if higher FHB severity and increased anther retention of genotypes with *Rht-D1b* is due to a pleiotropic effect, meaning that infection conditions within the florets are promoted by retained anthers, or if there is linkage with undesirable genes (Buerstmayr and Buerstmayr, 2016).

In conclusion, by using *Rht-D1b* in breeding, lines with moderate FHB resistance can be obtained, but the negative effects should be compensated by using positive QTL alleles. With *Rht-B1b* it is possible to breed lines with very good FHB resistance, therefore it should be the mean of choice in resistance breeding for FHB (Buerstmayr and Buerstmayr, 2016; Steiner et al., 2017). Also, rapid and complete anther extrusion can be used as a supportive indirect trait in resistance breeding. However, the negative effect of *Rht* alleles on disease resistance has to be balanced by the efficient implementation of resistance QTL, such as *Fhb1* or *Qfhs.ifa-5A* (Buerstmayr and Buerstmayr, 2015; Saville et al., 2012; Steiner et al., 2017).

Influence of semi-dwarfing *Rht1* genes on gibberellin signalling pathway

Gibberellic acid (GA) is a plant hormone essential for plant growth and development (Hooley, 1994). The central regulators of the GA signalling pathway are DELLA proteins, which act as repressors of plant growth, whereas GA promotes plant growth by overcoming DELLA-mediated growth restraint (Achard and Genschik, 2009; Bolle, 2004). DELLA proteins are named after their conserved N-terminal amino acid sequence, namely aspartic acid, glutamic acid, leucine, leucine, alanine (Hedden, 2003).

The *Rht1* semi-dwarfing alleles reduce stem elongation, because of insensitivity to GA. This altered response is caused by a mutation in the homologous DELLA genes *Rht-B1* and *Rht-D1*. The *Rht1* alleles contain a nucleotide, which is a point mutation that leads to the introduction of a premature stop codon within the DELLA region. It is hypothesized that translation might restart after the stop codon, which results in shortened proteins that are less sensitive to degradation by endogenous GA. The mutant DELLA protein is accumulated and causes growth inhibition, which leads to the semi-dwarf phenotype (Peng et al., 1999).

The evidence that mutant DELLA proteins are accumulated, supports a pleiotropic effect on disease resistance of *Rht* alleles rather than linkage to a susceptibility factor. Also, it is known that DELLA proteins in *Arabidopsis* are involved in regulation of several genes, for example in response to diseases and pathogens, toxin catabolism or multidrug transport (Cao et al., 2006).

Therefore, DELLA encoding genes could play a role in disease resistance in cereals and must be considered in plant breeding, where *Rht* alleles are used to achieve plants with stronger stems (Navarro et al., 2008; Saville et al., 2012). The shorter cultivars show advantageous agronomic traits, such as lower tendency to lodging due to decreased internode lengths and stronger stems, also higher number of spikelets and grains per spike which results in increased yield compared to taller cultivars (Allan, 1986; Flintham et al., 1997; Sial et al., 2002).

1.3.5. Identification of Fusarium Head Blight resistance genes

The major QTL *Fhb1* was originally mapped by Restriction Fragment Length Polymorphism (RFLP) analysis (Waldron et al., 1999) and is located on chromosome 3BS between microsatellite markers GWM493 and GWM533 (Anderson et al., 2001).

Until now *Fhb1* is the most important large effect QTL conferring type II FHB resistance (resistance to fungal spread within the spike) and also has the ability to detoxify DON into less toxic D3G (Lemmens et al., 2005; Schweiger et al., 2013, 2016). This QTL region was fine-mapped and sequenced, revealing that it consists of 28 genes assumed to be responsible for FHB and DON resistance. The whole list of genes located in *Fhb1* region is depicted in Schweiger et al. (2016).

Rawat et al. (2016) shows that a pore-forming toxin-like (PFT) gene at *Fhb1* confers FHB resistance, but not DON resistance. According to Schweiger et al. (2016) gene #22 encodes a chimeric lectin with agglutinin domains and a pore-forming toxin-like (PFT) domain on chromosome 3BS. It is only weakly expressed and might have direct antifungal activity by binding to fungal cell wall carbohydrate structures and permeating the membranes.

In the following the three candidate genes which are of importance for this master thesis are briefly described. However, it is still unknown which of these specific gene(s) contribute to DON resistance. Schweiger et al. (2016) hypothesizes that only one gene confers both, *Fusarium* and DON resistance. It is also possible that two different co-segregating resistance genes are involved.

Glycosyltransferase hga7 (gene #6)

Glycosylation is one of the mechanisms plants use to adapt rapidly to their environment. Glycosyltransferases (GTFs) recognize hormones, secondary metabolites, biotic and abiotic chemicals and toxins in the environment. Typically, GTFs transfer sugars to lipophilic acceptors. This glycosylation changes their chemical properties and bioactivity, which enables the altered acceptor to access membrane-bound transporter systems, such as ABC transporters. The glycoside is transported out of the cytosol, e.g. into the vacuole for detoxification (Bowles et al., 2006; Poppenberger et al., 2003). Gene #6 is annotated as Homogalacturonan (HGA)-like UDP-Glycosyltransferase (UGT).

It shares similarities to the large superfamily encoding small molecule conjugating UGTs (Ross et al., 2001), but most likely acts on formation of homogalacturonan as part of the cell wall (Yin et al., 2010).

Ubiquitin-2 like Rad60 SUMO-like protein (gene #17)

Ubiquitin is important in hormone synthesis, hormonal signalling cascades and plant defence mechanisms. Further, the ubiquitin/proteasome system (UPS) targets proteins in plants for degradation (Dreher and Callis, 2007).

Hypothetical calcium binding protein (gene #20)

Calcium is important for cellular signalling pathways and acts as a second messenger in plant cells. Usually Ca^{2+} concentrations are at lower levels but change rapidly in response to environmental or endogenous impacts. During pathogen infection the Ca^{2+} concentrations in plant cells are increased as a first step of plant defence. These changes are sensed by Ca^{2+} binding proteins and interpreted into specific physiological responses to cope with pathogen attacks (Zhang et al., 2014).

1.3.6. Targeting induced local lesions in genomes

A promising approach for the characterization of the actual genes of these resistance QTL, especially in *Fhb1* region, is the reverse genetics approach Targeting Induced Local Lesions in Genomes (TILLING). The strategy of reverse genetics is based on the alteration of a gene structure or activity, followed by analysis of the associated change in its plant phenotype (Kurowska et al., 2011). TILLING allows the identification of mutations in DNA sections of the genome. This method combines the high density of point mutations provided by mutagenesis with Ethyl-methane-sulfonate (EMS) or other chemicals, with a rapid mutational screening for induced lesions. The mutagenic chemical EMS tends to produce a certain type of random point mutation in DNA duplexes (McCallum et al., 2000; Slade et al., 2005).

The advantage of this method is its applicability for any type of plant, regardless of the size of genome or ploidy level, as well as for small- and large-scale screening. Further, relatively few individual plants are needed, because of the high density of point mutations (McCallum et al., 2000; Slade et al., 2005).

The basic principle of TILLING is shown in Figure 4. It involves the following steps:

a. Creation of a mutated population.
   - Mutagenesis of seeds with EMS to produce M_0.
   - M_0 seeds are planted to produce M_1. In M_1 generation chimerism occurs very frequently. Self-fertilization of M_1 generation and propagation by Single Seed Descent until M_2 or higher.
   - DNA extraction from an individual plant, e.g. M_2 plants, self-fertilization and storage of seeds for further phenotyping after screening for mutations in M_3 and M_5 generation.

b. Detection of mutations in M_2 and M_4 generation.

- DNA of individual M_2 and M_4 plants is extracted (e.g. of leaves).
- Mutation detection in M_2 generation and upwards is done by high-throughput analysis of DNA samples by pooling of 5-8 M_2 plants (Kurowska et al., 2011; McCallum et al., 2000; Till et al., 2003). For the detection of mutations in a targeted sequence a polymerase chain reaction (PCR) is used to amplify the targeted DNA sequence using pool DNA as a template (e.g. wild-type and mutant-type DNA) (Seidl, 2017). The three steps of a PCR are denaturation of double-stranded DNA, annealing of primers, elongation of primers catalyzed by DNA polymerase (Johnson et al., 1991).
- The mutations can be detected by different procedures, e.g. cleavage by specific endonucleases at mutation site and mutation detection by fragment analysis (e.g. electrophoreses), denaturing high-performance liquid chromatography (DHPLC) or high-throughput sequencing.
- M_2 and M_4 plants carrying a point mutation in the gene of interest are identified and the target gene is sequenced to confirm the mutation. Further, the type of nucleotide change and prediction of the effect of the mutation are determined (Kurowska et al., 2011; McCallum et al., 2000).

c. Analysis of mutant phenotype in M_3 and M_5 generation.

- The plants carrying the mutation are grown in the greenhouse or on the field to evaluate the effect of the mutation on the phenotype during the trial.

  To test the candidate genes in wheat for DON/*Fusarium* resistance, selected plants of a resistant cultivar carrying a mutation at the *Fhb1* QTL are used. In the mutated plants the suspected resistance gene is shut off. The plants are treated with a suspension of DON or *Fusarium* inoculum, either in the greenhouse or field. If plants show symptoms after infection, it can be assumed that the candidate gene, which was affected by the mutation, is connected to the resistance mechanism (Rawat et al., 2016; Seidl, 2017).

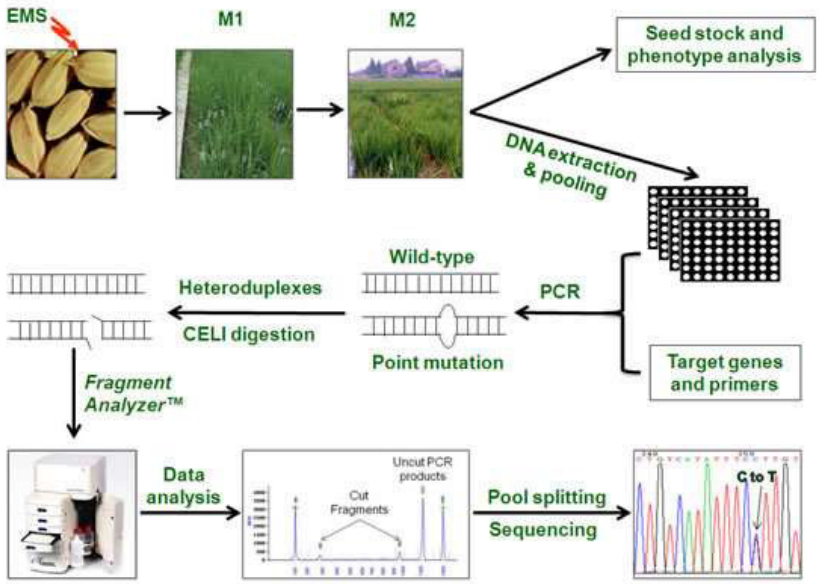

Figure 4: Schematic illustration of the TILLING workflow applied in rice (Institute of Botany, CAS, n.d.). The steps are: EMS mutagenesis. Growth of M_1 and M_2, both propagated by Single Seed Descent (SSD) until M_4. DNA extraction of leaves of individual M_2 and M_4 plant. DNA preparation and pooling of individuals. PCR amplification of a region of interest. Screening for mutations in genes of interest and selection of lines with mutations in *Fhb1* region. Mutation detection based on fragment analysis (e.g. electrophoreses). Or in DHPLC, the presence of a heteroduplex in a pool is detected as an extra peak in the chromatogram. Identification of mutant individual and further phenotyping.

2. Research questions and aims

The main goal of this master thesis was to broaden the general knowledge on FHB resistance. The thesis is divided into two parts:

(1) The first part was about the evaluation of near isogenic lines (NILs) carrying natural *Rht1* alleles or the transgenic *Rht-D1b* allele.

It has already been proven that smaller plants are more susceptible to FHB than taller plants (Draeger et al., 2007; Holzapfel et al., 2008; Srinivasachary et al., 2008, 2009; Voss et al., 2008). However, it is still not clear, whether wheat cultivars harboring plant height reducing *Rht1* alleles are more susceptible to FHB due to close linkage with a susceptibility factor, or due to a pleiotropic effect of *Rht1* semi-dwarfing alleles (Parry et al., 1995). My part of the study was:

- The identification of NILs being homozygous for natural *Rht-B1a*, *Rht-B1b*, *Rht-D1a* and *Rht-D1b* alleles in genetic backgrounds of C1, CM-82036, E4-61-T and Michael using allele specific markers.
- The identification of transgenic NILs being homozygous for the *Rht-D1b* transgene or homozygous for no transgene, in genetic backgrounds of C1, CM-82036, E4-61-T and Michael. In the transgenic plants the assumed linkage of *Rht-D1b* allele to a susceptibility factor was broken. The transgene is linked to green fluorescent protein (GFP) and used for visual screening of transformed plants.

The detection of positive transformants harboring the GFP gene, combined with an expected semi-dwarf phenotype provides good evidence that the transformed gene is expressed. The selected homozygous natural and transgenic NILs will be used for phenotyping and allow a direct comparison of the effect of transgenic *Rht-D1b* and natural *Rht-D1b* semi-dwarfing genes.

If NILs containing transgenic *Rht-D1b* alleles react like NILs with natural *Rht-D1b* alleles a pleiotropic effect can be assumed. However, if linkage is the reason for increased FHB susceptibility of small plants, it is expected that NILs with natural *Rht-D1b* alleles are susceptible, whereas NILs with transgenic *Rht-D1b* alleles are expected to be no longer susceptible, because only *Rht-D1b* alleles were introduced.

(2) The second part of the thesis was about the phenotypic evaluation of lines carrying deleterious mutations in candidate genes for DON and *Fusarium* resistance identified in a *T. aestivum* mutant population by the reverse genetics approach TILLING.

Fhb1, a major resistance QTL in bread wheat (*T. aestivum*) provides a moderately high resistance against the pathogen *Fusarium spp.* and contributes to detoxification of DON to D3G.

The genetic locus *Fhb1* has been fine-mapped and harbors 28 genes. However, the causal gene of DON resistance has not been identified yet (Rawat et al., 2016; Schweiger et al., 2016). In this thesis mutant lines, each harboring a mutation in one of three candidate genes have been tested for DON and FHB resistance. These mutations in the coding region of the gene could lead to a dysfunctional protein, namely Glycosyltransferase hga7 (gene #6), Ubiquitin-2 like Rad60 SUMO-like protein (gene #17), and Hypothetical calcium binding protein (gene #20) (Schweiger et al., 2016).

The aim was to identify the causal resistance gene(s) by testing lines carrying deleterious mutations in three promising candidate genes by inoculation with a spore suspension of *F. graminearum* and the mycotoxin DON. The mutant lines carrying a mutation in this specific gene region should show the typical symptoms of FHB spreading or DON induced bleaching, as the protein function is shut down.

Evaluation of Fusarium Head Blight susceptibility by genotyping NILs for natural *Rht-B1*, *Rht-D1* and transgenic *Rht-D1* alleles

3. Materials and methods

3.1. Plant material

NILs containing natural *Rht1* semi-dwarfing genes

Four sets of NILs were produced by crossing the four tall wheat cultivars C1, CM-82036, E4-61-T, Michael as recurrent parent to Monsun and to Bobwhite. C1 and CM-82036 are carriers of the *Fusarium* resistance QTL *Fhb1* and *Qfhs.ifa-5A* and show good resistance to FHB. The spring wheat cultivar Monsun was used as donor for the native *Rht-D1b* gene, and the spring wheat cultivar Bobwhite is the native *Rht-B1b* donor. After five times backcrossing (BC_5) to the respective recurrent parent, the genetic background is 97% as the recurrent parent, but with either *Rht-B1b*, or *Rht-D1b* instead of *Rht-B1a*, or *Rht-D1a*.

Successful introgression of *Rht1* genes was confirmed using allele specific markers. These lines are described in Table 4. Fourteen progeny of two plants of each cross (BC_5F_2=F_3) were genotyped to select NILs that contrasted for the *Rht-B1a/b* or *Rht-D1a/b* alleles. BC_5F_2=F_3 was analyzed using PCR-based markers. Plants homozygous for *Rht-B1a*, *Rht-B1b, Rht-D1a* and *Rht-D1b* were selected and propagated to produce seeds for phenotyping.

NILs containing transgenic *Rht-D1b* semi-dwarfing gene

The cloning and production of transgenic plants is described by Teutschl (2016). By using particle bombardment, the *Rht-D1b* gene has been introduced into Bobwhite. This is done by millions of DNA-coated gold particles that are shot at target cells or tissues, e.g. wheat embryos, by a gene gun. Once the DNA-coated particles enter the cells, they elute off the DNA. If foreign DNA reaches the nucleus, then transient expression is likely to occur and the transgene may be stably incorporated into host chromosomes (Kikkert et al., 2005).

By random inclusion of the transgenic allele into chromosomes the assumed linkage between FHB susceptibility and plant height is eliminated (Teutschl, 2016).

Four transgenic events (DN1, DN2, DN3 and DN4) were successfully introgressed into Bobwhite and have been positively screened for harboring *Rht-D1b* (Teutschl, 2016). The transgenic plants with DN1, DN2, DN3, DN4 were crossed to the four tall wheat cultivars C1, CM-82036, E4-61-T and Michael, and three to five times backcrossed to the respective parent to produce transgenic NILs. In Table 5 the transgenic NILs are further described. The presence of the transgene was confirmed by GFP and EXON specific markers. Further, in transgenic plants the *Rht-D1b* allele is linked to GFP. This can be used for visual screening of transgenic plants being homozygous or heterozygous for the transgene. If a transgenic plant is homozygous all pollen appears fluorescent under the fluorescence microscope.

As recurrent parent the four tall wheat cultivars C1, CM-82036, E4-61-T and Michael were used. The lines C1 and CM-82036 are highly resistant to FHB and carry the two major resistance QTL *Fhb1* and *Qfhs.ifa-5A.* Michael is highly susceptible to *Fusarium spp.* and the line E4-61-T is moderately susceptible.

Table 4: Summary of natural NILs. The line with respective natural *Rht1* gene and generation is given.

Line	Natural *Rht1* genes	Generation
C1	*Rht-D1b*	BC_5F_2=F3
C1	*Rht-B1b*	BC_5F_2=F3
CM-82036	*Rht-D1b*	BC_5F_2=F3
CM-82036	*Rht-B1b*	BC_5F_2=F3
E4-61-T	*Rht-D1b*	BC_5F_2=F3
E4-61-T	*Rht-B1b*	BC_5F_2=F3
Michael	*Rht-D1b*	BC_5F_2=F3
Michael	*Rht-B1b*	BC_5F_2=F3

Table 5: Summary of transgenic NILs. The line with respective transgenic *Rht1* gene and generation is given.

Line	Transgenic *Rht1* genes	Generation
C1	DN1	BC_5F_2=F3
C1	DN2	BC_3F_2=F3
C1	DN3	BC_4F_2=F3
CM-82036	DN1	BC_5F_2=F3
CM-82036	DN2	BC_5F_2=F3
CM-82036	DN3	BC_5F_2=F3
CM-82036	DN3	BC_4F_2=F3
Michael	DN1	BC_5F_2=F3
Michael	DN2	BC_5F_2=F3
Michael	DN3	BC_5F_2=F3
Michael	DN4	BC_5F_2=F3
E4-61-T	DN1	BC_5F_2=F3
E4-61-T	DN2	BC_5F_2=F3
E4-61-T	DN4	BC_4F_2=F3
E4-61-T	DN4	BC_5F_1=F2

3.2. DNA extraction

The selected plants are grown in the greenhouse (Figure 5). Leaves of young plants are harvested, which are then put into tubes and dried at a temperature of about 35°C for two days. DNA extraction was done according to IFA DNA extraction protocol.

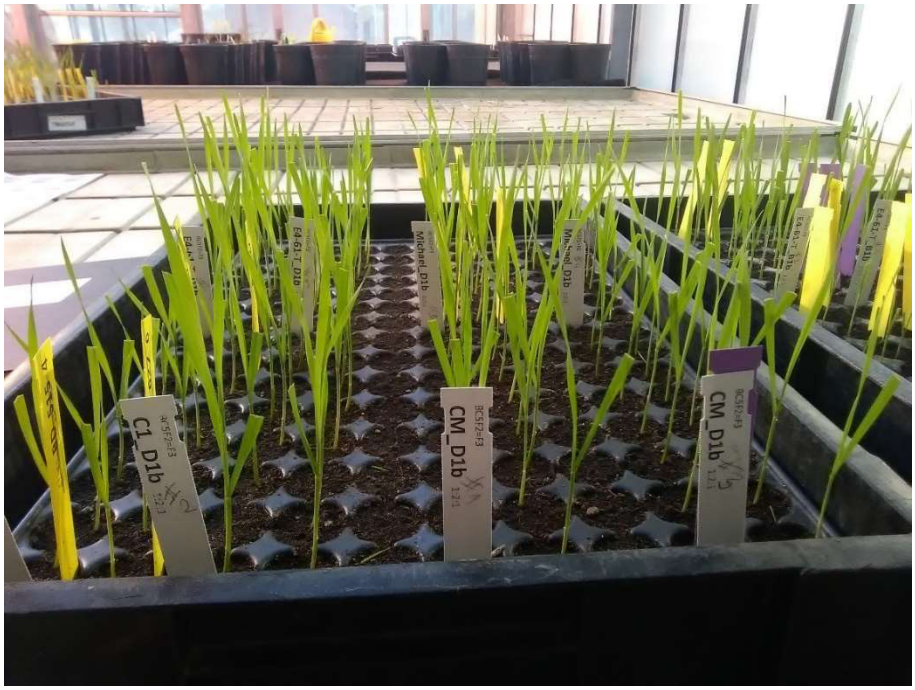

Figure 5: Wheat plants are grown in small pots in the greenhouse. The tips of young leaves are cut for DNA extraction.

A 96-plate with 1.2 ml 8-stripe tubes is prepared and each tube is filled with 3 small glass beads. The dry leaf-material is cut into the tubes and grinded in the Retsch-Mill for about 10 minutes. The finer the powder the better the amount of extracted DNA. The composition of CTAB-Buffer is listed in Table 6 and prepared for a final amount of 50 ml. Before adding CTAB and BME the buffer is warmed to 60-65°C. The BME is added under a fume hood.

Table 6: Composition of CTAB-Buffer. EDTA … Ethylenediaminetetraacetic acid, CTAB … Mixed alkyltrimethyl-ammonium bromide, BME … ß-Mercaptoethanol

Stock	Final	50 ml
dH_2O		32.5 ml
1 M Tris-7.5	100 mM	5 ml
0.5 M EDTA-8.0	50 mM	5 ml
5 M NaCl	700 mM	7 ml
CTAB	1%	0.5 g
14 M BME	140 mM	0.5 ml

Now the stripes are centrifuged at very low rpm to get the powder down from the covers, but not too strong, because otherwise the powder sticks on the bottom and cannot be mixed properly with the buffer (2,500 rpm for 3 minutes).

The stripes are opened carefully to avoid scattering the leaf powder or contamination of nearby tubes. To each well 700 µl of CTAB-Buffer is added under the fume hood. The stripes are closed tightly and mixed by inversion. The rack with the tubes is placed into a water bath at 65°C for 60-90 minutes with gentle rocking.

The stripes are put out and cooled down to room temperature. Under the fume hood 300 µl of Chloroform and Isoamyl alcohol (24:1) is added into to each well.

Stripes are closed and put on a plate fastener, tightly screwed and shaked by gentle inversion for 10 minutes.

Then, the probes are centrifuged for 10 minutes at 3,800 rcf. After centrifugation clearly separated layers become visible as shown in Figure 6. The top aqueous layer contains DNA, a small white layer containing the protein fraction and the lower fraction contains chloroplasts and other components.

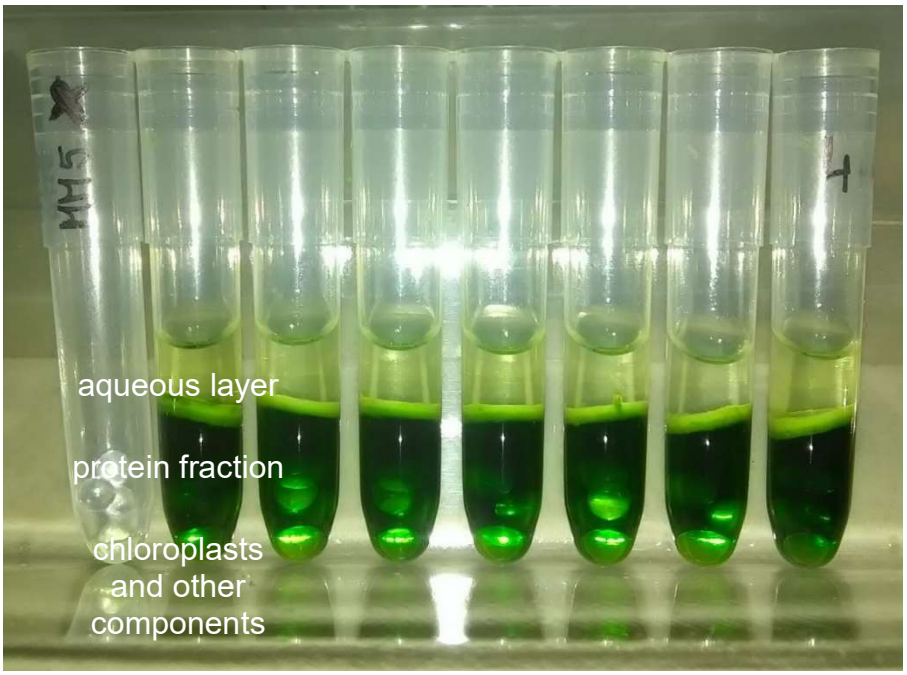

Figure 6: Different layers are visible in the probes. After centrifugation the aqueous layer, protein fraction, and chloroplasts become visible.

The top aqueous layer is pipetted off (about 300 µl) into a new stripe containing RNase A (5 µl, 10 µg/ml), which is then mixed and incubated at room temperature for 30 minutes. After incubation, 300 µl of Isopropyl alcohol is added and mixed well by gentle inversion to precipitate the DNA. In Figure 7 the DNA fibers are visible.

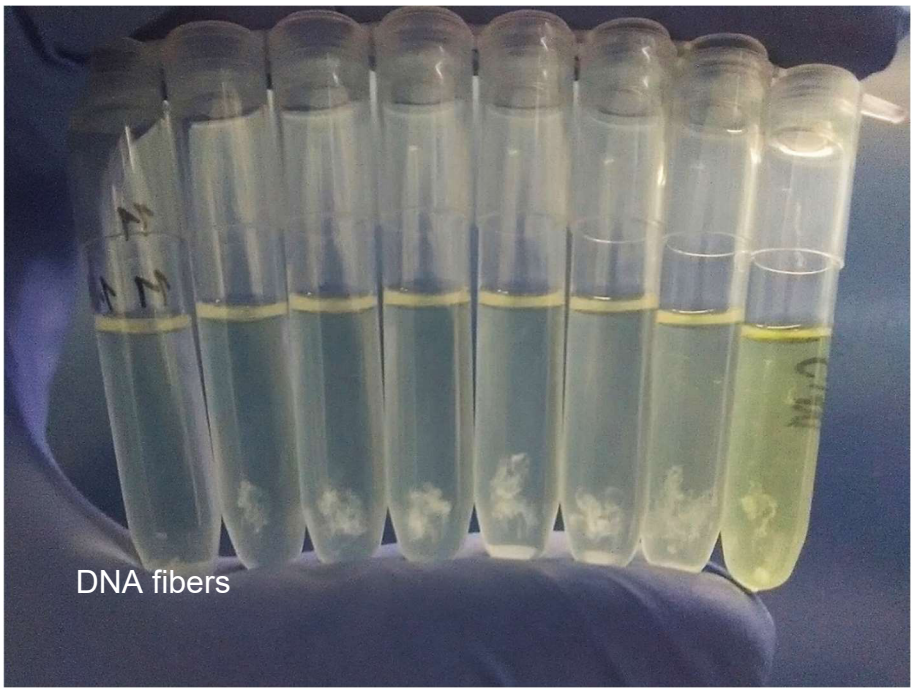

Figure 7: DNA fibers are visible in the probes. After addition of alcohol and gentle inversion of probes the DNA fibers become visible as white clouds.

The probes are centrifuged 8 minutes at low rpm (about 1,200 rcf). The DNA is now spun down, forming a DNA pellet at the bottom of each tube as shown in Figure 8. The DNA pellet should stick to the bottom of the tube and the liquid is poured off quickly.

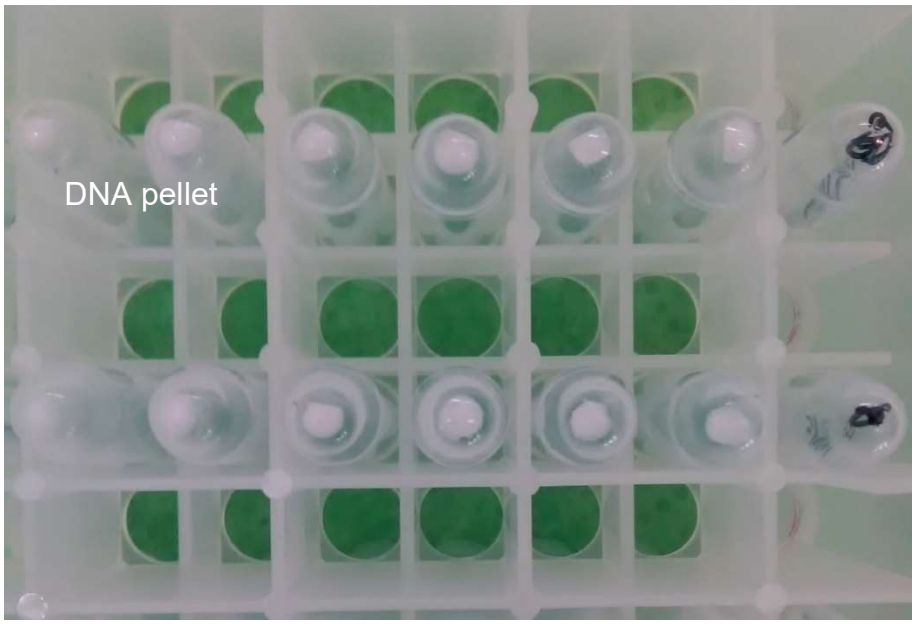

Figure 8: DNA pellets is visible at the bottom of the tube. After centrifugation the DNA pellet sticks to the bottom of the 8-stripe tubes and liquid is poured off quickly.

Two washing steps are carried out to clean the DNA from unwanted components. After these steps the DNA pellets are dried over night at room temperature.

On the following day the DNA pellet is dissolved in 100 µl of TE-8 Buffer and gently rocked for several hours at room temperature. The plate can then be stored at 4°C.

3.3. Determination of DNA concentration

First, the concentration and purity of several randomly selected probes was measured to estimate the approximate range of DNA concentration of the whole plate. For this, the BioSpecNano was used, which is a micro-volume spectrophotometer. It is based on the principle that DNA absorbs UV light, when exposed to it. A photo-detector then measures the light that passes through the sample. The more light absorbed, the higher the DNA concentration in the sample. Less light reaches the photodetector and results in a higher optical density (Heptinstall and Rapley, 2000). The BioSpecNano automatically calculates the DNA concentration in ng/µl.

At an average concentration of about 300 ng/µl a 1:1 dilution is done. In each well of the photometer plate (96-well plate) 50 µl of distilled water is pipetted. Also, about 3 blanks per photometer plate (50 µl distilled water + 50 µl TE-8) are set. In the remaining wells the DNA probes (50 µl) are pipetted. The plate is then sealed with an adhesive foil, to prevent the probes from evaporation and contamination. Then probes are centrifuged to make sure they are at the bottom of the well. Everything is mixed well with a shaker and centrifuged again.

The DNA concentration of the whole photometer plate is quantified with a Tecan plate reader. The results are summarized in an Excel file. To achieve a DNA concentration of 100 ng/µl the amount of distilled water in µl is calculated and added to each well.

After adjustment of DNA concentration of the probes, 2 µl of DNA are pipetted into a PCR plate (384-well plate), which is cooled by a frozen metal plate. After centrifugation of the PCR plate there should be a probe in each well. If not, the probe is pipetted into the well individually.

3.4. Preparation of mastermix

The Mastermix was prepared according to IFA SSR protocol. The composition of PCR Mastermix for screening natural and transgenic lines for *Rht-B1a/Rht-B1b* and *Rht-D1a/Rht-D1b* is given in Table 7. The primer sequences used to detect lines containing natural *Rht1* semi-dwarfing genes are shown in Table 8 (Ellis et al., 2002). The primers used for the transgenic lines are listed in Table 9.

Table 7: Composition of Mastermix for screening of lines with natural and transgenic *Rht1* genes.

	Stock	Final	10 µl/RXN
DNA template	10 ng/µl	2 ng/µl	2 µl
goTAQ (Green Mastermix)	2x	1x	5 µl
Forward primer	10 µM	0.4 µM	0.4 µl
Reverse primer	10 µM	0.4 µM	0.4 µl
PCR H_2O	-	-	2.2 µl

Table 8: Primers used for screening of NILs containing natural *Rht1* genes.

Primer		Sequence (5‘ - 3‘)
Rht-B1 primers:		
BF (forward)		GGT-AGG-GAG-GCG-AGA-GGC-GAG
WR1 (reverse)	*Rht-B1a*	CAT-CCC-CAT-GGC-CAT-CTC-GAG-CTG
MR1 (reverse)	*Rht-B1b*	CAT-CCC-CAT-GGC-CAT-CTC-GAG-CTA
Rht-D1 primers:		
DF (forward)		CGC-GCA-ATT-ATT-GGC-CAG-AGA-TAG
WR2 (reverse)	*Rht-D1a*	GGC-CAT-CTC-GAG-CTG-CAC
MR2 (reverse)	*Rht-D1b*	CCC-CAT-GGC-CAT-CTC-GAG-CTG-CTA

Table 9: Primers used for screening of NILs containing transgenic *Rht1* genes.

Primer	Sequence (5‘ - 3‘)
EX1 (forward)	GTG-GAG-GAG-AAG-GAA-GGC-TG
EX3 (reverse)	CGG-GGA-AAT-TCG-AGT-CGA-CA
GFP (forward)	GGT-CAC-GAA-CTC-CAG-CAG-GA
GFP (reverse)	GAC-CAC-ATG-AAG-CAG-CAC-GA

Rht-D1b and *Rht-B1b* need different markers and are therefore examined separately. The forward primers BF and DF are genome specific and bind to a region of the promoter with no sequence homology between *Rht-B1b* and *Rht-D1b*. BF and DF are each the same for mutant-type and wild-type. The reverse primer is allele specific and different for both mutant- and wild-type. For *Rht-B1* the primers BF and MR1 have been used for the mutant-type, for the wild-type BF and WR1. For *Rht-D1* the primers DF and MR2 have been used for the mutant-type, for the wild-type BF and WR2 (Ellis et al., 2002).

The transgenic lines were screened with two different primer sets EX1 and EX3, and GFP-f and GFP-r.

For each primer combination the Mastermix was prepared for an appropriate amount of DNA probes and needs to be cooled during the process. The Mastermix was prepared on ice and in following order: PCR H_2O, forward and reverse Primer, goTAQ. The goTAQ contains Taq-Polymerase, reaction buffer, deoxynucleoside triphosphate (dNTPs) and $MgCl_2$. $MgCl_2$ works as a co-factor for the thermostable DNA polymerase and is also important for primer annealing. dNTPs are single units of the bases Adenine, Guanine, Thymine, Cytosine, which are necessary for new DNA strands.

The Mastermix was vortexed and 8 µl Mastermix per well are pipetted into the PCR plate, with 2 µl of DNA probe in each well already. The PCR plate was sealed with a PCR foil, which prevents the content from evaporation and contamination. After centrifugation it is visually checked if there is Mastermix in each well. As soon as DNA and Mastermix are mixed together, they start to react. The steps of PCR are further described in chapter 3.5.

Marker-assisted screening for *Fhb1* and *Qfhs.ifa-5A*

The transgenic lines C1 and CM-82036 have been screened for the two major FHB resistance QTL by using linked microsatellite markers, namely Umn10 (*Fhb1*) and Gwm304 (*Qfhs.ifa-5A*). The plants were backcrossed up to 5 times with either CM-82036 or C1 and were screened for the presence of the resistance QTL. Both, CM-82036 and C1 are carriers of the resistance QTL *Fhb1* and *Qfhs.ifa-5A*.

The composition of the PCR Mastermix for screening for *Fhb1* on chromosome 3B and *Qfhs.ifa-5A* on chromosome 5A is given in Table 10.

Table 10: Composition of Mastermix for screening for *Fhb1* and *Qfhs.ifa-5A*.

	Stock	Final	10 µl/RXN
DNA template	10 ng/µl	2 ng/µl	2 µl
Reaction buffer (incl. 1.5 mM $MgCl_2$)	10x	1x	1 µl
dNTP Mix	2 mM	0.2 mM	1 µl
Forward primer	10 µM	0.02 µM	0.02 µl
Reverse primer	10 µM	0.2 µM	0.2 µl
M13 primer	10 µM	0.18 µM	0.18 µl
Taq polymerase	5 U/µl	0.05 U/µ	0.1 µl
PCR H_2O	-	-	5.5 µl

The transgenic lines are screened with two different primer sets, namely Gwm304-f and Gwm304-r, and Umn10-f and Umn10-r. The primer sets are extended by a M13 sequence at the 5' end. This M13 primer consists of a short sequence and a FAM (6-Carboxyfluorescein) or a Cy5 (Cyanin 5) fluorescence tail. In Table 11 the primer sequences of Gwm304, Umn10 and M13 are listed.

Table 11: Primers used for NILs containing natural *Rht1* genes. Amino acids marked in bold indicate the M13 primer tail sequence.

Primer	Sequence (5' - 3')
Gwm304 (forward)	**CCC-AGT-CAC-GAC-GTT-G** AGG-AAA-CAG-AAA-TAT-CGC-GG
Gwm304 (reverse)	AGG-ACT-GTG-GGG-AAT-GAA-TG
Umn10 (forward)	**CCC-AGT-CAC-GAC-GTT-G** CGT-GGT-TCC-ACG-TCT-TCT-A
Umn10 (reverse)	TGA-AGT-TCA-TGC-CAC-GCA-TA
M13 primer[1]	CCC-AGT-CAC-GAC-GTT-G

[1] either labelled with Fam or Cy5

3.5. Polymerase chain reaction

In Table 12 the PCR program for the NILs containing natural *Rht1* semi-dwarfing genes is shown, and in Table 13 the PCR program for the NILs containing transgenic *Rht-D1b* semi-dwarfing genes. In Table 14 the hot-start touchdown PCR program for the M13 primer sequence is shown.

Table 12: PCR program for NILs containing natural *Rht1* genes.

Steps	Temperature (°C)	Time (min)	Number of cycles
Initialization	94	02:00	1
Denaturation	94	00:30	
Annealing	63	00:30	35
Elongation	72	00:30	
Final elongation	72	05:00	1
Final hold	10	∞	1

Table 13: PCR program for NILs containing transgenic *Rht-D1b* genes.

Steps	Temperature (°C)	Time (min)	Number of cycles
Initialization	95	03:00	1
Denaturation	95	00:30	
Annealing	57	00:30	30
Elongation	72	00:30	
Final elongation	72	10:00	1
Final hold	10	∞	1

Table 14: Hot-start touchdown PCR program for M13 primer.

Steps	Temperature (°C)	Time (min)	Number of cycles
Pre-Denaturation	94	04:00	1
Denaturation	94	00:50	7 touchdown cycles with a temperature decrement of 2°C per cycle during Annealing
Annealing	65-51	01:00	
Elongation	72	00:60	
Denaturation	94	00:30	25
Annealing	51	00:30	
Elongation	72	00:30	
Final Elongation	72	05:00	1
Final hold	14	∞	1

The reaction chamber is heated up to 94°C before the plates are placed into the cycler to simulate a hot-start (manual hot-start). When the cold plate is placed into the pre-heated cycler immediate denaturation occurs. During denaturation the double-stranded DNA is separated into complementary strands by breaking the hydrogen bonds. For primer annealing the temperature is lowered, allowing the primers to attach to the single-stranded DNA templates. Two primers are used, a forward and a reverse primer, each complementary to the denatured single-stranded DNA. For the extension reaction the polymerase attaches to these primers and synthesizes a new DNA strand complementary to the DNA template. The polymerase adds fitting nucleotides (dNTPs) from the reaction mix to the DNA template in 5'-3' direction, which is called primer extension reaction. After this step the cycle is repeated several times. The final elongation is an optional step to ensure that the remaining single-stranded DNA is fully elongated (Johnson et al., 1991).

The hot-start touchdown PCR is a modified cycling program in which the initial annealing temperature is several degrees above the estimated primer melting temperature (T_m) and is gradually reduced (e.g. 1-2°C per cycle) over several cycles, until T_m is reached. Amplification is then continued at this annealing temperature. The advantage of a hot-start touchdown PCR is to reduce non-specific priming to sequences with low homology, primer-dimer formation prior to start of PCR and reaction setup at room temperature. The advantage of the touchdown program is to increase yield, sensitivity and specificity of the initial primer–template duplex formation and therefore specificity of the final PCR product (Ault et al., 1994; Korbie and Mattick, 2008). Once the PCR program is finished, the PCR cycler cools the plates for final hold. The plates are then stored in the fridge at 4°C.

3.6. Electrophoresis on agarose gel

The NILs are analyzed by Agarose Gel Electrophoresis and ultraviolet (UV) transilluminator to prove whether the lines contain the desired natural *Rht-D1b/Rht-B1b* or transgenic *Rht-D1b* semi-dwarfing genes.

The advantages of agarose gels are that it is a nontoxic gel medium, which is easy and quick to cast. It is good to separate medium to large DNA molecules, but only shows poor separation of samples with low molecular weight. It only has low resolving power, which results in fuzzy bands that tend to spread apart (Stellwagen, 1998).

For a gel of the size 11x14 cm 100 ml gel is needed. Therefore, 100 ml TAE buffer and 1.7 g Agarose (1.7%) is mixed and carefully melted in the microwave until the liquid is clear. Then the gel is cooled down to a temperature of < 60°C and as loading dye 5 ml SYBR Safe is added. SYBR Safe is a DNA gel stain that binds to DNA and makes is visible using UV excitation. SYBR Safe stain is a less hazardous alternative to ethidium bromide, which acts as a mutagen. Ethidium bromide intercalates with the DNA, for example inserting itself between the base pairs in the double helix, deforming the DNA and disrupting biological processes (Thermo Fisher Scientific, n.d.).

The agarose gel is poured into the tray and combs are inserted. After cooling down for about 30 min. the combs are removed. The tray is filled up with TAE buffer, so that the gel is covered by at least 0.5 cm. Now 10 µl of the samples are loaded into the gel with a pipette. In Figure 9, the lid is put on the tray and an electric current is applied, which carries the DNA from negative (black) to positive (red) electrode. The samples are now run at 30 V for 10 min., and at 80 V for 30 min. The fragments are separated according to their size: small fragments move faster through the gel than larger fragments. The gel electrophoresis is working properly when bubbles climb up the border of the tray.

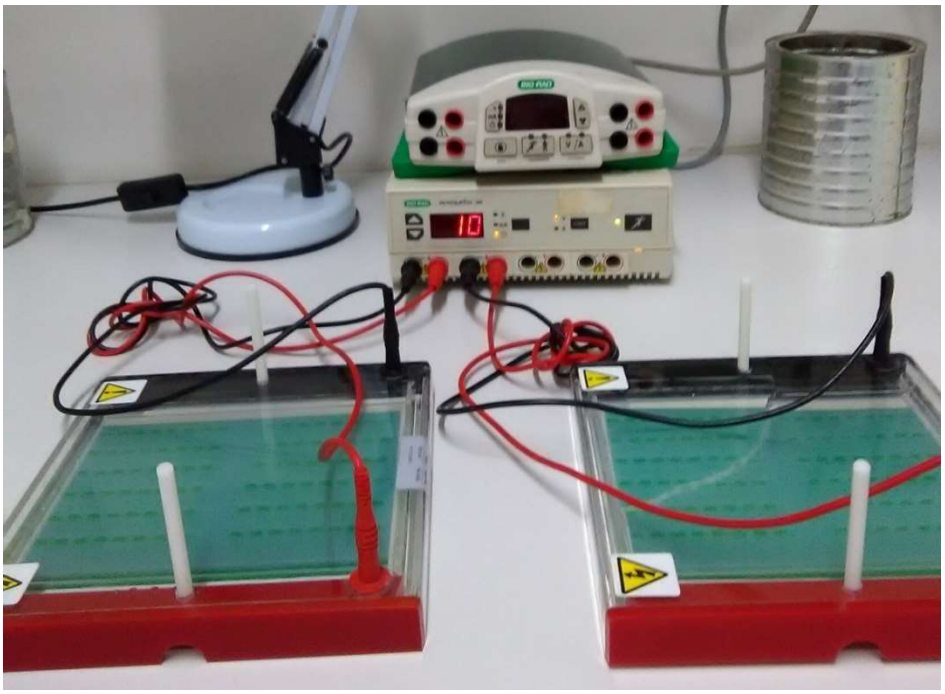

Figure 9: Experimental setup of agarose gel electrophoresis. Samples are run at 30 V for 10 min. and at 80 V for 30 min. Fragments separate according to their size and bands can be seen.

Once the gel electrophoresis is finished, the buffer is poured off and the agarose gel is analyzed in the UV transilluminator. The UV-light visualizes the separated DNA fragments, which appear as dark bands in the gel on the computer. A picture of the gel is taken and evaluated with Photoshop. The picture is checked for marker-specific bands. If a dark band is shown, it is evaluated with ‘1’, otherwise with ‘0’. An example can be seen in Figure 12. Data is entered in an Excel sheet and further analyzed.

3.7. Electrophoresis on polyacrylamide gel

The polyacrylamide gel electrophoresis (PAGE) is done to screen the transgenic lines C1 and CM-82036 for the presence of the two major FHB resistance QTL by using the microsatellite markers Umn10 and Gwm304. After PCR amplification, the final PCR product will be labelled with the fluorescent dye.

A polyacrylamide gel consists of toxic monomers. The gel preparation needs more time than agarose gels and is more difficult to prepare and handle. However, there are some main advantages. For example, this gel has a greater resolving power and shows sharp bands. It can separate fragments with low molecular weight and accommodate large quantities of DNA (Stellwagen, 1998). Two different probes can be loaded onto one gel by using a fluorescent labelled M13 primer with the dyes Cy5 and Fam.

A 7% polyacrylamide gel is prepared. The polyacrylamide gel has two different layers, the stacking gel where the loaded sample is stacked and the separating or running gel where the sample migrates according to its molecular weight. The gel is poured between two glass plates, the comb which creates the chambers for the probes is inserted.

After polymerisation of the gel, the comb is removed and TBE running buffer is added. To each sample 2.5 µl loading dye is added. The probes are pipetted into the vertically positioned gel, which is mounted between two buffer chambers. An electrical current is applied across the buffer chambers, which forces the DNA to migrate through the gel from negative (top) to positive (bottom) pole. Smaller molecules travel faster than larger molecules. The experimental setup is illustrated in Figure 10.

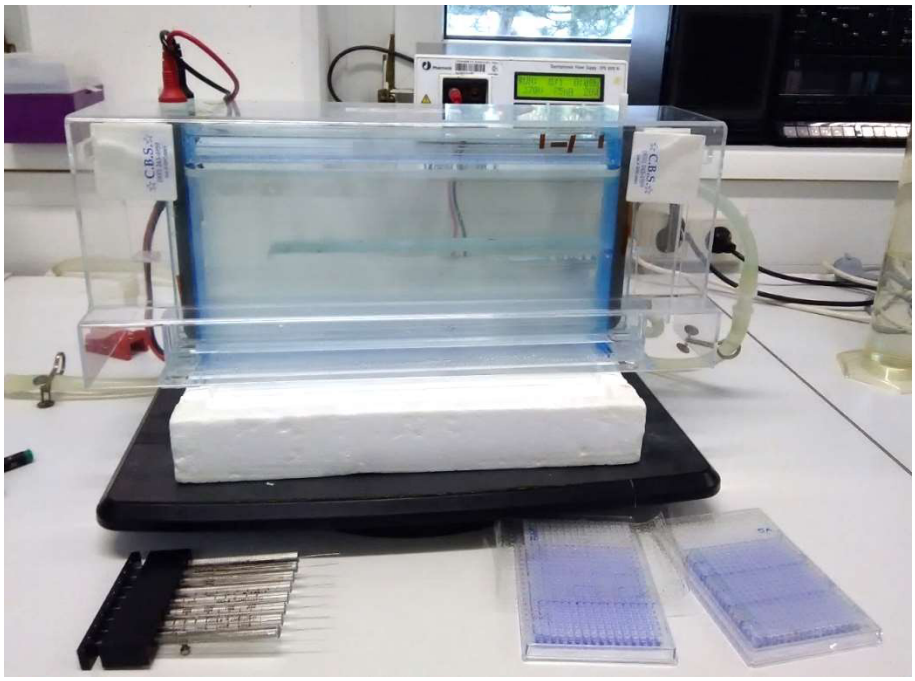

Figure 10: Experimental setup of PAGE. An electric current is applied, and fragments separate according to their size.

After separation by PAGE the fragments are visualized with a Typhoon Trio gel image scanner where the two fluorochromes are detected at different wavelengths, FAM is detected at 520 nm and Cy5 at 670 nm (Schuelke, 2000). The computer creates an image for each fluorochrome where the DNA fragments can be scored and further analyzed.

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3.8. Fluorescence microscopy

Homozygous transgenic lines cannot be distinguished from heterozygous transgenic lines by PCR and electrophoresis. Therefore, pollen of positive transformants, where transgenic *Rht-D1b* alleles could be confirmed after electrophoresis, were further examined by fluorescence microscopy.

Pollen of the flowering plants was collected and mixed with 20 µl of water and 0.4 µl mannitol. On the object slide 3 µl of the pollen mix is pipetted and screened for protein expression using 'Olympus IX-81 inverted fluorescence microscope', with the following settings: objective for brightfield (BF) with condenser DIC40 and lamp 1.9, objective for fluorescence microscopy 494 FITC with condenser DIC40 and lamp 1.9.

The transgene construct contains a protein that exhibits bright green fluorescence when exposed to UV or blue light. The GFP originates from the jellyfish *Aequorea victoria* and is widely used as a directly visible marker to detect transformed cells and issues in wheat. The big advantage is that no exogenously added substrate or co-factor is needed (Chalfie et al., 1994; Jordan, 2000). This GFP does not have any specific function in wheat but is useful for detection of the transgene construct.

As pollen is haploid, we can use it to distinguish between homozygous and heterozygous plants, as shown in Figure 11. If pollen derives from a homozygous transgenic plant, all collected and examined pollen is fluorescing. If pollen derives from plants heterozygous for the transgene, about half of the pollen is fluorescing, and the other half not. Pollen of plants without the transgene does not fluoresce at all. However, the intensity of fluorescence can differ between mutant transgenic genotypes and sometimes zygosity is not clear to identify. The detection of fluorescent pollen combined with an expected semi-dwarfed phenotype provides good evidence that the transformed gene is expressed.

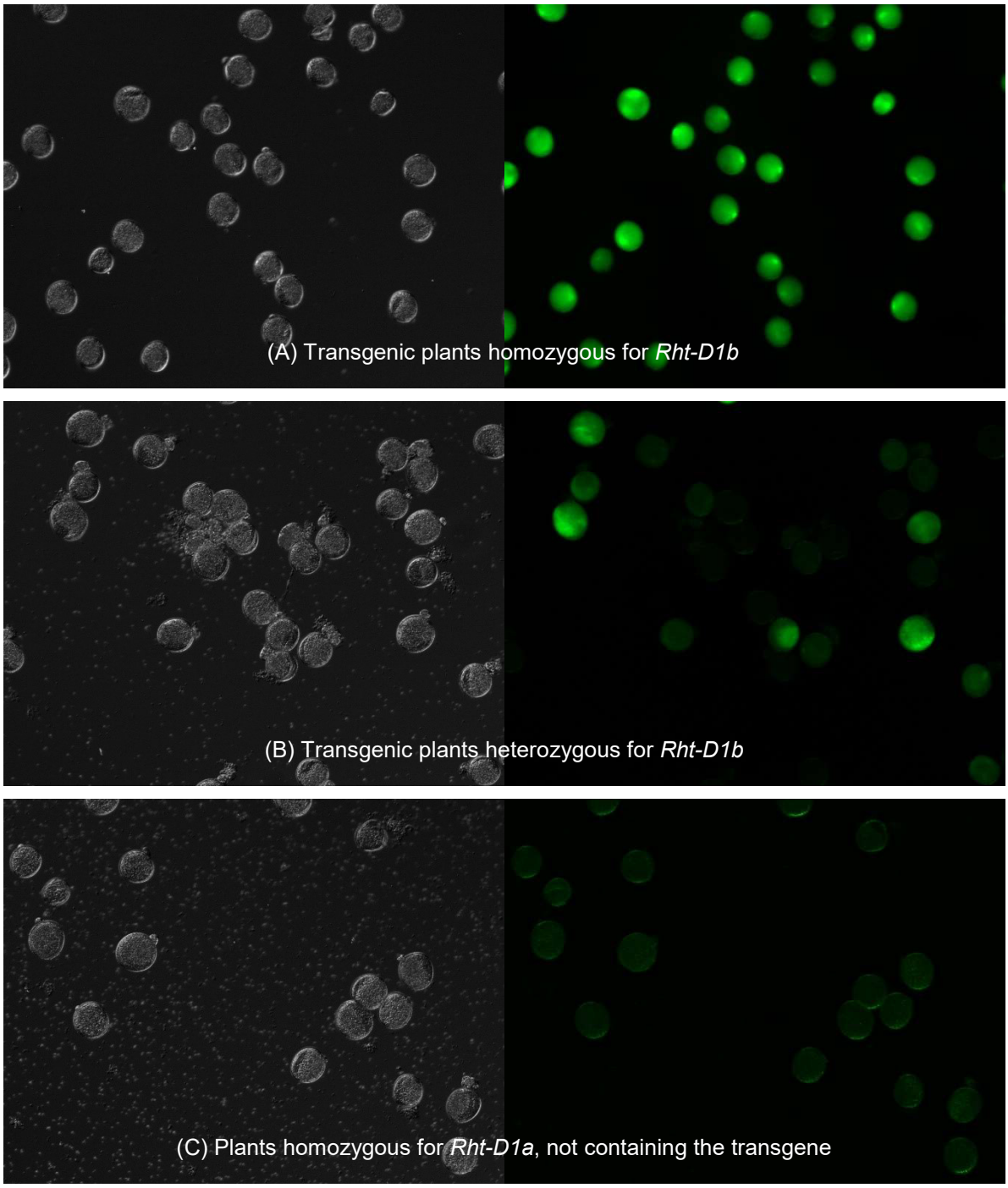

Figure 11: Fluorescence images of different CM mutants. (A) Transgenic plants that are homozygous for *Rht-D1b*. (B) Transgenic plants that are heterozygous for *Rht-D1b*. (C) Plants that are homozygous for *Rht-D1a*, and do not contain the transgene. Visualized with GFP and detected by fluorescence microscopy. Pictures on the left observed with BF, Pictures on the right observed with 494 FITC.

3.9. Data analysis

Pictures of the electrophoresis gels are taken, evaluated and entered into an Excel file. Also, results of fluorescence microscopy are entered in an Excel file.

All plants are derived from F_2 plants of BC_3, BC_4 or BC_5 generation. For the natural lines a segregation of the *Rht1* alleles of 1:2:1 ratio for homozygous wild-type : heterozygous : homozygous mutant (a/a : a/b : b/b) is expected – a is for the tall allele, b for the dwarfing allele. For the transgenic lines the results of genotyping are expected to show a segregation of 1:3 (NN : NT, TT) – N is for non-transgenic or wild-type *Rht-D1a* allele, T for transgenic *Rht-D1b* allele. The ratio of 1:3 is assumed because the heterozygous and homozygous mutants cannot be distinguished by PCR and electrophoresis. For the transgenic lines the results of microscopic analysis are expected to show a segregation of 1:2 (TT : NT). Not all plants homozygous for the wild-type (NN) have been evaluated by fluorescence microscopy. Therefore, they are not considered in the evaluations of microscopic analysis, as homozygous NN can already be unambiguously determined by PCR through absence of PCR amplified product.

A Chi^2 test is performed to test how well the observed segregation of the lines fits to the expected segregation or how likely it is that the observed segregation is due to chance.

H_0 = Observed segregation does not differ from expected segregation.

If $p \leq 0.05$ the results are significant and H_0 is rejected. This means that the expected segregation of 1:2:1 does not match the observed segregation, showing that the observed segregation is not due to chance.

If $p > 0.05$ the results are not significant and H_0 is accepted. This indicates that expected and observed segregation do not differ.

Due to small sample size the plant 1 and plant 2 of each genotype have been calculated together. A higher number of individual values increases degree of significance for Chi^2 test. The *p*-values of the different natural lines have been calculated in Excel and are shown in Table 15. Also, the *p*-values of the different transgenic lines have been calculated in Excel and are shown in Table 16 and 17.

4. Results

NILs containing natural semi-dwarfing genes are characterized according to their allelic status at *Rht-B1* and *Rht-D1* gene.

For PCR different controls have been used: Remus (*Rht-B1a*/*D1a*), Monsun, Bobwhite (*Rht-B1b/D1a*), Toras (*Rht-B1a/D1b*), Hermann (*Rht-B1b/Rht-D1a*), Skalmeje (*Rht-B1a/Rht-D1b*).

An example of an analyzed agarose gel picture is depicted in Figure 12.

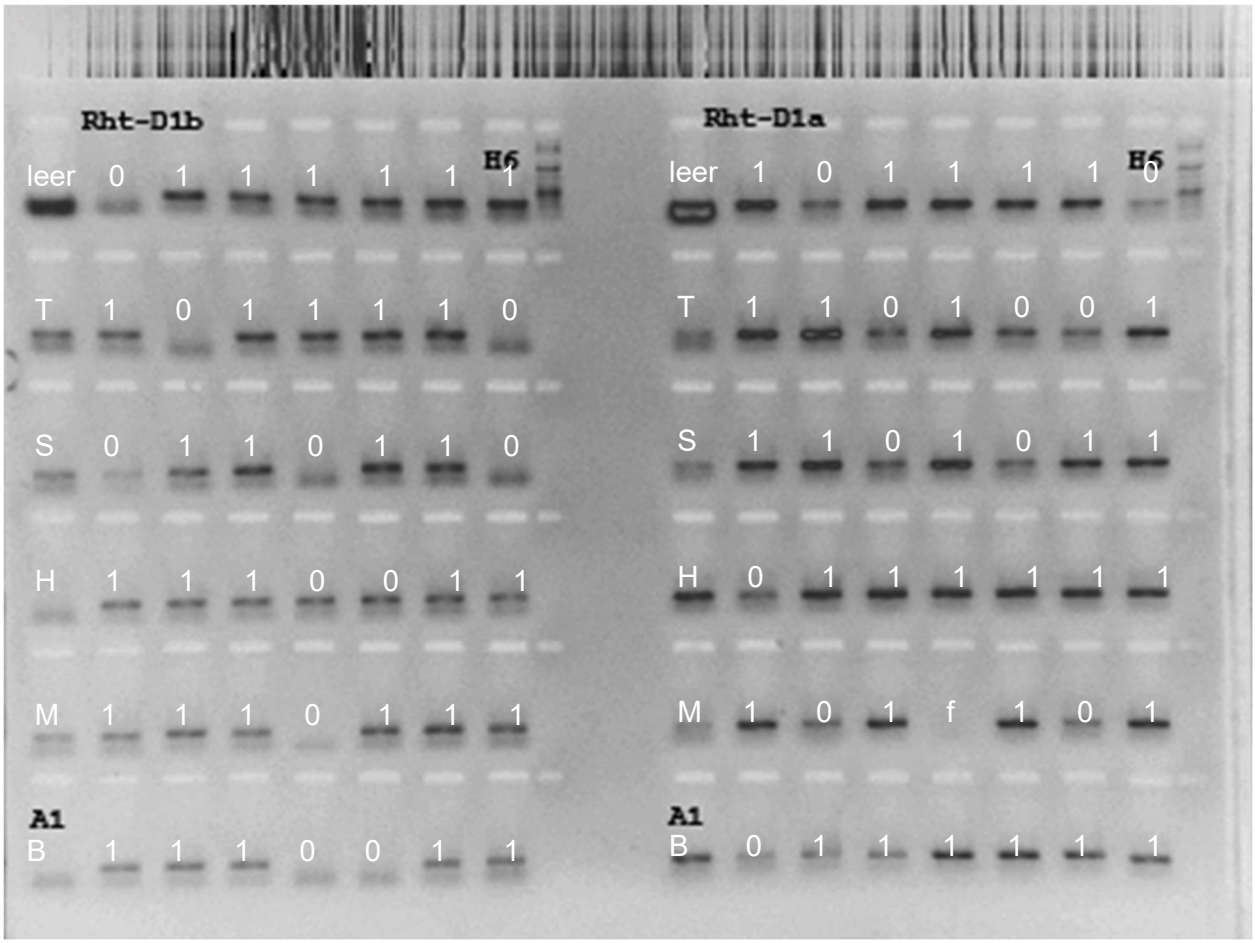

Figure 12: Evaluation of an agarose gel picture of natural line C1. 1 … allele detected, 0 … no allele detected, B … Bobwhite, M … Monsun, H … Hermann, S … Skalmeje, T … Toras, f … missing value

All gel pictures are evaluated and entered in an Excel sheet. For every plant the heterozygous or homozygous mutant- or wild-type was assessed.

4.1. NILs containing natural *Rht1* semi-dwarfing genes

The evaluation of data for the NILs that contain natural *Rht1* genes is shown in Table 15. The expected segregation pattern of a F_2 generation according to Mendel should show a ratio of 1:2:1 for NN : NT : TT.

Table 15: Chi² Test for NILs with different genetic background (C1, CM-82036, E4-61-T, Michael). Expected segregation of 1:2:1 (NN : NT : TT) ratio, for plant 1 and 2 calculated together. Type of allele, line, observed number of individual plants with wild-type, hetero-type or mutant-type is given. Compared to their respective expected number. P-values highlighted in bold are $p \leq 0.05$.

allele	line	obs wild	obs hetero	obs mutant	total	exp wild	exp hetero	exp mutant	total	*p*-value
Rht-D1b	C1	4	14	6	24	6.00	12.00	6.00	18.00	0.53
Rht-D1b	**CM-82036**	**4**	**8**	**15**	**27**	**6.75**	**13.50**	**6.75**	**20.25**	**0.00**
Rht-D1b	**E4-61-T**	**9**	**4**	**14**	**27**	**6.75**	**13.50**	**6.75**	**20.25**	**0.00**
Rht-D1b	Michael	7	13	8	28	7.00	14.00	7.00	21.00	0.61
Rht-B1b	E4-61-T	7	14	7	28	7.00	14.00	7.00	21.00	0.75
Rht-B1b	Michael	7	13	8	28	7.00	14.00	7.00	21.00	0.15
Rht-B1b	C1	4	15	4	23	5.75	11.50	5.75	17.25	0.18
Rht-B1b	CM-82036	4	10	11	25	6.25	12.50	6.25	18.75	0.08

The results show that only for two lines H_0 is rejected, namely for CM-82036 and E4-61-T which contain the natural *Rht-D1b* allele.

For the lines C1 and Michael, carrying the natural *Rht-D1b* allele H_0 is accepted, because expected and observed segregation do not differ. Also, all lines carrying the natural *Rht-B1b* allele match the expected segregation pattern of a 1:2:1 ratio.

4.2. NILs containing transgenic *Rht-D1b* semi-dwarfing genes

For the marker analysis the transgenic lines are expected to segregate in 1:3 ratio (NN : NT, TT), because the heterozygous and homozygous mutant lines cannot be distinguished by PCR and electrophoresis. The results are shown in Table 16.

For microscopic analysis transgenic lines are expected to show a segregation of 1:2 (TT : NT). The results are given in Table 17. Not all plants homozygous for the wild-type have been repotted after genotyping for evaluation with fluorescence microscopy, and therefore these are not considered in the evaluations of microscopic analysis. The heterozygous and homozygous transgenic plants were distinguished using fluorescence microscopy.

It was tested by a Chi^2 test, if the observed and expected segregation pattern differs or not. Plants were grouped by genotype and DN-event.

Table 16: Chi^2 Test for genotyping data of NILs with different genetic background (C1, CM-82036, E4-61-T, Michael). Expected segregation of 1:3 (NN : NT, TT) ratio, grouped by genotype and DN-event. Type of DN-event, line, observed number of individual plants with wild-type, and hetero-type or mutant-type is given. Compared to their respective expected number. P-values highlighted in red color are $p \leq 0.05$.

transgene	line	obs NN	obs TT, NT	total	exp NN	exp TT, NT	total	*p*-value
DN1	C1	18	23	41	13.67	27.33	41	0.06
DN2	C1	10	32	42	14.00	28.00	42	0.14
DN3	C1	17	25	42	14.00	28.00	42	0.30
DN1	CM-82036	14	27	41	13.67	27.33	41	0.58
DN2	CM-82036	10	32	42	14.00	28.00	42	0.06
DN3	CM-82036	13	27	40	13.33	26.67	40	0.22
DN1	E4-61-T	13	29	42	14.00	28.00	42	0.30
DN2	E4-61-T	16	26	42	14.00	28.00	42	0.35
DN4	**E4-61-T**	**25**	**17**	**42**	**14.00**	**28.00**	**42**	**0.00**
DN1	Michael	13	27	40	13.33	26.67	40	0.09
DN2	Michael	12	29	41	13.67	27.33	41	0.18
DN3	Michael	18	24	42	14.00	28.00	42	0.06
DN4	Michael	13	29	42	14.00	28.00	42	0.64

The results of genotyping show that H_0 is accepted for the majority of tested lines, and observed segregation pattern matches expected segregation pattern of 1:3 (NN : NT, TT). The only exception is the transgene DN4 of line E4-61-T, in which distinctly more homozygous non-transgenic plants were found.

Table 17: Chi² Test for data of microscopic evaluation of NILs with different genetic background (C1, CM-82036, E4-61-T, Michael). Expected segregation of 1:2 (TT : NT), grouped by genotype and DN-event. Type of DN-event, line, observed number of individual plants with mutant-type or hetero-type is given. Compared to their respective expected number. P-values highlighted in bold are $p \leq 0.05$.

transgene	line	obs TT	obs NT	total	exp TT	exp NT	total	*p*-value
DN1	C1	9	15	24	8.00	16.00	24	0.06
DN2	C1	9	19	28	9.33	18.67	28	0.08
DN3	**C1**	**3**	**21**	**24**	**8.00**	**16.00**	**24**	**0.03**
DN1	CM-82036	11	28	39	13.00	26.00	39	0.25
DN2	**CM-82036**	**8**	**39**	**47**	**15.67**	**31.33**	**47**	**0.01**
DN3	**CM-82036**	**3**	**25**	**28**	**9.33**	**18.67**	**28**	**0.01**
DN1	**E4-61-T**	**6**	**24**	**30**	**10.00**	**20.00**	**30**	**0.02**
DN2	E4-61-T	8	19	27	9.00	18.00	27	0.54
DN4	**E4-61-T**	**0**	**19**	**19**	**6.33**	**12.67**	**19**	**0.00**
DN1	**Michael**	**2**	**26**	**28**	**9.33**	**18.67**	**28**	**0.00**
DN2	**Michael**	**3**	**26**	**29**	**9.67**	**19.33**	**29**	**0.01**
DN3	**Michael**	**2**	**17**	**19**	**6.33**	**12.67**	**19**	**0.03**
DN4	**Michael**	**2**	**21**	**23**	**7.67**	**15.33**	**23**	**0.01**

After data evaluation of fluorescence microscopy, it was shown that H_0 can be rejected for the transgene DN3 of line C1, for transgenes DN2 and DN3 of line CM-82036, for transgenes DN1 and DN4 of line E4-61-T, and for all transgenes of line Michael. The expected and observed segregation pattern did not match.

Only for transgenes DN1 and DN2 of line C1, DN1 of CM-82036 and DN2 of E4-61-T the expected and observed segregation pattern match.

In Table 18 the lines where observed and expected segregation pattern did not match, and therefore H_0 was rejected, are summarized.

Table 18: H_0 is rejected for the following genotypes.

Natural NILs (1:2:1)		Genotyping of transgenic NILs (1:3)		Microscopic analysis of transgenic NILs (1:2)	
allele	line	transgene	line	transgene	line
Rht-D1b	CM-82036	DN4	E4-61-T	DN3	C1
Rht-D1b	E4-61-T			DN2, DN3	CM-82036
				DN1, DN4	E4-61-T
				DN1, DN2, DN3, DN4	Michael

4.3. Marker-assisted screening for *Fhb1* and *Qfhs.ifa-5A*

To determine if C1 and CM-82036 plants carry the resistance QTL *Fhb1* and *Qfhs.ifa-5A* marker-assisted screening using PAGE was performed. In Figure 13 and Figure 14 examples of a band pattern of markers Gwm304 and Umn10 can be seen. It is highly probable that they carry these QTL, because C1 and CM-82036 are already in BC_5. Nevertheless, it may be due to chance that these QTL are not included. However, MAS was performed to exclude this.

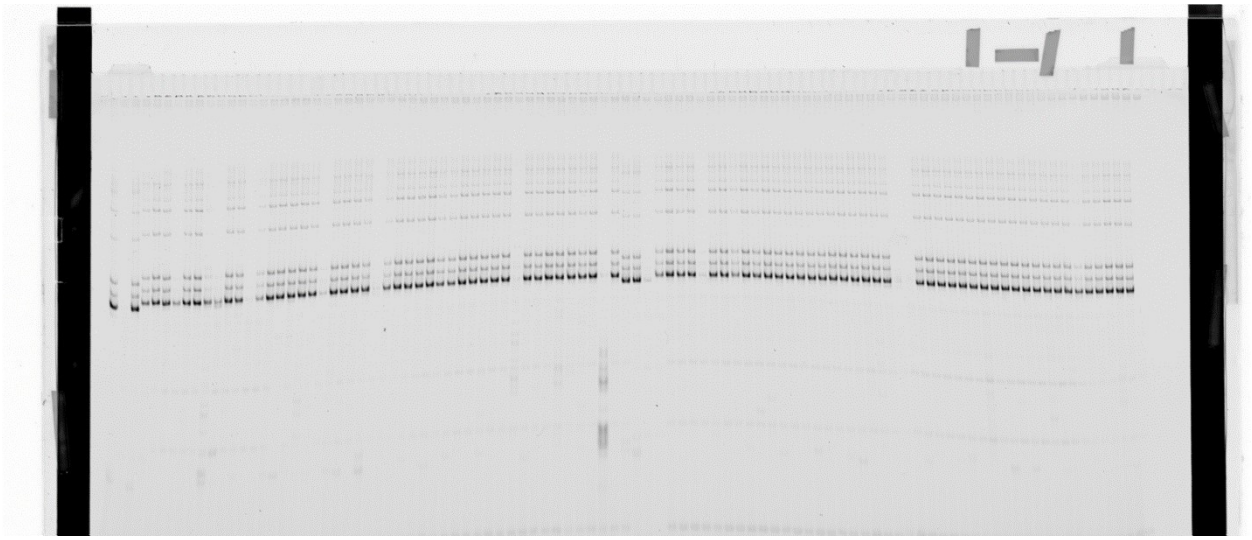

Figure 13: Example of a band pattern of marker Gwm304 (Qfhs.ifa-5A).

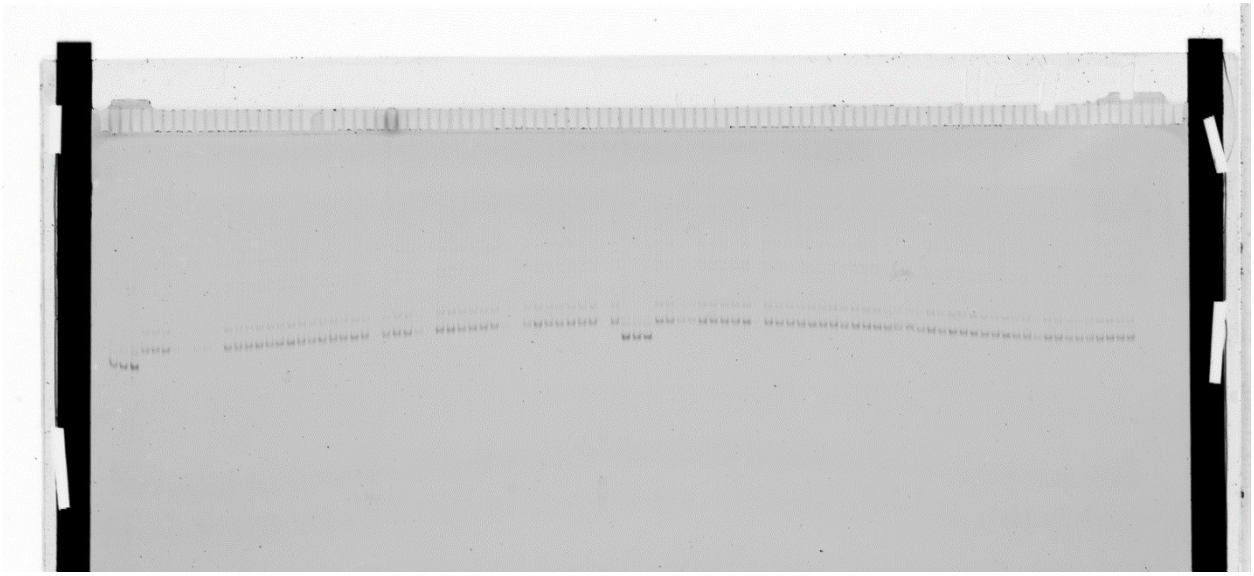

Figure 14: Example of a band pattern of marker Umn10 (Fhb1).

5. Discussion

In general, the agarose gel pictures were clearly to evaluate. However, in some cases lines were not evaluable or missing. This could be due to an error in preparation of probes, e.g. contamination, or gel, e.g. missing peqGREEN.

5.1. Identification of NILs containing natural *Rht-D1b/Rht-B1b* or transgenic *Rht-D1b* semi-dwarfing genes

Heterozygous and homozygous mutants cannot be distinguished by genotyping. To distinguish between NT and TT the progeny of these transgenic plants must be analyzed. If all progeny of these plants contains the transgene after PCR, then it can be validated that the parental plant was homozygous for the transgene. However, this does not automatically mean that these homozygous transgenic plants also show a small plant height, because genes cannot always be expressed. Expression of a transgene is dependent on the location where it is randomly incorporated.

To avoid the time intensive step of producing progeny, a fluorescence microscopic evaluation was done to distinguish between heterozygous and homozygous mutants. However, this method does not always deliver reliable data because of possibly weak fluorescence signals or different individual interpretation of the microscopic image of fluorescing pollen.

Lines, where observed and expected segregation pattern did not match, and therefore H_0 was rejected, are summarized in Table 18. Only for two natural NILs H_0 is rejected, namely for CM-82036 and E4-61-T which contain the natural *Rht-D1b* allele. In these NILs the *Rht-D1b* alleles did not segregate in 1:2:1 ratio according to Mendel. However, for the majority the mendelian segregation pattern holds true. Also, in NILs containing the transgenic *Rht-D1b* alleles one NIL did not segregate according to the expected segregation pattern of 1:3, namely E4-61-T with transgene DN4. The result of dissenting segregation pattern is not desired and may be caused by an error of PCR, whereby the gel pictures were not evaluable or difficult to evaluate.

Microscopic analysis of transgenic NILs show that in each line at least one progeny of one transgenic event (either DN1, DN2, DN3, or DN4) did not match the expected segregation pattern of 1:2, in Michael even none of the transgenic NILs observed matched the expected segregation. This could be, because in some cases pollen only showed weak fluorescence signals. Therefore, it was not clear to classify zygosity of these lines. However, the detection of fluorescent pollen combined with an expected dwarfed phenotype provides good evidence that the transformed gene is expressed.

Generally, it can be said that expected and observed segregation pattern of genotyping match better than these of microscopic evaluation. This can be due to problems in fluorescence microscopy, especially as already mentioned the signal strength of fluorescent pollen. To be on the safe side, these lines were classified as heterozygous. This may be the reason why many more heterozygous lines were found by fluorescence microscopy than by genotyping.

Further, it can be said that data of genotyping is more reliable. Even if expected and observed segregation pattern do not coincide in all cases, at least one homozygous transgenic plant per genotype could be found, except for the transgene DN4 of lines E4-61-T.

It must be pointed out that due to the small sample size, data should be interpreted with caution and no general conclusion for this trial can be drawn.

5.2. Validation of *Fhb1* and *Qfhs.ifa-5A by* marker-assisted screening

In general, the genotyping with the markers (Umn10 and Gwm304) worked well. The successful introgression of the resistance QTL *Fhb1* and *Qfhs.ifa-5A* could be verified in all plants. This is also highly probable in BC_5 generation as the recurrent parents C1 and CM-82036 are carriers of *Fhb1* and *Qfhs.ifa-5A* resistance QTL.

6. Conclusions and outlook

Despite the many published papers on enhancing FHB resistance, it is still not clear whether the connection between cultivars harboring *Rht1* dwarfing alleles are more susceptible to FHB due to close linkage with an undesirable gene, or due to a pleiotropic effect of plant height.

Short plant height means a shorter distance of wheat heads to the soil, where the fungus lives on crop debris. Rebounding raindrops from the ground can take up spores to flowering wheat heads. Furthermore, the canopy structure of shorter plants reduces air circulation, and plants need longer to dry up after dew or rainfalls. The higher chance of spores to reach the heads and the increased humidity around wheat head support infection and may reason the higher susceptibility of short plants compared to tall plants (Mesterházy, 1995; Parry et al., 1995; Rossi et al., 2002).

The *Rht1* alleles not only reduce plant height, but also have an impact on anther extrusion. In many studies the correlation between FHB severity and anther retention was evaluated. It was shown that retained anthers have a strong negative impact on *Fusarium* resistance, as colonization of the fungus often starts on retained or partially extruded anthers and serve as reservoir for further colonization of the surrounding tissue (Buerstmayr and Buerstmayr, 2015; Lu et al., 2013; Skinnes et al., 2010).

In this trial the assumed linkage between small plant height and increased FHB susceptibility was broken by a transgenic approach. The tested and selected homozygous NILs of this master thesis will be used for further phenotyping at the Institute of Biotechnology in Plant Production.

If NILs containing transgenic *Rht-D1b* alleles react like NILs with natural *Rht-D1b* alleles a pleiotropic effect can be assumed. However, if linkage is the reason for increased FHB susceptibility of plants carrying the semi-dwarfing alleles, it is expected that NILs with natural *Rht-D1b* alleles are susceptible, whereas NILs with transgenic *Rht-D1b* alleles are expected to be no longer susceptible, because the *Rht-D1b* alleles were randomly introduced and are thus unlinked with possible resistance/susceptibility genes.

This work may be seen as preparatory work for further trials.

Phenotyping of wheat mutant lines treated with the *Fusarium* mycotoxin deoxynivalenol

7. Materials and methods

7.1. Plant material

7.1.1. Mutant lines deriving from the highly resistant *Fhb1* donor line CM_82036

CM_82036 derives from a cross Sumai-3 x Thornbird-S and was developed in a shuttle breeding program between CIMMYT in Mexico and South America. It is highly resistant to FHB and carries both major QTL for *Fusarium* resistance, namely *Fhb1* and *Qfhs.ifa-5A* (Buerstmayr et al., 2002).

A TILLING population, which derives from the cultivar CM_82036, was mutagenized with EMS in 2009. From the M_1 generation, which grew from the EMS treated seeds, one head was harvested each and only one single seed of these heads was planted again to gain the M_2 generation. This process was repeated until M_4 generation. Leaves of M_2 and M_4 were harvested and DNA was extracted. Finally, 3,500 lines of M_2 and 3,050 lines of M_4 were obtained.

Seidl (2017) screened this mutant population of 6,000 lines (3,500 lines of M_2 generation, and 3,050 lines of M_4 generation) for mutations in the genes of interest. These mutations can lead to loss of functionality of the corresponding protein. The genes of interest are gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein), gene #20 (Hypothetical calcium binding protein) and gene #6 (Glycosyltransferase hga7). Seidl (2017) identified 157 lines with deleterious mutations in the three aforementioned candidate genes.

After screening of M_2 and M_4 generation, the seeds of selected lines in M_3 and M_5 generation were harvested and used for phenotyping of FHB resistance. Previously, it was important to know which of the selected lines carry the mutation in homozygous or heterozygous state. Only plants homozygous for the mutation can be used for phenotyping.

Thus, for the heterozygous lines 48 kernels of M_3 or M_5 per line were sown and sequenced for the respective gene. Plants, which are homozygous for the mutation or homozygous for the wild-type were selected. These individual plants have been analyzed in this greenhouse trial for susceptibility or resistance against DON and FHB. If the mutation of the investigated gene causes a dysfunction of a specific protein, this could indicate that the aforementioned gene(s) might be involved in the resistance mechanism.

The selected lines are listed in Table 19. In total 41 lines were selected for the greenhouse trial, whereby 28 lines harbor the mutation in homozygous state, and 13 lines in heterozygous state.

Table 19: List of selected lines with mutations in candidate genes based on the work of Seidl (2017).

	Lines with homozygous mutations		Lines with heterozygous mutations	
Lines carrying mutations in gene #6 (Glycosyltransferase hga7)	723_Glyco 940_Glyco 1148_Glyco 1369_Glyco 3093_Glyco	738_Glyco 1111_Glyco 1345_Glyco 1616_Glyco 3632_Glyco	88_Glyco 1500_Glyco 3094_Glyco	
Lines carrying mutations in gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein)	182_Ubi 481_Ubi* 869_Ubi 1254_Ubi 1632_Ubi 3746_Ubi	342_Ubi 721_Ubi 1047_Ubi 1325_Ubi 3045_Ubi	48_Ubi 448_Ubi 641_Ubi 2187_Ubi	
Lines carrying mutations in gene #20 (Hypothetical calcium binding protein)	98_Ca 784_Ca 6293_Ca 8892_Ca	219_Ca 1914_Ca 8406_Ca	1722_Ca 6504_Ca 6688_Ca 8032_Ca	6046_Ca 6551_Ca 8023_Ca

7.1.2. Control lines

CM_Nil38, CM_Nil43, CM_Nil47 and CM_Nil51 are near isogenic lines (NILs) and all four derive from the cross CM_82036 x Remus. The NILs share almost the same genetic background, due to five times backcrossing to CM_82036. They only differ in the loci that harbor the two resistance QTL *Fhb1* and *Qfhs.ifa-5A*. CM_Nil38 carries both resistance alleles, such as CM_82036, and was also used as positive control. CM_Nil43 only carries *Fhb1*, but not *Qfhs.ifa-5A*. CM_Nil47 only carries *Qfhs.ifa-5A* and CM_Nil51 lacks both QTL regions. The control lines used in this trial are summarized in Table 20.

Remus is a spring wheat cultivar and highly susceptible to FHB. However, it is well adapted to the conditions in central Europe. It was developed at the Bavarian State Institute for Agronomy in Freising, Germany (Buerstmayr et al., 2002, 2003).

The radiation hybrid (RH) deletion lines RH_CM_643, RH_CM_651 and RH_CM_83 all have a deletion at *Fhb1*, and have been identified in the master thesis of Allerstorfer (2017). These RH deletion lines were used as negative control.

Table 20: Summary of control lines.

Control Line	Resistance QTL Status	Parental Lines
CM_82036	*Fhb1* and *Qfhs.ifa-5A*	Sumai-3 x Thornbird-S
CM_Nil38	*Fhb1* and *Qfhs.ifa-5A*	CM_82036 x Remus
CM_Nil43	*Fhb1*	
CM_Nil47	*Qfhs.ifa-5A*	
CM_Nil51	Susceptible alleles	
Remus	Susceptible line	-
RH_CM_643	Deletion at *Fhb1*	gamma-irradiation-induced mutant lines of CM_82036
RH_CM_651	Deletion at *Fhb1*	
RH_CM_83	Deletion at *Fhb1*	

7.1.3. Other *Fhb1* donors

Sumai-3 is a famous Chinese spring wheat cultivar and was selected from a cross Funo x Taiwan-Wheat at the Suzhou Institute of Agricultural Science in Jiangsu province of China in 1972 (Yang, 1994). It carries the resistance QTL *Fhb1* and confers mainly type II resistance (Cuthbert et al., 2006). Today, it is widely used in breeding programs as a source of resistance.

Ning_7840 and W14 are also resistant Chinese wheat genotypes.

The Asian landraces Wangshuibai and Nobeokabozu_U are highly resistant to FHB, but they only show poor agronomic traits (Buerstmayr et al., 2012).

ND_2710 is a FHB resistant spring wheat cultivar, which was developed at North Dakota State University (NDSU) from a cross ND_2603 x Grandin, made in 1991. The cultivar was released by North Dakota Agricultural Experiment Station (NDAES) in June 1998 (Frohberg et al., 2004).

7.1.4. Verification and characterization of mutations in three tested candidate genes in *Fhb1* locus, according to Seidl (2017)

Seidl (2017) screened the mutant population for mutations in the *Fhb1* region with primers that are highly specific for the corresponding region of *Fhb1* locus. The mutations were verified and characterized. The mutant lines used for phenotyping in this work are summarized in Table 21, 23, 25.

The effects of the mutation on protein functionality were predicted using SIFT and PROVEAN. Further, a protein BLAST search was carried out and homologous sequences were compared with the amino acid sequence of the investigated gene. The results of the prediction for the effect of the mutation are summarized in Table 22, 24, 26.

The classification of mutations is the following: silent, missense, nonsense. In a silent mutation the corresponding codon still encodes for the same amino acid after base exchange by the mutation and no effect on the protein is detectable. Lines with a silent mutation were not selected for phenotyping.

However, all missense mutations detected by Seidl (2017) were used in this trial. In a missense mutation a nucleotide change in a base triplet occurs and changes the codon to encode for another amino acid. The effect on the resulting protein depends on the type of exchange and position. The effect can be total loss of protein functionality or only a very weak effect, if the changed amino acid has similar properties as the amino acid of the wild-type and therefore the protein is only slightly affected. However, the desired mutation is a nonsense mutation, which leads to a total loss of protein function. The amino acids tryptophan (W), arginine (R) or glutamine (Q) could potentially be converted into stop codons by mutagenesis with the chemical EMS. This would result in a nonsense mutation (Maquat, 2001).

Glycosyltransferase hga7 (gene #6)

Only missense mutations could be found in gene #6, encoding for Glycosyltransferase hga7. The tested lines and information on their mutation is given in Table 21. The prediction of the effect of the mutation is shown in Table 22.

Table 21: List of mutations verified by sequencing for gene #6 (Glycosyltransferase hga7) according to (Seidl, 2017). The following information is given: Plant ID, location of mutation site (coding or non-coding), type of nucleic acid exchange, zygosity status of mutation, type of amino acid exchange, classification of mutation (missense, nonsense or silent) and position of mutation in nucleic acid sequence.

Plant ID	Mutation site	Mutation	Zygosity	Amino acid change	Type	Nucleic acid position
723_Glyco	Exon 2	C → T	homo	A → T	missense	1251
738_Glyco	Exon 2	G → A	homo	P → S	missense	981
940_Glyco	Exon 2	G → A	homo	L → F	missense	27
1111_Glyco	Exon 2	G → A	homo	P → L	missense	1117
1148_Glyco	Exon 2	C → T	homo	V → M	missense	1155
1345_Glyco	Exon 2	G → A	homo	P → S	missense	111
1369_Glyco	Exon 2	G → A	homo	A → V	missense	1207
1616_Glyco	Exon 2	G → A	homo	P → L	missense	991
3093_Glyco	Exon 2	G → A	homo	L → F	missense	828
3093_Glyco	Exon 2	C → T	homo	A → T	missense	585
3632_Glyco	Exon 2	C → T	homo	R → Q	missense	1012
88_Glyco	Exon 2	G → A	hetero	P → S	missense	537
1500_Glyco	Exon 2	G → A	hetero	H → Y	missense	495
3094_Glyco	Exon 2	C → T	hetero	V → M	missense	1188

Table 22: Prediction of mutation effects on the resulting protein using SIFT, PROVEAN and BLAST alignment for gene #6 (Glycosyltransferase hga7) according to (Seidl, 2017). The following information is given: Plant ID, SIFT code, PROVEAN code, PROVEAN prediction, SIFT prediction, SIFT score, median sequence conservation, sequence represented, and alignment.

Plant ID	SIFT code	PROVEAN score	PROVEAN prediction	SIFT predicition	SIFT score	Median sequence conservation	Sequence represented	Alignment
723_Glyco	A375T	0.283	neutral	tolerated	0.15	3.01	16	0.67
738_Glyco	P287S	0.546	neutral	tolerated	0.15	3.23	13	0.67
940_Glyco	L10F	0.544	neutral	tolerated	0.92	4.32	3	0.83
1111_Glyco	P332L	-1.401	neutral	affect protein function	0.05	3.01	16	0.50
1148_Glyco	V345M	0.029	neutral	tolerated	1.00	3.01	16	0.50
1345_Glyco	P38S	-1.907	neutral	tolerated	0.12	3.33	8	0.83
1369_Glyco	A362V	-3.933	deleterious	affect protein function	0.00	3.01	16	0.50
1616_Glyco	P290L	-9.410	deleterious	affect protein function	0.00	3.03	15	0.83
3093_Glyco	A155T	-3.510	deleterious	affect protein function	0.02	3.01	16	0.67
3093_Glyco	L236F	-2.992	deleterious	affect protein function	0.02	3.01	16	0.67
3632_Glyco	R297Q	-3.975	deleterious	affect protein function	0.00	3.01	16	0.67
88_Glyco	P139S	0.372	neutral	tolerated	0.98	3.29	10	0.67
1500_Glyco	H125Y	0.411	neutral	tolerated	1.00	3.01	16	0.67
3094_Glyco	V356M	-1.592	neutral	tolerated	0.10	3.01	16	0.50

For five mutant lines PROVEAN predicts a deleterious mutation and SIFT predicts an effect on protein function.

Ubiquitin-2 like Rad60 SUMO-like protein (gene #17)

For gene #17, encoding for Ubiquitin-2 like Rad60 SUMO-like protein, two gene models were available, namely gene model 51 and 17_ub1-2. Seidl (2017) evaluated the found mutations once for each gene model. However, in Table 23 only the results of the second gene model, named 17_ub1-2, are summarized.

Table 23: List of mutations verified by sequencing for gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein) according to (Seidl, 2017). The following information is given: Plant ID, location of mutation site (coding or non-coding), type of nucleic acid exchange, zygosity status of mutation, type of amino acid exchange, classification of mutation (missense, nonsense or silent) and position of mutation in nucleic acid sequence.

Plant ID	Mutation site	Mutation	Zygosity	Amino acid change	Type	Nucleic acid position
182_Ubi	Exon 2	G → A	homo	P → S	missense	1048
342_Ubi	Exon 1	C → T	homo	R → H	missense	16
481_Ubi*	Exon 2	C → T	homo	W → stop codon	nonsense	1440
721_Ubi	Exon 2	C → T	homo	G → E	missense	1223
869_Ubi	Exon 1	C → T	homo	R → H	missense	16
1047_Ubi	Exon 2	C → T	homo	D → N	missense	1114
1254_Ubi	Exon 2	C → T	homo	D → N	missense	1009
1325_Ubi	Exon 2	C → T	homo	G → E	missense	1148
1632_Ubi	Exon 1	C → T	homo	E → D	missense	82
3045_Ubi	Exon 2	G → A	homo	S → F	missense	1190
3746_Ubi	Exon 2	G → A	homo	P → S	missense	1258
448_Ubi	Exon 2	C → T	hetero	V → M	missense	1321
641_Ubi	Exon 2	G → A	hetero	P → L	missense	1331
2187_Ubi	Exon 2	C → T	hetero	D → N	missense	1285

Thirteen missense mutations were discovered and have a possibly deleterious effect on the protein as summarized in the following Table 24.

In line 481_Ubi a nonsense mutation was discovered, which has the most prominent effect on protein function. The nucleotide exchange results in a codon for T that is changed to a stop codon. This mutation leads to a truncated protein, which is most likely not functional anymore. Therefore, this line is the most promising candidate for this phenotyping experiment to find out whether gene #17 is involved in DON or *Fusarium* resistance (Seidl, 2017).

Table 24: Prediction of mutation effects on the resulting protein using SIFT, PROVEAN and BLAST alignment for gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein) according to (Seidl, 2017). The following information is given: Plant ID, SIFT code, PROVEAN code, PROVEAN prediction, SIFT prediction, SIFT score, median sequence conservation, sequence represented, and alignment.

Plant ID	SIFT code	PROVEAN score	PROVEAN prediction	SIFT predicition	SIFT score	Median sequence conservation	Sequence represented	Alignment
182_Ubi	P76S	0.270	neutral	tolerated	0.10	3.83	4	0.67
342_Ubi	R6H	-0.024	neutral	affect protein function	0.00	4.32	1	0.00
481_Ubi*	W207_A245del	-22.818	deleterious	n/a	n/a	n/a	n/a	n/a
721_Ubi	G134E	-5.003	deleterious	tolerated	0.08	3.06	61	0.83
869_Ubi	R6H	-0.024	neutral	affect protein function	0.00	4.32	1	0.00
1047_Ubi	D98N	-3.167	deleterious	tolerated	0.24	3.10	62	1.00
1254_Ubi	D63N	-0.810	neutral	affect protein function	0.00	4.32	1	0.00
1325_Ubi	G109E	1.603	neutral	tolerated	0.19	3.06	63	0.17
1632_Ubi	E29D	-0.024	neutral	affect protein function	0.00	4.32	1	0.00
3045_Ubi	S123F	-1.833	neutral	affect protein function	0.02	3.12	61	0.33
3746_Ubi	P146S	0.841	neutral	tolerated	0.37	3.15	29	0.33
448_Ubi	V167M	-0.256	neutral	tolerated	0.09	3.09	30	0.67
641_Ubi	P170L	-2.745	deleterious	affect protein function	0.02	3.15	18	1.00
2187_Ubi	D155N	-0.947	neutral	affect protein function	0.02	3.06	62	0.50

The predictions for the mutations from PROVEAN and SIFT gave contradictory results for most of the missense mutations. For three mutations PROVEAN predicted a deleterious effect on the resulting protein, and for seven mutations SIFT predicted an effect on protein function. Only in line 641_Ubi PROVEAN and SIFT corresponded. The contradictory results can be due to the use of different databases of both programs for protein sequence search (Seidl, 2017).

Hypothetical calcium binding protein (gene #20)

For gene #20, encoding for Hypothetical calcium binding protein, two different gene models were available. Seidl (2017) evaluated the found mutations once for each gene model. In Table 25 the results for the mutant lines are summarized.

Table 25: List of mutations verified by sequencing for gene #20 (Hypothetical calcium binding protein) according to (Seidl, 2017). The following information is given: Plant ID, location of mutation site (coding or non-coding), type of nucleic acid exchange, zygosity status of mutation, type of amino acid exchange, classification of mutation (missense, nonsense or silent) and position of mutation in nucleic acid sequence.

Plant ID	Mutation site	Mutation	Zygosity	Amino acid change	Type	Nucleic acid position
98_Ca	Exon	C → T	homo	S → N	missense	n/a
219_Ca	Exon	C → T	homo	R → K	missense	n/a
784_Ca	Exon	G → A	homo	L → F	missense	n/a
1914_Ca	Exon	C → T	homo	V → M	missense	n/a
6293_Ca	n/a	C → T	homo	R → K	missense	n/a
8406_Ca	n/a	C → T	homo	D → N	missense	n/a
8892_Ca	n/a	C → T	homo	R → K	missense	n/a
1722_Ca	Exon 3	G → A	hetero	S → F	missense	1500
6046_Ca	n/a	C → T	hetero	R → K	missense	n/a
6504_Ca	n/a	G → A	hetero	H → Y	missense	n/a
6551_Ca	n/a	G → A	hetero	H → Y	missense	n/a
6688_Ca	n/a	C → T	hetero	E → K	missense	n/a
8023_Ca	n/a	C → T	hetero	S → L	missense	n/a
8032_Ca	n/a	C → T	hetero	R → K	missense	n/a

Only missense mutations could be found in gene #20. The predicted effect of the found missense mutations on the protein is summarized in Table 26.

Table 26: Prediction of mutation effects on the resulting protein using SIFT, PROVEAN and BLAST alignment for gene #20 (Hypothetical calcium binding protein) according to (Seidl, 2017). The following information is given: Plant ID, SIFT code, PROVEAN code, PROVEAN prediction, SIFT prediction, SIFT score, median sequence conservation, sequence represented, and alignment.

Plant ID	SIFT code	PROVEAN score	PROVEAN prediction	SIFT predicition	SIFT score	Median sequence conservation	Sequence represented	Alignment
98_Ca	S131N	-0.478	neutral	n/a	0.02	n/a	n/a	n/a
219_Ca	R146K	0.167	neutral	n/a	0.29	n/a	n/a	n/a
784_Ca	L32F	-1.089	neutral	n/a	0.03	n/a	n/a	n/a
1914_Ca	V21M	-0.267	neutral	n/a	0.24	n/a	n/a	n/a
6293_Ca	R173K	0.250	neutral	n/a	0.74	n/a	n/a	n/a
8406_Ca	D91N	-0.456	neutral	n/a	0.47	n/a	n/a	n/a
8892_Ca	R154K	-0.111	neutral	n/a	0.64	n/a	n/a	n/a
1722_Ca	S227F	-2.600	deleterious	n/a	n/a	n/a	n/a	0.67
6046_Ca	R108K	0.200	neutral	n/a	0.33	n/a	n/a	n/a
6504_Ca	H217Y	-2.578	deleterious	n/a	0.07	n/a	n/a	n/a
6551_Ca	H176Y	-2.633	deleterious	n/a	0.63	n/a	n/a	n/a
6688_Ca	E128K	-1.011	neutral	n/a	0.04	n/a	n/a	n/a
8023_Ca	S113L	-2.722	deleterious	n/a	0.11	n/a	n/a	n/a
8032_Ca	R65K	0.267	neutral	n/a	0.72	n/a	n/a	n/a

For line 1722_Ca, 6504_Ca, 6551_Ca and 8023_Ca a mutation with deleterious effect on the protein function was predicted by PROVEAN. Unfortunately, there were no values available for SIFT prediction.

7.2. Conduction of the greenhouse trial

The trial was conducted in Tulln at the Institute of Biotechnology in Plant Production, Department of Agrobiotechnology (IFA), University of Natural Resources and Life Sciences Vienna (BOKU).

The greenhouse trial consisted of two individual experiments, with the same plant material for both experiments. It was designed as a randomized complete block experiment with two blocks as replications and genotypes (pots) within the blocks were randomized.

For the phenotyping experiment it is important to know whether the mutant lines are homozygous or heterozygous for the mutation. According to the law of segregation of Mendelian inheritance, in the next generation of heterozygous plants one quarter will be homozygous for the mutation, one quarter will not harbor the mutation (homozygous for wild-type allele) and one half will be heterozygous for the mutation (ratio of 1:2:1).

Only plants that are homozygous for the mutation can be used for phenotyping. Therefore, for the heterozygous plants more seeds had to be planted. Also, the heterozygous plants allow for a direct phenotypic comparison between wild-type allele and mutant allele. For the mutations in heterozygous state the M_2/M_4 genotyping generation was screened before phenotyping to identify the allelic status. Lines in M_3/M_5 generation either homozygous for the mutation or homozygous for the wild-type allele were selected and planted in pots together according to their allelic status and the respective mutation.

For the homozygous mutant lines and the control lines four pots per line with 20 kernels each were sown, to obtain at least five plants per pot. One pot for both replications of the DON and *Fusarium* experiment resulting in four pots per genotype with five plants each.

During anthesis of wheat plants, the temperature at daytime was constantly held at 22°C and at nighttime temperature was at 18°C. The plants had about 16 h of light per day at 15,000 lux.

7.3. Treatment of plants with the mycotoxin DON

7.3.1. Preparation of DON solution

The mycotoxin DON was produced according to Standard Operating Procedure SOP 10-00 of the Institute of Biotechnology in Plant Production and was provided by M. Lemmens. The desired *Fusarium* strain was grown on SNA-agar and was used to inoculate liquid SNA-medium. An aliquot of this culture was transferred on freshly prepared rice-medium and incubated for several weeks. After chemical and mechanical treatment of the culture, the suspension was poured through a filter. By ion exchange chromatography the mycotoxin DON could be crystallized.

Just before treatment of plants DON crystals were dissolved in ddH_2O by heating and stirring for about five minutes. The solution was mixed with ddH_2O to achieve a DON concentration of 12 g/l DON and one droplet of 0.1% Tween 20 was added to reduce surface tension.

7.3.2. Preparation of *Fusarium* inoculum

F. graminearum (Isolate IFA65) inoculum, with a concentration of 50,000 conidia/ml, was prepared according to Standard Operating Procedure SOP 3-01 of the Institute of Biotechnology in Plant Production and was provided by M. Lemmens.

7.3.3. Infiltration of flowering wheat plants with DON

Infiltration with DON was done at time of anthesis. Before the treatment with DON all flowering heads were tagged with numbered labels in distinct colors, attached on the stem. The key for the numbered labels with infiltration/inoculation date and evaluation date is shown in Table 27 and is valid for DON and *Fusarium* experiment.

On each flowering head two spikelets (four florets) were infiltrated with 20 µl per floret of DON solution, which makes a total of 0.96 g DON/80 µl per head. The spore suspension of 12 g/l is pipetted between palea and lemma of the spikelets. The maximum of treated heads was about 5 heads per pot. After treatment a plastic bag sprayed with water was put over the infiltrated heads for 48 h (Figure 15). This was done to make sure that the mycotoxin is provided with optimal conditions for absorption by the wheat head.

Table 27: Date of infiltration, respectively inoculation. A number from 1 to 16 on the labels was used as abbreviation for the infiltration/inoculation date, and to mark all flowering wheat heads with the same treatment date. Evaluation date was determined 20 and 25 days after inoculation (dai).

Label	Infiltration/ inoculation date	Evaluation date	
		20 dai	25 dai
1	15.11.2017	05.12.2017	10.12.2017
2	17.11.2017	07.12.2017	12.12.2017
3	20.11.2017	10.12.2017	15.12.2017
4	22.11.2017	12.12.2017	17.12.2017
5	24.11.2017	14.12.2017	19.12.2017
6	27.11.2017	17.12.2017	22.12.2017
7	29.11.2017	19.12.2017	24.12.2017
8	01.12.2017	21.12.2017	26.12.2017
9	04.12.2017	24.12.2017	29.12.2017
10	06.12.2017	26.12.2017	31.12.2017
11	08.12.2017	28.12.2017	02.01.2018
12	11.12.2017	31.12.2017	05.01.2018
13	13.12.2017	02.01.2018	07.01.2018
14	15.12.2017	04.01.2018	09.01.2018
15	18.12.2017	07.01.2018	12.01.2018
16	20.12.2017	09.01.2018	14.01.2018

7.3.4. Inoculation of flowering wheat plants with spore suspension of *F. graminearum*

At anthesis the wheat heads were inoculated with a spore suspension of *F. graminearum* (Isolate IFA65) with a concentration of 50,000 conidia/ml. On flowering heads two spikelets (four florets) were inoculated with 10 µl per floret, which makes a total of 2,000 conidia/40 µl per head and covered with a plastic bag (Figure 15). About 10 heads per pot were inoculated with a spore suspension of *F. graminearum*, sometimes even more.

Figure 15: Treated wheat heads covered with plastic bag.

7.3.5. Evaluation of DON severity

Phenotyping was done two times, 20 and 25 days after inoculation (dai), whereas in the last rating premature DON bleaching and natural yellowing caused by ripening was difficult to distinguish. Infected spikelets show premature bleaching whereas the rest of the spike is still green.

The number of DON bleached spikelets per pot and head/plant was counted after 20 and 25 dai. Data was entered in a greenhouse book. The greenhouse book is provided in the Appendix.

7.3.6. Evaluation of Fusarium Head Blight severity

Phenotypic evaluations were performed two times, 20 and 25 dai, as described for the DON trial. The resistance to fungal spread from an infected floret along the rachis (type II resistance) was measured.

The number of *Fusarium* bleached spikelets per pot and head/plant was counted after 20 and 25 dai, whereas the two inoculated spikelets should always show symptoms of *Fusarium* infection in susceptible and resistant varieties. This confirms the effectiveness of the used inoculation method to measure disease spreading. Data was entered in a greenhouse book. The greenhouse book is provided in the Appendix.

7.4. Data analysis

The date of inoculation and infected spikelets per head were recorded in a greenhouse book. The data was then entered in an Excel sheet, and additionally edited with the free software 'R Project for Statistical Computing'. The R-protocol is listed in the Appendix.

For each pot (= replication) the DON severity, respectively *Fusarium* severity was calculated as the mean number of symptomatic spikelets per head (number of bleached spikelets per line/number of treated heads per line) 20 dai and 25 dai.

The Pearson Correlation Coefficient for first and second rating, for infiltration/inoculation date and number of DON-bleached/FHB-symptomatic spikelets per individually treated head was calculated.

The repeatability (REP) was calculated for the two replications for DON and FHB of the greenhouse trial (Formula 1). The values range from 0 to 1. A value close to 1 indicates high REP, which means that replication one and two lead to similar results.

$$REP = 1 - \frac{MS_{residuals}}{MS_{lines}}$$

Formula 1: Formula for repeatability. $MS_{residuals}$ … mean square of residuals from calculated ANOVAs; MS_{lines} … mean square of lines from calculated ANOVAs.

A Students' t-test (unpaired, two-sided, α = 0.05) was calculated to identify significant differences between genotypes.

$$t = \frac{\bar{x}_1 - \bar{x}_2}{\sqrt{s^2\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}}$$

$$s^2 = \frac{\sum_{i=1}^{n_1}(x_i - \bar{x}_1)^2 + \sum_{j=1}^{n_2}(x_j - \bar{x}_2)^2}{n_1 + n_2 - 2}$$

Formula 2: Formula for unpaired t-test. $\bar{x}_1, \bar{x}_2$ … sample means; s^2 … pooled sample variance; n_1, n_2 … sample size

8. Results

In this experiment lines with homozygous and heterozygous mutations have been tested for DON/FHB resistance and were evaluated separately. For the lines with homozygous mutations a pot mean was calculated and for the lines with heterozygous mutations a plant mean was calculated.

First, a general overview on the number of treated lines is provided. Then, the correlation coefficient between the first (20 dai) and second rating (25 dai) was calculated. Also, REP was calculated to assess if both replications within an experiment lead to the same results.

The homozygous and heterozygous lines are interpreted separately. The correlation coefficient for infiltration date and number of DON bleached spikelets as well as the inoculation date and FHB symptomatic spikelets per individually treated head 20 dai is calculated.

Also, general statistics (min, max, mean, SD) for DON and *Fusarium* severity is calculated. Symptom severity is the number of DON bleached or FHB bleached spikelets per individually treated head.

Boxplots for all lines with homozygous or heterozygous mutations, separate for treatment with DON and *Fusarium* inoculum, summarize the results of the greenhouse trial.

8.1. General overview of mutant population

For the lines with homozygous mutations 305 heads were infiltrated with DON and 497 heads with spore suspension of *F. graminearum*. The line 98_Ca was only treated with DON, as in the *Fusarium* experiment the plants did not grow. A more detailed list is given in Table 28, where the number of lines, the number of pots and infiltrated/inoculated heads for each candidate gene is provided.

Table 28: Summary of treated lines with homozygous mutations.

	Gene #6			Gene #17			Gene #20		
	lines	pots	heads	lines	pots	heads	lines	pots	heads
Infiltration with DON*	10	20	103	11	22	114	9	18	88
Inoculation with *Fusarium**	10	20	181	11	21	184	8	16	132

* replication 1 and 2 summarized

In total, 467 heads of lines with heterozygous mutations were infiltrated with DON and 221 heads with *Fusarium* inoculum. Although seeds of every line, for each mutant and wild-type, were sown, several seeds did not germinate, or some plants did not grow heads. In the DON experiment the wild-type of lines 48_Ubi and 8023_Ca were missing. In the *Fusarium* experiment the mutant-type of line 88_Glyco and wild-type of line 3094_Glyco were missing. Also, the plants for wild-type of lines 6046_Ca and 6688_Ca did not grow. In Table 29 a more detailed list on the treated lines, their number of genotypes and infiltrated/inoculated heads for each candidate gene is shown.

Table 29: Summary of lines with heterozygous mutations.

		Gene #6			Gene #17			Gene #20		
		lines	genotypes	heads	lines	genotypes	heads	lines	genotypes	heads
Infiltration with DON	mutant	3	20	55	5	32	77	7	39	103
	wild-type	3	21	59	4	30	78	6	36	95
Inoculation with *Fusarium*	mutant	2	15	35	4	22	53	6	17	36
	wild-type	2	10	27	4	21	44	4	12	26

8.1.1. Pictures of DON and *Fusarium* treated control lines

Pictures of the six control lines with (CM_82036, CM_Nil38, CM_Nil43) or without (CM_Nil47, CM_Nil51, Remus) *Fhb1* after DON treatment are shown in Figure 16.

Figure 16: Pictures of control lines after DON infiltration. Control lines harboring the resistant *Fhb1* alleles (CM_82036, CM_Nil38, CM_Nil43), control lines with the susceptible *Fhb1* alleles CM_NIL47, CM_NIL51) and highly susceptible cultivar Remus are shown. The different symptom severities can be clearly seen.

It can be clearly seen in Figure 16 that the resistant cultivars (CM_82036, CM_Nil38, CM_Nil43) do not show any infection. They all harbor *Fhb1* and are therefore resistant to DON bleaching. However, there are significant differences between cultivars that carry *Fhb1* and those without *Fhb1*. The susceptible cultivars (CM_Nil47, CM_Nil51, Remus) all show clear bleaching after DON infiltration.

Pictures of the six control lines with (CM_82036, CM_Nil38, CM_Nil43) or without (CM_Nil47, CM_Nil51, Remus) *Fhb1* after *Fusarium* treatment are shown in Figure 17.

Figure 17: Pictures of control lines after *Fusarium* inoculation. Control lines harboring the resistant *Fhb1* alleles (CM_82036, CM_Nil38, CM_Nil43), control lines with susceptible *Fhb1* alleles (CM_NIL47, CM_NIL51) and highly susceptible cultivar Remus are shown. In resistant cultivars there is an infection at inoculation site. The different spreading severities can be clearly seen.

All control lines that harbor *Fhb1* (CM_82036, CM_Nil38, CM_Nil43) do not show spreading of FHB. The control lines without *Fhb1* (CM_Nil47, CM_Nil51, Remus) show distinct different FHB spreading severities. The pictures also show that at the site of inoculation with *Fusarium* the spikelets are infected with the fungus, but no spreading can be observed.

8.1.2. Correlation coefficient of first and second rating

In Figure 16 the correlation of first and second rating for DON and *Fusarium* treatment is shown. The correlation coefficient between first and second rating for DON and *Fusarium* treated heads was calculated, to determine whether they differ in symptom severity.

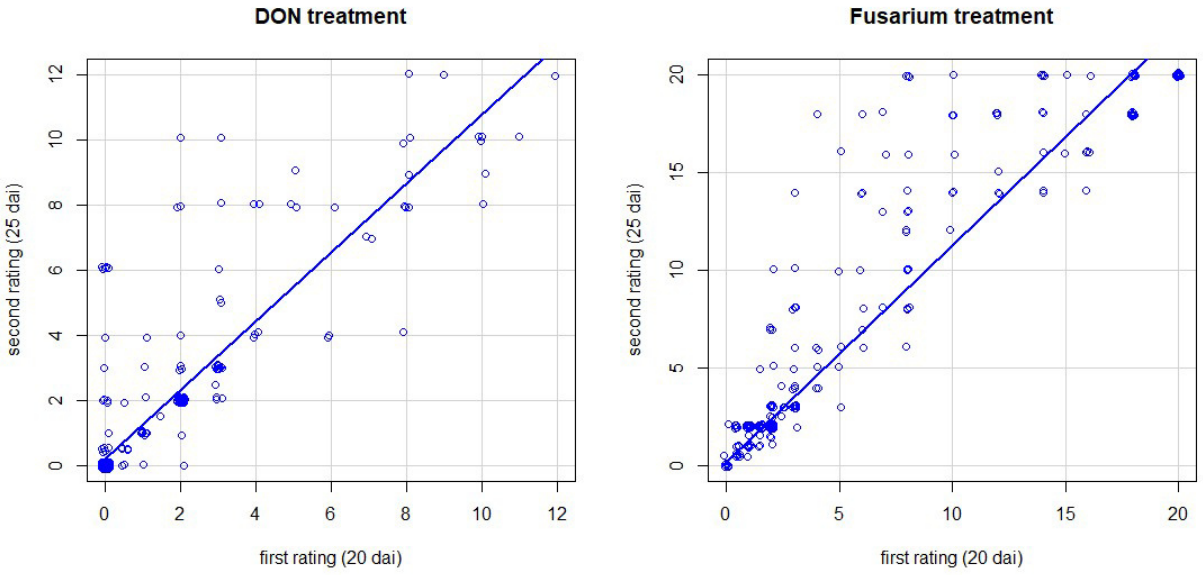

Figure 18: Scatterplot for the correlation of first rating (20 dai) and second rating (25 dai) for DON treatment (n = 305 heads) and *Fusarium* treatment (n = 497 heads). Calculation with data of single heads. r_{DON} = 0.88, r_{FUS} = 0.944.

The correlation between the first and second rating of DON treatment shows a correlation coefficient of r = 0.88 which is a strong positive and highly significant correlation.

Also, in the *Fusarium* treatment the correlation of first and second rating shows a correlation coefficient of r = 0.944 which is very strong positive correlation.

8.1.3. Repeatability of replication one and two

The one-way ANOVA for first and second rating showed significant differences in DON severity between genotypes. Data is shown in Table 30 and Table 31. The REP for first rating between the two replications amounted to 0.86. For second rating the REP between the two replications amounted to 0.90.

Table 30: One-way ANOVA for DON treatment, first rating (20 dai). Calculation with mean pot values.

	Df	Sum sq	Mean sq	F-value	*p*-value
Genotype	47	212.16	4.514	7.2	< 0.001***
Residuals	45	28.21	0.627		

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
Df = degrees of freedom; Sum sq = sum of squares; Mean sq = mean of squares

Table 31: One-way ANOVA for DON treatment, second rating (25 dai). Calculation with mean pot values.

	Df	Sum sq	Mean sq	F-value	*p*-value
Genotype	47	320.1	6.81	9.732	< 0.001***
Residuals	45	31.5	0.70		

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
Df = degrees of freedom; Sum sq = sum of squares; Mean sq = mean of squares

For FHB severity the one-way ANOVA showed significant differences between genotypes in both ratings (20 dai, 25 dai). Data can be seen in Table 32 and Table 33. The REP for first rating between the two replications amounted to 0.96. For second rating the REP between the two replications amounted to 0.95.

Table 32: One-way ANOVA for *Fusarium* treatment, first rating (20 dai). Calculation with mean pot values.

	Df	Sum sq	Mean sq	F-value	*p*-value
Genotype	44	1384.0	31.454	26.84	< 0.001***
Residuals	45	52.7	1.172		

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
Df = degrees of freedom; Sum sq = sum of squares; Mean sq = mean of squares

Table 33: One-way ANOVA for *Fusarium* treatment, second rating (25 dai). Calculation with mean pot values.

	Df	Sum sq	Mean sq	F-value	*p*-value
Genotype	44	1744.3	39.64	19.52	< 0.001***
Residuals	45	91.4	2.03		

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
Df = degrees of freedom; Sum sq = sum of squares; Mean sq = mean of squares

8.2. Evaluation of lines with homozygous mutations

8.2.1. Correlation coefficient for infiltration date and number of DON bleached spikelets per head

A scatterplot (Figure 17) shows the correlation for the trait combination infiltration date and number of DON bleached spikelets per individually treated head 20 dai.

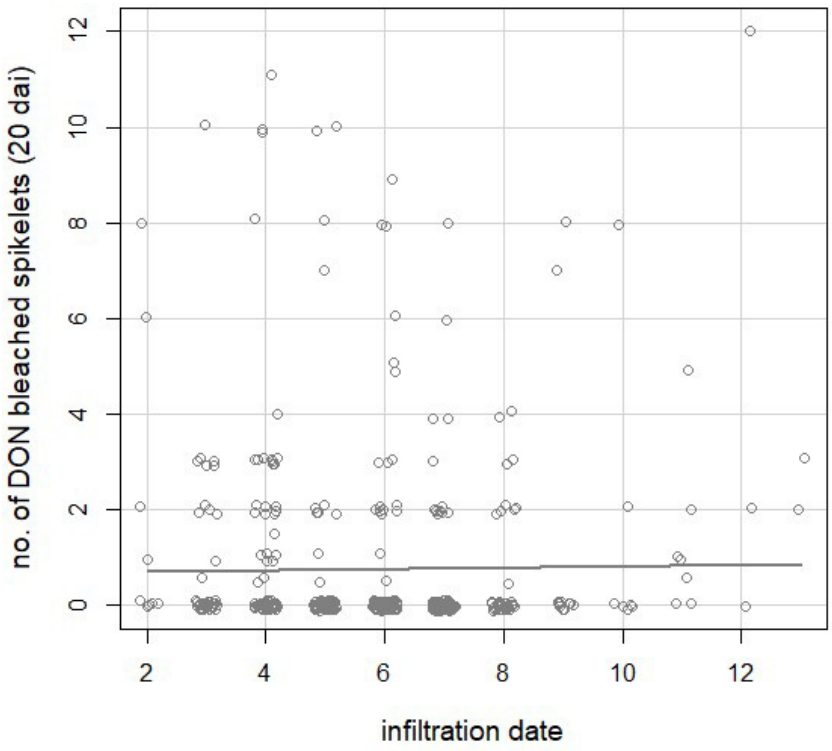

Figure 19: Scatterplot for the correlation of infiltration date and number of DON bleached spikelets per individually treated head 20 dai (n = 305 heads). Calculation with data of single heads. r = 0.015.

No linear correlation could be found (r = 0.015). Above all, low levels of DON bleaching could be found over the entire treatment period. Wheat heads with high levels of DON bleaching were less common but could also be found throughout the treatment period.

8.2.2. Symptom severity of DON treated control and homozygous mutant lines

The boxplots of all control lines treated with the mycotoxin DON are shown in Figure 18.

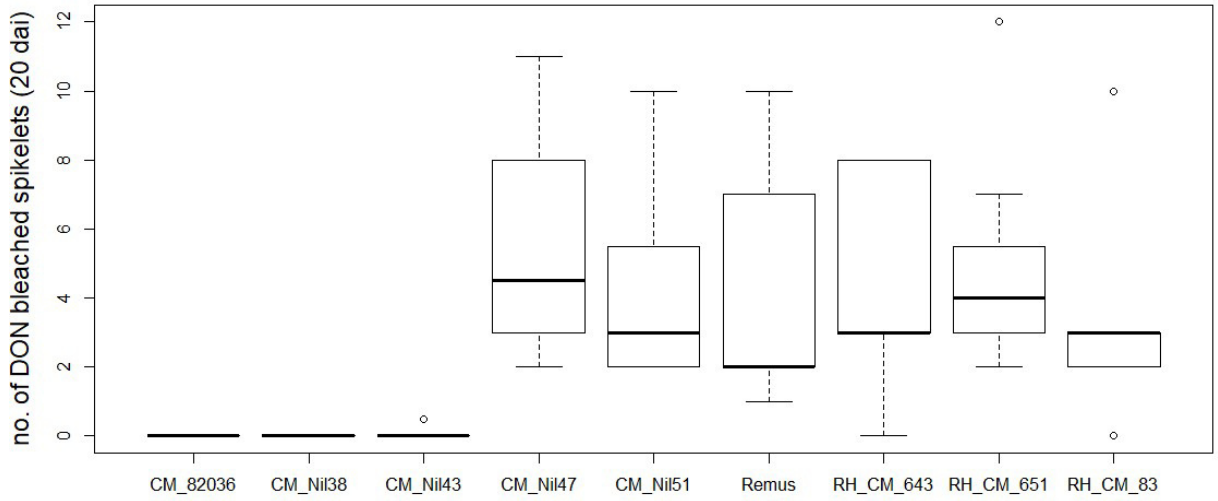

Figure 20: Boxplot for number of DON bleached spikelets (20 dai) of control lines. Control lines harboring the resistant *Fhb1* alleles (CM_82036, CM_Nil38, CM_Nil43), control lines with the susceptible *Fhb1* alleles (CM_NIL47, CM_NIL51), highly susceptible cultivar Remus, and control lines with deletions at Fbh1 (RH_CM_643, RH_CM_651, RH_CM_83) are shown. Calculation with data of single heads.

The resistant and susceptible phenotypes clearly separate in DON symptoms. Lines with resistant *Fhb1* alleles (CM_82036, CM_Nil38, CM_Nil43) show low or no DON symptoms. The control lines harboring susceptible *Fhb1* alleles (CM_Nil47, CM_Nil51) show on average 5.4 and 4.45 DON bleached spikelets per head. The highly susceptible cultivar Remus shows a DON severity of 4. The DON severity of the radiation hybrids (RH_CM_643, RH_CM_651, RH_CM_83) ranges from 3.6 to 4.73.

In Table 34 the general statistics (Minimum, Maximum, Mean, Standard Deviation) is shown. Also, DON severity for 20 dai was calculated and number of infiltrated heads is given. CM_82036 shows no symptoms in all treated heads and CM_Nil47 shows the highest DON severity of control lines.

Table 34: General statistics (Min, Max, Median, SD) for symptom severity of DON treated heads. Symptom severity is calculated with number of infected spikelets divided by number of inoculated spikelets and was assessed 20 dai.

Control line	Min	Max	Mean	SD	DON severity (20 dai)	Number of heads
CM_82036	0	0	0	0.00	0.00	12
CM_Nil38	0	0	0	0.00	0.00	11
CM_Nil43	0	1	0	0.14	0.04	12
CM_Nil47	2	11	5	3.04	5.40	10
CM_Nil51	2	10	4	2.90	4.45	11
Remus	1	10	4	3.14	4.00	18
RH_CM_643	0	8	4	3.14	4.40	5
RH_CM_651	2	12	5	2.73	4.73	11
RH_CM_83	0	10	4	3.38	3.60	5

SD … standard deviation, DON severity … mean number of symptomatic spikelets per head

In Table 35 the DON severity and the results of the two-sided t-test (α = 0.05) can be seen. The *p*-values have been calculated compared to CM_82036, which carries both resistance QTL *Fhb1* and *Qfhs.ifa-5A,* and compared to CM_Nil47, which only carries *Qfhs.ifa-5A.*

Table 35: DON severity (20 dai) and *p*-values compared to CM_82036 and CM_Nil47. DON severity is calculated with number of infected spikelets divided by number of inoculated spikelets and was assessed 20 days after inoculation. Lines with a statistical non-significant value compared to CM_Nil47 are highlighted in bold. '---' no calculation possible.

Line	DON severity (20 dai)	*p*-value (CM_82036)	*p*-value (CM_Nil47)
CM_82036	0.00	---	< 0.001
CM_Nil38	0.00	---	< 0.001
CM_Nil43	0.04	0.341	< 0.001
CM_Nil47	5.40	< 0.001	---
CM_Nil51	**4.45**	**< 0.001**	**0.645**
Remus	**4.00**	**< 0.0001**	**0.284**
RH_CM_643	**4.40**	**< 0.01**	**0.630**
RH_CM_651	**4.73**	**< 0.001**	**0.664**
RH_CM_83	**3.60**	**0.100**	**0.392**

Table continued on next page.

Table 35: Continued from previous page.

Line	DON severity (20 dai)	*p*-value (CM_82036)	*p*-value (CM_Nil47)
ND_2710	2.10	< 0.0001	< 0.05
Ning_7840	0.00	---	< 0.001
Nobeokabozu_U	0.60	0.089	< 0.001
Sumai3_MX	0.95	< 0.05	< 0.001
Sumai3_Ung	0.40	0.168	< 0.01
W14	0.60	0.051	< 0.01
Wangshuibai	0.15	0.193	< 0.001
723_Glyco	0.00	---	< 0.001
738_Glyco	0.05	0.343	< 0.001
940_Glyco	0.00	---	< 0.001
1111_Glyco	0.00	---	< 0.001
1148_Glyco	0.00	---	< 0.001
1345_Glyco	0.00	---	< 0.001
1369_Glyco	0.50	0.096	< 0.01
1616_Glyo	0.20	0.343	< 0.01
3093_Glyco	0.00	---	< 0.001
3632_Glyco	0.00	---	< 0.001
182_Ubi	0.00	---	< 0.001
342_Ubi	0.00	---	< 0.001
481_Ubi*	0.00	---	< 0.001
721_Ubi	0.00	---	< 0.001
869_Ubi	0.10	0.343	< 0.001
1047_Ubi	0.00	---	< 0.001
1254_Ubi	0.00	---	< 0.001
1325_Ubi	0.00	---	< 0.001
1632_Ubi	0.00	---	< 0.001
3045_Ubi	0.00	---	< 0.001
3746_Ubi	0.00	---	< 0.001
98_Ca	0.08	0.363	< 0.01
219_Ca	0.00	---	< 0.001
784_Ca	0.45	0.121	< 0.001
1914_Ca	0.17	0.343	< 0.001
6293_Ca	0.09	0.343	< 0.001
8406_Ca	0.00	---	< 0.001
8892_Ca	0.00	---	< 0.001
2203_Ca	0.00	---	< 0.001
3028_Ca	0.00	---	< 0.001

All susceptible control lines, except for RH_CM_83, showed a statistically significant difference in DON severity compared to the resistant control line CM_82036. The control lines carrying *Fhb1* showed a statistically significant difference compared to CM_Nil47.

Further, all tested mutant lines showed statistically significant differences in DON severity compared to CM_Nil47 and no mutant lines showed a statistically significant difference in DON severity compared to CM_82036.

The boxplots in Figure 19-21 show the comparison between gene #6, gene#17, gene #20 mutants and CM_82036, CM_Nil47. The gene #6 mutants show no DON bleaching, except for 1369_Glyco which shows a DON severity of on average 0.5 DON bleached spikelets. Further, none of the gene #17 mutants show symptoms. In gene #20 mutants only 784_Ca shows a DON severity of on average 0.45 DON bleached spikelets.

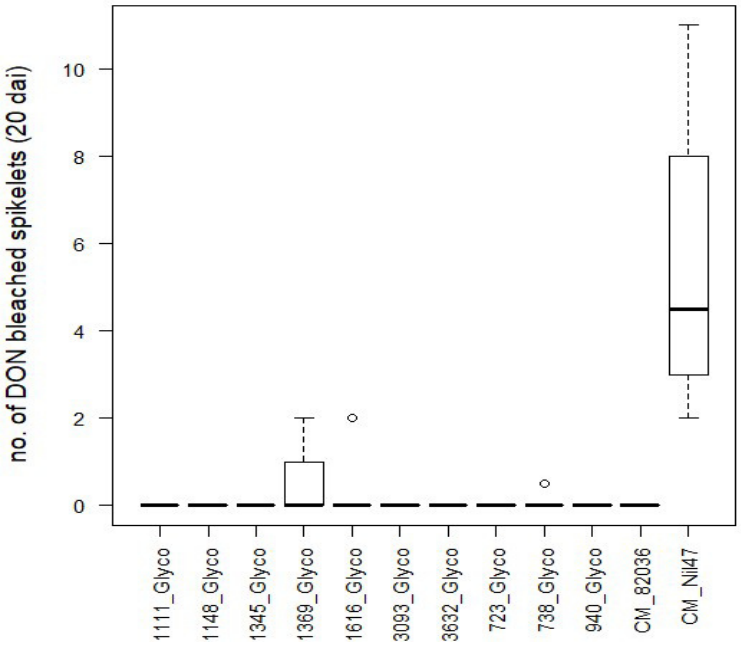

Figure 21: Boxplot for number of DON bleached spikelets (20 dai) of gene #6 (Glycosyltransferase hga7) mutants. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads.

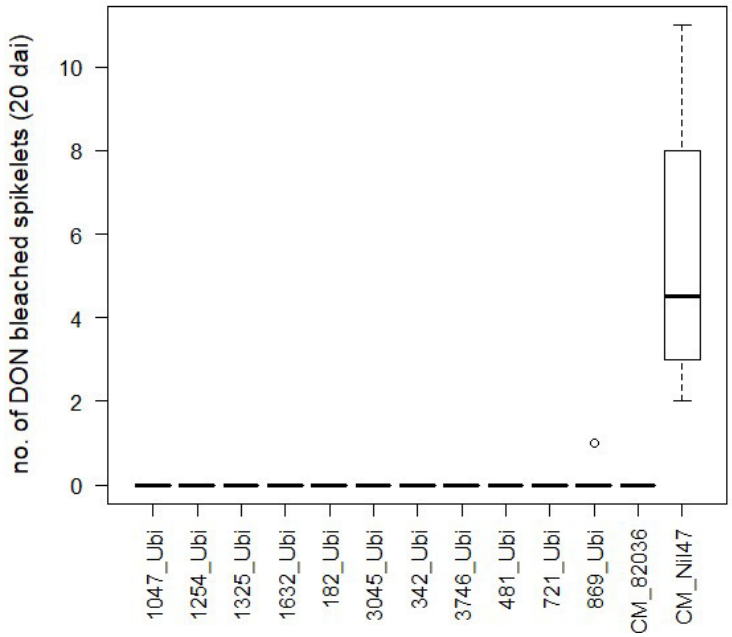

Figure 22: Boxplot for number of DON bleached spikelets (20 dai) of gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein) mutants. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads.

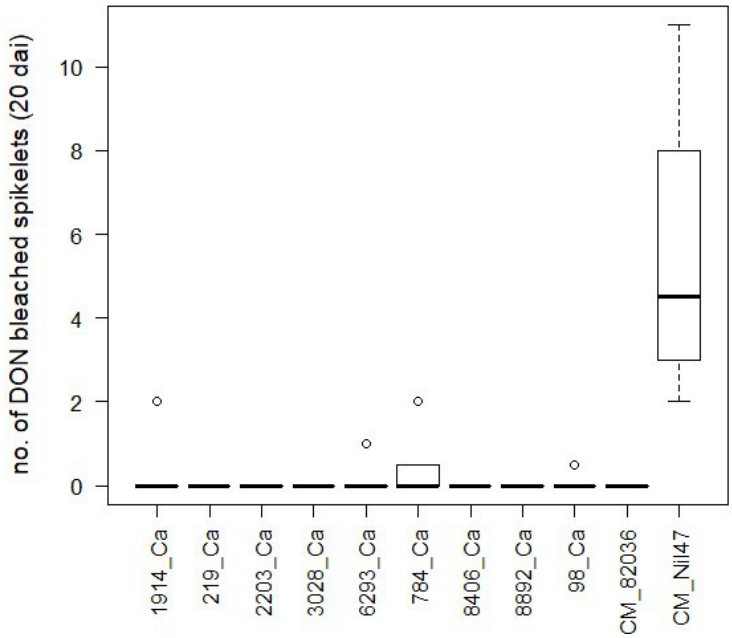

Figure 23: Boxplot for number of DON bleached spikelets (20 dai) of gene #20 (Hypothetical calcium binding protein) mutants. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads.

8.2.3. Correlation coefficient for inoculation date and number of FHB bleached spikelets per head

The scatterplot (Figure 22) shows the correlation for the trait combination inoculation date and number of FHB bleached spikelets per individually treated head 20 dai.

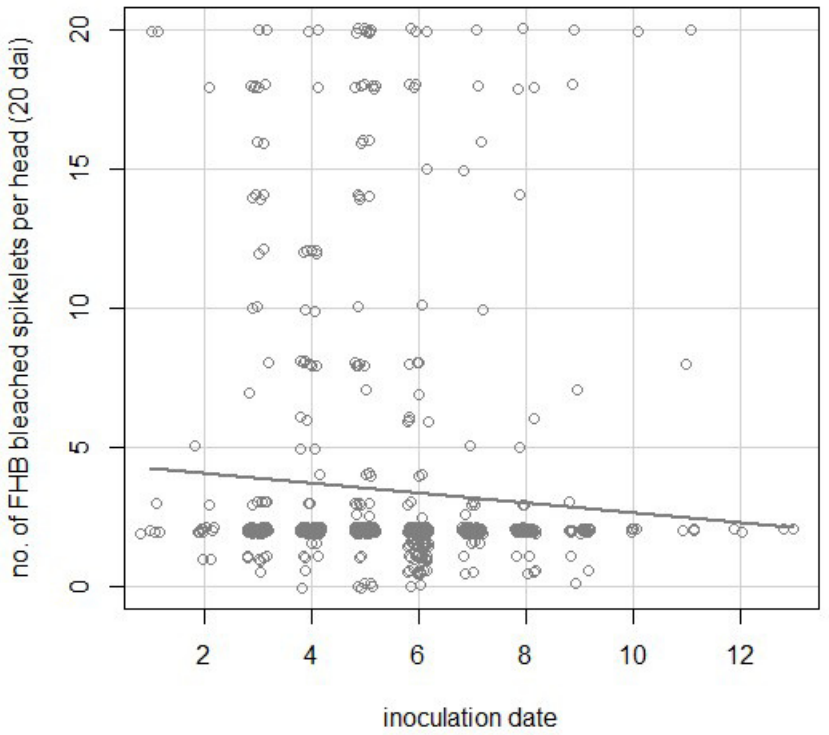

Figure 24: Scatterplot for the correlation of inoculation date and number of FHB bleached spikelets per individually treated head 20 dai (n = 497 heads). Calculation with data of single heads. r = -0.075.

A slight negative correlation could be found (r = -0.075). The majority of wheat heads shows low levels of FHB symptoms in intermediate treated heads. However, wheat heads with medium to high levels of FHB bleaching were less common and can also be found mostly in early and intermediate treated heads.

8.2.4. Disease severity of *Fusarium* treated control and homozygous mutant lines

In Figure 23 the boxplots of all control lines inoculated with a spore suspension of *F. graminearum* are shown.

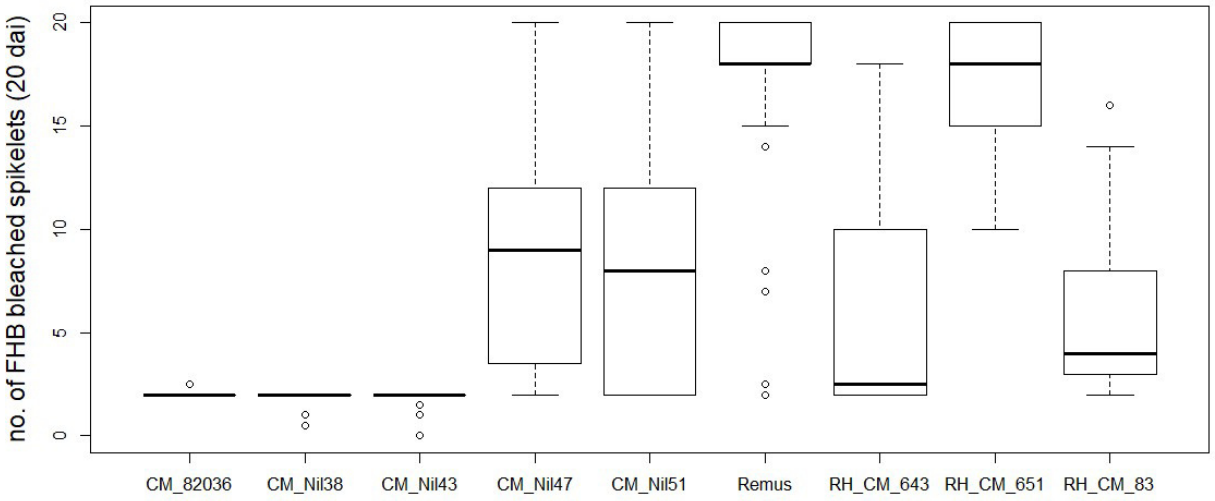

Figure 25: Boxplot for number of FHB bleached spikelets (20 dai) of control lines. Control lines harboring the resistant *Fhb1* alleles (CM_82036, CM_Nil38, CM_Nil43), control lines with the susceptible *Fhb1* alleles (CM_NIL47, CM_NIL51), highly susceptible cultivar Remus, and control lines with deletions at *Fbh1* (RH_CM_643, RH_CM_651, RH_CM_83) are shown. Calculation with data of single heads.

The difference between resistant and susceptible phenotypes can clearly be seen in *Fusarium* symptoms. Lines with resistant *Fhb1* alleles (CM_82036, CM_Nil38, CM_Nil43) show low symptoms, ranging from 1.76 to 2.03 bleached spikelets per head on average. The control lines harboring susceptible *Fhb1* alleles (CM_Nil47, CM_Nil51) show on average 9.1 and 8.19 *Fusarium* bleached spikelets per head. The highly susceptible cultivar Remus shows a very high symptom severity of 16.48 number of bleached spikelets. The radiation hybrids RH_CM_643 and RH_CM_83 show an average *Fusarium* severity of about 6 bleached spikelets per head, whereby RH_CM_651 shows an upwards outlier of 17.21.

The general statistics (Minimum, Maximum, Mean, Standard Deviation) of the *F. graminearum* inoculated control lines is shown in Table 36. Further, *Fusarium* severity for 20 dai was calculated and number of inoculated heads is also given. CM_Nil43 shows the lowest symptoms in all treated heads and RH_CM_651 shows the highest *Fusarium* severity of control lines.

Table 36: General statistics (Min, Max, Median, SD) for symptom severity of *Fusarium* treated heads. Symptom severity is calculated with number of infected spikelets divided by number of inoculated spikelets and was assessed 20 days after inoculation.

Control line	Min	Max	Mean	SD	*Fusarium* severity (20 dai)	Number of heads
CM_82036	2	3	2	0.11	2.03	20
CM_Nil38	1	2	2	0.41	1.84	22
CM_Nil43	0	2	2	0.52	1.76	19
CM_Nil47	2	20	9	5.67	9.10	20
CM_Nil51	2	20	8	5.59	8.19	21
Remus	2	20	16	5.27	16.48	26
RH_CM_643	2	18	6	5.73	6.44	18
RH_CM_651	10	20	17	3.03	17.21	14
RH_CM_83	2	16	6	4.43	6.21	14

A two-sided t-test (α = 0.05) was used to calculate the *p*-values compared to CM_82036, which carries both resistance QTL *Fhb1* and *Qfhs.ifa-5A,* and compared to CM_Nil47, which only carries *Qfhs.ifa-5A.* The *Fusarium* severity and *p*-values of all lines inoculated with *F. graminearum* are listed in Table 37.

Table 37: *Fusarium* severity (20 dai) and *p*-values compared to CM_82036 and CM_Nil47. *Fusarium* severity is calculated with number of infected spikelets divided by number of inoculated spikelets and was assessed 20 days after inoculation. Lines with a statistical non-significant value compared to CM_Nil47 are highlighted in bold. '---' no calculation possible.

Line	*Fusarium* severity (20 dai)	*p*-value (CM_82036)	*p*-values (CM_Nil47)
CM_82036	2.03	---	< 0.0001
CM_Nil38	1.84	0.057	< 0.0001
CM_Nil43	1.76	< 0.05	< 0.0001
CM_Nil47	9.10	< 0.0001	---
CM_Nil51	**8.19**	**< 0.0001**	**0.617**
Remus	16.48	< 0.0001	< 0.01
RH_CM_643	6.44	< 0.01	< 0.05
RH_CM_651	17.21	< 0.0001	< 0.01
RH_CM_83	6.21	< 0.01	< 0.05

Table continued on next page.

Table 37: Continued from previous page.

Line	*Fusarium* severity (20 dai)	*p*-value (CM_82036)	*p*-values (CM_Nil47)
ND_2710	2.86	0.157	< 0.001
Ning_7840	1.76	0.143	< 0.0001
Nobeokabozu_U	1.95	0.422	< 0.0001
Sumai3_MX	1.95	0.187	< 0.0001
Sumai3_Ung	2.44	0.287	< 0.0001
W14	2.23	0.387	< 0.001
Wangshuibai	1.90	0.235	< 0.0001
723_Glyco	2.28	0.440	< 0.0001
738_Glyco	1.95	0.087	< 0.0001
940_Glyco	2.00	0.330	< 0.0001
1111_Glyco	2.00	0.727	< 0.0001
1148_Glyco	2.07	0.903	< 0.0001
1345_Glyco	2.53	0.169	< 0.001
1369_Glyco	2.00	0.331	< 0.001
1616_Glyo	1.82	0.341	< 0.001
3093_Glyco	1.90	0.099	< 0.0001
3632_Glyco	1.43	0.174	< 0.0001
182_Ubi	1.85	0.104	< 0.001
342_Ubi	18.29	< 0.0001	< 0.0001
481_Ubi*	1.97	0.792	< 0.0001
721_Ubi	1.85	0.104	< 0.001
869_Ubi	1.94	0.188	< 0.0001
1047_Ubi	1.90	0.217	< 0.001
1254_Ubi	1.71	< 0.05	< 0.0001
1325_Ubi	2.00	0.330	< 0.0001
1632_Ubi	2.47	0.251	< 0.0001
3045_Ubi	1.76	< 0.05	< 0.0001
3746_Ubi	2.22	0.515	< 0.0001
98_Ca	---	---	---
219_Ca	1.97	0.333	< 0.0001
784_Ca	4.36	0.067	< 0.01
1914_Ca	1.90	0.099	< 0.0001
6293_Ca	2.00	0.331	< 0.0001
8406_Ca	1.95	0.187	< 0.0001
8892_Ca	2.00	0.331	< 0.0001
2203_Ca	2.00	0.330	< 0.0001
3028_Ca	1.73	0.192	< 0.001

All control lines, except for CM_Nil38, showed a statistically significant difference in DON severity compared to the resistant control line CM_82036. Further, only CM_Nil51 showed a statistically significant difference compared to CM_Nil47.

All tested mutant lines showed statistically significant differences in DON severity compared to CM_Nil47 and except for 342_Ubi, 1254_Ubi and 3045_Ubi no other mutant line showed statistically significant differences compared to CM_82036.

The boxplots in Figure 24-26 show the comparison between gene #6, gene#17, gene #20 mutants and CM_82036, CM_Nil47. In general, the gene #6 mutants show no *Fusarium* bleached spikelets, except for the two inoculated spikelets that should always show symptoms. None of the gene #17 mutants show symptoms beyond the two inoculated spikelets, except for 342_Ubi which shows on average 18.29 *Fusarium* bleached spikelets. In the gene #20 mutants only 784_Ca shows a *Fusarium* severity of on average 4.36 *Fusarium* bleached spikelets.

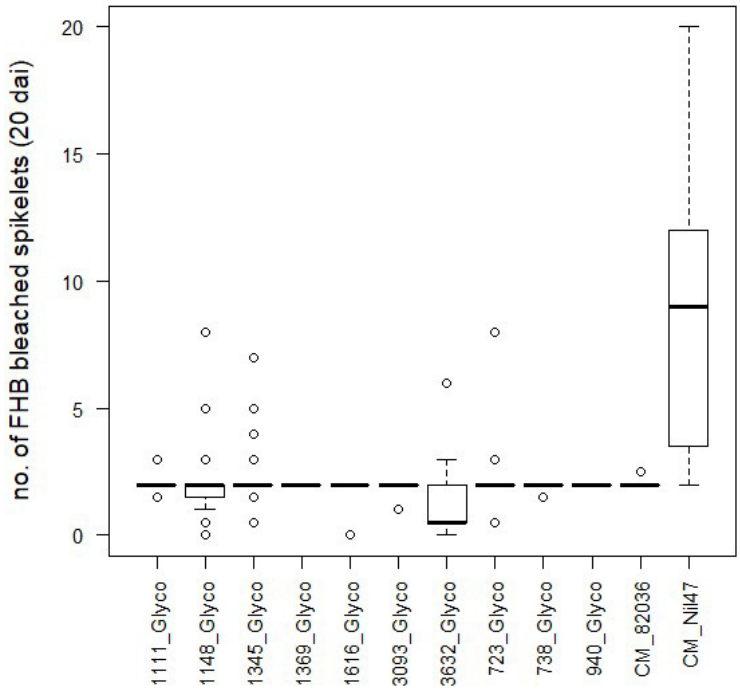

Figure 26: Boxplot for number of FHB bleached spikelets (20 dai) of gene #6 (Glycosyltransferase hga7) mutants. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads.

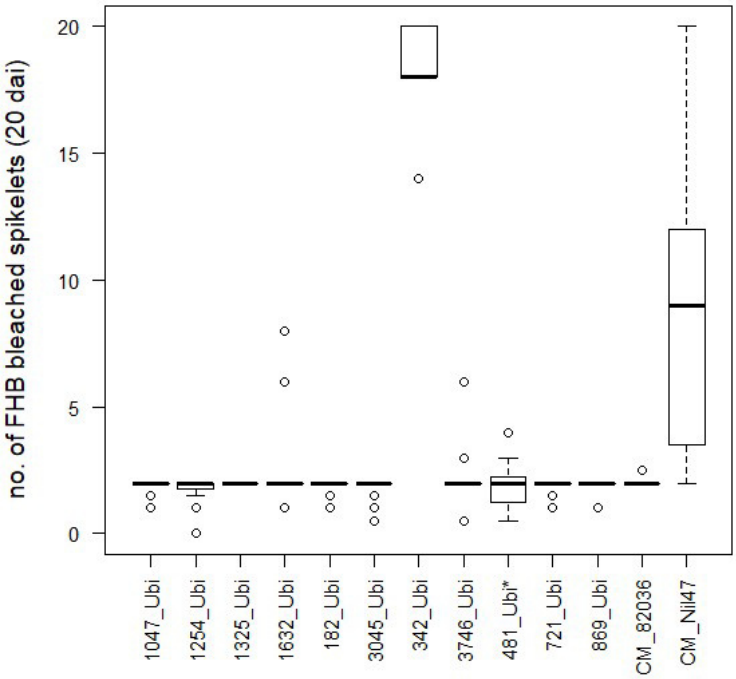

Figure 27: Boxplot for number of FHB bleached spikelets (20 dai) of gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein) mutants. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads.

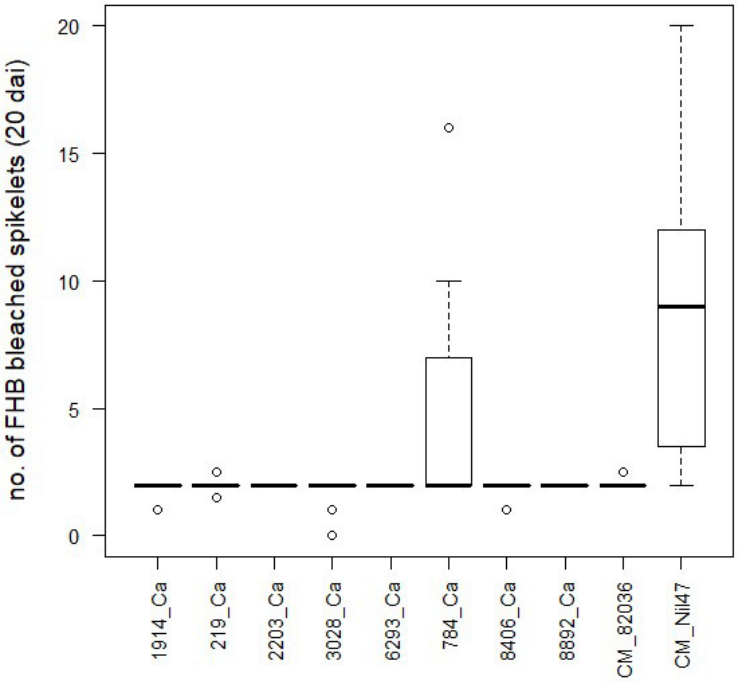

Figure 28: Boxplot for number of FHB bleached spikelets (20 dai) of gene #20 (Hypothetical calcium binding protein) mutants. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads.

8.3. Evaluation of lines with heterozygous mutations

8.3.1. Correlation coefficient for inoculation date and number of DON bleached spikelets per head

In Figure 27 the correlation for infiltration date and number of DON bleached spikelets per individually treated head 20 dai is shown.

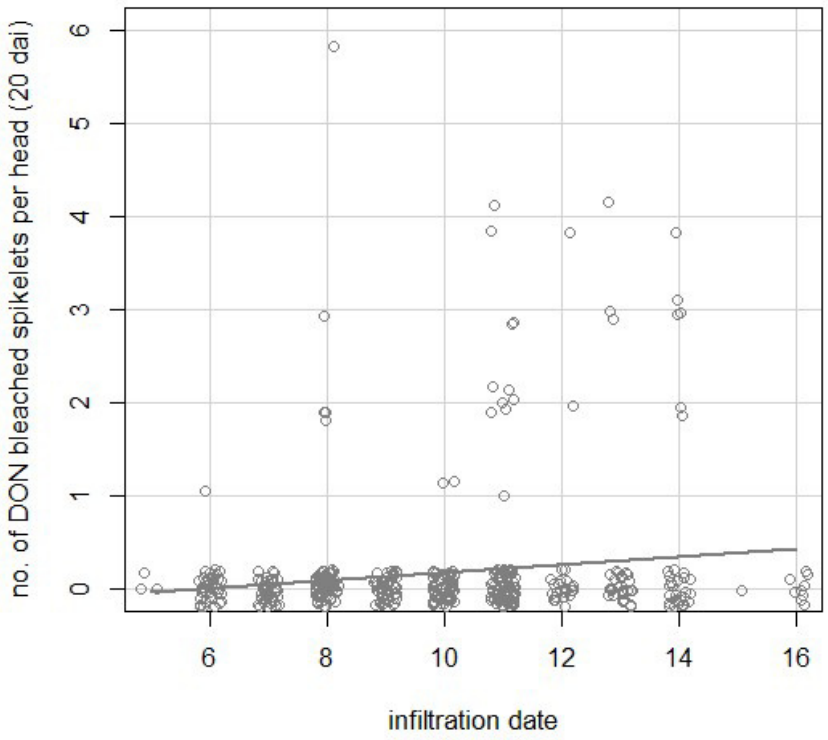

Figure 29: Scatterplot for the correlation of inoculation date and number of DON bleached spikelets per individually treated head 20 dai (n = 467 heads). Calculation with data of single heads. r = 0.148.

A slight positive linear correlation (r = 0.148) between number of DON beached spikelets and infiltration date could be found. Most of the heterozygous mutant lines treated with DON show no symptoms of DON bleaching throughout the entire treatment period. However, several wheat heads with medium to high levels of DON induced bleaching could be found.

8.3.2. Disease severity of DON treated heterozygous mutant lines

The mutant lines carrying the heterozygous mutations have been analyzed by a two-sided t-test (α = 0.05), compared to their respective wild-type and in Table 38 the results are listed.

Table 38: DON severity (20 dai) and *p*-value compared to wild-type of each line. DON severity is calculated with number of infected spikelets divided by number of infiltrated spikelets and was assessed 20 days after infiltration. Lines with a statistically significant value compared to their wild-type are highlighted in bold. '---' no calculation possible.

Line	DON severity (20 dai) (mutant-type)	DON severity (20 dai) (wild-type)	*p*-value (compared to wild-type)
88_Glyco	0.00	0.00	0
1500_Glyco	0.00	0.00	0
3094_Glyco	0.06	0.17	0.30
48_Ubi	0.00	---	---
256_Ubi	**10.00**	**0.00**	**< 0.0001**
448_Ubi	0.00	0.00	0
641_Ubi	0.00	0.00	0
2187_Ubi	0.00	0.00	0
1722_Ca	0.00	0.00	0
6046_Ca	0.00	0.00	0
6504_Ca	0.00	0.00	0
6551_Ca	0.00	0.00	0
6688_Ca	0.00	0.00	0
8023_Ca	0.00	---	---
8032_Ca	0.00	0.00	0

Most heterozygous mutant lines show no DON bleaching, no matter if they have the mutant or wild-type alleles, and therefore no DON severity could be assessed. The exception is line 3094_Glyco, where mutant and wild-type show DON bleaching, and a *p*-value of 0.30, which means there is no significant difference between mutant wild-type alleles. Also, line 256_Ubi shows clear DON bleaching and a significant difference to its wild-type.

Figure 28-30 illustrate the number of DON bleached spikelets (20 dai) of lines carrying the mutations in heterozygous form, comparing mutant and wild-type, as well as compared to CM_82036 and CM_Nil47. For gene #6 and gene #20 no line shows any DON bleached spikelets. For gene #17 only the mutant-type of 256_Ubi shows DON bleaching, on average 10 DON bleached spikelets per head.

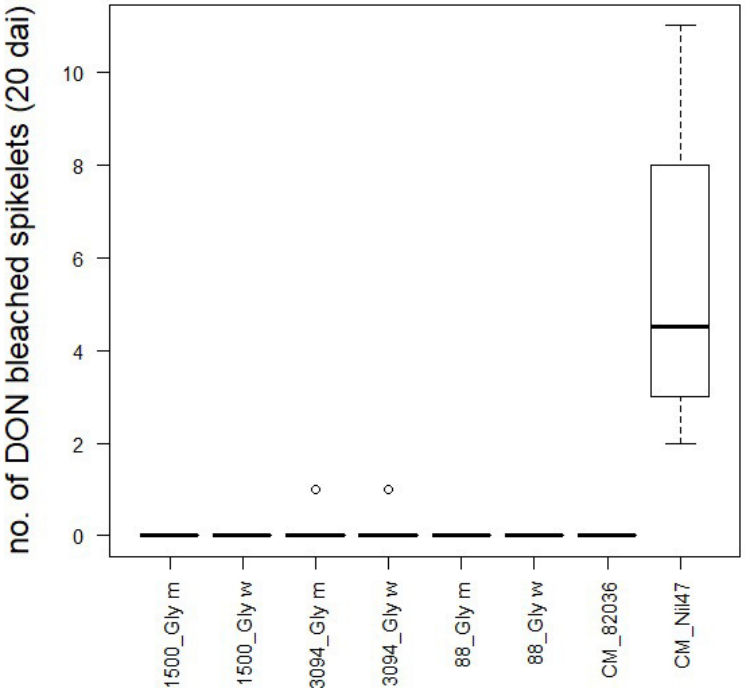

Figure 30: Boxplot for number of DON bleached spikelets (20 dai) of lines carrying gene #6 (Glycosyltransferase hga7) in heterozygous condition. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads. m … mutant-type, w … wild-type.

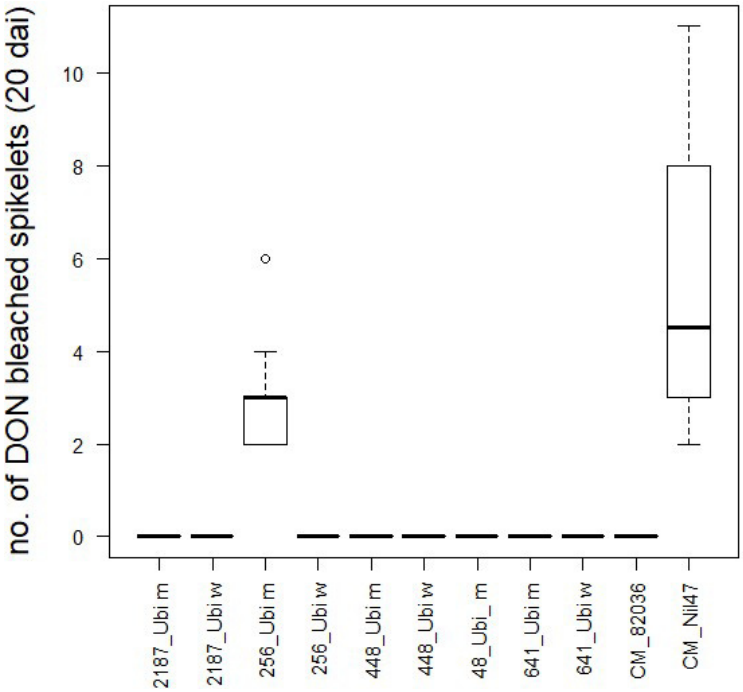

Figure 31: Boxplot for number of DON bleached spikelets (20 dai) of lines carrying gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein) in heterozygous condition. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads. m … mutant-type, w … wild-type.

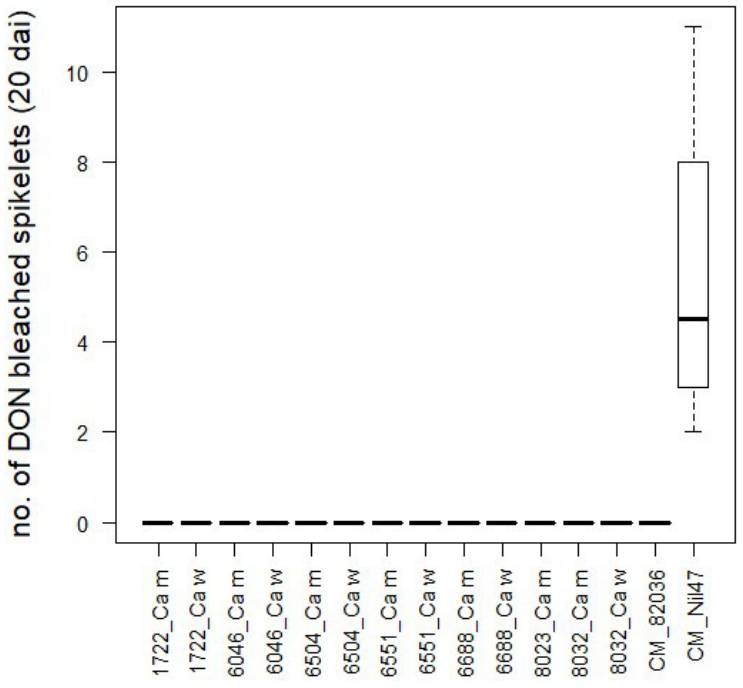

Figure 32: Boxplot for number of DON spikelets (20 dai) of lines carrying gene #20 (Hypothetical calcium binding protein) in heterozygous condition. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads. m … mutant-type, w … wild-type.

8.3.3. Correlation coefficient for inoculation date and number of FHB bleached spikelets

Figure 31 shows the correlation for inoculation date and number of FHB bleached spikelets per individually treated head 20 dai.

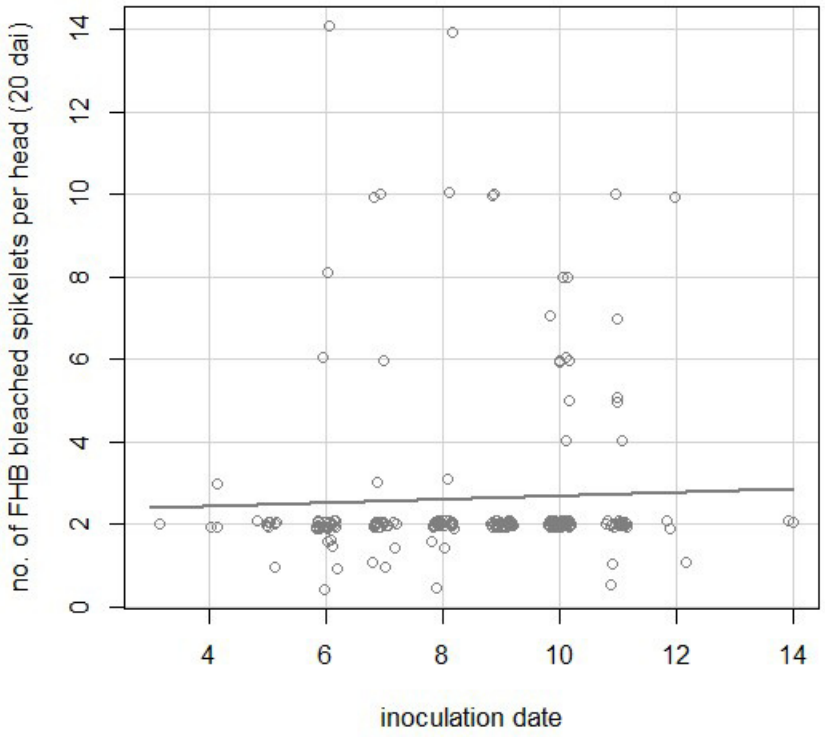

Figure 33: Scatterplot for the correlation of inoculation date and number of FHB bleached spikelets per individually treated head 20 dai (n = 310 heads). Calculation with data of single heads. r = 0.042.

A very slight positive correlation could be found (r = 0.042). The majority of the heterozygous mutant lines treated with *Fusarium* inoculum only show symptoms on the two inoculated spikelets. However, some wheat heads show medium to high levels of FHB bleaching.

8.3.4. Disease severity of *Fusarium* treated control and heterozygous mutant lines

In Table 39 the results of the two-sided t-test (α = 0.05) for the comparison of the mutant lines carrying the heterozygous mutations and their respective wild-type are listed. Only one line showed a statistically significant difference to its respective wild-type, namely mutant line 256_Ubi.

The pots with the wild-type for lines 3094_Glyco, 6046_Ca, 6688_Ca and the pot with the mutant-type for 88_Glyco were not evaluable, therefore no *p*-value could be calculated.

Table 39: *Fusarium* severity (20 dai) and *p*-value compared to wild-type of each line. *Fusarium* severity is calculated with number of infected spikelets divided by number of inoculated spikelets and was assessed 20 days after inoculation. Lines with a statistically significant value compared to their wild-type are highlighted in bold. '---' no calculation possible.

Line	*Fusarium* severity (20 dai) (mutant-type)	*Fusarium* severity (20 dai) (wild-type)	*p*-value (compared to wild-type)
88_Glyco	---	2.36	---
1500_Glyco	2.00	2.66	0.23
3094_Glyco	3.41	---	---
256_Ubi	**5.90**	**2.00**	**< 0.0001**
448_Ubi	2.50	1.64	0.31
641_Ubi	3.55	2.00	0.20
2187_Ubi	2.00	2.00	0
1722_Ca	1.80	2.00	0.37
6046_Ca	1.93	---	---
6551_Ca	2.10	2.00	0.34
6688_Ca	1.89	---	---
8023_Ca	1.33	1.83	0.36
8032_Ca	2.00	2.00	0

Only mutant line 256_Ubi shows clear FHB bleaching, resulting in an increased *Fusarium* severity in the mutant-type, but not in the wild-type.

In Figure 32-34 the number of FHB bleached spikelets (20 dai) of lines carrying the mutations in heterozygous form are illustrated, comparing their mutant and wild-type, as well as compared to CM_82036 and CM_Nil47. The two inoculated spikelets showing FHB bleaching can be clearly seen in each line. For gene #6 only the mutant-type of line 3094_Gly shows increased FHB bleaching, on average 3.41 FHB bleached spikelets per head. For gene #17 only the mutant-type of 256_Ubi shows FHB bleaching, on average 5.90 FHB bleached spikelets per head. And for gene #20 no line showed increased FHB bleaching after *Fusarium* inoculation.

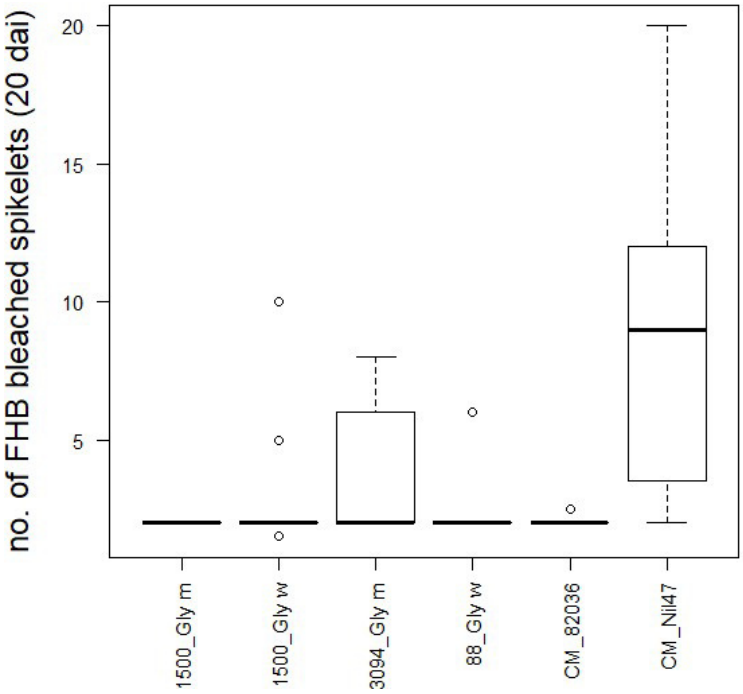

Figure 34: Boxplot for number of FHB bleached spikelets (20 dai) of lines carrying gene #6 (Glycosyltransferase hga7) in heterozygous condition. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads. m … mutant-type, w … wild-type.

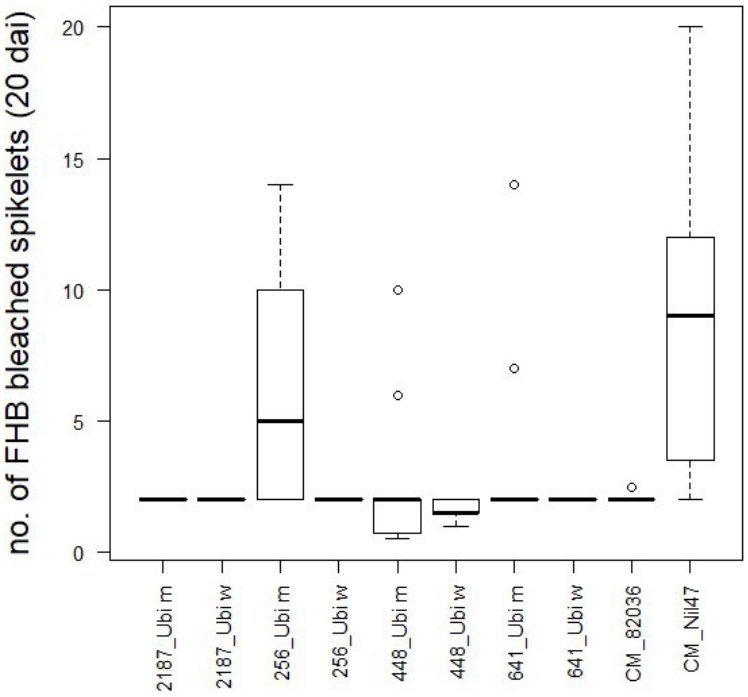

Figure 35: Boxplot for number of FHB bleached spikelets (20 dai) of lines carrying gene #17 (Ubiquitin-2 like Rad60 SUMO-like protein) in heterozygous condition. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads. m … mutant-type, w … wild-type.

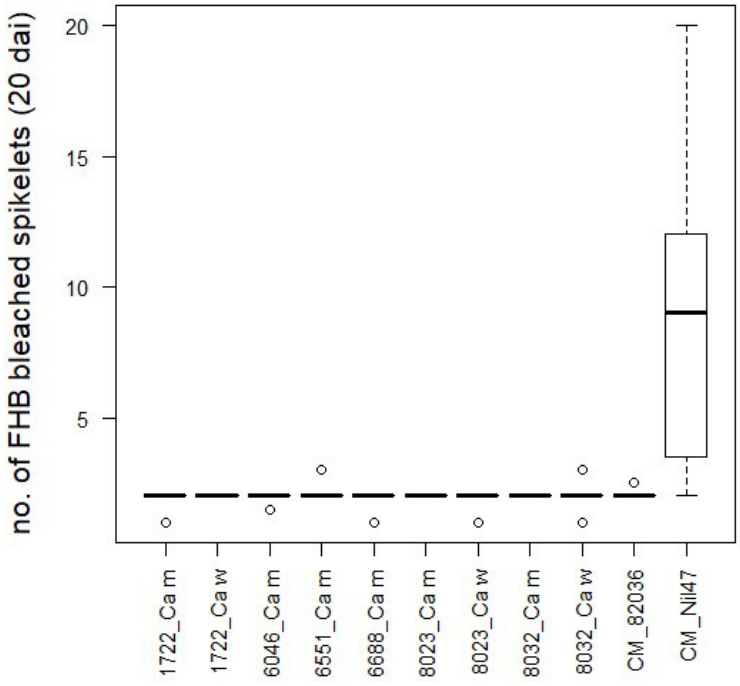

Figure 36: Boxplot for number of FHB bleached spikelets (20 dai) of lines carrying gene #20 (Hypothetical calcium binding protein) in heterozygous condition. Compared to CM_82036 and CM_Nil47. Calculation with data of single heads. m … mutant-type, w … wild-type.

8.4. Connection between DON severity and FHB severity

8.4.1. Mutant lines with homozygous mutations

Table 40 summarizes DON and FHB severity of control and mutant lines with homozygous mutations of the greenhouse trial. Also, a scatterplot for the correlation of DON and FHB severity of these lines is shown in Figure 35.

Table 40: Comparison of DON and *Fusarium* severity (20 dai) of control and mutant lines with homozygous mutations of the greenhouse trial. Symptom severity is calculated with number of infected spikelets divided by number of inoculated spikelets and was assessed 20 days after inoculation. '---' no calculation possible.

Line	DON severity (20 dai)	*Fusarium* severity (20 dai)
CM_82036	0.00	2.03
CM_Nil38	0.00	1.84
CM_Nil43	0.04	1.76
CM_Nil47	5.40	9.10
CM_Nil51	4.45	8.19
Remus	4.00	16.48
RH_CM_643	4.40	6.44
RH_CM_651	4.73	17.21
RH_CM_83	3.60	6.21
ND_2710	2.10	2.86
Ning_7840	0.00	1.76
Nobeokabozu_U	0.60	1.95
Sumai3_MX	0.95	1.95
Sumai3_Ung	0.40	2.44
W14	0.60	2.23
Wangshuibai	0.15	1.90
723_Glyco	0.00	2.28
738_Glyco	0.05	1.95
940_Glyco	0.00	2.00
1111_Glyco	0.00	2.00
1148_Glyco	0.00	2.07
1345_Glyco	0.00	2.53
1369_Glyco	0.50	2.00
1616_Glyo	0.20	1.82
3093_Glyco	0.00	1.90
3632_Glyco	0.00	1.43

Table continued on next page.

Table 40: Continued from previous page.

Line	DON severity (20 dai)	*Fusarium* severity (20 dai)
182_Ubi	0.00	1.85
342_Ubi	0.00	**18.29**
481_Ubi*	0.00	1.97
721_Ubi	0.00	1.85
869_Ubi	0.10	1.94
1047_Ubi	0.00	1.90
1254_Ubi	0.00	1.71
1325_Ubi	0.00	2.00
1632_Ubi	0.00	2.47
3045_Ubi	0.00	1.76
3746_Ubi	0.00	2.22
98_Ca	0.08	---
219_Ca	0.00	1.97
784_Ca	0.45	4.36
1914_Ca	0.17	1.90
6293_Ca	0.09	2.00
8406_Ca	0.00	1.95
8892_Ca	0.00	2.00
2203_Ca	0.00	2.00
3028_Ca	0.00	1.73

Control lines responded to the treatment with DON solution or spore suspension of *F. graminearum* according to their genetic background. The resistant control lines (CM_82036, CM_Nil38, CM_Nil43) show no symptoms, except on the two spikelets inoculated with the spore suspension of *F. graminearum*. The susceptible control lines show clear symptoms of both FHB and DON bleaching.

For the mutant lines with homozygous mutations, only low DON severity could be found, whereby 1369_Glyco shows the highest mean of DON bleached spikelets per head (0.50). Also, mostly low *Fusarium* severity could be recorded. The mutant lines 342_Ubi shows highest mean of FHB bleached spikelets per head (18.29) and 784_Ca shows the second highest mean (4.36).

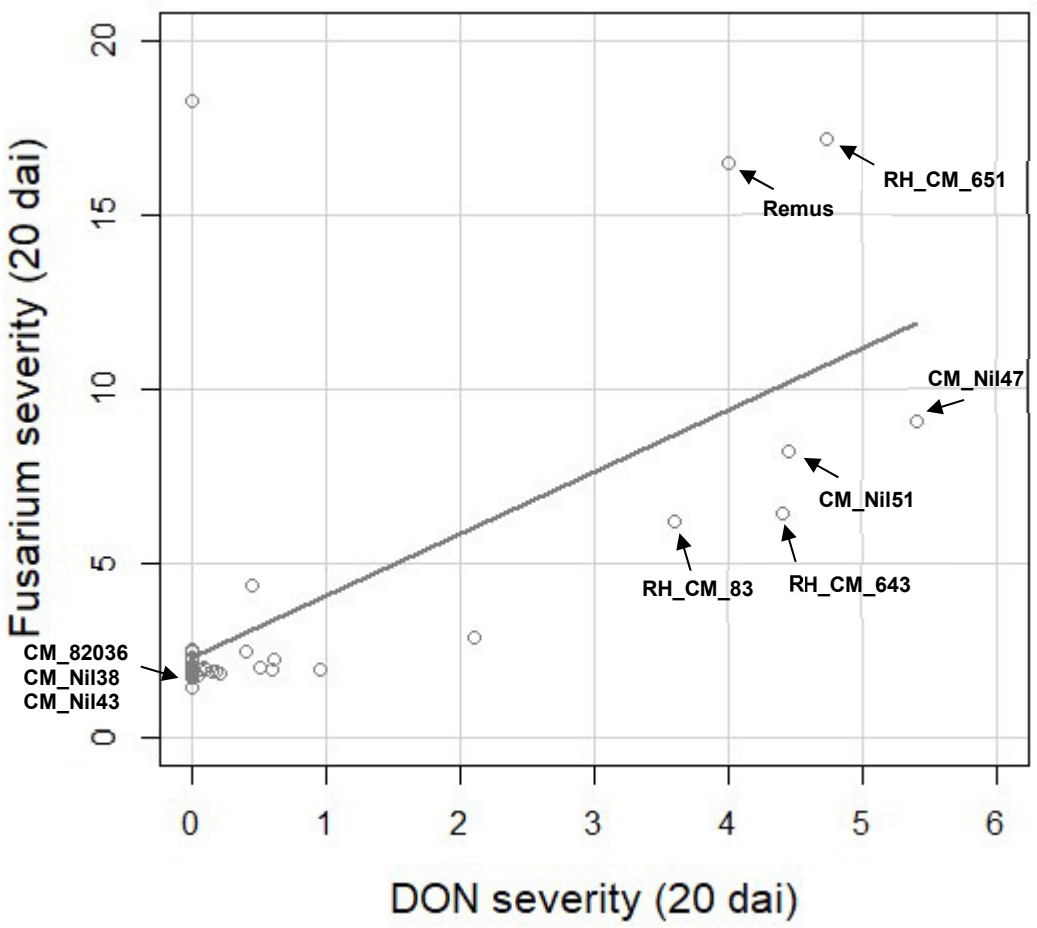

Figure 37: Scatterplot for the comparison of *Fusarium* and DON severity (20 dai) of lines carrying homozygous mutations (n = 46). Symptom severity is calculated with number of infected spikelets divided by number of inoculated spikes. Control lines are identified in the scatterplot. r = 0.666

The traits *Fusarium* severity and DON severity show a strong positive linear correlation (r = 0.666). Resistant control lines (CM_82036, CM_Nil38, CM_Nil43) show no symptoms, except for the two inoculated and FHB bleached spikelets. The highly susceptible control lines Remus shows high *Fusarium* and DON severity. Radiation hybrid RH_CM_651 shows even higher symptom severity than Remus. Also, CM_Nil51 and RH_CM_83, RH_CM_643 show symptoms of FHB and DON bleaching. However, no mutant line carrying a homozygous mutation showed a strong positive correlation between *Fusarium* and DON severity.

8.4.2. Mutant lines with heterozygous mutations

In Table 41 the comparison of DON and FHB severity of mutant lines with heterozygous mutations, each for mutant- and wild-type, are shown. In the scatterplot of Figure 36 the correlation between DON and FHB severity of the lines with wild-type and in Figure 37 the mutant-type is shown.

Table 41: Comparison of DON and *Fusarium* severity (20 dai) of mutant lines with heterozygous mutations of the greenhouse trial, each for mutant and wild-type. Symptom severity is calculated with number of infected spikelets divided by number of inoculated spikelets and was assessed 20 days after inoculation. Mutant lines with a statistically significant value or higher symptom severity compared to their wild-type are highlighted in bold. '---' no calculation possible.

Line	DON severity (mutant-type)	DON severity (wild-type)	*Fusarium* severity (mutant-type)	*Fusarium* severity (wild-type)
88_Glyco	0.00	0.00	---	2.36
1500_Glyco	0.00	0.00	2.00	2.66
3094_Glyco	0.06	0.17	3.41	---
48_Ubi	0.00	---	---	---
256_Ubi	**10.00**	**0.00**	**5.90**	**2.00**
448_Ubi	0.00	0.00	2.50	1.64
641_Ubi	0.00	0.00	3.55	2.00
2187_Ubi	0.00	0.00	2.00	2.00
1722_Ca	0.00	0.00	1.80	2.00
6046_Ca	0.00	0.00	1.93	---
6504_Ca	0.00	0.00	---	---
6551_Ca	0.00	0.00	2.10	2.00
6688_Ca	0.00	0.00	1.89	---
8023_Ca	0.00	---	1.33	1.83
8032_Ca	0.00	0.00	2.00	2.00

The lines carrying heterozygous mutations show mostly low DON severity in both mutant- and wild-type, except for the mutant-type of 256_Ubi, which shows also a high *Fusarium* severity. All other lines, mutant- and wild-type, show a *Fusarium* severity of about 2.00 which is the intended result. The mutant-type of 3094_Glyco and 641_Ubi show only slightly higher *Fusarium* severity than the two inoculated and bleached spikelets per head.

Figure 36 and Figure 37 illustrate the Table shown above. Again, no wild-type of lines with a heterozygous mutation shows notably high DON severity or a *Fusarium* severity of beyond 2 bleached kernels per head. However, the mutant-type of line 256_Ubi shows high DON severity, namely on average 10 DON bleached spikelets per head.

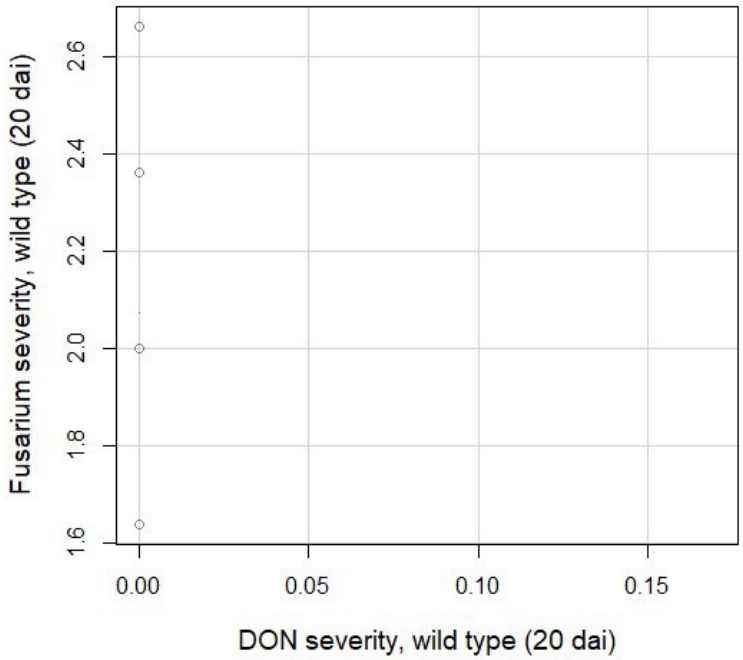

Figure 38: Scatterplot for the comparison of *Fusarium* and DON severity (20 dai) of the wild-types of lines with heterozygous mutations (n = 15). Symptom severity is calculated with number of infected spikelets divided by number of inoculated spikes. r = 1

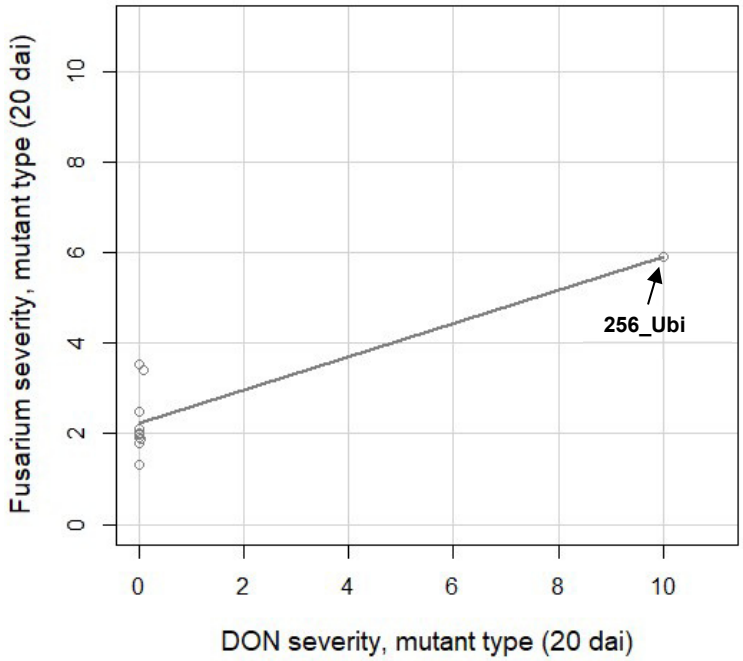

Figure 39: Scatterplot for the comparison of *Fusarium* and DON severity (20 dai) of lines with homozygous mutations (n = 15). Symptom severity is calculated with number of infected spikelets divided by number of inoculated spikes. Outliers are identified in the scatterplot. r = 0.856

9. Discussion

This part of the master thesis is focused on testing three of in total 28 candidate genes located in *Fhb1* region, namely gene #6, encoding for Glycosyltransferase hga7, gene #17, encoding for Ubiquitin-2 like Rad60 SUMO-like protein, and gene #20, encoding for Hypothetical calcium binding protein. The *Fhb1* region leads to an increase in overall resistance to *F. graminearum* and the mycotoxin DON (Schweiger et al., 2016).

It is still unclear, which gene(s) contribute to DON resistance. Further, it is hypothesized that DON resistance plays an important role in FHB resistance, as resistant lines are able to convert DON into less toxic D3G. This detoxification process plays an important role in disease resistance (Lemmens et al., 2005).

Seidl (2017) screened a mutant population by using the reverse genetics approach TILLING to find lines with deleterious mutations in the candidate genes. Finally, in this trial the screened, evaluated and selected mutant lines were planted in the greenhouse and tested for their phenotype. The greenhouse trial consisted of two individual experiments, with two replications and the same plant material for both experiments. One experiment was the inoculation with a spore suspension of *F. graminearum* to evaluate type II resistance, which is resistance to pathogen spreading in infected tissue. The other experiment was to assess DON bleaching by infiltration of flowering wheat heads with a DON solution. If a mutant line shows typical symptoms of FHB spreading or DON induced bleaching, it can be assumed that one of these genes is involved in the resistance mechanism of *Fhb1*. However, it remains unclear if one gene is responsible for both FHB and DON resistance. It could also be that two genes closely located together are also inherited together, therefore co-segregation of the traits took place – one gene for DON resistance and one for FHB resistance (Schweiger et al., 2016).

9.1. General evaluation of the mutant lines for FHB and DON resistance

Inoculations with *F. graminearum* and infiltration with DON were done quickly and efficient. About 99% (Table 45) of the *Fusarium* inoculated heads of mutant lines developed disease symptoms, which is a prerequisite to measure resistance to fungal spread. Of the 99% about 7% showed spreading of the fungus. As heads inoculated with *F. graminearum* do not always show a uniform phenotype, several heads of each line were inoculated to obtain a meaningful result, on average 17 heads per line were inoculated. This could also explain the several outliers in the results. These outliers are single heads of several different lines. Treatment was done about every two days, because time of flowering is not uniform among the tested lines. About 95% (Table 46) of DON treated heads of mutant lines showed no DON induced bleaching.

At the first rating the susceptible control lines showed distinct symptoms of DON bleaching upwards the infiltration site and spreading of the disease after inoculation with *F. graminearum*. The resistant controls showed no DON bleaching, or only FHB bleaching of the two inoculated spikelets, but no spreading (type II resistance). The results also show that the experimental setup in the greenhouse worked well, because all control lines showed spreading symptoms according to their genotype.

Each experiment consisted of two replications and was evaluated twice (20 and 25 dai). Therefore, first and second rating were compared to assess which rating shows the most significant differences between genotypes. REP was calculated to see if both replications within an experiment show the same results. However, it must be considered that at the second rating natural ripening of the heads was already progressed so far that it was difficult to distinguish natural yellowing and DON/FHB induced bleaching. Therefore, the first rating was considered in the calculations, because the difference between healthy and bleached spikelets was clear to see. Also, there was high REP, which means that both replications lead to similar results.

9.2. Identification of susceptible lines with homozygous mutations

The correlation between infiltration/inoculation date and number of bleached spikelets per head was tested. The idea behind the greenhouse trial is not only to speed up the breeding progress, but also to exclude environmental conditions with significant genotype-by-environment interactions (Miedaner et al., 2001). Overall, no or very low correlation between infiltration/inoculation date and number of bleached spikelets per head could be found, which shows that the greenhouse setting worked well.

For the DON treated control lines the results show significant differences in DON bleaching between lines carrying *Fhb1* (CM_82036, CM_Nil38, CM_Nil43) and without *Fhb1* (CM_Nil47, CM_Nil51, Remus, RH_CM_643, RH_CM_651, RH_CM_83). Also, for the *Fusarium* treated control lines the results show clear FHB bleaching of lines without *Fhb1* and no symptoms in lines that harbor *Fhb1*, except for the two inoculated spikelets that should always show symptoms to be able to assess type II resistance. The significant difference between the control lines with and without *Fhb1* indicate that the greenhouse trial worked well.

In this trial also other important *Fhb1* donors were analyzed. Ning_7840, Nobeokabozu_U, Sumai-3, W14 and Wangshuibai are all resistant to FHB and therefore these lines show on average below one DON bleached spikelet per head, except for ND_2710 shows on average 2.10 DON bleached spikelets per head. After *Fusarium* inoculation all other *Fhb1* donors show about on average two infected spikelets per head and no FHB spreading, which is the expected result.

For the DON treated homozygous mutant lines it was shown that no mutant line reached a significant difference to CM_82036. Also, many mutant lines did not show any symptoms and therefore had a DON severity of 0.00 infected spikelets per head. However, all mutant lines showed a statistically significant difference to the susceptible control line CM_Nil47. Summarized, no mutant line developed a DON susceptible phenotype, although some single heads of individual plants showed DON bleaching. These are interpreted as outliers, which could be caused by accidental inoculation of one spikelet twice. All DON treated mutant lines are classified as resistant lines. All three tested candidate genes (gene #6, gene #17, gene #20) can be excluded as the causal resistance genes to contribute to DON resistance.

For the *Fusarium* treated homozygous mutant lines most lines showed no significant difference to CM_82036, except for line 342_Ubi. This line carries a missense mutation and showed very high *Fusarium* severity (20 dai) of on average 18.29 infected spikelets per head, which exceeds the control line Remus with 16.48 infected spikelets per head. However, the effect of a missense mutation on the resulting protein is determined by the type of exchange and position. PROVEAN prediction of mutation effect is neutral, but SIFT prediction indicates an effect on protein function. The promising line 481_Ubi, discovered by Seidl (2017), was also tested in this trial. This line carries a nonsense mutation that leads to a premature stop codon and results in a non-functional protein. However, in this trial this promising line showed no symptoms of bleaching and therefore no altered phenotype compared to the wild-type.

Mutant line 784_Ca was the only line that showed symptoms of FHB bleaching beyond the two inoculated spikelets (on average 4.36 infected spikelets per head). Therefore, this line can be classified as FHB susceptible phenotype. The type of mutation is a missense mutation and PROVEAN prediction of the mutation effect is neutral. However, it was found out that the mutation was not homozygous as previously expected. Therefore, more F_2 lines need to be evaluated to assess if the SNP co-segregates with the susceptible phenotype.

In conclusion, the results indicate that all three candidate genes can be excluded as the causal gene for DON resistance. For FHB resistance none of the three tested genes can be identified with certainty as the resistance gene for spreading of the fungus.

9.3. Identification of susceptible lines with heterozygous mutations

Again, the correlation between infiltration/inoculation date and number of bleached spikelets per head was tested and only very low not significant correlation could be found. This could also be due to chance. Therefore, it can be said that the greenhouse setting worked well.

The heterozygous plants evaluated in this trial allow a direct phenotypic comparison between wild-type allele and mutant allele.

The mutant-type should show symptoms in this trial, whereas the wild-type should not show any symptoms of bleaching or spreading. The line 3094_Glyco showed faint bleaching in the mutant and wild-type lines, meaning that the altered phenotype is most likely controlled by a background mutation. In the DON experiment, only the mutant-type of line 256_Ubi showed on average 10 DON bleached spikelets per head and the wild-type showed no bleaching. In the *Fusarium* experiment, this line was the only one that showed FHB bleaching and a statistically significant difference to its wild-type. This line shows good co-segregation of the SNP in gene #17 and the phenotype. Therefore, it can be confirmed that a mutation in the *Fhb1* interval is responsible for the susceptible DON and FHB phenotypes. To validate that gene #17 is the causal resistance gene further analysis is needed. However, the mutant line 481_Ubi with a premature stop codon mutation in gene #17 revealed no DON bleaching and FHB spreading pointing towards a different genetic control.

10. Conclusions and outlook

Many questions remain concerning resistance mechanisms of FHB and identification of genes behind the resistance QTL in wheat is quite challenging, due to the large genome size of wheat and polyploidy. Also, many of the identified QTL still have to be fully validated and the number of ready-to use QTL is limited. Not only is FHB resistance a polygenic trait, but also other agronomical and quality traits. Further, environmental influences play an important role on disease development. Breeding for all important traits at once is very difficult. Therefore, selection of major resistance QTL by MAS is suggested before selection for other traits (Jia et al., 2018).

Schweiger et al. (2016) were able to identify 28 promising candidate genes for FHB and DON resistance in *Fhb1* region, on chromosome 3BS. Rawat et al. (2016) identified a gene encoding a chimeric lectin with agglutinin domains and a PFT domain on chromosome 3BS. This gene is assumed to confer FHB resistance, but not DON resistance. However, it is still not proven that PFT contributes to resistance. It is hypothesized that the association of PFT and FHB resistance is due to tight linkage to resistance genes in some germplasm (Jia et al., 2018). Further, it is still unclear if one gene confers both, FHB and DON resistance, or if two co-segregating genes confer each FHB and DON resistance (Schweiger et al., 2016). Further research must be done to clarify these questions.

In the past years genomic approaches have progressed and now allow rapid and cost-efficient gene identification based on induced mutagenesis. However, plants with a mutant phenotype still need to be identified on the field or in the greenhouse, among the most will show the wild-type for the trait of interest (Jia et al., 2018). Phenotyping is very time, labour and cost intensive, due to artificial inoculation systems, mist irrigation systems in the field or lighting systems. This is all done to provide optimal conditions for the fungus to grow on the field or in the greenhouse. Further, the influence of environment and genotype-by-environment interactions can be challenging to achieve reliable data. That is the reason why these experiments are repeated over several years.

However, over the past 5 years huge advances have been made in genomics that allow rapid and cost-efficient gene cloning. In the past years positional cloning was the method of choice for gene cloning. Positional cloning builds on genetic mapping and molecular approaches with stepwise localization of the gene. A segregating high-resolution mapping population that can be genotyped with molecular markers and phenotyped for the gene of interest is very important (Bettgenhaeuser and Krattinger, 2018). Double haploid production (Niu et al., 2014), speed breeding and high-throughput molecular marker technologies have significantly sped up this process. Speed breeding means that the photoperiod is prolonged and light spectrum optimized, thus the generation time of crop plants can be halved (Watson et al., 2018).

Mapping populations can then be rapidly genotyped with molecular markers with SNP arrays and genotyping-by-sequencing (GBS) protocols. The physical interval of the mapped gene then needs to be pieced together by chromosome walking. This takes several rounds of BAC library screening. BAC clones can store 100-200 kb of DNA. For hexaploid wheat around 500,000 BAC clones are required. Therefore, in BAC clones the major limitation is the size of foreign DNA that can be taken up by the vector to be stored in *E. coli* culture (Bettgenhaeuser and Krattinger, 2018).

There are several new approaches to rapidly clone a gene of interest in cereals. With ‘Targeted chromosome-based cloning vial long-range assembly’ (TACCA) the rapid establishment of physical intervals from any donor line is possible. Instead of spending several years with BAC clones, TACCA can generate mega-sized scaffolds spanning the target region only within a few months. This method is suitable for any gene, it is very cost-intensive (Bettgenhaeuser and Krattinger, 2018)..

Exome capture assay is used to sequence coding regions of EMS mutants. This approach allows to drastically reduce the genome size. Nonsense and missense mutations can be identified very fast. Compared to whole-genome resequencing, the success of this approach is dependent on the quality of the capture array (transcriptomics data or gene annotations that are used to design the array) (Bettgenhaeuser and Krattinger, 2018).

Also, based on whole genome-sequencing or reduced representation libraries TILLING populations can be resequenced and bring new possibilities for gene cloning. Usually, TILLING populations are used in reverse genetics approaches, but the availability of sequence information of entire mutant populations allows TILLING populations to be used for forward genetics approaches, too. In near future it is possible to start from an interesting phenotype detected in mutant lines and clone underlying genes by association mapping (MutMap) (Bettgenhaeuser and Krattinger, 2018).

Another strategy is the candidate gene approach to reduce complexity of large cereal genomes. This system is based on the knowledge that, e.g. many resistance genes code for intracellular immune receptors of NLR family. These NLRs trigger a strong defence reaction, known as hypersensitive reaction. In 'Resistance gene enrichment sequencing' (RenSeq) protocol the knowledge of NLR-triggered immunity for plant defence is used. RenSeq is based on capture array and allows an enrichment of NLR sequences. MutRenSeq combines RenSeq with forward genetics using EMS mutagenesis. With RenSeq it is possible to further reduce genome fraction, and consequently reduce costs for sequencing – compared to exome capture. However, not all disease resistance genes in cereals encode for NLRs (Bettgenhaeuser and Krattinger, 2018).

Also, natural DNA partitioning (e.g. chromosomes) can be used to reduce genomes of many cereal species. Single chromosomes of a cereal species can be isolated at high purity by flow cytometry. This approach is named MutChromSeq. It starts with an EMS-treated population, where loss-of-function mutants are identified. Then chromosomes, which harbour the gene of interest, are isolated from mutant and wild-type parent. Contigs with independent sequence polymorphisms in all mutants will be used for further validation (Vrána et al., 2000).

In future it may be possible to cost-efficiently resequence large genomes, such as maize, barley, wheat. Still, the identification and cloning of minor effect QTL, that are the rule in resistance breeding, is challenging because they do not show an easily to detect phenotypic effect. The best way until now is positional cloning, following a QTL or genome-wide association study (Bettgenhaeuser and Krattinger, 2018).

The main goal of this thesis, to evaluate three candidate genes for DON and FHB resistance, has been achieved, even if all three candidate genes can be excluded to contribute to DON resistance. For *Fusarium* resistance none of three candidate genes could be identified with certainty to contribute to FHB resistance. The mutant lines/genes that showed a susceptible phenotype after *Fusarium* inoculation need to be further characterized.

Until now, the only promising way for farmers to prevent the mycotoxin problem is to set up an IPM plan and combine crop rotation, cultural practices and resistant cultivars to reduce the threat FHB. Resistance to FHB in wheat cultivars is the most sustainable, economic and environmentally-friendly way to ensure healthful and safe food for a growing world population.

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12. Appendix

Table 42: Composition of TE-8 Buffer.

Name	Volume (ml)
1M TRIS	1 ml
0,5 M EDTA	0,2 ml
dH_2O	Fill up to 100 ml

Table 43: Composition of Wash 1.

Name	Volume (ml)
EtOH absolut	380 ml
2,5 M NaOAc	40 ml
dH_2O	80 ml

Table 44: Composition of Wash 2.

Name	Volume (ml)
EtOH absolut	380 ml
1 M NH_4OAc	5 ml
dH_2O	115 ml

Table 45: Evaluation of *Fusarium* bleached spikelets of mutant lines. The total number of Fusarium inoculated heads of homozygous and heterozygous lines is given. No. of heads with less than 2 symptomatic spikelets, no. of heads with 2 symptomatic spikelets and more than 2 symptomatic spikelets (= FHB spreading) are further listed.

	Homozygous lines	Heterozygous lines
no. of inoculated heads	487	221
0 bleached spikelets per head	5	0
	1%	-
0.5 - 2 bleached spikelets per head	450	192
	92%	87%
> 2 bleached spikelets per head	32	29
	7%	13%

Table 46: Evaluation of DON bleached spikelets per head of mutant lines. The total number of DON infiltrated heads of homozygous and heterozygous lines is given. No. of heads with 0 bleached spikelets and no. of heads more than 0 bleached spikelets are further listed.

	Homozygous lines	Heterozygous lines
no. of infiltrated heads	305	467
0 bleached spikelets per head	293	437
	96%	94%
>0 bleached spikelets per head	12	30
	4%	6%

R protocol:

Set data matrix:

```
Dataset <-
  read.table("C:/Users/Maria/Documents/Masterarbeit/Data.csv",
   header=TRUE, sep=";", na.strings="NA", dec=".", strip.white=TRUE)
```

Correlation between B1 and B2:

```
cor(Dataset[,c("B1","B2")], use="complete")
```

Scatterplot with linear regression for DON experiment:

```
scatterplot(B1~date | treat, regLine=TRUE, smooth=FALSE, boxplots=FALSE,
xlab="infiltration date", ylab="no. of DON-bleached spikelets per head
20 dai",
  by.groups=TRUE, data=Dataset)
```

```
scatterplot(B1~date,   regLine=TRUE,   smooth=FALSE,   boxplots=FALSE,
jitter=list(x=1, y=1),
xlab="infiltration date", ylab="no. of DON bleached spikelets per head
20 dai", col=c('gray50'), cex.lab=1.2,
   data=Dataset_DON)
```

Boxplot for control and mutant lines:

```
Boxplot(B1~genotype, data=Dataset_ubi, id=list(method="none"), xlab="",
ylab="no. of Fusarium bleached spikelets (20 dai)",
main="", cex.axis=1.1, cex.lab=1.5, las=2)
```

Comparison between DON and *Fusarium* experiment of homozygous lines:

```
scatterplot(Fus~DON,   regLine=TRUE,   smooth=FALSE,   boxplots=FALSE,
jitter=list(x=1, y=1), data=Dataset,
xlab="DON  severity  (20  dai)",  ylab="Fusarium  severity  (20  dai)",
col=c('gray50'), cex.lab=1.3, xlim=c(0,6), ylim=c(0,20))
```

Comparison between DON and *Fusarium* experiment of heterozygous lines:

```
scatterplot(Fus~DON,   regLine=TRUE,   smooth=FALSE,   boxplots=FALSE,
jitter=list(x=1, y=1), data=Dataset_Vgl_mut,
xlab="DON severity, wild type (20 dai)", ylab="Fusarium severity, wild
type   (20   dai)",   col=c('gray50'),   cex.lab=1.2,   xlim=c(0,11),
ylim=c(0,11))
```

Table 47: Greenhouse book of the homozygous mutant lines. The treatment (DON infiltration, Fusarium inoculation), number of replication, and genotype can be seen. Further, the pot number, number of infiltrated/inoculated heads, date of treatment and date of evaluation (B1, B2) is given. The respective dates are listed in Table 27.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	CM_Nil38	1	1	5	0			0		
DON	1	CM_Nil38	1	2	6	0			0		
DON	1	CM_Nil38	1	3	7	0			0		
DON	1	CM_Nil38	1	4	7	0			0		
DON	1	CM_Nil38	1	5	7	0			0		
DON	1	CM_Nil38	1	6	10	0			0		
DON	1	CM_Nil38	1	7							
DON	1	CM_Nil38	1	8							
DON	1	CM_Nil38	1	9							
DON	1	CM_Nil38	1	10							
DON	1	RH_CM_651	2	1	8	3	2		3		
DON	1	RH_CM_651	2	2	9	7			7		
DON	1	RH_CM_651	2	3	11	2			3		
DON	1	RH_CM_651	2	4	12	12			12		
DON	1	RH_CM_651	2	5	13	3			5		
DON	1	RH_CM_651	2	6							
DON	1	RH_CM_651	2	7							
DON	1	RH_CM_651	2	8							
DON	1	RH_CM_651	2	9							
DON	1	RH_CM_651	2	10							
DON	1	CM_Nil47	3	1	7	8			10		
DON	1	CM_Nil47	3	2	7	3			3		
DON	1	CM_Nil47	3	3	8	2	1		2	3	
DON	1	CM_Nil47	3	4	9	8			8		
DON	1	CM_Nil47	3	5	10	8			12		
DON	1	CM_Nil47	3	6							
DON	1	CM_Nil47	3	7							
DON	1	CM_Nil47	3	8							
DON	1	CM_Nil47	3	9							
DON	1	CM_Nil47	3	10							
DON	1	CM_Nil43	4	1	6	0,5?			0.5		
DON	1	CM_Nil43	4	2	6	0			0		
DON	1	CM_Nil43	4	3	6	0			0		
DON	1	CM_Nil43	4	4	6	0			0		
DON	1	CM_Nil43	4	5	7	0			0		
DON	1	CM_Nil43	4	6	8	0			0		
DON	1	CM_Nil43	4	7							
DON	1	CM_Nil43	4	8							
DON	1	CM_Nil43	4	9							
DON	1	CM_Nil43	4	10							
DON	1	Remus	5	1	10	2			2		
DON	1	Remus	5	2	11	1	1		2		
DON	1	Remus	5	3	11	1	1		4		
DON	1	Remus	5	4	11	5	3		9		
DON	1	Remus	5	5	12	2			4		
DON	1	Remus	5	6	13	2			2		
DON	1	Remus	5	7							
DON	1	Remus	5	8							
DON	1	Remus	5	9							
DON	1	Remus	5	10							
DON	1	CM_Nil51	6	1	4	3	8		3	8	
DON	1	CM_Nil51	6	2	6	5	2		8		
DON	1	CM_Nil51	6	3	6	2			2	5	
DON	1	CM_Nil51	6	4	7	6			4	3	
DON	1	CM_Nil51	6	5	7	2	2		2		
DON	1	CM_Nil51	6	6	7	2	5		2	5	
DON	1	CM_Nil51	6	7							
DON	1	CM_Nil51	6	8							
DON	1	CM_Nil51	6	9							
DON	1	CM_Nil51	6	10							
DON	1	CM_82036	7	1	8	0			0		
DON	1	CM_82036	7	2	8	0			0		
DON	1	CM_82036	7	3	8	0			0		

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	CM_82036	7	4	9	0			0		
DON	1	CM_82036	7	5	9	0			0		
DON	1	CM_82036	7	6	10	0			0		
DON	1	CM_82036	7	7	10	0			0		
DON	1	CM_82036	7	8							
DON	1	CM_82036	7	9							
DON	1	CM_82036	7	10							
DON	1	RM_CM_83	8	1	5	2			2		
DON	1	RM_CM_83	8	2	6	8			10		
DON	1	RM_CM_83	8	3	6	8			9		
DON	1	RM_CM_83	8	4	6	3	2		2	6	
DON	1	RM_CM_83	8	5	7	2	1		2		
DON	1	RM_CM_83	8	6	8	2			2	1	
DON	1	RM_CM_83	8	7							
DON	1	RM_CM_83	8	8							
DON	1	RM_CM_83	8	9							
DON	1	RM_CM_83	8	10							
DON	1	RM_CM_643	9	1	6	2	5		8		
DON	1	RM_CM_643	9	2	8	3	4		6	1	
DON	1	RM_CM_643	9	3	8	2			2		
DON	1	RM_CM_643	9	4	8	4			4		
DON	1	RM_CM_643	9	5	8	2	4		10		
DON	1	RM_CM_643	9	6							
DON	1	RM_CM_643	9	7							
DON	1	RM_CM_643	9	8							
DON	1	RM_CM_643	9	9							
DON	1	RM_CM_643	9	10							
DON	1	GA_3B_18_1	10	1	6	2	2		2		
DON	1	GA_3B_18_1	10	2	7	4			3		
DON	1	GA_3B_18_1	10	3	8	10			10		
DON	1	GA_3B_18_1	10	4	9	0	3		6		
DON	1	GA_3B_18_1	10	5	9	8			8		
DON	1	GA_3B_18_1	10	6							
DON	1	GA_3B_18_1	10	7							
DON	1	GA_3B_18_1	10	8							
DON	1	GA_3B_18_1	10	9							
DON	1	GA_3B_18_1	10	10							
DON	1	GA_3B_18_3	11	1	5	2	3		2	3	
DON	1	GA_3B_18_3	11	2	7	2	1		2		
DON	1	GA_3B_18_3	11	3	8	3			3	1	
DON	1	GA_3B_18_3	11	4	8	2			2	1	
DON	1	GA_3B_18_3	11	5	9	8			5		
DON	1	GA_3B_18_3	11	6							
DON	1	GA_3B_18_3	11	7							
DON	1	GA_3B_18_3	11	8							
DON	1	GA_3B_18_3	11	9							
DON	1	GA_3B_18_3	11	10							
DON	1	GA_3B_18_22	12	1	5	0			0		
DON	1	GA_3B_18_22	12	2	5	0			0		
DON	1	GA_3B_18_22	12	3	5	0			0		
DON	1	GA_3B_18_22	12	4	6	0			0		
DON	1	GA_3B_18_22	12	5	6	0			0		
DON	1	GA_3B_18_22	12	6	8	0			0		
DON	1	GA_3B_18_22	12	7							
DON	1	GA_3B_18_22	12	8							
DON	1	GA_3B_18_22	12	9							
DON	1	GA_3B_18_22	12	10							
DON	1	98_Ca	13	1	8	0			0		
DON	1	98_Ca	13	2	8	0			0		
DON	1	98_Ca	13	3	11	0,5?			0.5		
DON	1	98_Ca	13	4							
DON	1	98_Ca	13	5							
DON	1	98_Ca	13	6							
DON	1	98_Ca	13	7							
DON	1	98_Ca	13	8							
DON	1	98_Ca	13	9							

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	98_Ca	13	10							
DON	1	GA_3B_18_15	14	1	4	0			0		
DON	1	GA_3B_18_15	14	2	5	0			0		
DON	1	GA_3B_18_15	14	3	5	0			0		
DON	1	GA_3B_18_15	14	4	5	0			0		
DON	1	GA_3B_18_15	14	5	6	0			0		
DON	1	GA_3B_18_15	14	6							
DON	1	GA_3B_18_15	14	7							
DON	1	GA_3B_18_15	14	8							
DON	1	GA_3B_18_15	14	9							
DON	1	GA_3B_18_15	14	10							
DON	1	784_Ca	15	1	3	?			2b		
DON	1	784_Ca	15	2	3	0			0		
DON	1	784_Ca	15	3	5	2b			2b	2b	
DON	1	784_Ca	15	4	5	2b			2b		
DON	1	784_Ca	15	5	6	0	2b		0	2b	
DON	1	784_Ca	15	6							
DON	1	784_Ca	15	7							
DON	1	784_Ca	15	8							
DON	1	784_Ca	15	9							
DON	1	784_Ca	15	10							
DON	1	219_Ca	16	1	5	0			0		
DON	1	219_Ca	16	2	6	0			0		
DON	1	219_Ca	16	3	6	0			0	4	
DON	1	219_Ca	16	4	7	0			0		
DON	1	219_Ca	16	5	7	0			0		
DON	1	219_Ca	16	6							
DON	1	219_Ca	16	7							
DON	1	219_Ca	16	8							
DON	1	219_Ca	16	9							
DON	1	219_Ca	16	10							
DON	1	GA_3B_18_9	17	1	4	2			2		
DON	1	GA_3B_18_9	17	2	4	2			2		
DON	1	GA_3B_18_9	17	3	4	2	5		4	4	
DON	1	GA_3B_18_9	17	4	5	4			3	1	
DON	1	GA_3B_18_9	17	5	7	10			10		
DON	1	GA_3B_18_9	17	6	8	7			8		
DON	1	GA_3B_18_9	17	7							
DON	1	GA_3B_18_9	17	8							
DON	1	GA_3B_18_9	17	9							
DON	1	GA_3B_18_9	17	10							
DON	1	GA_3B_18_16	18	1	5	0			0		
DON	1	GA_3B_18_16	18	2	5	0			0	2	
DON	1	GA_3B_18_16	18	3	5	0	0.5		0	2	
DON	1	GA_3B_18_16	18	4	6	0			0		
DON	1	GA_3B_18_16	18	5	6	0			0		
DON	1	GA_3B_18_16	18	6							
DON	1	GA_3B_18_16	18	7							
DON	1	GA_3B_18_16	18	8							
DON	1	GA_3B_18_16	18	9							
DON	1	GA_3B_18_16	18	10							
DON	1	1399	19	1	6	2	3		2		
DON	1	1399	19	2	6	4	5		8		
DON	1	1399	19	3	7	8			10		
DON	1	1399	19	4	7	3	3		2	5	
DON	1	1399	19	5	7	3			2		
DON	1	1399	19	6	10	6			9		
DON	1	1399	19	7	10	6			3	5	
DON	1	1399	19	8							
DON	1	1399	19	9							
DON	1	1399	19	10							
DON	1	256	20	1	8	2			2		
DON	1	256	20	2	8	2	1		2		
DON	1	256	20	3	10	2			2		
DON	1	256	20	4	10	2	2		3	1	
DON	1	256	20	5	11	1	2		3	1	

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	256	20	6	11	2			2		
DON	1	256	20	7	11	4			3		
DON	1	256	20	8	13	3			3		
DON	1	256	20	9							
DON	1	256	20	10							
DON	1	177	21	1	8	2			2		
DON	1	177	21	2	8	2			2		
DON	1	177	21	3	8	2			2		
DON	1	177	21	4	8	2	1		2		
DON	1	177	21	5	9	2			2		
DON	1	177	21	6	10	2	1		3		
DON	1	177	21	7	10	3			3		
DON	1	177	21	8	11	3			3		
DON	1	177	21	9	11	2			2		
DON	1	177	21	10							
DON	1	7919_no_1_Hyd	22	1	8	0			0		
DON	1	7919_no_1_Hyd	22	2	9	0			0		
DON	1	7919_no_1_Hyd	22	3	9	0			0		
DON	1	7919_no_1_Hyd	22	4	9	0			0		
DON	1	7919_no_1_Hyd	22	5	10	0			0		
DON	1	7919_no_1_Hyd	22	6	7	0			0		
DON	1	7919_no_1_Hyd	22	7							
DON	1	7919_no_1_Hyd	22	8							
DON	1	7919_no_1_Hyd	22	9							
DON	1	7919_no_1_Hyd	22	10							
DON	1	1903	23	1	6	0			0		
DON	1	1903	23	2	7	0			0		
DON	1	1903	23	3	7	0			0		
DON	1	1903	23	4	7	0			0		
DON	1	1903	23	5	7	0			0		
DON	1	1903	23	6							
DON	1	1903	23	7							
DON	1	1903	23	8							
DON	1	1903	23	9							
DON	1	1903	23	10							
DON	1	Sumai3_MX	24	1	2	0			0		
DON	1	Sumai3_MX	24	2	2	0?			0		
DON	1	Sumai3_MX	24	3	4	0			0		
DON	1	Sumai3_MX	24	4	4	3			3		
DON	1	Sumai3_MX	24	5	4	0			0		
DON	1	Sumai3_MX	24	6	4	2			2		
DON	1	Sumai3_MX	24	7							
DON	1	Sumai3_MX	24	8							
DON	1	Sumai3_MX	24	9							
DON	1	Sumai3_MX	24	10							
DON	1	W14	25	1	3	0			0		
DON	1	W14	25	2	4	2			2		
DON	1	W14	25	3	4	1			1		
DON	1	W14	25	4	5	0			0	0.5	
DON	1	W14	25	5	5	0			0	0.1	
DON	1	W14	25	6							
DON	1	W14	25	7							
DON	1	W14	25	8							
DON	1	W14	25	9							
DON	1	W14	25	10							
DON	1	2203_Ca	26	1	5	0			0		
DON	1	2203_Ca	26	2	5	0			0	6	
DON	1	2203_Ca	26	3	5	0			0		
DON	1	2203_Ca	26	4	7	0			0		
DON	1	2203_Ca	26	5	7	0			0		
DON	1	2203_Ca	26	6							
DON	1	2203_Ca	26	7							
DON	1	2203_Ca	26	8							
DON	1	2203_Ca	26	9							
DON	1	2203_Ca	26	10							
DON	1	1914_Ca	27	1	5	2b			0,5*2b		

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	1914_Ca	27	2	6	0			0		
DON	1	1914_Ca	27	3	6	0			0		
DON	1	1914_Ca	27	4	6	0			0		
DON	1	1914_Ca	27	5	6	0			0		
DON	1	1914_Ca	27	6							
DON	1	1914_Ca	27	7							
DON	1	1914_Ca	27	8							
DON	1	1914_Ca	27	9							
DON	1	1914_Ca	27	10							
DON	1	6293_Ca	28	1	4	1			1		
DON	1	6293_Ca	28	2	5	0			0		
DON	1	6293_Ca	28	3	5	0			0	2	
DON	1	6293_Ca	28	4	6	0			0		
DON	1	6293_Ca	28	5	6	0			0		
DON	1	6293_Ca	28	6							
DON	1	6293_Ca	28	7							
DON	1	6293_Ca	28	8							
DON	1	6293_Ca	28	9							
DON	1	6293_Ca	28	10							
DON	1	8406_Ca	29	1	5	0			0		
DON	1	8406_Ca	29	2	5	0			0		
DON	1	8406_Ca	29	3	6	0			-		
DON	1	8406_Ca	29	4	7	0			0		
DON	1	8406_Ca	29	5	7	0			0		
DON	1	8406_Ca	29	6							
DON	1	8406_Ca	29	7							
DON	1	8406_Ca	29	8							
DON	1	8406_Ca	29	9							
DON	1	8406_Ca	29	10							
DON	1	940_Glyco	30	1	5	0			0		
DON	1	940_Glyco	30	2	8	0			0		
DON	1	940_Glyco	30	3	8	0			0		
DON	1	940_Glyco	30	4	8	0			0		
DON	1	940_Glyco	30	5	8	0			0		
DON	1	940_Glyco	30	6							
DON	1	940_Glyco	30	7							
DON	1	940_Glyco	30	8							
DON	1	940_Glyco	30	9							
DON	1	940_Glyco	30	10							
DON	1	738_Glyco	31	1	4	0			0		
DON	1	738_Glyco	31	2	4	0			0		
DON	1	738_Glyco	31	3	4	0			0		
DON	1	738_Glyco	31	4	5	0			0		
DON	1	738_Glyco	31	5	6	0			0		
DON	1	738_Glyco	31	6							
DON	1	738_Glyco	31	7							
DON	1	738_Glyco	31	8							
DON	1	738_Glyco	31	9							
DON	1	738_Glyco	31	10							
DON	1	1369_Glyco	32	1	5	2?			3	3	
DON	1	1369_Glyco	32	2	5	2?			2	2	
DON	1	1369_Glyco	32	3	5	1?			3	3	
DON	1	1369_Glyco	32	4	6	0	1		0	7	
DON	1	1369_Glyco	32	5	6	0	0.5		0		
DON	1	1369_Glyco	32	6							
DON	1	1369_Glyco	32	7							
DON	1	1369_Glyco	32	8							
DON	1	1369_Glyco	32	9							
DON	1	1369_Glyco	32	10							
DON	1	1345_Glyco	33	1	4	0			0		
DON	1	1345_Glyco	33	2	4	0			0		
DON	1	1345_Glyco	33	3	4	0			0		
DON	1	1345_Glyco	33	4	5	0			0		
DON	1	1345_Glyco	33	5	5	0			0,5b		
DON	1	1345_Glyco	33	6							
DON	1	1345_Glyco	33	7							

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	1345_Glyco	33	8							
DON	1	1345_Glyco	33	9							
DON	1	1345_Glyco	33	10							
DON	1	481_Ubi	34	1	5	0			0		
DON	1	481_Ubi	34	2	6	0			0		
DON	1	481_Ubi	34	3	6	0			0		
DON	1	481_Ubi	34	4	6	0			0		
DON	1	481_Ubi	34	5	6	0			0		
DON	1	481_Ubi	34	6							
DON	1	481_Ubi	34	7							
DON	1	481_Ubi	34	8							
DON	1	481_Ubi	34	9							
DON	1	481_Ubi	34	10							
DON	1	3632_Glyco	35	1	4	0			0		
DON	1	3632_Glyco	35	2	6	0			0		
DON	1	3632_Glyco	35	3	6	0			0		
DON	1	3632_Glyco	35	4	7	0			0		
DON	1	3632_Glyco	35	5	7	0			0		
DON	1	3632_Glyco	35	6							
DON	1	3632_Glyco	35	7							
DON	1	3632_Glyco	35	8							
DON	1	3632_Glyco	35	9							
DON	1	3632_Glyco	35	10							
DON	1	182_Ubi	36	1	6	0			0		
DON	1	182_Ubi	36	2	6	0			0		
DON	1	182_Ubi	36	3	7	0			0		
DON	1	182_Ubi	36	4	7	0			0		
DON	1	182_Ubi	36	5	8	0			0		
DON	1	182_Ubi	36	6							
DON	1	182_Ubi	36	7							
DON	1	182_Ubi	36	8							
DON	1	182_Ubi	36	9							
DON	1	182_Ubi	36	10							
DON	1	342_Ubi	37	1	3	0			0		
DON	1	342_Ubi	37	2	4	0			0		
DON	1	342_Ubi	37	3	5	0			0	4	
DON	1	342_Ubi	37	4	6	0			0,5b		
DON	1	342_Ubi	37	5	7	0			0		
DON	1	342_Ubi	37	6							
DON	1	342_Ubi	37	7							
DON	1	342_Ubi	37	8							
DON	1	342_Ubi	37	9							
DON	1	342_Ubi	37	10							
DON	1	3045_Ubi	38	1	3	0			0		
DON	1	3045_Ubi	38	2	4	0	1		0	1	
DON	1	3045_Ubi	38	3	4	0	0.5		0	0.5	
DON	1	3045_Ubi	38	4	5	0			0		
DON	1	3045_Ubi	38	5	5	0			0	4	
DON	1	3045_Ubi	38	6							
DON	1	3045_Ubi	38	7							
DON	1	3045_Ubi	38	8							
DON	1	3045_Ubi	38	9							
DON	1	3045_Ubi	38	10							
DON	1	2119	39	1	7	0			0		
DON	1	2119	39	2	7	0			0		
DON	1	2119	39	3	7	0			0		
DON	1	2119	39	4	8	0			0		
DON	1	2119	39	5	9	0			0		
DON	1	2119	39	6	10	0			0		
DON	1	2119	39	7	10	0			0		
DON	1	2119	39	8	10	0			0		
DON	1	2119	39	9	11	0			0		
DON	1	2119	39	10							
DON	1	3093_Glyco	40	1	3	0			0		
DON	1	3093_Glyco	40	2	3	0			0	2	
DON	1	3093_Glyco	40	3	3	0	1		0	2	

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	3093_Glyco	40	4	4	0			0		
DON	1	3093_Glyco	40	5	4	0			0		
DON	1	3093_Glyco	40	6							
DON	1	3093_Glyco	40	7							
DON	1	3093_Glyco	40	8							
DON	1	3093_Glyco	40	9							
DON	1	3093_Glyco	40	10							
DON	1	869_Ubi	41	1	4	0			0		
DON	1	869_Ubi	41	2	4	0			0	2	
DON	1	869_Ubi	41	3	5	0			0		
DON	1	869_Ubi	41	4	5	0			0		
DON	1	869_Ubi	41	5	6	0			0		
DON	1	869_Ubi	41	6							
DON	1	869_Ubi	41	7							
DON	1	869_Ubi	41	8							
DON	1	869_Ubi	41	9							
DON	1	869_Ubi	41	10							
DON	1	721_Ubi	42	1	6	0			0		
DON	1	721_Ubi	42	2	6	0			0		
DON	1	721_Ubi	42	3	7	0			0		
DON	1	721_Ubi	42	4	7	0			0		
DON	1	721_Ubi	42	5	8	0			0		
DON	1	721_Ubi	42	6							
DON	1	721_Ubi	42	7							
DON	1	721_Ubi	42	8							
DON	1	721_Ubi	42	9							
DON	1	721_Ubi	42	10							
DON	1	1616_Glyco	43	1	3	0			0		
DON	1	1616_Glyco	43	2	3	0			0		
DON	1	1616_Glyco	43	3	3	2?			?		
DON	1	1616_Glyco	43	4	3	0			0		
DON	1	1616_Glyco	43	5	4	0			0		
DON	1	1616_Glyco	43	6							
DON	1	1616_Glyco	43	7							
DON	1	1616_Glyco	43	8							
DON	1	1616_Glyco	43	9							
DON	1	1616_Glyco	43	10							
DON	1	1148_Glyco	44	1	4	0			0		
DON	1	1148_Glyco	44	2	4	0			0		
DON	1	1148_Glyco	44	3	5	0			0	1	
DON	1	1148_Glyco	44	4	5	0			0	2	
DON	1	1148_Glyco	44	5	6	0			0		
DON	1	1148_Glyco	44	6							
DON	1	1148_Glyco	44	7							
DON	1	1148_Glyco	44	8							
DON	1	1148_Glyco	44	9							
DON	1	1148_Glyco	44	10							
DON	1	1111_Glyco	45	1	4	0			0		
DON	1	1111_Glyco	45	2	4	0			0		
DON	1	1111_Glyco	45	3	6	0			0		
DON	1	1111_Glyco	45	4	6	0			0		
DON	1	1111_Glyco	45	5	6	0			0		
DON	1	1111_Glyco	45	6							
DON	1	1111_Glyco	45	7							
DON	1	1111_Glyco	45	8							
DON	1	1111_Glyco	45	9							
DON	1	1111_Glyco	45	10							
DON	1	3028_Ca	46	1	5	0			0		
DON	1	3028_Ca	46	2	5	0			0		
DON	1	3028_Ca	46	3	6	0			0		
DON	1	3028_Ca	46	4	6	0			0		
DON	1	3028_Ca	46	5	6	0			0		
DON	1	3028_Ca	46	6							
DON	1	3028_Ca	46	7							
DON	1	3028_Ca	46	8							
DON	1	3028_Ca	46	9							

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	3028_Ca	46	10							
DON	1	723_Glyco	47	1	4	0			0		
DON	1	723_Glyco	47	2	4	0			0		
DON	1	723_Glyco	47	3	5	0			0		
DON	1	723_Glyco	47	4	6	0			0		
DON	1	723_Glyco	47	5	6	0			0		
DON	1	723_Glyco	47	6							
DON	1	723_Glyco	47	7							
DON	1	723_Glyco	47	8							
DON	1	723_Glyco	47	9							
DON	1	723_Glyco	47	10							
DON	1	8892_Ca	48	1	5	0			0		
DON	1	8892_Ca	48	2	5	0			0		
DON	1	8892_Ca	48	3	5	0			0		
DON	1	8892_Ca	48	4	6	0			0		
DON	1	8892_Ca	48	5	6	0			0		
DON	1	8892_Ca	48	6							
DON	1	8892_Ca	48	7							
DON	1	8892_Ca	48	8							
DON	1	8892_Ca	48	9							
DON	1	8892_Ca	48	10							
DON	1	Wangshuibai	49	1	3	0	1		?		
DON	1	Wangshuibai	49	2	4	0			0		
DON	1	Wangshuibai	49	3	4	0			0		
DON	1	Wangshuibai	49	4	4	0.5			0.5		
DON	1	Wangshuibai	49	5	5	0			0		
DON	1	Wangshuibai	49	6							
DON	1	Wangshuibai	49	7							
DON	1	Wangshuibai	49	8							
DON	1	Wangshuibai	49	9							
DON	1	Wangshuibai	49	10							
DON	1	ND_2710	50	1	2	1			1		
DON	1	ND_2710	50	2	2	2			2		
DON	1	ND_2710	50	3	3	3			3b		
DON	1	ND_2710	50	4	3	1			1		
DON	1	ND_2710	50	5	3	3			3b		
DON	1	ND_2710	50	6							
DON	1	ND_2710	50	7							
DON	1	ND_2710	50	8							
DON	1	ND_2710	50	9							
DON	1	ND_2710	50	10							
DON	1	Sumai3_Ung1	51	1	4	0			0		
DON	1	Sumai3_Ung1	51	2	6	0			0	3	
DON	1	Sumai3_Ung1	51	3	6	0	3		0	3	
DON	1	Sumai3_Ung1	51	4	6	0			0		
DON	1	Sumai3_Ung1	51	5	6	0			0		
DON	1	Sumai3_Ung1	51	6							
DON	1	Sumai3_Ung1	51	7							
DON	1	Sumai3_Ung1	51	8							
DON	1	Sumai3_Ung1	51	9							
DON	1	Sumai3_Ung1	51	10							
DON	1	1632_Ubi	52	1	6	0			0		
DON	1	1632_Ubi	52	2	7	0			0		
DON	1	1632_Ubi	52	3	8	0			0		
DON	1	1632_Ubi	52	4	8	0			0		
DON	1	1632_Ubi	52	5	8	0			0		
DON	1	1632_Ubi	52	6							
DON	1	1632_Ubi	52	7							
DON	1	1632_Ubi	52	8							
DON	1	1632_Ubi	52	9							
DON	1	1632_Ubi	52	10							
DON	1	1325_Ubi	53	1	6	0			0		
DON	1	1325_Ubi	53	2	6	0			0		
DON	1	1325_Ubi	53	3	6	0			0		
DON	1	1325_Ubi	53	4	7	0			0		
DON	1	1325_Ubi	53	5	7	0			0		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	1325_Ubi	53	6							
DON	1	1325_Ubi	53	7							
DON	1	1325_Ubi	53	8							
DON	1	1325_Ubi	53	9							
DON	1	1325_Ubi	53	10							
DON	1	5863	54	1	5	9			6		
DON	1	5863	54	2	6	4			4	5	
DON	1	5863	54	3	6	7			10		
DON	1	5863	54	4	6	6			8		
DON	1	5863	54	5	7	4			8		
DON	1	5863	54	6							
DON	1	5863	54	7							
DON	1	5863	54	8							
DON	1	5863	54	9							
DON	1	5863	54	10							
DON	1	5527	55	1	7	0			0		
DON	1	5527	55	2	7	2b			2b		
DON	1	5527	55	3	8	0	0.5		?	?	
DON	1	5527	55	4	8	0			?	?	
DON	1	5527	55	5	8	0			?	?	
DON	1	5527	55	6	8	0			?	?	
DON	1	5527	55	7	10	0			0		
DON	1	5527	55	8	10	0			0		
DON	1	5527	55	9	11	0.5			0.5		
DON	1	5527	55	10	13	0,5 braun			0,5b		
DON	1	1254_Ubi	56	1	3	0			0		
DON	1	1254_Ubi	56	2	3	0			0		
DON	1	1254_Ubi	56	3	3	0			0		
DON	1	1254_Ubi	56	4	4	0			0		
DON	1	1254_Ubi	56	5	4	0			0		
DON	1	1254_Ubi	56	6							
DON	1	1254_Ubi	56	7							
DON	1	1254_Ubi	56	8							
DON	1	1254_Ubi	56	9							
DON	1	1254_Ubi	56	10							
DON	1	1047_Ubi	57	1	4	0			0	2	
DON	1	1047_Ubi	57	2	4	0	2		0	2	
DON	1	1047_Ubi	57	3	5	0			0	1	
DON	1	1047_Ubi	57	4	5	0			0	1	
DON	1	1047_Ubi	57	5	5	0			0	3	
DON	1	1047_Ubi	57	6							
DON	1	1047_Ubi	57	7							
DON	1	1047_Ubi	57	8							
DON	1	1047_Ubi	57	9							
DON	1	1047_Ubi	57	10							
DON	1	1882	58	1	3	0	2		0.5	0.5	
DON	1	1882	58	2	5	1.5			2		
DON	1	1882	58	3	5	0.5			0	2	
DON	1	1882	58	4	6	2			2		
DON	1	1882	58	5	6	4	2		8		
DON	1	1882	58	6	15	3			4		
DON	1	1882	58	7	15	3			3		
DON	1	1882	58	8							
DON	1	1882	58	9							
DON	1	1882	58	10							
DON	1	1745	59	1	6	0			0		
DON	1	1745	59	2	7	0			0		
DON	1	1745	59	3	8	0			0		
DON	1	1745	59	4	8	0			0		
DON	1	1745	59	5	8	0			0		
DON	1	1745	59	6	10	0			0		
DON	1	1745	59	7	10	0			0		
DON	1	1745	59	8							
DON	1	1745	59	9							
DON	1	1745	59	10							
DON	1	Ning_7840	66	1	2	0			0		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	Ning_7840	66	2	2	0			0		
DON	1	Ning_7840	66	3	2	0			0		
DON	1	Ning_7840	66	4	3	0			0		
DON	1	Ning_7840	66	5	3	0			0		
DON	1	Ning_7840	66	6	3	0			0		
DON	1	Ning_7840	66	7							
DON	1	Ning_7840	66	8							
DON	1	Ning_7840	66	9							
DON	1	Ning_7840	66	10							
DON	1	Nobeokabozu_U	67	1	3	3b			3b		
DON	1	Nobeokabozu_U	67	2	3	0			?		
DON	1	Nobeokabozu_U	67	3	4	1b			1b		
DON	1	Nobeokabozu_U	67	4	4	1,5b			1,5b		
DON	1	Nobeokabozu_U	67	5	5	0,5b			0	0,5b	
DON	1	Nobeokabozu_U	67	6							
DON	1	Nobeokabozu_U	67	7							
DON	1	Nobeokabozu_U	67	8							
DON	1	Nobeokabozu_U	67	9							
DON	1	Nobeokabozu_U	67	10							
DON	1	2259	68	1	4	0			2+4		
DON	1	2259	68	2	5	0	2		2		
DON	1	2259	68	3	6	0			0		
DON	1	2259	68	4	7	2*0,5			0		
DON	1	2259	68	5	7	0.5			0		
DON	1	2259	68	6	8	0			7		
DON	1	2259	68	7	11	0	0.5		0	0.5	
DON	1	2259	68	8							
DON	1	2259	68	9							
DON	1	2259	68	10							
DON	1	3746_Ubi	69	1	7	0			0		
DON	1	3746_Ubi	69	2	7	0			0		
DON	1	3746_Ubi	69	3	7	0			0		
DON	1	3746_Ubi	69	4	7	0			0		
DON	1	3746_Ubi	69	5	7	0			0		
DON	1	3746_Ubi	69	6							
DON	1	3746_Ubi	69	7							
DON	1	3746_Ubi	69	8							
DON	1	3746_Ubi	69	9							
DON	1	3746_Ubi	69	10							
DON	1	3060	70	1	5	0			0		
DON	1	3060	70	2	5	0			0		
DON	1	3060	70	3	6	0			0		
DON	1	3060	70	4	6	0			0		
DON	1	3060	70	5	6	0			0		
DON	1	3060	70	6	8	0,5*2			0		
DON	1	3060	70	7							
DON	1	3060	70	8							
DON	1	3060	70	9							
DON	1	3060	70	10							
DON	1	3219	71	1	7	0			0		
DON	1	3219	71	2	8	0			0		
DON	1	3219	71	3	9	0			0		
DON	1	3219	71	4	9	0			0		
DON	1	3219	71	5	10	0			0		
DON	1	3219	71	6	10	0			0		
DON	1	3219	71	7							
DON	1	3219	71	8							
DON	1	3219	71	9							
DON	1	3219	71	10							
DON	1	3144	72	1	7	0			0		
DON	1	3144	72	2	8	0			0		
DON	1	3144	72	3	9	0			0		
DON	1	3144	72	4	9	0			0		
DON	1	3144	72	5	9	0			0		
DON	1	3144	72	6	10	0			0		
DON	1	3144	72	7	10	0			0		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	1	3144	72	8							
DON	1	3144	72	9							
DON	1	3144	72	10							
DON	1	5485	73	1	6	0	1		0	1	
DON	1	5485	73	2	6	0			0		
DON	1	5485	73	3	7	0			0		
DON	1	5485	73	4	7	0			0		
DON	1	5485	73	5	7	0			0		
DON	1	5485	73	6							
DON	1	5485	73	7							
DON	1	5485	73	8							
DON	1	5485	73	9							
DON	1	5485	73	10							
DON	1	5108	74	1	5	0			0		
DON	1	5108	74	2	6	0			0		
DON	1	5108	74	3	7	0			0		
DON	1	5108	74	4	7	0			0		
DON	1	5108	74	5	7	0			0		
DON	1	5108	74	6							
DON	1	5108	74	7							
DON	1	5108	74	8							
DON	1	5108	74	9							
DON	1	5108	74	10							
DON	2	CM_Nil47	131	1	2	6	4		4	5	
DON	2	CM_Nil47	131	2	3	3	4		8		
DON	2	CM_Nil47	131	3	3	3			3		
DON	2	CM_Nil47	131	4	4	11			10		
DON	2	CM_Nil47	131	5	4	2			3		
DON	2	CM_Nil47	131	6							
DON	2	CM_Nil47	131	7							
DON	2	CM_Nil47	131	8							
DON	2	CM_Nil47	131	9							
DON	2	CM_Nil47	131	10							
DON	2	CM_82036	132	1	4	0			0	0.5	
DON	2	CM_82036	132	2	5	0			0		
DON	2	CM_82036	132	3	6	0			0		
DON	2	CM_82036	132	4	7	0			0		
DON	2	CM_82036	132	5	7	0			0		
DON	2	CM_82036	132	6							
DON	2	CM_82036	132	7							
DON	2	CM_82036	132	8							
DON	2	CM_82036	132	9							
DON	2	CM_82036	132	10							
DON	2	CM_Nil43	133	1	4	0			0		
DON	2	CM_Nil43	133	2	4	0			0	2	
DON	2	CM_Nil43	133	3	4	0			0	0.5	
DON	2	CM_Nil43	133	4	5	0	0.5		0		
DON	2	CM_Nil43	133	5	5	0	2		0		
DON	2	CM_Nil43	133	6	6	0			0		
DON	2	CM_Nil43	133	7							
DON	2	CM_Nil43	133	8							
DON	2	CM_Nil43	133	9							
DON	2	CM_Nil43	133	10							
DON	2	CM_Nil51	134	1	3	10			10		
DON	2	CM_Nil51	134	2	4	10			10		
DON	2	CM_Nil51	134	3	4	4	4		8		
DON	2	CM_Nil51	134	4	4	3	4		2	5	
DON	2	CM_Nil51	134	5	4	2			2		
DON	2	CM_Nil51	134	6							
DON	2	CM_Nil51	134	7							
DON	2	CM_Nil51	134	8							
DON	2	CM_Nil51	134	9							
DON	2	CM_Nil51	134	10							
DON	2	CM_Nil38	135	1	3	0			0	2	
DON	2	CM_Nil38	135	2	3	0	0.5		0	2	
DON	2	CM_Nil38	135	3	3	0			0	2	

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	CM_Nil38	135	4	4	0			0		
DON	2	CM_Nil38	135	5	4	0			0		
DON	2	CM_Nil38	135	6							
DON	2	CM_Nil38	135	7							
DON	2	CM_Nil38	135	8							
DON	2	CM_Nil38	135	9							
DON	2	CM_Nil38	135	10							
DON	2	Remus	136	1	6	2	8		8		
DON	2	Remus	136	2	7	2			2		
DON	2	Remus	136	3	7	2	4		2	4	
DON	2	Remus	136	4	7	2	3		2	6	
DON	2	Remus	136	5	7	2			2	5	
DON	2	Remus	136	6							
DON	2	Remus	136	7							
DON	2	Remus	136	8							
DON	2	Remus	136	9							
DON	2	Remus	136	10							
DON	2	5485	137	1	4	0.5			0.5		
DON	2	5485	137	2	5	0			0		
DON	2	5485	137	3	5	0			0?		
DON	2	5485	137	4	5	0	0.5		0	0.5	
DON	2	5485	137	5	5	0			0		
DON	2	5485	137	6							
DON	2	5485	137	7							
DON	2	5485	137	8							
DON	2	5485	137	9							
DON	2	5485	137	10							
DON	2	5108	138	1	5	0			0	1	
DON	2	5108	138	2	6	0			0		
DON	2	5108	138	3	6	0			0		
DON	2	5108	138	4	6	0	1		0	1	
DON	2	5108	138	5	7	0			0		
DON	2	5108	138	6	7	0			0		
DON	2	5108	138	7							
DON	2	5108	138	8							
DON	2	5108	138	9							
DON	2	5108	138	10							
DON	2	1745	139	1	6	0			0		
DON	2	1745	139	2	6	0			0		
DON	2	1745	139	3	7	0			0		
DON	2	1745	139	4	7	0			0		
DON	2	1745	139	5	7	0			0		
DON	2	1745	139	6	7	0			0		
DON	2	1745	139	7							
DON	2	1745	139	8							
DON	2	1745	139	9							
DON	2	1745	139	10							
DON	2	5527	140	1	8	0			0		
DON	2	5527	140	2	8	0			0		
DON	2	5527	140	3	9	0			0		
DON	2	5527	140	4	9	0			0		
DON	2	5527	140	5	9	0			0		
DON	2	5527	140	6	11	3			3		
DON	2	5527	140	7	11	0b			0b		
DON	2	5527	140	8	11	0b			0b		
DON	2	5527	140	9	13	0			0	2?	
DON	2	5527	140	10							
DON	2	1882	141	1	4	2.5			2.5		
DON	2	1882	141	2	5	2b			b		
DON	2	1882	141	3	5	2			1.5		
DON	2	1882	141	4	6	2b			0		
DON	2	1882	141	5	7	2b			0		
DON	2	1882	141	6							
DON	2	1882	141	7							
DON	2	1882	141	8							
DON	2	1882	141	9							

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	1882	141	10							
DON	2	3060	142	1	6	0			0		
DON	2	3060	142	2	6	0			0		
DON	2	3060	142	3	6	0			0		
DON	2	3060	142	4	7	0			0		
DON	2	3060	142	5	8	0			0		
DON	2	3060	142	6							
DON	2	3060	142	7							
DON	2	3060	142	8							
DON	2	3060	142	9							
DON	2	3060	142	10							
DON	2	3219	143	1	6	0			0		
DON	2	3219	143	2	6	0			0		
DON	2	3219	143	3	6	0			0		
DON	2	3219	143	4	7	0			0		
DON	2	3219	143	5	7	0			0		
DON	2	3219	143	6							
DON	2	3219	143	7							
DON	2	3219	143	8							
DON	2	3219	143	9							
DON	2	3219	143	10							
DON	2	3144	144	1	6	0			0		
DON	2	3144	144	2	6	0			0		
DON	2	3144	144	3	6	0			0		
DON	2	3144	144	4	7	0			0		
DON	2	3144	144	5	7	0			0		
DON	2	3144	144	6							
DON	2	3144	144	7							
DON	2	3144	144	8							
DON	2	3144	144	9							
DON	2	3144	144	10							
DON	2	2119	145	1	6	0			0		
DON	2	2119	145	2	7	0			0		
DON	2	2119	145	3	7	0			0		
DON	2	2119	145	4	8	0			0		
DON	2	2119	145	5	8	0			0		
DON	2	2119	145	6	10	0			0.5		
DON	2	2119	145	7							
DON	2	2119	145	8							
DON	2	2119	145	9							
DON	2	2119	145	10							
DON	2	5863	146	1	4	5			6		
DON	2	5863	146	2	5	3	5		4	3	
DON	2	5863	146	3	5	3	2		3	2	
DON	2	5863	146	4	5	8			8		
DON	2	5863	146	5	5	8			8		
DON	2	5863	146	6							
DON	2	5863	146	7							
DON	2	5863	146	8							
DON	2	5863	146	9							
DON	2	5863	146	10							
DON	2	1903	147	1	5	3			6	1	
DON	2	1903	147	2	5	0			6	2	
DON	2	1903	147	3	5	0	2		4	2	
DON	2	1903	147	4	5	0	5		4	2	
DON	2	1903	147	5	5	2	3		4	2	
DON	2	1903	147	6							
DON	2	1903	147	7							
DON	2	1903	147	8							
DON	2	1903	147	9							
DON	2	1903	147	10							
DON	2	721_Ubi	148	1	6	0			0		
DON	2	721_Ubi	148	2	6	0?			0		
DON	2	721_Ubi	148	3	7	0			0		
DON	2	721_Ubi	148	4	7	0			0		
DON	2	721_Ubi	148	5	7	0			0	2	

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	721_Ubi	148	6	8	0			0		
DON	2	721_Ubi	148	7							
DON	2	721_Ubi	148	8							
DON	2	721_Ubi	148	9							
DON	2	721_Ubi	148	10							
DON	2	481_Ubi	149	1	5	0			0		
DON	2	481_Ubi	149	2	6	0			0		
DON	2	481_Ubi	149	3	7	0			0		
DON	2	481_Ubi	149	4	7	0			0		
DON	2	481_Ubi	149	5	8	0			0		
DON	2	481_Ubi	149	6							
DON	2	481_Ubi	149	7							
DON	2	481_Ubi	149	8							
DON	2	481_Ubi	149	9							
DON	2	481_Ubi	149	10							
DON	2	3632_Glyco	150	1	3	0			0		
DON	2	3632_Glyco	150	2	5	0			0		
DON	2	3632_Glyco	150	3	5	0			0		
DON	2	3632_Glyco	150	4	5	0			0		
DON	2	3632_Glyco	150	5	6	0			0		
DON	2	3632_Glyco	150	6	6	0			0		
DON	2	3632_Glyco	150	7	8	0			0	2	
DON	2	3632_Glyco	150	8							
DON	2	3632_Glyco	150	9							
DON	2	3632_Glyco	150	10							
DON	2	869_Ubi	151	1	4	1			1b	3	
DON	2	869_Ubi	151	2	6	0			0		
DON	2	869_Ubi	151	3	6	0			0		
DON	2	869_Ubi	151	4	6	0			0		
DON	2	869_Ubi	151	5	7	0			0		
DON	2	869_Ubi	151	6							
DON	2	869_Ubi	151	7							
DON	2	869_Ubi	151	8							
DON	2	869_Ubi	151	9							
DON	2	869_Ubi	151	10							
DON	2	3093_Glyco	152	1	4	0			0		
DON	2	3093_Glyco	152	2	4	0			0		
DON	2	3093_Glyco	152	3	5	0			0	5	
DON	2	3093_Glyco	152	4	5	0	2		0	7	
DON	2	3093_Glyco	152	5	5	0			6	1	
DON	2	3093_Glyco	152	6							
DON	2	3093_Glyco	152	7							
DON	2	3093_Glyco	152	8							
DON	2	3093_Glyco	152	9							
DON	2	3093_Glyco	152	10							
DON	2	1254_Ubi	153	1	5	0			0		
DON	2	1254_Ubi	153	2	5	0			0		
DON	2	1254_Ubi	153	3	5	0			0		
DON	2	1254_Ubi	153	4	5	0			0		
DON	2	1254_Ubi	153	5	6	0			0		
DON	2	1254_Ubi	153	6							
DON	2	1254_Ubi	153	7							
DON	2	1254_Ubi	153	8							
DON	2	1254_Ubi	153	9							
DON	2	1254_Ubi	153	10							
DON	2	1047_Ubi	154	1	5	0			0		
DON	2	1047_Ubi	154	2	6	0			0		
DON	2	1047_Ubi	154	3	6	0			0		
DON	2	1047_Ubi	154	4	6	0			0		
DON	2	1047_Ubi	154	5	7	0			0		
DON	2	1047_Ubi	154	6							
DON	2	1047_Ubi	154	7							
DON	2	1047_Ubi	154	8							
DON	2	1047_Ubi	154	9							
DON	2	1047_Ubi	154	10							
DON	2	3045_Ubi	155	1	5	0			3		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	3045_Ubi	155	2	5	0	1		0	9?	
DON	2	3045_Ubi	155	3	6	0			0		
DON	2	3045_Ubi	155	4	6	0			0		
DON	2	3045_Ubi	155	5	6	0			0		
DON	2	3045_Ubi	155	6							
DON	2	3045_Ubi	155	7							
DON	2	3045_Ubi	155	8							
DON	2	3045_Ubi	155	9							
DON	2	3045_Ubi	155	10							
DON	2	2259	156	1	8	0			0		
DON	2	2259	156	2	9	0			0	3	
DON	2	2259	156	3	9	0			0	2	
DON	2	2259	156	4	9	0			0		
DON	2	2259	156	5	10	0			0		
DON	2	2259	156	6	10	0			0		
DON	2	2259	156	7	11	0			0	2	
DON	2	2259	156	8							
DON	2	2259	156	9							
DON	2	2259	156	10							
DON	2	182_Ubi	157	1	6	0			0		
DON	2	182_Ubi	157	2	7	0			0		
DON	2	182_Ubi	157	3	7	0			0		
DON	2	182_Ubi	157	4	8	0			0		
DON	2	182_Ubi	157	5	9	0			0		
DON	2	182_Ubi	157	6							
DON	2	182_Ubi	157	7							
DON	2	182_Ubi	157	8							
DON	2	182_Ubi	157	9							
DON	2	182_Ubi	157	10							
DON	2	342_Ubi	158	1	3	0			0		
DON	2	342_Ubi	158	2	6	0			0		
DON	2	342_Ubi	158	3	6	0			0		
DON	2	342_Ubi	158	4	6	0			0		
DON	2	342_Ubi	158	5	8	0			0		
DON	2	342_Ubi	158	6							
DON	2	342_Ubi	158	7							
DON	2	342_Ubi	158	8							
DON	2	342_Ubi	158	9							
DON	2	342_Ubi	158	10							
DON	2	8892_Ca	159	1	6	0			0		
DON	2	8892_Ca	159	2	6	0			0		
DON	2	8892_Ca	159	3	6	0			0		
DON	2	8892_Ca	159	4	6	0			0		
DON	2	8892_Ca	159	5	6	0			0		
DON	2	8892_Ca	159	6							
DON	2	8892_Ca	159	7							
DON	2	8892_Ca	159	8							
DON	2	8892_Ca	159	9							
DON	2	8892_Ca	159	10							
DON	2	3028_Ca	160	1	7	0			0		
DON	2	3028_Ca	160	2	7	0			0		
DON	2	3028_Ca	160	3	10	0			0		
DON	2	3028_Ca	160	4							
DON	2	3028_Ca	160	5							
DON	2	3028_Ca	160	6							
DON	2	3028_Ca	160	7							
DON	2	3028_Ca	160	8							
DON	2	3028_Ca	160	9							
DON	2	3028_Ca	160	10							
DON	2	1325_Ubi	161	1	6	0			0		
DON	2	1325_Ubi	161	2	7	0			0		
DON	2	1325_Ubi	161	3	7	0			0		
DON	2	1325_Ubi	161	4	7	0			0		
DON	2	1325_Ubi	161	5	7	0			0		
DON	2	1325_Ubi	161	6	7	0			0		
DON	2	1325_Ubi	161	7	8	0			0		

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	1325_Ubi	161	8	8	0			0		
DON	2	1325_Ubi	161	9							
DON	2	1325_Ubi	161	10							
DON	2	1632_Ubi	162	1	5	0			0		
DON	2	1632_Ubi	162	2	5	0	0.5		2	2	
DON	2	1632_Ubi	162	3	6	0			0		
DON	2	1632_Ubi	162	4	7	0			0		
DON	2	1632_Ubi	162	5	7	0			0		
DON	2	1632_Ubi	162	6							
DON	2	1632_Ubi	162	7							
DON	2	1632_Ubi	162	8							
DON	2	1632_Ubi	162	9							
DON	2	1632_Ubi	162	10							
DON	2	1914_Ca	163	1	3	0			0		
DON	2	1914_Ca	163	2	4	0			0		
DON	2	1914_Ca	163	3	4	0			0	2	
DON	2	1914_Ca	163	4	5	0	2		6		
DON	2	1914_Ca	163	5	6	0			0		
DON	2	1914_Ca	163	6	6	0			0		
DON	2	1914_Ca	163	7	9	0			0 Tb		
DON	2	1914_Ca	163	8							
DON	2	1914_Ca	163	9							
DON	2	1914_Ca	163	10							
DON	2	6293_Ca	164	1	4	0	0.5		0	2	
DON	2	6293_Ca	164	2	5	0			0	3	
DON	2	6293_Ca	164	3	5	0	2		0	2	
DON	2	6293_Ca	164	4	5	0	0.5		0	3	
DON	2	6293_Ca	164	5	6	0			0		
DON	2	6293_Ca	164	6	6	0			0		
DON	2	6293_Ca	164	7							
DON	2	6293_Ca	164	8							
DON	2	6293_Ca	164	9							
DON	2	6293_Ca	164	10							
DON	2	1616_Glyco	165	1	4	0			0		
DON	2	1616_Glyco	165	2	4	0			0	2	
DON	2	1616_Glyco	165	3	5	0			0		
DON	2	1616_Glyco	165	4	5	0	7		4	1	
DON	2	1616_Glyco	165	5	6	0			0		
DON	2	1616_Glyco	165	6							
DON	2	1616_Glyco	165	7							
DON	2	1616_Glyco	165	8							
DON	2	1616_Glyco	165	9							
DON	2	1616_Glyco	165	10							
DON	2	1369_Glyco	166	1	5	0	7		6	1	
DON	2	1369_Glyco	166	2	6	0			0		
DON	2	1369_Glyco	166	3	6	0			0		
DON	2	1369_Glyco	166	4	7	0			0		
DON	2	1369_Glyco	166	5	7	0			0		
DON	2	1369_Glyco	166	6							
DON	2	1369_Glyco	166	7							
DON	2	1369_Glyco	166	8							
DON	2	1369_Glyco	166	9							
DON	2	1369_Glyco	166	10							
DON	2	1345_Glyco	167	1	4	0			0		
DON	2	1345_Glyco	167	2	5	0			0		
DON	2	1345_Glyco	167	3	5	0			0?		
DON	2	1345_Glyco	167	4	6	0			0		
DON	2	1345_Glyco	167	5	6	0			0		
DON	2	1345_Glyco	167	6							
DON	2	1345_Glyco	167	7							
DON	2	1345_Glyco	167	8							
DON	2	1345_Glyco	167	9							
DON	2	1345_Glyco	167	10							
DON	2	1148_Glyco	168	1	5	0			0		
DON	2	1148_Glyco	168	2	5	0			0		
DON	2	1148_Glyco	168	3	6	0			0		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	1148_Glyco	168	4	6	0			0		
DON	2	1148_Glyco	168	5	7	0			0		
DON	2	1148_Glyco	168	6	8	0			0		
DON	2	1148_Glyco	168	7							
DON	2	1148_Glyco	168	8							
DON	2	1148_Glyco	168	9							
DON	2	1148_Glyco	168	10							
DON	2	738_Glyco	169	1	3	0			0		
DON	2	738_Glyco	169	2	3	0			0		
DON	2	738_Glyco	169	3	3	0.5			0.5		
DON	2	738_Glyco	169	4	4	0			0		
DON	2	738_Glyco	169	5	4	0			0		
DON	2	738_Glyco	169	6							
DON	2	738_Glyco	169	7							
DON	2	738_Glyco	169	8							
DON	2	738_Glyco	169	9							
DON	2	738_Glyco	169	10							
DON	2	98_Ca	170	1	9	0			0		
DON	2	98_Ca	170	2	11	0			0		
DON	2	98_Ca	170	3	12	0			0		
DON	2	98_Ca	170	4							
DON	2	98_Ca	170	5							
DON	2	98_Ca	170	6							
DON	2	98_Ca	170	7							
DON	2	98_Ca	170	8							
DON	2	98_Ca	170	9							
DON	2	98_Ca	170	10							
DON	2	940_Glyco	171	1	6	0			0		
DON	2	940_Glyco	171	2	6	0			0		
DON	2	940_Glyco	171	3	6	0			0		
DON	2	940_Glyco	171	4	6	0			0		
DON	2	940_Glyco	171	5	7	0			0		
DON	2	940_Glyco	171	6							
DON	2	940_Glyco	171	7							
DON	2	940_Glyco	171	8							
DON	2	940_Glyco	171	9							
DON	2	940_Glyco	171	10							
DON	2	177	172	1	6	2			2		
DON	2	177	172	2	6	2*0,5			2		
DON	2	177	172	3	6	2*0,5			2		
DON	2	177	172	4	6	1	1		1		
DON	2	177	172	5	7	2	1		2		
DON	2	177	172	6							
DON	2	177	172	7							
DON	2	177	172	8							
DON	2	177	172	9							
DON	2	177	172	10							
DON	2	784_Ca	173	1	5	0			0		
DON	2	784_Ca	173	2	6	0			2b		
DON	2	784_Ca	173	3	7	0			0		
DON	2	784_Ca	173	4	8	0.5			0		
DON	2	784_Ca	173	5	8	0	0.5		0		
DON	2	784_Ca	173	6							
DON	2	784_Ca	173	7							
DON	2	784_Ca	173	8							
DON	2	784_Ca	173	9							
DON	2	784_Ca	173	10							
DON	2	GA_3B_18_16	174	1	5	0			0		
DON	2	GA_3B_18_16	174	2	5	0			5	1	
DON	2	GA_3B_18_16	174	3	5	0	1		2	1	
DON	2	GA_3B_18_16	174	4	6	0	1		0	2	
DON	2	GA_3B_18_16	174	5	6	0			0		
DON	2	GA_3B_18_16	174	6							
DON	2	GA_3B_18_16	174	7							
DON	2	GA_3B_18_16	174	8							
DON	2	GA_3B_18_16	174	9							

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	GA_3B_18_16	174	10							
DON	2	GA_3B_18_1	175	1	4	3			4		
DON	2	GA_3B_18_1	175	2	5	2.5			2	1	
DON	2	GA_3B_18_1	175	3	5	6			6		
DON	2	GA_3B_18_1	175	4	5	2			2		
DON	2	GA_3B_18_1	175	5	5	2			2	1	
DON	2	GA_3B_18_1	175	6							
DON	2	GA_3B_18_1	175	7							
DON	2	GA_3B_18_1	175	8							
DON	2	GA_3B_18_1	175	9							
DON	2	GA_3B_18_1	175	10							
DON	2	1111_Glyco	176	1	5	0			2		
DON	2	1111_Glyco	176	2	5	0	2		0	2	
DON	2	1111_Glyco	176	3	6	0			0		
DON	2	1111_Glyco	176	4	7	0			0		
DON	2	1111_Glyco	176	5	7	0			0		
DON	2	1111_Glyco	176	6							
DON	2	1111_Glyco	176	7							
DON	2	1111_Glyco	176	8							
DON	2	1111_Glyco	176	9							
DON	2	1111_Glyco	176	10							
DON	2	GA_3B_18_3	177	1	5	7			7		
DON	2	GA_3B_18_3	177	2	5	8			8		
DON	2	GA_3B_18_3	177	3	6	2	4		8		
DON	2	GA_3B_18_3	177	4	6	4	4		8		
DON	2	GA_3B_18_3	177	5	7	2	5		3	6	
DON	2	GA_3B_18_3	177	6	7	2	1		3		
DON	2	GA_3B_18_3	177	7							
DON	2	GA_3B_18_3	177	8							
DON	2	GA_3B_18_3	177	9							
DON	2	GA_3B_18_3	177	10							
DON	2	2203_Ca	178	1	6	0			0		
DON	2	2203_Ca	178	2	7	0			0		
DON	2	2203_Ca	178	3	7	0			0		
DON	2	2203_Ca	178	4	7	0			0		
DON	2	2203_Ca	178	5	7	0			0		
DON	2	2203_Ca	178	6		0			0		
DON	2	2203_Ca	178	7							
DON	2	2203_Ca	178	8							
DON	2	2203_Ca	178	9							
DON	2	2203_Ca	178	10							
DON	2	8406_Ca	179	1	6	0			0		
DON	2	8406_Ca	179	2	6	0			0		
DON	2	8406_Ca	179	3	7	0			0		
DON	2	8406_Ca	179	4	7	0			0		
DON	2	8406_Ca	179	5	9	0			0		
DON	2	8406_Ca	179	6							
DON	2	8406_Ca	179	7							
DON	2	8406_Ca	179	8							
DON	2	8406_Ca	179	9							
DON	2	8406_Ca	179	10							
DON	2	GA_3B_18_15	180	1	6	0			0		
DON	2	GA_3B_18_15	180	2	6	0			0		
DON	2	GA_3B_18_15	180	3	7	0			0		
DON	2	GA_3B_18_15	180	4	7	0			0		
DON	2	GA_3B_18_15	180	5	7	0			0		
DON	2	GA_3B_18_15	180	6							
DON	2	GA_3B_18_15	180	7							
DON	2	GA_3B_18_15	180	8							
DON	2	GA_3B_18_15	180	9							
DON	2	GA_3B_18_15	180	10							
DON	2	723_Glyco	181	1	6	0			0		
DON	2	723_Glyco	181	2	7	0			0		
DON	2	723_Glyco	181	3	7	0			0		
DON	2	723_Glyco	181	4	7	0			0.5		
DON	2	723_Glyco	181	5	8	0			0		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	723_Glyco	181	6							
DON	2	723_Glyco	181	7							
DON	2	723_Glyco	181	8							
DON	2	723_Glyco	181	9							
DON	2	723_Glyco	181	10							
DON	2	3746_Ubi	182	1	9	0			0		
DON	2	3746_Ubi	182	2	9	0			0		
DON	2	3746_Ubi	182	3	9	0			0		
DON	2	3746_Ubi	182	4	10	0			0		
DON	2	3746_Ubi	182	5	11	0			0		
DON	2	3746_Ubi	182	6							
DON	2	3746_Ubi	182	7							
DON	2	3746_Ubi	182	8							
DON	2	3746_Ubi	182	9							
DON	2	3746_Ubi	182	10							
DON	2	219_Ca	183	1	6	0			0		
DON	2	219_Ca	183	2	6	0			0		
DON	2	219_Ca	183	3	7	0			0		
DON	2	219_Ca	183	4	7	0			0		
DON	2	219_Ca	183	5	7	0			0		
DON	2	219_Ca	183	6							
DON	2	219_Ca	183	7							
DON	2	219_Ca	183	8							
DON	2	219_Ca	183	9							
DON	2	219_Ca	183	10							
DON	2	7919_no_1_Hyd	184	1	6	0			0		
DON	2	7919_no_1_Hyd	184	2	7	0			0b		
DON	2	7919_no_1_Hyd	184	3	7	0			0b		
DON	2	7919_no_1_Hyd	184	4	7	0			0b		
DON	2	7919_no_1_Hyd	184	5	8	0b			0		
DON	2	7919_no_1_Hyd	184	6	8	0b			0		
DON	2	7919_no_1_Hyd	184	7	10	0			0b		
DON	2	7919_no_1_Hyd	184	8							
DON	2	7919_no_1_Hyd	184	9							
DON	2	7919_no_1_Hyd	184	10							
DON	2	256	185	1	8	2	4		2	2	
DON	2	256	185	2	8	2	2		3	1	
DON	2	256	185	3	10	2	1		2	1	
DON	2	256	185	4	11	2			2		
DON	2	256	185	5	12	3			3		
DON	2	256	185	6	12	2			3		
DON	2	256	185	7	13	4			4		
DON	2	256	185	8	13	2			2		
DON	2	256	185	9	13	4			4		
DON	2	256	185	10	14	3			3		
DON	2	1399	186	1	6	1	3		2		
DON	2	1399	186	2	6	2			2	5	
DON	2	1399	186	3	6	6			3		
DON	2	1399	186	4	7	5			2		
DON	2	1399	186	5	7	3			3		
DON	2	1399	186	6	8	8			8		
DON	2	1399	186	7	10	4	1		4	1	
DON	2	1399	186	8							
DON	2	1399	186	9							
DON	2	1399	186	10							
DON	2	GA_3B_18_22	187	1	5	0			0.5		
DON	2	GA_3B_18_22	187	2	6	0			0		
DON	2	GA_3B_18_22	187	3	6	0			0		
DON	2	GA_3B_18_22	187	4	6	3			3		
DON	2	GA_3B_18_22	187	5	6	0			0		
DON	2	GA_3B_18_22	187	6							
DON	2	GA_3B_18_22	187	7							
DON	2	GA_3B_18_22	187	8							
DON	2	GA_3B_18_22	187	9							
DON	2	GA_3B_18_22	187	10							
DON	2	GA_3B_18_9	188	1	6	4	1		3	5	

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	GA_3B_18_9	188	2	6	2	2		8		
DON	2	GA_3B_18_9	188	3	7	4	1		8		
DON	2	GA_3B_18_9	188	4	7	2			2		
DON	2	GA_3B_18_9	188	5	7	4	2		8		
DON	2	GA_3B_18_9	188	6							
DON	2	GA_3B_18_9	188	7							
DON	2	GA_3B_18_9	188	8							
DON	2	GA_3B_18_9	188	9							
DON	2	GA_3B_18_9	188	10							
DON	2	Wangshuibai	189	1	5	0			0		
DON	2	Wangshuibai	189	2	5	0			0		
DON	2	Wangshuibai	189	3	6	0			0		
DON	2	Wangshuibai	189	4	6	1	1		0	2	
DON	2	Wangshuibai	189	5	6	0	0.5		0	0.5	
DON	2	Wangshuibai	189	6							
DON	2	Wangshuibai	189	7							
DON	2	Wangshuibai	189	8							
DON	2	Wangshuibai	189	9							
DON	2	Wangshuibai	189	10							
DON	2	Sumai3_Ung1	190	1	6	2			2		
DON	2	Sumai3_Ung1	190	2	6	2			2		
DON	2	Sumai3_Ung1	190	3	7	0			0		
DON	2	Sumai3_Ung1	190	4	7	0			0		
DON	2	Sumai3_Ung1	190	5	7	0	2		0	2	
DON	2	Sumai3_Ung1	190	6							
DON	2	Sumai3_Ung1	190	7							
DON	2	Sumai3_Ung1	190	8							
DON	2	Sumai3_Ung1	190	9							
DON	2	Sumai3_Ung1	190	10							
DON	2	Nobeokabozu_U	191	1	6	0			0.5		
DON	2	Nobeokabozu_U	191	2	7	0			0		
DON	2	Nobeokabozu_U	191	3	8	0	2?		0	3?	
DON	2	Nobeokabozu_U	191	4	8	0			0		
DON	2	Nobeokabozu_U	191	5	9	0			0		
DON	2	Nobeokabozu_U	191	6							
DON	2	Nobeokabozu_U	191	7							
DON	2	Nobeokabozu_U	191	8							
DON	2	Nobeokabozu_U	191	9							
DON	2	Nobeokabozu_U	191	10							
DON	2	Sumai3_MX	192	1	3	0			0	2	
DON	2	Sumai3_MX	192	2	3	0			0	2	
DON	2	Sumai3_MX	192	3	4	0.5			2b		
DON	2	Sumai3_MX	192	4	6	3	5		2		
DON	2	Sumai3_MX	192	5	6	2			0	2	
DON	2	Sumai3_MX	192	6							
DON	2	Sumai3_MX	192	7							
DON	2	Sumai3_MX	192	8							
DON	2	Sumai3_MX	192	9							
DON	2	Sumai3_MX	192	10							
DON	2	Ning_7840	193	1	5	0			0		
DON	2	Ning_7840	193	2	5	0			0		
DON	2	Ning_7840	193	3	5	0			0		
DON	2	Ning_7840	193	4	5	0			0		
DON	2	Ning_7840	193	5	6	0			0		
DON	2	Ning_7840	193	6							
DON	2	Ning_7840	193	7							
DON	2	Ning_7840	193	8							
DON	2	Ning_7840	193	9							
DON	2	Ning_7840	193	10							
DON	2	ND_2710	194	1	3	2			2		
DON	2	ND_2710	194	2	3	2			2b		
DON	2	ND_2710	194	3	3	2			2b		
DON	2	ND_2710	194	4	4	2			2b		
DON	2	ND_2710	194	5	4	3			3b		
DON	2	ND_2710	194	6							
DON	2	ND_2710	194	7							

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
DON	2	ND_2710	194	8							
DON	2	ND_2710	194	9							
DON	2	ND_2710	194	10							
DON	2	W14	195	1	4	0			0,5b		
DON	2	W14	195	2	4	1			1b		
DON	2	W14	195	3	4	2			2b		
DON	2	W14	195	4	6	0			0		
DON	2	W14	195	5	6	0			0		
DON	2	W14	195	6							
DON	2	W14	195	7							
DON	2	W14	195	8							
DON	2	W14	195	9							
DON	2	W14	195	10							
DON	2	RH_CM_83	196	1	4	3	4		5	2	
DON	2	RH_CM_83	196	2	4	2			2		
DON	2	RH_CM_83	196	3	4	3			3		
DON	2	RH_CM_83	196	4	5	10			8	2	
DON	2	RH_CM_83	196	5	7	0	5		6		
DON	2	RH_CM_83	196	6							
DON	2	RH_CM_83	196	7							
DON	2	RH_CM_83	196	8							
DON	2	RH_CM_83	196	9							
DON	2	RH_CM_83	196	10							
DON	2	RH_CM_651	197	1	6	2	2		2		
DON	2	RH_CM_651	197	2	6	5	3		8		
DON	2	RH_CM_651	197	3	6	6	2		8		
DON	2	RH_CM_651	197	4	7	4	2		8		
DON	2	RH_CM_651	197	5	7	4			4		
DON	2	RH_CM_651	197	6	8	4			4		
DON	2	RH_CM_651	197	7							
DON	2	RH_CM_651	197	8							
DON	2	RH_CM_651	197	9							
DON	2	RH_CM_651	197	10							
DON	2	RH_CM_643	198	1	4	8			8		
DON	2	RH_CM_643	198	2	4	3			2.5		
DON	2	RH_CM_643	198	3	4	3	5		3	5	
DON	2	RH_CM_643	198	4	5	8			4	2	
DON	2	RH_CM_643	198	5	5	0	5		6	1	
DON	2	RH_CM_643	198	6							
DON	2	RH_CM_643	198	7							
DON	2	RH_CM_643	198	8							
DON	2	RH_CM_643	198	9							
DON	2	RH_CM_643	198	10							
Fus	1	6293_Ca	199	1	5	2			2.5		
Fus	1	6293_Ca	199	2	5	2			2		
Fus	1	6293_Ca	199	3	5	2			2		
Fus	1	6293_Ca	199	4	5	2			2		
Fus	1	6293_Ca	199	5	6	2			2		
Fus	1	6293_Ca	199	6	7	2			2		
Fus	1	6293_Ca	199	7	8	2			2		
Fus	1	6293_Ca	199	8	8	2			2		
Fus	1	6293_Ca	199	9	10	2			2		
Fus	1	6293_Ca	199	10							
Fus	1	8892_Ca	200	1	6	2			2		
Fus	1	8892_Ca	200	2	6	2			2		
Fus	1	8892_Ca	200	3	8	2			2		
Fus	1	8892_Ca	200	4	8	2			2		
Fus	1	8892_Ca	200	5	8	2			2		
Fus	1	8892_Ca	200	6	9	2			2		
Fus	1	8892_Ca	200	7	9	2			2		
Fus	1	8892_Ca	200	8	9	2			2		
Fus	1	8892_Ca	200	9							
Fus	1	8892_Ca	200	10							
Fus	1	RH_CM_643	201	1	6	2			3		
Fus	1	RH_CM_643	201	2	6	8			20		
Fus	1	RH_CM_643	201	3	6	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	RH_CM_643	201	4	6	8			10		
Fus	1	RH_CM_643	201	5	7	5			3		
Fus	1	RH_CM_643	201	6	9	2			2		
Fus	1	RH_CM_643	201	7	9	2			2		
Fus	1	RH_CM_643	201	8	9	3			3		
Fus	1	RH_CM_643	201	9							
Fus	1	RH_CM_643	201	10							
Fus	1	RH_CM_83	202	1	5	8			12		
Fus	1	RH_CM_83	202	2	5	8			6		
Fus	1	RH_CM_83	202	3	6	10			14		
Fus	1	RH_CM_83	202	4	8	2			3		
Fus	1	RH_CM_83	202	5							
Fus	1	RH_CM_83	202	6							
Fus	1	RH_CM_83	202	7							
Fus	1	RH_CM_83	202	8							
Fus	1	RH_CM_83	202	9							
Fus	1	RH_CM_83	202	10							
Fus	1	CM_Nil38	203	1	4	2			2		
Fus	1	CM_Nil38	203	2	4	2*0,5			2		
Fus	1	CM_Nil38	203	3	5	2			2		
Fus	1	CM_Nil38	203	4	5	2			2		
Fus	1	CM_Nil38	203	5	5	2			2		
Fus	1	CM_Nil38	203	6	6	2			2		
Fus	1	CM_Nil38	203	7	6	2			2		
Fus	1	CM_Nil38	203	8	6	2			2		
Fus	1	CM_Nil38	203	9	6	2			2		
Fus	1	CM_Nil38	203	10	6	2			2		
Fus	1	Remus	204	1	7	18			20		
Fus	1	Remus	204	2	8	18			18		
Fus	1	Remus	204	3	8	20			20		
Fus	1	Remus	204	4	9	20			20		
Fus	1	Remus	204	5	9	18			18		
Fus	1	Remus	204	6	9	7			16		
Fus	1	Remus	204	7	10	20			20		
Fus	1	Remus	204	8	11	8			12		
Fus	1	Remus	204	9	12	2			2		
Fus	1	Remus	204	10							
Fus	1	CM_82036	205	1	6	2			2		
Fus	1	CM_82036	205	2	7	2			2		
Fus	1	CM_82036	205	3	7	2			2		
Fus	1	CM_82036	205	4	7	2			2		
Fus	1	CM_82036	205	5	7	2			2		
Fus	1	CM_82036	205	6	8	2			2		
Fus	1	CM_82036	205	7	8	2			2		
Fus	1	CM_82036	205	8	9	2			2		
Fus	1	CM_82036	205	9	9	2			2		
Fus	1	CM_82036	205	10	10	2			2		
Fus	1	CM_Nil43	206	1	5	2			2		
Fus	1	CM_Nil43	206	2	5	2			2		
Fus	1	CM_Nil43	206	3	6	2*0,5			2		
Fus	1	CM_Nil43	206	4	6	2			2		
Fus	1	CM_Nil43	206	5	6	2*0,5			2		
Fus	1	CM_Nil43	206	6	6	1.5			2		
Fus	1	CM_Nil43	206	7	7	2			2		
Fus	1	CM_Nil43	206	8	7	2			2		
Fus	1	CM_Nil43	206	9	8	2			2		
Fus	1	CM_Nil43	206	10							
Fus	1	CM_Nil47	207	1	4	10			16		
Fus	1	CM_Nil47	207	2	4	12			15		
Fus	1	CM_Nil47	207	3	5	2			3		
Fus	1	CM_Nil47	207	4	5	4			4		
Fus	1	CM_Nil47	207	5	6	6			10		
Fus	1	CM_Nil47	207	6	6	8			16		
Fus	1	CM_Nil47	207	7	6	2			2		
Fus	1	CM_Nil47	207	8	6	7			8		
Fus	1	CM_Nil47	207	9	7	3			5		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	CM_Nil47	207	10	7	2			2		
Fus	1	RH_CM_651	208	1	7	20			20		
Fus	1	RH_CM_651	208	2	8	14			20		
Fus	1	RH_CM_651	208	3	8	18			20		
Fus	1	RH_CM_651	208	4	11	20			20		
Fus	1	RH_CM_651	208	5							
Fus	1	RH_CM_651	208	6							
Fus	1	RH_CM_651	208	7							
Fus	1	RH_CM_651	208	8							
Fus	1	RH_CM_651	208	9							
Fus	1	RH_CM_651	208	10							
Fus	1	CM_Nil51	209	1	4	8			14		
Fus	1	CM_Nil51	209	2	4	4			18		
Fus	1	CM_Nil51	209	3	4	12			18		
Fus	1	CM_Nil51	209	4	5	20			20		
Fus	1	CM_Nil51	209	5	5	18			18		
Fus	1	CM_Nil51	209	6	5	16			16		
Fus	1	CM_Nil51	209	7	6	2			2		
Fus	1	CM_Nil51	209	8	6	6			14		
Fus	1	CM_Nil51	209	9	6	2			2		
Fus	1	CM_Nil51	209	10	6	2			2		
Fus	1	CM_Nil51	209	11	6	6			18		
Fus	1	7919_no_1_Hyd	210	1	5	2			2		
Fus	1	7919_no_1_Hyd	210	2	6	2			2		
Fus	1	7919_no_1_Hyd	210	3	6	2			2		
Fus	1	7919_no_1_Hyd	210	4	7	2			2		
Fus	1	7919_no_1_Hyd	210	5	7	2			2		
Fus	1	7919_no_1_Hyd	210	6	7	2			2		
Fus	1	7919_no_1_Hyd	210	7	9	2			2		
Fus	1	7919_no_1_Hyd	210	8							
Fus	1	7919_no_1_Hyd	210	9							
Fus	1	7919_no_1_Hyd	210	10							
Fus	1	1903	211	1	5	2			2		
Fus	1	1903	211	2	5	2			2		
Fus	1	1903	211	3	5	2			2		
Fus	1	1903	211	4	6	2			2		
Fus	1	1903	211	5	6	2			2		
Fus	1	1903	211	6	6	2			2.5		
Fus	1	1903	211	7	6	2			2		
Fus	1	1903	211	8	6	2			2		
Fus	1	1903	211	9	7	2			2		
Fus	1	1903	211	10	7	2			2		
Fus	1	1903	211	11	7	2			2		
Fus	1	1903	211	12	8	2			2		
Fus	1	1399	212	1	5	2			3		
Fus	1	1399	212	2	6	2			2		
Fus	1	1399	212	3	6	2			2		
Fus	1	1399	212	4	6	2			2		
Fus	1	1399	212	5	6	2			2		
Fus	1	1399	212	6	7	2			3		
Fus	1	1399	212	7	7	2			2		
Fus	1	1399	212	8	8	2			2		
Fus	1	1399	212	9	8	3			3		
Fus	1	1399	212	10							
Fus	1	256	213	1	8	2			2		
Fus	1	256	213	2	8	2			2		
Fus	1	256	213	3	8	2			2		
Fus	1	256	213	4	8	8			8		
Fus	1	256	213	5	8	4			9		
Fus	1	256	213	6	9	4			4		
Fus	1	256	213	7	11	2			2		
Fus	1	256	213	8	11	8			8		
Fus	1	256	213	9							
Fus	1	256	213	10							
Fus	1	177	214	1	6	18			20		
Fus	1	177	214	2	8	18			20		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	177	214	3	11	12			12		
Fus	1	177	214	4	11	8			12		
Fus	1	177	214	5	11	8			18		
Fus	1	177	214	6	11	10			20		
Fus	1	177	214	7							
Fus	1	177	214	8							
Fus	1	177	214	9							
Fus	1	177	214	10							
Fus	1	1155_mutant	215	1	6	2			2		
Fus	1	1155_mutant	215	2	7	2			2		
Fus	1	1155_mutant	215	3	8	2			2		
Fus	1	1155_mutant	215	4	8	2			2		
Fus	1	1155_mutant	215	5	9	0			0		
Fus	1	1155_mutant	215	6	9	2			2		
Fus	1	1155_mutant	215	7	9	0			0		
Fus	1	1155_mutant	215	8	11	2			2		
Fus	1	1155_mutant	215	9							
Fus	1	1155_mutant	215	10							
Fus	1	1155_no_mutation	216	1	8	2			2		
Fus	1	1155_no_mutation	216	2	11	2			2		
Fus	1	1155_no_mutation	216	3	11	2			2		
Fus	1	1155_no_mutation	216	4	11	2			2		
Fus	1	1155_no_mutation	216	5	9	0			0		
Fus	1	1155_no_mutation	216	6	9	0			0		
Fus	1	1155_no_mutation	216	7							
Fus	1	1155_no_mutation	216	8							
Fus	1	1155_no_mutation	216	9							
Fus	1	1155_no_mutation	216	10							
Fus	1	219_Ca	217	1	5	2			2		
Fus	1	219_Ca	217	2	5	2			1.5		
Fus	1	219_Ca	217	3	6	2			2		
Fus	1	219_Ca	217	4	7	1.5			2		
Fus	1	219_Ca	217	5	7	2			2		
Fus	1	219_Ca	217	6	9	2			2		
Fus	1	219_Ca	217	7							
Fus	1	219_Ca	217	8							
Fus	1	219_Ca	217	9							
Fus	1	219_Ca	217	10							
Fus	1	784_Ca	218	1	4	2			2		
Fus	1	784_Ca	218	2	5	2			2		
Fus	1	784_Ca	218	3	5	2			2		
Fus	1	784_Ca	218	4	7	2			2		
Fus	1	784_Ca	218	5	8	2			2		
Fus	1	784_Ca	218	6	8	2			2		
Fus	1	784_Ca	218	7							
Fus	1	784_Ca	218	8							
Fus	1	784_Ca	218	9							
Fus	1	784_Ca	218	10							
Fus	1	2203_Ca	219	1	5	2			2		
Fus	1	2203_Ca	219	2	5	2			2		
Fus	1	2203_Ca	219	3	6	2			2		
Fus	1	2203_Ca	219	4							
Fus	1	2203_Ca	219	5							
Fus	1	2203_Ca	219	6							
Fus	1	2203_Ca	219	7							
Fus	1	2203_Ca	219	8							
Fus	1	2203_Ca	219	9							
Fus	1	2203_Ca	219	10							
Fus	1	8406_Ca	220	1	3	2			2		
Fus	1	8406_Ca	220	2	5	2			2*0,5		
Fus	1	8406_Ca	220	3	5	2			2		
Fus	1	8406_Ca	220	4	6	2*0,5			2		
Fus	1	8406_Ca	220	5	8	2			2		
Fus	1	8406_Ca	220	6	8	2			2		
Fus	1	8406_Ca	220	7	8	2			2		
Fus	1	8406_Ca	220	8	9	2			2		

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	8406_Ca	220	9	11	2			2		
Fus	1	8406_Ca	220	10	11	2			2		
Fus	1	3028_Ca	221	1	3	2			2		
Fus	1	3028_Ca	221	2	5	2			1.5		
Fus	1	3028_Ca	221	3	5	2			2		
Fus	1	3028_Ca	221	4	5	2			2		
Fus	1	3028_Ca	221	5	5	2			2		
Fus	1	3028_Ca	221	6	5	0			0		
Fus	1	3028_Ca	221	7							
Fus	1	3028_Ca	221	8							
Fus	1	3028_Ca	221	9							
Fus	1	3028_Ca	221	10							
Fus	1	721_Ubi	222	1	5	2			2		
Fus	1	721_Ubi	222	2	5	2			2		
Fus	1	721_Ubi	222	3	6	1			2		
Fus	1	721_Ubi	222	4	6	2			2		
Fus	1	721_Ubi	222	5	8	2			2		
Fus	1	721_Ubi	222	6	8	2			2		
Fus	1	721_Ubi	222	7							
Fus	1	721_Ubi	222	8							
Fus	1	721_Ubi	222	9							
Fus	1	721_Ubi	222	10							
Fus	1	869_Ubi	223	1	5	2			2		
Fus	1	869_Ubi	223	2	5	2			2		
Fus	1	869_Ubi	223	3	6	2			2		
Fus	1	869_Ubi	223	4	9	2			2		
Fus	1	869_Ubi	223	5	9	2			2		
Fus	1	869_Ubi	223	6	9	2			2		
Fus	1	869_Ubi	223	7	10	2			2		
Fus	1	869_Ubi	223	8							
Fus	1	869_Ubi	223	9							
Fus	1	869_Ubi	223	10							
Fus	1	1345_Glyco	224	1	3	7			18		
Fus	1	1345_Glyco	224	2	3	3			14		
Fus	1	1345_Glyco	224	3	3	2			2		
Fus	1	1345_Glyco	224	4	4	2			2		
Fus	1	1345_Glyco	224	5	5	2			2		
Fus	1	1345_Glyco	224	6	5	2			2		
Fus	1	1345_Glyco	224	7	6	2			2		
Fus	1	1345_Glyco	224	8	6	4			5		
Fus	1	1345_Glyco	224	9	6	2			2		
Fus	1	1345_Glyco	224	10	7	2			2		
Fus	1	1148_Glyco	225	1	5	2			2		
Fus	1	1148_Glyco	225	2	6	1.5			2		
Fus	1	1148_Glyco	225	3	6	1.5			2		
Fus	1	1148_Glyco	225	4	6	2			2		
Fus	1	1148_Glyco	225	5	6	1.5			2		
Fus	1	1148_Glyco	225	6	6	-			2		
Fus	1	1148_Glyco	225	7	7	2			2		
Fus	1	1148_Glyco	225	8	8	2			2		
Fus	1	1148_Glyco	225	9	8	2			2		
Fus	1	1148_Glyco	225	10	8	2			2		
Fus	1	1111_Glyco	226	1	6	2			2		
Fus	1	1111_Glyco	226	2	6	2			2		
Fus	1	1111_Glyco	226	3	6	2			2		
Fus	1	1111_Glyco	226	4	6	2			2		
Fus	1	1111_Glyco	226	5	6	2			2		
Fus	1	1111_Glyco	226	6	7	2			2		
Fus	1	1111_Glyco	226	7	7	2			2		
Fus	1	1111_Glyco	226	8	8	2			2		
Fus	1	1111_Glyco	226	9							
Fus	1	1111_Glyco	226	10							
Fus	1	738_Glyco	227	1	3	2			2		
Fus	1	738_Glyco	227	2	3	2			2		
Fus	1	738_Glyco	227	3	3	2			2		
Fus	1	738_Glyco	227	4	4	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	738_Glyco	227	5	6	2			2		
Fus	1	738_Glyco	227	6	6	2			2		
Fus	1	738_Glyco	227	7	6	2			2		
Fus	1	738_Glyco	227	8	6	2			2		
Fus	1	738_Glyco	227	9	6	2			2		
Fus	1	738_Glyco	227	10	5	2			2		
Fus	1	940_Glyco	228	1	4	2			2		
Fus	1	940_Glyco	228	2	4	2			2		
Fus	1	940_Glyco	228	3	4	2			2		
Fus	1	940_Glyco	228	4	5	2			2		
Fus	1	940_Glyco	228	5	5	2			2		
Fus	1	940_Glyco	228	6	6	2			2		
Fus	1	940_Glyco	228	7	6	2			2		
Fus	1	940_Glyco	228	8	7	2			2		
Fus	1	940_Glyco	228	9	7	2			2		
Fus	1	940_Glyco	228	10	8	2			2		
Fus	1	940_Glyco	228	11	8	2			2		
Fus	1	1616_Glyco	229	1	4	2			2		
Fus	1	1616_Glyco	229	2	5	2			2		
Fus	1	1616_Glyco	229	3	6	2			2		
Fus	1	1616_Glyco	229	4	6	2			2		
Fus	1	1616_Glyco	229	5	6	2			2		
Fus	1	1616_Glyco	229	6	9	0			0		
Fus	1	1616_Glyco	229	7							
Fus	1	1616_Glyco	229	8							
Fus	1	1616_Glyco	229	9							
Fus	1	1616_Glyco	229	10							
Fus	1	1369_Glyco	230	1	5	2			2		
Fus	1	1369_Glyco	230	2	5	2			2		
Fus	1	1369_Glyco	230	3	5	2			2		
Fus	1	1369_Glyco	230	4	6	2			2		
Fus	1	1369_Glyco	230	5	6	2			2		
Fus	1	1369_Glyco	230	6	6	2			2		
Fus	1	1369_Glyco	230	7	7	2			2		
Fus	1	1369_Glyco	230	8	7	2			2		
Fus	1	1369_Glyco	230	9	7	2			2		
Fus	1	1369_Glyco	230	10	8	2			2		
Fus	1	1914_Ca	231	1	3	2			2		
Fus	1	1914_Ca	231	2	3	2			2		
Fus	1	1914_Ca	231	3	3	1			1		
Fus	1	1914_Ca	231	4	3	2			2		
Fus	1	1914_Ca	231	5	4	2			2		
Fus	1	1914_Ca	231	6	4	2			2		
Fus	1	1914_Ca	231	7	5	2			2		
Fus	1	1914_Ca	231	8	5	2			2		
Fus	1	1914_Ca	231	9	5	2			2		
Fus	1	1914_Ca	231	10	5	2			2		
Fus	1	1914_Ca	231	11	6	2			2		
Fus	1	723_Glyco	232	1	4	8			20		
Fus	1	723_Glyco	232	2	4	2			2		
Fus	1	723_Glyco	232	3	4	3			3		
Fus	1	723_Glyco	232	4	5	2			2		
Fus	1	723_Glyco	232	5	5	2			2		
Fus	1	723_Glyco	232	6	6	2			2		
Fus	1	723_Glyco	232	7	6	2			2		
Fus	1	723_Glyco	232	8	6	2			2		
Fus	1	723_Glyco	232	9	7	2			2		
Fus	1	723_Glyco	232	10	7	2			2		
Fus	1	Sumai3_Ung1	233	1	5	2			2		
Fus	1	Sumai3_Ung1	233	2	6	2			2		
Fus	1	Sumai3_Ung1	233	3	6	2			2		
Fus	1	Sumai3_Ung1	233	4	6	2			2		
Fus	1	Sumai3_Ung1	233	5	6	2			2		
Fus	1	Sumai3_Ung1	233	6	6	2			2		
Fus	1	Sumai3_Ung1	233	7	6	2			2		
Fus	1	Sumai3_Ung1	233	8							

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	Sumai3_Ung1	233	9							
Fus	1	Sumai3_Ung1	233	10							
Fus	1	481_Ubi*	234	1	5	2			2		
Fus	1	481_Ubi*	234	2	5	2.5			3		
Fus	1	481_Ubi*	234	3	5	3			4		
Fus	1	481_Ubi*	234	4	6	2			2		
Fus	1	481_Ubi*	234	5	6	1.5			2		
Fus	1	481_Ubi*	234	6	7	2			2		
Fus	1	481_Ubi*	234	7							
Fus	1	481_Ubi*	234	8							
Fus	1	481_Ubi*	234	9							
Fus	1	481_Ubi*	234	10							
Fus	1	342_Ubi	235	1	3	14			16		
Fus	1	342_Ubi	235	2	3	20			20		
Fus	1	342_Ubi	235	3	3	18			20		
Fus	1	342_Ubi	235	4	5	18			18		
Fus	1	342_Ubi	235	5	6	20			20		
Fus	1	342_Ubi	235	6	6	18			20		
Fus	1	342_Ubi	235	7	6	20			20		
Fus	1	342_Ubi	235	8							
Fus	1	342_Ubi	235	9							
Fus	1	342_Ubi	235	10							
Fus	1	182_Ubi	236	1	6	2			2		
Fus	1	182_Ubi	236	2	6	2			2		
Fus	1	182_Ubi	236	3	7	2			2		
Fus	1	182_Ubi	236	4	7	2			2		
Fus	1	182_Ubi	236	5	7	2			2		
Fus	1	182_Ubi	236	6	8	2			2		
Fus	1	182_Ubi	236	7	8	1			2		
Fus	1	182_Ubi	236	8							
Fus	1	182_Ubi	236	9							
Fus	1	182_Ubi	236	10							
Fus	1	3632_Glyco	237	1	5	2			2		
Fus	1	3632_Glyco	237	2	5	2			2		
Fus	1	3632_Glyco	237	3	6	0.5			0.5		
Fus	1	3632_Glyco	237	4	6	0.5			0.5		
Fus	1	3632_Glyco	237	5	6	0.5			0.5		
Fus	1	3632_Glyco	237	6	6	0			0		
Fus	1	3632_Glyco	237	7	8	0.5			2		
Fus	1	3632_Glyco	237	8	9	1			2		
Fus	1	3632_Glyco	237	9							
Fus	1	3632_Glyco	237	10							
Fus	1	3093_Glyco	238	1	3	2			2		
Fus	1	3093_Glyco	238	2	3	2			2		
Fus	1	3093_Glyco	238	3	3	1			1		
Fus	1	3093_Glyco	238	4	3	2			2		
Fus	1	3093_Glyco	238	5	4	2			2		
Fus	1	3093_Glyco	238	6	5	2			2		
Fus	1	3093_Glyco	238	7	5	2			2		
Fus	1	3093_Glyco	238	8	5	2			2		
Fus	1	3093_Glyco	238	9	5	2			2		
Fus	1	3093_Glyco	238	10	5	2			2		
Fus	1	1632_Ubi	239	1	4	2			3		
Fus	1	1632_Ubi	239	2	5	2			2		
Fus	1	1632_Ubi	239	3	5	2			7		
Fus	1	1632_Ubi	239	4	6	2			2		
Fus	1	1632_Ubi	239	5	6	2			2		
Fus	1	1632_Ubi	239	6	8	2			2		
Fus	1	1632_Ubi	239	7	8	2			2		
Fus	1	1632_Ubi	239	8							
Fus	1	1632_Ubi	239	9							
Fus	1	1632_Ubi	239	10							
Fus	1	1325_Ubi	240	1	4	2			2		
Fus	1	1325_Ubi	240	2	4	2			2		
Fus	1	1325_Ubi	240	3	5	2			2		
Fus	1	1325_Ubi	240	4	5	2			2		

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	1325_Ubi	240	5	5	2			2		
Fus	1	1325_Ubi	240	6	5	2			2		
Fus	1	1325_Ubi	240	7	6	2			2		
Fus	1	1325_Ubi	240	8	6	2			2		
Fus	1	1325_Ubi	240	9	6	2			2		
Fus	1	1325_Ubi	240	10	8	2			2		
Fus	1	1254_Ubi	241	1	2	2*0,5			2		
Fus	1	1254_Ubi	241	2	4	2			2		
Fus	1	1254_Ubi	241	3	4	2*0,5			2		
Fus	1	1254_Ubi	241	4	4	0			0		
Fus	1	1254_Ubi	241	5	4	2			2		
Fus	1	1254_Ubi	241	6	5	2			2		
Fus	1	1254_Ubi	241	7	6	1			2		
Fus	1	1254_Ubi	241	8	6	2			2		
Fus	1	1254_Ubi	241	9							
Fus	1	1254_Ubi	241	10							
Fus	1	1047_Ubi	242	1	4	2			2		
Fus	1	1047_Ubi	242	2	4	2			2		
Fus	1	1047_Ubi	242	3	5	2			2		
Fus	1	1047_Ubi	242	4	5	2			2		
Fus	1	1047_Ubi	242	5	5	2			2		
Fus	1	1047_Ubi	242	6	6	2			2		
Fus	1	1047_Ubi	242	7	6	1.5			2		
Fus	1	1047_Ubi	242	8							
Fus	1	1047_Ubi	242	9							
Fus	1	1047_Ubi	242	10							
Fus	1	3045_Ubi	243	1	5	2			2		
Fus	1	3045_Ubi	243	2	5	2			2		
Fus	1	3045_Ubi	243	3	5	2			2		
Fus	1	3045_Ubi	243	4	5	2			2		
Fus	1	3045_Ubi	243	5	6	0.5			2		
Fus	1	3045_Ubi	243	6	6	2*0,5			2		
Fus	1	3045_Ubi	243	7	6	0.5			2		
Fus	1	3045_Ubi	243	8	6	2			2		
Fus	1	3045_Ubi	243	9	6	1.5			2		
Fus	1	3045_Ubi	243	10							
Fus	1	3746_Ubi	244	1	5	2			2		
Fus	1	3746_Ubi	244	2	7	2			2		
Fus	1	3746_Ubi	244	3	7	2			2		
Fus	1	3746_Ubi	244	4	8	2			2		
Fus	1	3746_Ubi	244	5	8	2			2		
Fus	1	3746_Ubi	244	6	11	2			2		
Fus	1	3746_Ubi	244	7							
Fus	1	3746_Ubi	244	8							
Fus	1	3746_Ubi	244	9							
Fus	1	3746_Ubi	244	10							
Fus	1	Nobeokabozu_U	245	1	3	2			2		
Fus	1	Nobeokabozu_U	245	2	3	2			2		
Fus	1	Nobeokabozu_U	245	3	3	3			3		
Fus	1	Nobeokabozu_U	245	4	4	2			2		
Fus	1	Nobeokabozu_U	245	5	4	2			2		
Fus	1	Nobeokabozu_U	245	6	4	2			2		
Fus	1	Nobeokabozu_U	245	7	4	2			2		
Fus	1	Nobeokabozu_U	245	8	5	2			2		
Fus	1	Nobeokabozu_U	245	9	5	1			1		
Fus	1	Nobeokabozu_U	245	10							
Fus	1	Wangshuibai	246	1	2	2			2		
Fus	1	Wangshuibai	246	2	2	2			2		
Fus	1	Wangshuibai	246	3	2	2			2		
Fus	1	Wangshuibai	246	4	2	2			2		
Fus	1	Wangshuibai	246	5	3	2			2		
Fus	1	Wangshuibai	246	6	3	2			2		
Fus	1	Wangshuibai	246	7	3	2			2		
Fus	1	Wangshuibai	246	8	3	2			2		
Fus	1	Wangshuibai	246	9	3	2			2		
Fus	1	Wangshuibai	246	10	5	0			0		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	Sumai3_MX	247	1	2	2			2		
Fus	1	Sumai3_MX	247	2	2	2			2		
Fus	1	Sumai3_MX	247	3	3	2			2		
Fus	1	Sumai3_MX	247	4	3	2			2		
Fus	1	Sumai3_MX	247	5	3	1			1		
Fus	1	Sumai3_MX	247	6	4	2			2		
Fus	1	Sumai3_MX	247	7	4	2			2		
Fus	1	Sumai3_MX	247	8	4	2			2		
Fus	1	Sumai3_MX	247	9	4	2			5		
Fus	1	Sumai3_MX	247	10	5	2			2		
Fus	1	Sumai3_MX	247	11	6	2			2		
Fus	1	3060	248	1	5	2			2		
Fus	1	3060	248	2	6	2			2		
Fus	1	3060	248	3	6	2			2		
Fus	1	3060	248	4	7	2			2		
Fus	1	3060	248	5	7	2			3		
Fus	1	3060	248	6	8	2			2		
Fus	1	3060	248	7	8	2			2		
Fus	1	3060	248	8							
Fus	1	3060	248	9							
Fus	1	3060	248	10							
Fus	1	5485	249	1	3	2			2		
Fus	1	5485	249	2	3	2			2		
Fus	1	5485	249	3	3	1			1		
Fus	1	5485	249	4	3	2			2		
Fus	1	5485	249	5	4	2			2		
Fus	1	5485	249	6	5	2			2		
Fus	1	5485	249	7	6	2.5			2.5		
Fus	1	5485	249	8							
Fus	1	5485	249	9							
Fus	1	5485	249	10							
Fus	1	2119	250	1	6	2			2		
Fus	1	2119	250	2	7	2			2		
Fus	1	2119	250	3	7	2			2		
Fus	1	2119	250	4	8	2			2		
Fus	1	2119	250	5	8	2			2		
Fus	1	2119	250	6	8	2			10		
Fus	1	2119	250	7	9	2			2		
Fus	1	2119	250	8	9	2			2		
Fus	1	2119	250	9	9	2			2		
Fus	1	2119	250	10							
Fus	1	5527	251	1	6	3			3		
Fus	1	5527	251	2	7	3			8		
Fus	1	5527	251	3	8	2.5			3		
Fus	1	5527	251	4	8	2			2		
Fus	1	5527	251	5	8	2			2		
Fus	1	5527	251	6	9	18			20		
Fus	1	5527	251	7	9	3			3		
Fus	1	5527	251	8	9	2			2		
Fus	1	5527	251	9	10	2			2		
Fus	1	5527	251	10	10	3			3		
Fus	1	2259	252	1	6	2			2		
Fus	1	2259	252	2	7	2.5			2.5		
Fus	1	2259	252	3	7	2			2		
Fus	1	2259	252	4	7	18			20		
Fus	1	2259	252	5	7	16			20		
Fus	1	2259	252	6	7	12			16		
Fus	1	2259	252	7	8	20			20		
Fus	1	2259	252	8	8	2			2		
Fus	1	2259	252	9	8	2			3		
Fus	1	2259	252	10	9	2			2		
Fus	1	5108	253	1	5	2			2		
Fus	1	5108	253	2	6	3			3		
Fus	1	5108	253	3	6	2			2		
Fus	1	5108	253	4	6	2			2		
Fus	1	5108	253	5	6	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	5108	253	6	7	2			2		
Fus	1	5108	253	7	8	2			2		
Fus	1	5108	253	8	8	2			2		
Fus	1	5108	253	9							
Fus	1	5108	253	10							
Fus	1	3144	254	1	6	2			2		
Fus	1	3144	254	2	6	2			2		
Fus	1	3144	254	3	6	1.5			2		
Fus	1	3144	254	4	6	2			2		
Fus	1	3144	254	5	7	2			2		
Fus	1	3144	254	6	7	2			2		
Fus	1	3144	254	7	7	2			2		
Fus	1	3144	254	8	7	1.5			2		
Fus	1	3144	254	9	8	2			2		
Fus	1	3144	254	10	8	2			2		
Fus	1	3144	254	11	9	2			2		
Fus	1	1882	263	1	3	10			18		
Fus	1	1882	263	2	3	10			20		
Fus	1	1882	263	3	3	10			18		
Fus	1	1882	263	4	3	12			16		
Fus	1	1882	263	5	4	10			16		
Fus	1	1882	263	6	4	3			3		
Fus	1	1882	263	7	4	8			20		
Fus	1	1882	263	8	5	4			10		
Fus	1	1882	263	9	5	3			3		
Fus	1	1882	263	10	6	2			2		
Fus	1	1745	264	1	5	2			2		
Fus	1	1745	264	2	5	2			1.5		
Fus	1	1745	264	3	6	2			2		
Fus	1	1745	264	4	6	1.5			2		
Fus	1	1745	264	5	6	1.5			2		
Fus	1	1745	264	6	6	2			2		
Fus	1	1745	264	7	6	2			2		
Fus	1	1745	264	8	7	1.5			2		
Fus	1	1745	264	9	7	2			2		
Fus	1	1745	264	10	8	2			2		
Fus	1	3219	265	1	5	2			2		
Fus	1	3219	265	2	6	2			2		
Fus	1	3219	265	3	6	2			2		
Fus	1	3219	265	4	6	2			2		
Fus	1	3219	265	5	6	2			2		
Fus	1	3219	265	6	7	2			2		
Fus	1	3219	265	7	8	2			2		
Fus	1	3219	265	8	8	3			3		
Fus	1	3219	265	9	8	2			2		
Fus	1	3219	265	10	8	2			2		
Fus	1	5863	266	1	4	12			18		
Fus	1	5863	266	2	4	12			14		
Fus	1	5863	266	3	5	18			18		
Fus	1	5863	266	4	5	2			2		
Fus	1	5863	266	5	5	2.5			10		
Fus	1	5863	266	6	5	2			2		
Fus	1	5863	266	7	6	3			3		
Fus	1	5863	266	8	6	2			2		
Fus	1	5863	266	9	6	2			2		
Fus	1	5863	266	10	8	8			20		
Fus	1	ND_2710	267	1	1	2			2		
Fus	1	ND_2710	267	2	1	2			2		
Fus	1	ND_2710	267	3	1	2			2		
Fus	1	ND_2710	267	4	2	3			3		
Fus	1	ND_2710	267	5	3	2			2		
Fus	1	ND_2710	267	6	3	2			2		
Fus	1	ND_2710	267	7	3	2*0,5			1.5		
Fus	1	ND_2710	267	8	3	2			2		
Fus	1	ND_2710	267	9	3	2			2		
Fus	1	ND_2710	267	10	3	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	1	ND_2710	267	11	3	2			2		
Fus	1	Ning_7840	268	1	3	2			2		
Fus	1	Ning_7840	268	2	3	3			4		
Fus	1	Ning_7840	268	3	3	3			10		
Fus	1	Ning_7840	268	4	3	2			2.5		
Fus	1	Ning_7840	268	5	4	0.5			0.5		
Fus	1	Ning_7840	268	6	4	2			2		
Fus	1	Ning_7840	268	7	5	2			2		
Fus	1	Ning_7840	268	8	5	2			2		
Fus	1	Ning_7840	268	9	5	2			2		
Fus	1	Ning_7840	268	10	5	2			2		
Fus	1	W14	269	1	2	5			16		
Fus	1	W14	269	2	3	2			2		
Fus	1	W14	269	3	3	2			2		
Fus	1	W14	269	4	3	2			2		
Fus	1	W14	269	5	3	2			2		
Fus	1	W14	269	6	4	1			1		
Fus	1	W14	269	7							
Fus	1	W14	269	8							
Fus	1	W14	269	9							
Fus	1	W14	269	10							
Fus	2	RH_CM_651	304	1	3	16			14		
Fus	2	RH_CM_651	304	2	4	20			20		
Fus	2	RH_CM_651	304	3	4	20			20		
Fus	2	RH_CM_651	304	4	4	18			18		
Fus	2	RH_CM_651	304	5	5	14			14		
Fus	2	RH_CM_651	304	6	5	20			20		
Fus	2	RH_CM_651	304	7	5	20			20		
Fus	2	RH_CM_651	304	8	5	16			16		
Fus	2	RH_CM_651	304	9	6	15			16		
Fus	2	RH_CM_651	304	10	7	10			14		
Fus	2	CM_82036	305	1	3	2			2		
Fus	2	CM_82036	305	2	4	2			2		
Fus	2	CM_82036	305	3	5	2			2		
Fus	2	CM_82036	305	4	5	2			2		
Fus	2	CM_82036	305	5	5	2			2		
Fus	2	CM_82036	305	6	5	2			2		
Fus	2	CM_82036	305	7	6	2			2		
Fus	2	CM_82036	305	8	6	2.5			2.5		
Fus	2	CM_82036	305	9	6	2			2		
Fus	2	CM_82036	305	10	7	2			2		
Fus	2	CM_Nil51	306	1	3	14			20		
Fus	2	CM_Nil51	306	2	3	14			20		
Fus	2	CM_Nil51	306	3	3	8			10		
Fus	2	CM_Nil51	306	4	3	2			2		
Fus	2	CM_Nil51	306	5	3	2			2		
Fus	2	CM_Nil51	306	6	4	12			18		
Fus	2	CM_Nil51	306	7	4	6			7		
Fus	2	CM_Nil51	306	8	4	8			8		
Fus	2	CM_Nil51	306	9	5	2			2		
Fus	2	CM_Nil51	306	10	5	8			13		
Fus	2	CM_Nil47	307	1	3	10			18		
Fus	2	CM_Nil47	307	2	3	18			20		
Fus	2	CM_Nil47	307	3	3	20			20		
Fus	2	CM_Nil47	307	4	3	12			18		
Fus	2	CM_Nil47	307	5	3	12			18		
Fus	2	CM_Nil47	307	6	3	3			8		
Fus	2	CM_Nil47	307	7	3	18			20		
Fus	2	CM_Nil47	307	8	4	5			5		
Fus	2	CM_Nil47	307	9	4	12			14		
Fus	2	CM_Nil47	307	10	5	16			18		
Fus	2	RH_CM_643	308	1	3	14			20		
Fus	2	RH_CM_643	308	2	5	18			20		
Fus	2	RH_CM_643	308	3	5	2			2		
Fus	2	RH_CM_643	308	4	5	10			18		
Fus	2	RH_CM_643	308	5	5	18			18		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	RH_CM_643	308	6	5	2			7		
Fus	2	RH_CM_643	308	7	5	14			18		
Fus	2	RH_CM_643	308	8	5	2			7		
Fus	2	RH_CM_643	308	9	6	2			2		
Fus	2	RH_CM_643	308	10	7	2			2		
Fus	2	CM_Nil43	309	1	3	2			2		
Fus	2	CM_Nil43	309	2	4	2			2		
Fus	2	CM_Nil43	309	3	4	2			2		
Fus	2	CM_Nil43	309	4	4	2			2		
Fus	2	CM_Nil43	309	5	4	2			2		
Fus	2	CM_Nil43	309	6	5	2			2		
Fus	2	CM_Nil43	309	7	5	2			2		
Fus	2	CM_Nil43	309	8	5	2			2		
Fus	2	CM_Nil43	309	9	5	0			0.5		
Fus	2	CM_Nil43	309	10	6	2			2		
Fus	2	RH_CM_83	310	1	3	16			20		
Fus	2	RH_CM_83	310	2	4	2			2		
Fus	2	RH_CM_83	310	3	4	3			8		
Fus	2	RH_CM_83	310	4	4	8			8		
Fus	2	RH_CM_83	310	5	5	3			3		
Fus	2	RH_CM_83	310	6	5	4			6		
Fus	2	RH_CM_83	310	7	5	14			14		
Fus	2	RH_CM_83	310	8	5	3			3		
Fus	2	RH_CM_83	310	9	5	4			6		
Fus	2	RH_CM_83	310	10	6	2			2		
Fus	2	CM_Nil38	311	1	2	2			2		
Fus	2	CM_Nil38	311	2	2	2*0,5			2		
Fus	2	CM_Nil38	311	3	2	2			2		
Fus	2	CM_Nil38	311	4	3	0.5			2		
Fus	2	CM_Nil38	311	5	3	2			2		
Fus	2	CM_Nil38	311	6	3	2			2		
Fus	2	CM_Nil38	311	7	3	2			2		
Fus	2	CM_Nil38	311	8	3	2			2		
Fus	2	CM_Nil38	311	9	4	2			2		
Fus	2	CM_Nil38	311	10	4	2			2		
Fus	2	CM_Nil38	311	11	4	2			2		
Fus	2	CM_Nil38	311	12	5	2			2		
Fus	2	Remus	312	1	5	18			18		
Fus	2	Remus	312	2	5	14			18		
Fus	2	Remus	312	3	5	20			20		
Fus	2	Remus	312	4	5	18			18		
Fus	2	Remus	312	5	5	20			20		
Fus	2	Remus	312	6	6	18			20		
Fus	2	Remus	312	7	6	18			20		
Fus	2	Remus	312	8	7	2.5			4		
Fus	2	Remus	312	9	7	15			20		
Fus	2	Remus	312	10							
Fus	2	3093_Glyco	319	1	3	2			2		
Fus	2	3093_Glyco	319	2	3	2			2		
Fus	2	3093_Glyco	319	3	3	2			2		
Fus	2	3093_Glyco	319	4	3	2*0,5			0.5		
Fus	2	3093_Glyco	319	5	4	2			2		
Fus	2	3093_Glyco	319	6	4	2			2		
Fus	2	3093_Glyco	319	7	4	2			2		
Fus	2	3093_Glyco	319	8	5	2			2		
Fus	2	3093_Glyco	319	9	5	2			2		
Fus	2	3093_Glyco	319	10	5	2			3		
Fus	2	3093_Glyco	319	11	5	2			2		
Fus	2	1325_Ubi	320	1	3	2			2		
Fus	2	1325_Ubi	320	2	3	2			2		
Fus	2	1325_Ubi	320	3	4	2			2		
Fus	2	1325_Ubi	320	4	4	2			2		
Fus	2	1325_Ubi	320	5	4	2			2		
Fus	2	1325_Ubi	320	6	5	2			2		
Fus	2	1325_Ubi	320	7	5	2			2		
Fus	2	1325_Ubi	320	8	5	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	1325_Ubi	320	9	5	2			2		
Fus	2	1325_Ubi	320	10	6	2			2		
Fus	2	1325_Ubi	320	11	6	2			2		
Fus	2	5485	321	1	4	2			2		
Fus	2	5485	321	2	4	2			2		
Fus	2	5485	321	3	5	2			2		
Fus	2	5485	321	4	5	2			2		
Fus	2	5485	321	5	5	2			2		
Fus	2	5485	321	6	6	2			2		
Fus	2	5485	321	7	6	2			2		
Fus	2	5485	321	8	6	2			2		
Fus	2	5485	321	9	7	2			2		
Fus	2	5485	321	10							
Fus	2	3219	322	1	5	2			2		
Fus	2	3219	322	2	6	2			2		
Fus	2	3219	322	3	6	2			2		
Fus	2	3219	322	4	7	2			2		
Fus	2	3219	322	5	8	2			2		
Fus	2	3219	322	6	8	5			8		
Fus	2	3219	322	7	8	2			2		
Fus	2	3219	322	8							
Fus	2	3219	322	9							
Fus	2	3219	322	10							
Fus	2	5108	323	1	6	2			2		
Fus	2	5108	323	2	6	2			2		
Fus	2	5108	323	3	7	2			2		
Fus	2	5108	323	4	7	2			2		
Fus	2	5108	323	5	7	2			2		
Fus	2	5108	323	6	8	2			2		
Fus	2	5108	323	7	8	2			2		
Fus	2	5108	323	8	8	2			2		
Fus	2	5108	323	9	8	2			2		
Fus	2	5108	323	10							
Fus	2	2259	324	1	7	2			2		
Fus	2	2259	324	2	7	16			18		
Fus	2	2259	324	3	7	2			6		
Fus	2	2259	324	4	8	2			2		
Fus	2	2259	324	5	8	2			2		
Fus	2	2259	324	6	8	2			2		
Fus	2	2259	324	7	9	2			2		
Fus	2	2259	324	8	9	2			14		
Fus	2	2259	324	9	10	3			3		
Fus	2	2259	324	10							
Fus	2	3060	325	1	5	2			2		
Fus	2	3060	325	2	6	1			2		
Fus	2	3060	325	3	6	2			2		
Fus	2	3060	325	4	6	2			2		
Fus	2	3060	325	5	7	2			2		
Fus	2	3060	325	6	7	2			2		
Fus	2	3060	325	7	7	2			2		
Fus	2	3060	325	8	8	2			2		
Fus	2	3060	325	9							
Fus	2	3060	325	10							
Fus	2	1254_Ubi	326	1	3	2			2		
Fus	2	1254_Ubi	326	2	3	2			2		
Fus	2	1254_Ubi	326	3	3	2			2		
Fus	2	1254_Ubi	326	4	4	2			2		
Fus	2	1254_Ubi	326	5	4	2			2		
Fus	2	1254_Ubi	326	6	4	2			2		
Fus	2	1254_Ubi	326	7	4	1.5			1		
Fus	2	1254_Ubi	326	8	4	2			2		
Fus	2	1254_Ubi	326	9	5	2			2		
Fus	2	1254_Ubi	326	10	5	2			2		
Fus	2	1254_Ubi	326	11	5	2			2		
Fus	2	3045_Ubi	327	1	3	2			2		
Fus	2	3045_Ubi	327	2	3	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	3045_Ubi	327	3	3	2			2		
Fus	2	3045_Ubi	327	4	4	2			2		
Fus	2	3045_Ubi	327	5	4	2			2		
Fus	2	3045_Ubi	327	6	4	2			2		
Fus	2	3045_Ubi	327	7	4	2			2		
Fus	2	3045_Ubi	327	8	5	2			2		
Fus	2	3045_Ubi	327	9	5	2			2		
Fus	2	3045_Ubi	327	10	5	2			2		
Fus	2	1882	328	1	3	12			14		
Fus	2	1882	328	2	3	2			11		
Fus	2	1882	328	3	5	2			2		
Fus	2	1882	328	4	5	3			3		
Fus	2	1882	328	5	6	3			3		
Fus	2	1882	328	6	8	2			2		
Fus	2	1882	328	7	8	3			6		
Fus	2	1882	328	8	9	3			12		
Fus	2	1882	328	9	11	2			4		
Fus	2	1882	328	10	11	2			2		
Fus	2	5863	329	1	4	12			14		
Fus	2	5863	329	2	4	8			18		
Fus	2	5863	329	3	5	4			5		
Fus	2	5863	329	4	5	14			16		
Fus	2	5863	329	5	5	2			2		
Fus	2	5863	329	6	5	3			3		
Fus	2	5863	329	7	6	2			2		
Fus	2	5863	329	8	6	5			8		
Fus	2	5863	329	9	6	2			2		
Fus	2	5863	329	10	6	2			3		
Fus	2	5863	329	11	6	2			3		
Fus	2	5863	329	12	6	1			2		
Fus	2	1745	330	1	6	1.5			2		
Fus	2	1745	330	2	7	2			2		
Fus	2	1745	330	3	7	1.5			2		
Fus	2	1745	330	4	7	2			2		
Fus	2	1745	330	5	7	0.5			2		
Fus	2	1745	330	6	7	0.5			2		
Fus	2	1745	330	7	8	1			1		
Fus	2	1745	330	8	8	2			2		
Fus	2	1745	330	9	9	2			2		
Fus	2	1745	330	10							
Fus	2	5527_2	331	1	5	2			2		
Fus	2	5527_2	331	2	6	2			2		
Fus	2	5527_2	331	3	6	2			3		
Fus	2	5527_2	331	4	6	2			2		
Fus	2	5527_2	331	5	6	3			4		
Fus	2	5527_2	331	6	6	2			2		
Fus	2	5527_2	331	7	7	4			6		
Fus	2	5527_2	331	8	8	4			9		
Fus	2	5527_2	331	9	8	3			3		
Fus	2	5527_2	331	10	9	2			2		
Fus	2	5527_2	331	11	9	2			2		
Fus	2	5527_2	331	12	9	3			3		
Fus	2	481_Ubi*	332	1	5	2			2		
Fus	2	481_Ubi*	332	2	5	2			2		
Fus	2	481_Ubi*	332	3	5	2			2		
Fus	2	481_Ubi*	332	4	6	4			4		
Fus	2	481_Ubi*	332	5	6	1			1		
Fus	2	481_Ubi*	332	6	7	3			3		
Fus	2	481_Ubi*	332	7	7	1			1		
Fus	2	481_Ubi*	332	8	8	1			2		
Fus	2	481_Ubi*	332	9	9	0.5			0.5		
Fus	2	481_Ubi*	332	10							
Fus	2	182_Ubi	333	1	6	2			2		
Fus	2	182_Ubi	333	2	6	2			2		
Fus	2	182_Ubi	333	3	7	2			2		
Fus	2	182_Ubi	333	4	7	1.5			1		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	182_Ubi	333	5	7	2			2		
Fus	2	182_Ubi	333	6	7	1.5			2		
Fus	2	182_Ubi	333	7							
Fus	2	182_Ubi	333	8							
Fus	2	182_Ubi	333	9							
Fus	2	182_Ubi	333	10							
Fus	2	721_Ubi	334	1	6	2			2		
Fus	2	721_Ubi	334	2	6	1.5			2		
Fus	2	721_Ubi	334	3	6	1.5			1.5		
Fus	2	721_Ubi	334	4	7	2			2		
Fus	2	721_Ubi	334	5	8	2			2		
Fus	2	721_Ubi	334	6	8	2			2		
Fus	2	721_Ubi	334	7	9	2			2		
Fus	2	721_Ubi	334	8							
Fus	2	721_Ubi	334	9							
Fus	2	721_Ubi	334	10							
Fus	2	869_Ubi	335	1	5	2			2		
Fus	2	869_Ubi	335	2	5	2			2		
Fus	2	869_Ubi	335	3	6	1			2		
Fus	2	869_Ubi	335	4	7	2			2		
Fus	2	869_Ubi	335	5	7	2			2		
Fus	2	869_Ubi	335	6	8	2			2		
Fus	2	869_Ubi	335	7	8	2			2		
Fus	2	869_Ubi	335	8	8	2			2		
Fus	2	869_Ubi	335	9	8	2			2		
Fus	2	869_Ubi	335	10	9	2			2		
Fus	2	1632_Ubi	336	1	4	6			14		
Fus	2	1632_Ubi	336	2	4	2			2		
Fus	2	1632_Ubi	336	3	4	8			20		
Fus	2	1632_Ubi	336	4	5	2			2		
Fus	2	1632_Ubi	336	5	5	2			2		
Fus	2	1632_Ubi	336	6	5	2			10	w	
Fus	2	1632_Ubi	336	7	5	2			2		
Fus	2	1632_Ubi	336	8	5	2			2		
Fus	2	1632_Ubi	336	9	6	1			2		
Fus	2	1632_Ubi	336	10	6	2			2		
Fus	2	1632_Ubi	336	11	6	2			2		
Fus	2	1632_Ubi	336	12	8	2			2		
Fus	2	1369_Glyco	337	1	5	2			2		
Fus	2	1369_Glyco	337	2	6	2			2		
Fus	2	1369_Glyco	337	3	6	2			2		
Fus	2	1369_Glyco	337	4	6	2			2		
Fus	2	1369_Glyco	337	5	7	2			2		
Fus	2	1369_Glyco	337	6	7	2			2		
Fus	2	1369_Glyco	337	7	7	2			2		
Fus	2	1369_Glyco	337	8	7	2			2		
Fus	2	1369_Glyco	337	9							
Fus	2	1369_Glyco	337	10							
Fus	2	2119	338	1	6	2			2		
Fus	2	2119	338	2	6	2			2		
Fus	2	2119	338	3	7	2			2		
Fus	2	2119	338	4	7	2			2		
Fus	2	2119	338	5	8	2			2		
Fus	2	2119	338	6	8	2			2		
Fus	2	2119	338	7	8	2			2		
Fus	2	2119	338	8	9	2			2		
Fus	2	2119	338	9	9	2			2		
Fus	2	2119	338	10	9	2			2		
Fus	2	3144	339	1	4	2			2		
Fus	2	3144	339	2	5	2			2		
Fus	2	3144	339	3	6	0.5			2		
Fus	2	3144	339	4	7	2			2		
Fus	2	3144	339	5	8	2			2		
Fus	2	3144	339	6	8	2			2		
Fus	2	3144	339	7	8	2			2		
Fus	2	3144	339	8	9	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	3144	339	9							
Fus	2	3144	339	10							
Fus	2	1903	340	1	5	2			2		
Fus	2	1903	340	2	5	2			2		
Fus	2	1903	340	3	5	2			2		
Fus	2	1903	340	4	5	2			2		
Fus	2	1903	340	5	6	2			2		
Fus	2	1903	340	6	6	2			2		
Fus	2	1903	340	7	6	2			2		
Fus	2	1903	340	8	6	1.5			2		
Fus	2	1903	340	9	7	2			2		
Fus	2	1903	340	10							
Fus	2	3632_Glyco	341	1	4	2			2		
Fus	2	3632_Glyco	341	2	7	0.5			2		
Fus	2	3632_Glyco	341	3	8	6			8		
Fus	2	3632_Glyco	341	4	8	0.5			1		
Fus	2	3632_Glyco	341	5	8	0.5			0.5		
Fus	2	3632_Glyco	341	6	8	2			2		
Fus	2	3632_Glyco	341	7	8	3			8		
Fus	2	3632_Glyco	341	8							
Fus	2	3632_Glyco	341	9							
Fus	2	3632_Glyco	341	10							
Fus	2	1616_Glyco	342	1	5	2			2		
Fus	2	1616_Glyco	342	2	6	2			2		
Fus	2	1616_Glyco	342	3	7	2			2		
Fus	2	1616_Glyco	342	4	8	2			2		
Fus	2	1616_Glyco	342	5	8	2			2		
Fus	2	1616_Glyco	342	6							
Fus	2	1616_Glyco	342	7							
Fus	2	1616_Glyco	342	8							
Fus	2	1616_Glyco	342	9							
Fus	2	1616_Glyco	342	10							
Fus	2	1914_Ca	343	1	3	2			2		
Fus	2	1914_Ca	343	2	3	2*0,5			1		
Fus	2	1914_Ca	343	3	3	2			2		
Fus	2	1914_Ca	343	4	3	2			2		
Fus	2	1914_Ca	343	5	5	2			2		
Fus	2	1914_Ca	343	6	5	2			2		
Fus	2	1914_Ca	343	7	5	2			2		
Fus	2	1914_Ca	343	8	5	2			2		
Fus	2	1914_Ca	343	9	5	2			2		
Fus	2	1914_Ca	343	10	5	2			2		
Fus	2	8892_Ca	344	1	5	2			2		
Fus	2	8892_Ca	344	2	5	2			2		
Fus	2	8892_Ca	344	3	6	2			2		
Fus	2	8892_Ca	344	4	6	2			2		
Fus	2	8892_Ca	344	5	6	2			2		
Fus	2	8892_Ca	344	6	7	2			2		
Fus	2	8892_Ca	344	7	7	2			2		
Fus	2	8892_Ca	344	8	7	2			2		
Fus	2	8892_Ca	344	9	8	2			2		
Fus	2	8892_Ca	344	10	8	2			2		
Fus	2	3746_Ubi	345	1	5	2			2		
Fus	2	3746_Ubi	345	2	5	2			2		
Fus	2	3746_Ubi	345	3	6	2			2		
Fus	2	3746_Ubi	345	4	6	0.5			1		
Fus	2	3746_Ubi	345	5	6	2			2		
Fus	2	3746_Ubi	345	6	6	6			6		
Fus	2	3746_Ubi	345	7	7	2			2		
Fus	2	3746_Ubi	345	8	7	2			2		
Fus	2	3746_Ubi	345	9	8	3			3		
Fus	2	3746_Ubi	345	10	8	2			2		
Fus	2	1047_Ubi	346	1	4	2			2		
Fus	2	1047_Ubi	346	2	4	2			2		
Fus	2	1047_Ubi	346	3	4	2			2		
Fus	2	1047_Ubi	346	4	4	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	1047_Ubi	346	5	5	2			2		
Fus	2	1047_Ubi	346	6	5	2			2		
Fus	2	1047_Ubi	346	7	5	2			2		
Fus	2	1047_Ubi	346	8	6	1			2		
Fus	2	1047_Ubi	346	9							
Fus	2	1047_Ubi	346	10							
Fus	2	1111_Glyco	347	1	4	2			2		
Fus	2	1111_Glyco	347	2	4	2			2		
Fus	2	1111_Glyco	347	3	4	2			2		
Fus	2	1111_Glyco	347	4	5	2			2		
Fus	2	1111_Glyco	347	5	5	2			2		
Fus	2	1111_Glyco	347	6	5	2			2		
Fus	2	1111_Glyco	347	7	6	2			2		
Fus	2	1111_Glyco	347	8	6	1.5			2		
Fus	2	1111_Glyco	347	9	6	3			3		
Fus	2	1111_Glyco	347	10	6	1.5			2		
Fus	2	1111_Glyco	347	11	6	2			2		
Fus	2	1345_Glyco	348	1	5	2			2		
Fus	2	1345_Glyco	348	2	6	2			2		
Fus	2	1345_Glyco	348	3	6	2			2		
Fus	2	1345_Glyco	348	4	6	0.5			1		
Fus	2	1345_Glyco	348	5	6	1.5			2		
Fus	2	1345_Glyco	348	6	6	2			2		
Fus	2	1345_Glyco	348	7	8	5			10		
Fus	2	1345_Glyco	348	8							
Fus	2	1345_Glyco	348	9							
Fus	2	1345_Glyco	348	10							
Fus	2	1148_Glyco	349	1	4	5			6		
Fus	2	1148_Glyco	349	2	4	8			10		
Fus	2	1148_Glyco	349	3	4	2			2		
Fus	2	1148_Glyco	349	4	5	2			2		
Fus	2	1148_Glyco	349	5	6	1.5			2		
Fus	2	1148_Glyco	349	6	6	0.5			2		
Fus	2	1148_Glyco	349	7	6	3			2		
Fus	2	1148_Glyco	349	8	6	1			2		
Fus	2	1148_Glyco	349	9	6	1.5			2		
Fus	2	1148_Glyco	349	10	6	0.5			2		
Fus	2	1148_Glyco	349	11	6	2			2		
Fus	2	1148_Glyco	349	12	8	2			2		
Fus	2	738_Glyco	350	1	3	2			2		
Fus	2	738_Glyco	350	2	4	1.5			2		
Fus	2	738_Glyco	350	3	5	2			2		
Fus	2	738_Glyco	350	4	5	2			2		
Fus	2	738_Glyco	350	5	5	2			2		
Fus	2	738_Glyco	350	6	5	2			2		
Fus	2	738_Glyco	350	7	5	2			2		
Fus	2	738_Glyco	350	8	5	2			2		
Fus	2	738_Glyco	350	9	6	1.5			2		
Fus	2	738_Glyco	350	10	6	2			2		
Fus	2	738_Glyco	350	11	6	2			2		
Fus	2	940_Glyco	351	1	4	2			2		
Fus	2	940_Glyco	351	2	5	2			2		
Fus	2	940_Glyco	351	3	5	2			2		
Fus	2	940_Glyco	351	4	6	2			2		
Fus	2	940_Glyco	351	5	7	2			2		
Fus	2	940_Glyco	351	6	7	2			2		
Fus	2	940_Glyco	351	7							
Fus	2	940_Glyco	351	8							
Fus	2	940_Glyco	351	9							
Fus	2	940_Glyco	351	10							
Fus	2	8406_Ca	352	1	4	2			2		
Fus	2	8406_Ca	352	2	4	2			2		
Fus	2	8406_Ca	352	3	4	2			2		
Fus	2	8406_Ca	352	4	5	2			2		
Fus	2	8406_Ca	352	5	5	2			2		
Fus	2	8406_Ca	352	6	6	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	8406_Ca	352	7	7	2			2		
Fus	2	8406_Ca	352	8	7	2			2		
Fus	2	8406_Ca	352	9	8	2			2		
Fus	2	8406_Ca	352	10							
Fus	2	6293_Ca	353	1	3	2			2		
Fus	2	6293_Ca	353	2	3	2			2		
Fus	2	6293_Ca	353	3	4	2			2		
Fus	2	6293_Ca	353	4	4	2			2		
Fus	2	6293_Ca	353	5	5	2			2		
Fus	2	6293_Ca	353	6	5	2			2		
Fus	2	6293_Ca	353	7	5	2			2		
Fus	2	6293_Ca	353	8	5	2			2		
Fus	2	6293_Ca	353	9	5	2			2		
Fus	2	6293_Ca	353	10	6	2			2		
Fus	2	3028_Ca	354	1	6	1			2		
Fus	2	3028_Ca	354	2	9	2			2		
Fus	2	3028_Ca	354	3	10	2			2		
Fus	2	3028_Ca	354	4	12	2			2		
Fus	2	3028_Ca	354	5	12	2			2		
Fus	2	3028_Ca	354	6							
Fus	2	3028_Ca	354	7							
Fus	2	3028_Ca	354	8							
Fus	2	3028_Ca	354	9							
Fus	2	3028_Ca	354	10							
Fus	2	256	355	1	6	7			12		
Fus	2	256	355	2	6	6			12		
Fus	2	256	355	3	7	2			2		
Fus	2	256	355	4	7	2			3		
Fus	2	256	355	5	7	2			2		
Fus	2	256	355	6	8	4			11		
Fus	2	256	355	7	8	10			14		
Fus	2	256	355	8	8	2			2		
Fus	2	256	355	9	9	5			10		
Fus	2	256	355	10	9	2			2		
Fus	2	256	355	11	9	2			2		
Fus	2	256	355	12	9	3			3		
Fus	2	256	355	13	10	2			2		
Fus	2	1399	356	1	4	8			12		
Fus	2	1399	356	2	5	4			4		
Fus	2	1399	356	3	5	20			20		
Fus	2	1399	356	4	6	2			2		
Fus	2	1399	356	5	6	3			8		
Fus	2	1399	356	6	6	7			10		
Fus	2	1399	356	7	6	2			2		
Fus	2	1399	356	8	6	2			2		
Fus	2	1399	356	9	7	2			2		
Fus	2	1399	356	10	8	2			2		
Fus	2	1399	356	11	8	8			11		
Fus	2	177	357	1	5	18			20		
Fus	2	177	357	2	5	20			20		
Fus	2	177	357	3	6	10			14		
Fus	2	177	357	4	6	18			12		
Fus	2	177	357	5	6	3			10		
Fus	2	177	357	6	6	5			8		
Fus	2	177	357	7	7	12			12		
Fus	2	177	357	8	7	18			16		
Fus	2	177	357	9	7	12			18		
Fus	2	177	357	10	8	12			12		
Fus	2	177	357	11	8	10			12		
Fus	2	177	357	12	8	8			12		
Fus	2	177	357	13	8	10			10		
Fus	2	1155_mutant	358	1	7	0			0		
Fus	2	1155_mutant	358	2	7	2			2		
Fus	2	1155_mutant	358	3	7	2.5			2.5		
Fus	2	1155_mutant	358	4	8	2			2		
Fus	2	1155_mutant	358	5	8	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	1155_mutant	358	6	9	2			2		
Fus	2	1155_mutant	358	7	9	2			2		
Fus	2	1155_mutant	358	8							
Fus	2	1155_mutant	358	9							
Fus	2	1155_mutant	358	10							
Fus	2	1155_no_mutation	359	1	7	2			2		
Fus	2	1155_no_mutation	359	2	7	2			2		
Fus	2	1155_no_mutation	359	3	7	2			2		
Fus	2	1155_no_mutation	359	4	7	2			2		
Fus	2	1155_no_mutation	359	5	7	2			2		
Fus	2	1155_no_mutation	359	6	8	2			2		
Fus	2	1155_no_mutation	359	7	8	2			2		
Fus	2	1155_no_mutation	359	8	9	2			2		
Fus	2	1155_no_mutation	359	9							
Fus	2	1155_no_mutation	359	10							
Fus	2	784_Ca	360	1	3	2			2		
Fus	2	784_Ca	360	2	4	2			2		
Fus	2	784_Ca	360	3	4	10			12		
Fus	2	784_Ca	360	4	5	7			13		
Fus	2	784_Ca	360	5	5	2			2		
Fus	2	784_Ca	360	6	5	8			13		
Fus	2	784_Ca	360	7	7	16			16		
Fus	2	784_Ca	360	8	8	2			2		
Fus	2	784_Ca	360	9							
Fus	2	784_Ca	360	10							
Fus	2	219_Ca	361	1	5	2.5			3		
Fus	2	219_Ca	361	2	5	2			2		
Fus	2	219_Ca	361	3	5	2			2		
Fus	2	219_Ca	361	4	6	2			3		
Fus	2	219_Ca	361	5	6	2			2		
Fus	2	219_Ca	361	6	6	1.5			2		
Fus	2	219_Ca	361	7	7	2			2		
Fus	2	219_Ca	361	8	7	2			2		
Fus	2	219_Ca	361	9	7	2			2		
Fus	2	219_Ca	361	10	9	2			2		
Fus	2	2203_Ca	362	1	5	2			2		
Fus	2	2203_Ca	362	2	5	2			2		
Fus	2	2203_Ca	362	3	6	2			2		
Fus	2	2203_Ca	362	4	6	2			2		
Fus	2	2203_Ca	362	5	7	2			2		
Fus	2	2203_Ca	362	6	7	2			2		
Fus	2	2203_Ca	362	7	7	2			2		
Fus	2	2203_Ca	362	8	8	2			3		
Fus	2	2203_Ca	362	9	8	2			2		
Fus	2	2203_Ca	362	10	8	2			2		
Fus	2	2203_Ca	362	11	9	2			2		
Fus	2	723_Glyco	363	1	6	2			2		
Fus	2	723_Glyco	363	2	6	2			2		
Fus	2	723_Glyco	363	3	7	0.5			2		
Fus	2	723_Glyco	363	4	8	2			2		
Fus	2	723_Glyco	363	5	8	2			2		
Fus	2	723_Glyco	363	6	8	2			2		
Fus	2	723_Glyco	363	7	8	2			2		
Fus	2	723_Glyco	363	8	9	2			2		
Fus	2	723_Glyco	363	9	13	2			?		
Fus	2	723_Glyco	363	10	13	2			?		
Fus	2	7919_no_1_Hyd	370	1	5	2			2		
Fus	2	7919_no_1_Hyd	370	2	5	2			2		
Fus	2	7919_no_1_Hyd	370	3	6	2			2		
Fus	2	7919_no_1_Hyd	370	4	6	2			2		
Fus	2	7919_no_1_Hyd	370	5	6	2			2		
Fus	2	7919_no_1_Hyd	370	6	7	2			2		
Fus	2	7919_no_1_Hyd	370	7	7	2			2		
Fus	2	7919_no_1_Hyd	370	8	8	2			2		
Fus	2	7919_no_1_Hyd	370	9	8	2			2		
Fus	2	7919_no_1_Hyd	370	10	9	2			2		

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Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	7919_no_1_Hyd	370	11	9	2			2		
Fus	2	Sumai3_Ung1	371	1	5	2			2		
Fus	2	Sumai3_Ung1	371	2	5	8			8		
Fus	2	Sumai3_Ung1	371	3	5	3			6		
Fus	2	Sumai3_Ung1	371	4	5	3			3		
Fus	2	Sumai3_Ung1	371	5	6	2			2		
Fus	2	Sumai3_Ung1	371	6	6	1			2		
Fus	2	Sumai3_Ung1	371	7	6	1.5			5	w	
Fus	2	Sumai3_Ung1	371	8	7	3			3		
Fus	2	Sumai3_Ung1	371	9	7	2			2		
Fus	2	Sumai3_Ung1	371	10	8	2			2		
Fus	2	Wangshuibai	372	1	4	2			2		
Fus	2	Wangshuibai	372	2	4	2			2		
Fus	2	Wangshuibai	372	3	4	2			2		
Fus	2	Wangshuibai	372	4	4	2			2		
Fus	2	Wangshuibai	372	5	4	2			2		
Fus	2	Wangshuibai	372	6	5	2			2		
Fus	2	Wangshuibai	372	7	5	2			2		
Fus	2	Wangshuibai	372	8	5	2			2		
Fus	2	Wangshuibai	372	9	5	2			2		
Fus	2	Wangshuibai	372	10	5	2			2		
Fus	2	Wangshuibai	372	11	6	2			2		
Fus	2	Ning_7840	373	1	4	2			2		
Fus	2	Ning_7840	373	2	4	2			2		
Fus	2	Ning_7840	373	3	5	2			2		
Fus	2	Ning_7840	373	4	5	2			2		
Fus	2	Ning_7840	373	5	5	0			0		
Fus	2	Ning_7840	373	6	5	0			0		
Fus	2	Ning_7840	373	7	5	2			2		
Fus	2	Ning_7840	373	8	5	2			2		
Fus	2	Ning_7840	373	9	5	2			2		
Fus	2	Ning_7840	373	10	6	2*0,5			2		
Fus	2	Ning_7840	373	11	6	1.5			2		
Fus	2	ND_2710	374	1	1	2			2		
Fus	2	ND_2710	374	2	1	3			3		
Fus	2	ND_2710	374	3	3	2			2		
Fus	2	ND_2710	374	4	3	10			20		
Fus	2	ND_2710	374	5	4	2			2		
Fus	2	ND_2710	374	6	4	2			2		
Fus	2	ND_2710	374	7	4	2			2		
Fus	2	ND_2710	374	8	4	12			14		
Fus	2	ND_2710	374	9	4	2			2		
Fus	2	ND_2710	374	10	5	2			2		
Fus	2	ND_2710	374	11	5	2			2		
Fus	2	W14	375	1	3	2			2		
Fus	2	W14	375	2	3	2			2		
Fus	2	W14	375	3	3	3			4		
Fus	2	W14	375	4	3	2			2		
Fus	2	W14	375	5	4	2			2		
Fus	2	W14	375	6	5	2			2		
Fus	2	W14	375	7	6	2			2		
Fus	2	W14	375	8							
Fus	2	W14	375	9							
Fus	2	W14	375	10							
Fus	2	Sumai3_MX	376	1	2	2			2		
Fus	2	Sumai3_MX	376	2	3	2			2		
Fus	2	Sumai3_MX	376	3	3	2			2		
Fus	2	Sumai3_MX	376	4	3	2			2		
Fus	2	Sumai3_MX	376	5	4	2			2		
Fus	2	Sumai3_MX	376	6	4	2			2		
Fus	2	Sumai3_MX	376	7	4	2			2		
Fus	2	Sumai3_MX	376	8	4	2			2		
Fus	2	Sumai3_MX	376	9	5	2			2		
Fus	2	Sumai3_MX	376	10	5	2			2		
Fus	2	Nobeokabozu_U	377	1	4	2			2		
Fus	2	Nobeokabozu_U	377	2	4	2			2		

Table continued on next page.

Table 47: Continued from previous page.

Treatment	Replication	Genotype	Pot	Head	Date	B1	weak B1	B1 all	B2	weak B2	B2 all
Fus	2	Nobeokabozu_U	377	3	5	2			2		
Fus	2	Nobeokabozu_U	377	4	5	2			2		
Fus	2	Nobeokabozu_U	377	5	5	1			1		
Fus	2	Nobeokabozu_U	377	6	6	2			2		
Fus	2	Nobeokabozu_U	377	7	6	2			2		
Fus	2	Nobeokabozu_U	377	8	6	2			2		
Fus	2	Nobeokabozu_U	377	9	6	2			2		
Fus	2	Nobeokabozu_U	377	10	7	2			2		
Fus	2	Nobeokabozu_U	377	11	8	2			2		
Fus	3	Remus	378	1	1	20			20		
Fus	3	Remus	378	2	1	20			20		
Fus	3	Remus	378	3	2	18			18		
Fus	3	Remus	378	4	3	18			20		
Fus	3	Remus	378	5	3	18			20		
Fus	3	Remus	378	6	5	20			20		
Fus	3	Remus	378	7	5	20			20		
Fus	3	Remus	378	8	6	20			20		
DON	3	Remus	378	9	8	2			2		
Fus	3	Remus	378	10							
DON	3	Remus	198B	1	2	8			8		
DON	3	Remus	198B	2	2		2w		10		
DON	3	Remus	198B	3	5	7	2		7	2	
DON	3	Remus	198B	4	5	10			9	2	
DON	3	Remus	198B	5	6	9			12		
DON	3	Remus	198B	6	6	3	3		10		
DON	3	Remus	198B	7	4	10			10		
DON	3	Remus	198B	8	4		2			2	

Table continued on next page.

Table 48: Greenhouse book of the heterozygous mutant lines. The treatment (DON infiltration, Fusarium inoculation), genotype and wild-type or mutant-type can be seen. Further, the pot number, number of infiltrated/inoculated heads, date of treatment and date of evaluation (B1, B2) is given. The respective dates are listed in Table 27.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	1500_Gly	355_wt	60	1	7	0		0	
DON	1500_Gly	355_wt	60	2	14	0		0	
DON	1500_Gly	355_wt	60	3					
DON	1500_Gly	356_wt	60	1	6	0		0	
DON	1500_Gly	356_wt	60	2	12	0		0	
DON	1500_Gly	356_wt	60	3	14	0		0	
DON	1500_Gly	357_wt	60	1	6	0		0	
DON	1500_Gly	357_wt	60	2	11	0		0	
DON	1500_Gly	357_wt	60	3	11	0		0	
DON	1500_Gly	357_wt	60	4	12	0		0	
DON	1500_Gly	342_mut	61	1	6	0		0	
DON	1500_Gly	342_mut	61	2	9	0		0	
DON	1500_Gly	342_mut	61	3	10	0		0	
DON	1500_Gly	343_mut	61	1	6	0		0	
DON	1500_Gly	343_mut	61	2	11	0		0	
DON	1500_Gly	343_mut	61	3					
DON	1500_Gly	344_mut	61	1	6	0		0	
DON	1500_Gly	344_mut	61	2	11	0		0	
DON	1500_Gly	344_mut	61	3	12	0		0	
DON	3094_Gly	253_wt	62	1	7	0		0	
DON	3094_Gly	253_wt	62	2	8	0		0	
DON	3094_Gly	253_wt	62	3	9	0		0	
DON	3094_Gly	254_wt	62	1	7	0		0	
DON	3094_Gly	254_wt	62	2	9	0		0	
DON	3094_Gly	254_wt	62	3	10	2*0,5		2*0,5	
DON	3094_Gly	254_wt	62	4	12	0		0	
DON	3094_Gly	255_wt	62	1	5	0		0	
DON	3094_Gly	255_wt	62	2	9	0		0	
DON	3094_Gly	255_wt	62	3	9	0		0	
DON	3094_Gly	255_wt	62	4	12	0		0	
DON	3094_Gly	250_mut	63	1	8	0		0	
DON	3094_Gly	250_mut	63	2	11	0		0	
DON	3094_Gly	250_mut	63	3	12	0		0	
DON	3094_Gly	251_mut	63	1	8	0		0	
DON	3094_Gly	251_mut	63	2	10	0		0	
DON	3094_Gly	251_mut	63	3					
DON	3094_Gly	252_mut	63	1	7	0		0	
DON	3094_Gly	252_mut	63	2	9	0		0	
DON	3094_Gly	252_mut	63	2	10	0		0	
DON	3094_Gly	252_mut	63	3	12	0		0	
DON	3094_Gly	256_wt	64	1	8	0		0	
DON	3094_Gly	256_wt	64	2	10	2*0,5		0	
DON	3094_Gly	256_wt	64	3					
DON	3094_Gly	257_wt	64	1	6	2*0,5		0	
DON	3094_Gly	257_wt	64	2	9	0		0	
DON	3094_Gly	257_wt	64	3					
DON	3094_Gly	258_wt	64	1					
DON	3094_Gly	258_wt	64	2					
DON	3094_Gly	258_wt	64	3					
DON	3094_Gly	259_wt	64	1	7	0		0	
DON	3094_Gly	259_wt	64	2	10	0		0	
DON	3094_Gly	259_wt	64	3	11	0		0	
DON	1500_Gly	358_wt	65	1	7	0		0	
DON	1500_Gly	358_wt	65	2	7	0		0	
DON	1500_Gly	358_wt	65	3	10	0		0	
DON	1500_Gly	358_wt	65	4	11	0		0	
DON	1500_Gly	359_wt	65	1	10	0		0	
DON	1500_Gly	359_wt	65	2	12	0		0	
DON	1500_Gly	359_wt	65	3					
DON	1500_Gly	366_wt	65	1	8	0		0	
DON	1500_Gly	366_wt	65	2	12	0		0	
DON	1500_Gly	366_wt	65	3	13	0		0	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	1500_Gly	366_wt	65	4	13	0		0	
DON	641_Ubi	283_mut	75	1	9	0		0	
DON	641_Ubi	283_mut	75	2					
DON	641_Ubi	283_mut	75	3					
DON	641_Ubi	284_mut	75	1	8	0		0	
DON	641_Ubi	284_mut	75	2	12	0		0	
DON	641_Ubi	284_mut	75	3	13	0		0	
DON	641_Ubi	285_mut	75	1	7	0		0	
DON	641_Ubi	285_mut	75	2	11	0		0	
DON	641_Ubi	285_mut	75	3					
DON	641_Ubi	286_mut	75	1	7	0		0	
DON	641_Ubi	286_mut	75	2	12	0		0	
DON	641_Ubi	286_mut	75	3					
DON	641_Ubi	293_wt	76	1	8	0		0	
DON	641_Ubi	293_wt	76	2	11	0		0	
DON	641_Ubi	293_wt	76	3	13	0		0	
DON	641_Ubi	294_wt	76	1	11	0		0	
DON	641_Ubi	294_wt	76	2	12	0		0	
DON	641_Ubi	294_wt	76	3					
DON	641_Ubi	295_wt	76	1	8	0		0	
DON	641_Ubi	295_wt	76	2	13	0		0	
DON	641_Ubi	295_wt	76	3					
DON	1500_Gly	345_mut	77	1	9	0		0	
DON	1500_Gly	345_mut	77	2	11	0		0	
DON	1500_Gly	345_mut	77	3	13	0		0	
DON	1500_Gly	346_mut	77	1	8	0		0	
DON	1500_Gly	346_mut	77	2					
DON	1500_Gly	346_mut	77	3					
DON	1500_Gly	353_mut	77	1	7	0		0	
DON	1500_Gly	353_mut	77	2	11	0		0	
DON	1500_Gly	353_mut	77	3	11	0		0	
DON	1500_Gly	353_mut	77	4	12	0		0	
DON	256_Ubi	22_wt	78	1	10	0		0	
DON	256_Ubi	22_wt	78	2	13	0		0	
DON	256_Ubi	22_wt	78	3	14	0		0	
DON	256_Ubi	23_wt	78	1	8	0		0	
DON	256_Ubi	23_wt	78	2	11	0		0	
DON	256_Ubi	23_wt	78	3	13	0		0	
DON	256_Ubi	24_wt	78	1					
DON	256_Ubi	24_wt	78	2					
DON	256_Ubi	24_wt	78	3					
DON	256_Ubi	25_wt	78	1	11	0		0	
DON	256_Ubi	25_wt	78	2	14	0		0	
DON	256_Ubi	25_wt	78	3	16	0		0	
DON	256_Ubi	26_wt	78	1	11	0		0	
1	256_Ubi	26_wt	78	2	14	0		0	
DON	256_Ubi	26_wt	78	3	16	0		0	
DON	256_Ubi	26_wt	78	4	16	0		0	
DON	256_Ubi	27_wt	79	1	11	0		0	
DON	256_Ubi	27_wt	79	2	14	0		0	
DON	256_Ubi	27_wt	79	3					
DON	256_Ubi	28_wt	79	1	8	0		0	
DON	256_Ubi	28_wt	79	2	11	0		0	
DON	256_Ubi	28_wt	79	3					
DON	256_Ubi	29_wt	79	1	14	0		0	
DON	256_Ubi	29_wt	79	2					
DON	256_Ubi	29_wt	79	3					
DON	256_Ubi	30_wt	79	1	10	0		0	
DON	256_Ubi	30_wt	79	2	12	0		0	
DON	256_Ubi	30_wt	79	3					
DON	256_Ubi	31_wt	79	1	11	0		0	
DON	256_Ubi	31_wt	79	2	11	0		0	
DON	256_Ubi	31_wt	79	3					
DON	256_Ubi	1_mut	80	1	11	3		3	
DON	256_Ubi	1_mut	80	2	13	3		3	5
DON	256_Ubi	1_mut	80	3	14	3		3	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	256_Ubi	2_mut	80	1	8	2		2	
DON	256_Ubi	2_mut	80	2	11	3	1	4	
DON	256_Ubi	2_mut	80	3	12	4		4	
DON	256_Ubi	3_mut	80	1	8	2	1	3	2
DON	256_Ubi	3_mut	80	2	11	2		2	
DON	256_Ubi	3_mut	80	3	11	2		3	
DON	256_Ubi	4_mut	80	1	11	2		2	
DON	256_Ubi	4_mut	80	2	13	3		3	
DON	256_Ubi	4_mut	80	3					
DON	256_Ubi	5_mut	80	1	8	3		3	2
DON	256_Ubi	5_mut	80	2	11	2		2	3
DON	256_Ubi	5_mut	80	3					
DON	256_Ubi	10_mut	81	1	8	2	1	3	2
DON	256_Ubi	10_mut	81	2	11	4		4	
DON	256_Ubi	10_mut	81	3					
DON	256_Ubi	6_mut	81	1	11	2		2	
DON	256_Ubi	6_mut	81	2	14	3		2	
DON	256_Ubi	6_mut	81	3					
DON	256_Ubi	7_mut	81	1	11	4		8	
DON	256_Ubi	7_mut	81	2	14	3		3	4
DON	256_Ubi	7_mut	81	3	14	2		2	5
DON	256_Ubi	8_mut	81	1	11	2		2	
DON	256_Ubi	8_mut	81	2	14	4		2	5
DON	256_Ubi	8_mut	81	3	14	2		3	
DON	256_Ubi	9_mut	81	1	8	6	1	6	1
DON	256_Ubi	9_mut	81	2	12	2		2	
DON	256_Ubi	9_mut	81	3	13	4		4	
DON	1399_Ca	47_mut	82	1	12	2		2	
DON	1399_Ca	47_mut	82	2					
DON	1399_Ca	47_mut	82	3					
DON	1399_Ca	48_mut	82	1	6	2		2	
DON	1399_Ca	48_mut	82	2	9	10		10	
DON	1399_Ca	48_mut	82	3	10	8		10	
DON	1399_Ca	49_mut	82	1	8	4	2	6	
DON	1399_Ca	49_mut	82	2	11	2		2	
DON	1399_Ca	49_mut	82	3	11	4		4	
DON	1399_Ca	50_mut	82	1	8	2	4	6	
DON	1399_Ca	50_mut	82	2	10	2	2	3	1
DON	1399_Ca	50_mut	82	3					
DON	1399_Ca	51_mut	82	1	8	2	1	6	
DON	1399_Ca	51_mut	82	2	10	3		2	
DON	1399_Ca	51_mut	82	3	10	3		2	
DON	1399_Ca	52_mut	83	1	7	3	1	3	1
DON	1399_Ca	52_mut	83	2	9	8		8	
DON	1399_Ca	52_mut	83	3	9	8		8	
DON	1399_Ca	53_mut	83	1	7	2	5	8	
DON	1399_Ca	53_mut	83	2	9	0		0	
DON	1399_Ca	53_mut	83	3	10	8	1	10	
DON	1399_Ca	54_mut	83	1	8	2	1	2	1
DON	1399_Ca	54_mut	83	2	9	8		8	
DON	1399_Ca	54_mut	83	3					
DON	1399_Ca	55_mut	83	1	7	2		2	
DON	1399_Ca	55_mut	83	2	8	8	1	8	2
DON	1399_Ca	55_mut	83	3					
DON	1399_Ca	56_mut	83	1	9	8		8	
DON	1399_Ca	56_mut	83	2	9	8		8	
DON	1399_Ca	56_mut	83	3					
DON	1399_Ca	71_wt	84	1	7	0		0	
DON	1399_Ca	71_wt	84	2	8	0		0?	
DON	1399_Ca	71_wt	84	3	9	0		?	
DON	1399_Ca	72_wt	84	1	8	0		0	3?
DON	1399_Ca	72_wt	84	2	10	0		0	
DON	1399_Ca	72_wt	84	3	11	0		0	
DON	1399_Ca	73_wt	84	1	8	0		?	2
DON	1399_Ca	73_wt	84	2					
DON	1399_Ca	73_wt	84	3					

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	1399_Ca	74_wt	84	1	7	0		0	
DON	1399_Ca	74_wt	84	2	9	0		?	4
DON	1399_Ca	74_wt	84	3	9	0		?	
DON	1399_Ca	75_wt	84	1	7	0		0	
DON	1399_Ca	75_wt	84	2	10	0	2	0	3
DON	1399_Ca	75_wt	84	3	10	0	2	0	3
DON	1399_Ca	76_wt	85	1	7	0		0	
DON	1399_Ca	76_wt	85	2	9	0		0	
DON	1399_Ca	76_wt	85	3	9	0		0	
DON	1399_Ca	77_wt	85	1	6	0		0	
DON	1399_Ca	77_wt	85	2	8	0		0	2
DON	1399_Ca	77_wt	85	3					
DON	1399_Ca	78_wt	85	1	5	0		0	
DON	1399_Ca	78_wt	85	2	9	0		0	
DON	1399_Ca	78_wt	85	3	9	0		0	
DON	1399_Ca	79_wt	85	1	6	0	0.5	0	0.5
DON	1399_Ca	79_wt	85	2					
DON	1399_Ca	79_wt	85	3					
DON	1399_Ca	80_wt	85	1	6	0		0	
DON	1399_Ca	80_wt	85	2	8	0		0	
DON	1399_Ca	80_wt	85	3	10	0		0	
DON	177_Ca	93_mut	86	1	6	2		2	
DON	177_Ca	93_mut	86	2	9	2		2	
DON	177_Ca	93_mut	86	3					
DON	177_Ca	94_mut	86	1	8	2		3	
DON	177_Ca	94_mut	86	2					
DON	177_Ca	94_mut	86	3					
DON	177_Ca	95_mut	86	1	7	2		2	
DON	177_Ca	95_mut	86	2	8	3	2	3	2
DON	177_Ca	95_mut	86	3	9	2		2	
DON	177_Ca	96_mut	86	1	6	2		2	
DON	177_Ca	96_mut	86	2	8	5	1	6	
DON	177_Ca	96_mut	86	3	8	3	1	3	
DON	177_Ca	100_mut	87	1	7	2		2	
DON	177_Ca	100_mut	87	2	10	2		2	
DON	177_Ca	100_mut	87	3	10	2		2	
DON	177_Ca	97_mut	87	1	6	2		3	
DON	177_Ca	97_mut	87	2	8	2		2	
DON	177_Ca	97_mut	87	3	9	2.5		2	
DON	177_Ca	98_mut	87	1	8	2		2	
DON	177_Ca	98_mut	87	2	11	3		3	
DON	177_Ca	98_mut	87	3	11	2		2	
DON	177_Ca	99_mut	87	1	7	2		2	
DON	177_Ca	99_mut	87	2	10	2		2	
DON	177_Ca	99_mut	87	3	10	4		4	
DON	177_Ca	109_wt	88	1	7	0		0	
DON	177_Ca	109_wt	88	2	10	0		0	
DON	177_Ca	109_wt	88	3	10	0		0	
DON	177_Ca	110_wt	88	1	6	0,5b		0.5	
DON	177_Ca	110_wt	88	2	8	0		0	
DON	177_Ca	110_wt	88	3	8	0		0	
DON	177_Ca	110_wt	88	4	9	0		0	
DON	177_Ca	111_wt	88	1	7	0		0	
DON	177_Ca	111_wt	88	2	9	0		0	
DON	177_Ca	111_wt	88	3					
DON	177_Ca	112_wt	88	1	10	2*0,5		0.5	
DON	177_Ca	112_wt	88	2	11	0		0	0.5
DON	177_Ca	112_wt	88	3					
DON	177_Ca	113_wt	88	1	7	0		0	
DON	177_Ca	113_wt	88	2	7	0		0	
DON	177_Ca	113_wt	88	3	10	0		0	
DON	177_Ca	113_wt	88	4	10	0		0	
DON	177_Ca	114_wt	89	1	7	0		0	
DON	177_Ca	114_wt	89	2	9	0		0	2?
DON	177_Ca	114_wt	89	3	10	0		0	
DON	177_Ca	115_wt	89	1	7	0		0	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	177_Ca	115_wt	89	2	9	0		0	2?
DON	177_Ca	115_wt	89	3					
DON	177_Ca	116_wt	89	1	8	0		0	2
DON	177_Ca	116_wt	89	2	9	0		0	2?
DON	177_Ca	116_wt	89	3					
DON	177_Ca	117_wt	89	1	8	0		0	3
DON	177_Ca	117_wt	89	2	10	0		0	
DON	177_Ca	117_wt	89	3	10	0		0	
DON	177_Ca	118_wt	89	1	6	0		0	
DON	177_Ca	118_wt	89	2	7	0		0	
DON	177_Ca	118_wt	89	3	9	0		0	2
DON	177_Ca	118_wt	89	4	9	0		0	2?
DON	448_Ubi	147_wt	90	1	6	0		0	
DON	448_Ubi	147_wt	90	2	8	0		0	
DON	448_Ubi	147_wt	90	3	9	0		0	
DON	448_Ubi	148_wt	90	1	6	0		0	
DON	448_Ubi	148_wt	90	2	8	0		0	
DON	448_Ubi	148_wt	90	3	8	0		0	
DON	448_Ubi	149_wt	90	1	6	0		0	
DON	448_Ubi	149_wt	90	2	9	0		0	
DON	448_Ubi	149_wt	90	3	9	0		0	
DON	448_Ubi	150_wt	91	1	6	0		0	
DON	448_Ubi	150_wt	91	2	8	0		0	
DON	448_Ubi	150_wt	91	3	9	0		0	
DON	448_Ubi	151_wt	91	1	6	0		0	
DON	448_Ubi	151_wt	91	2	10	0		0	
DON	448_Ubi	151_wt	91	3	10	0		0	
DON	448_Ubi	152_wt	91	1	7	0		0	
DON	448_Ubi	152_wt	91	2	8	0		0	
DON	448_Ubi	152_wt	91	3	11	0		0	
DON	2187_Ubi	156_mut	92	1	8	0		0	6
DON	2187_Ubi	156_mut	92	2	11	0		0	
DON	2187_Ubi	156_mut	92	3	11	0		0	
DON	2187_Ubi	157_mut	92	1	9	0		2*0,5	
DON	2187_Ubi	157_mut	92	2	10	0		0	
DON	2187_Ubi	157_mut	92	3					
DON	2187_Ubi	158_mut	92	1	8	0		2*0,5	
DON	2187_Ubi	158_mut	92	2	10	0		0	
DON	2187_Ubi	158_mut	92	3	11	0		0	
DON	2187_Ubi	159_mut	93	1	7	0		0	
DON	2187_Ubi	159_mut	93	2	8	0		0	
DON	2187_Ubi	159_mut	93	3	10	0		0	
DON	2187_Ubi	159_mut	93	4	11	0		0	
DON	2187_Ubi	160_mut	93	1	9	0		2*0,5	
DON	2187_Ubi	160_mut	93	2	13	0		0	
DON	2187_Ubi	160_mut	93	3	13	0		0	
DON	2187_Ubi	161_mut	93	1	8	0		0	
DON	2187_Ubi	161_mut	93	2	10	0		0.5	
DON	2187_Ubi	161_mut	93	3	11	0		0	
DON	2187_Ubi	166_wt	94	1	8	0		0	
DON	2187_Ubi	166_wt	94	2	13	0		0	2
DON	2187_Ubi	166_wt	94	3					
DON	2187_Ubi	167_wt	94	1	9	0		0	
DON	2187_Ubi	167_wt	94	2	13	0		0	
DON	2187_Ubi	167_wt	94	3					
DON	2187_Ubi	168_wt	94	1	7	0		0	
DON	2187_Ubi	168_wt	94	2	10	0		0	
DON	2187_Ubi	168_wt	94	3	10	0		0	
DON	2187_Ubi	169_wt	94	1	8	0		0	
DON	2187_Ubi	169_wt	94	2	10	0		0	
DON	2187_Ubi	169_wt	94	3	10	0		0	
DON	2187_Ubi	170_wt	95	1	8	0		0	
DON	2187_Ubi	170_wt	95	2	10	0		0	
DON	2187_Ubi	170_wt	95	3	11	0		0	
DON	2187_Ubi	171_wt	95	1	8	0		0	
DON	2187_Ubi	171_wt	95	2	11	0		0	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	2187_Ubi	171_wt	95	3	11	0		0	1
DON	2187_Ubi	172_wt	95	1	8	0		0	
DON	2187_Ubi	172_wt	95	2	10	0		0	
DON	2187_Ubi	172_wt	95	3	10	0		0	
DON	2187_Ubi	173_wt	95	1	8	0		0	
DON	2187_Ubi	173_wt	95	2	11	0		0	1
DON	2187_Ubi	173_wt	95	3	11	0		0	
DON	8032_Ca	190_mut	96	1	7	0		0	
DON	8032_Ca	190_mut	96	2	9	0		0	
DON	8032_Ca	190_mut	96	3	9	0		0	
DON	8032_Ca	190_mut	96	4	10	0		0	
DON	8032_Ca	191_mut	96	1	5	0		0	
DON	8032_Ca	191_mut	96	2	8	0		0	
DON	8032_Ca	191_mut	96	3	9	0		0	
DON	8032_Ca	191_mut	96	4	10	0		0	
DON	8032_Ca	192_mut	96	1	6	0		0	
DON	8032_Ca	192_mut	96	2	9	0		0	
DON	8032_Ca	192_mut	96	3	10	0		0	
DON	8032_Ca	193_mut	97	1	5	0		0	
DON	8032_Ca	193_mut	97	2	8	0		0	
DON	8032_Ca	193_mut	97	3	11	0		0	
DON	8032_Ca	194_mut	97	1	8	0		0	
DON	8032_Ca	194_mut	97	2	11	0		0	
DON	8032_Ca	194_mut	97	3	14	0		0	2
DON	8032_Ca	199_wt	98	1	6	0		0	
DON	8032_Ca	199_wt	98	2	8	0		0	
DON	8032_Ca	199_wt	98	3	13	0		0	
DON	8032_Ca	200_wt	98	1	8	0		0	
DON	8032_Ca	200_wt	98	2	8	0		0	
DON	8032_Ca	200_wt	98	3					
DON	8032_Ca	201_wt	98	1	6	0		0	
DON	8032_Ca	201_wt	98	2	8	0		0	
DON	8032_Ca	201_wt	98	3	14	0		0	
DON	8032_Ca	202_wt	98	1					
DON	8032_Ca	202_wt	98	2					
DON	8032_Ca	202_wt	98	3					
DON	8032_Ca	203_wt	99	1					
DON	8032_Ca	203_wt	99	2					
DON	8032_Ca	203_wt	99	3					
DON	8032_Ca	204_wt	99	1	6	0		0	
DON	8032_Ca	204_wt	99	2	8	0		0	
DON	8032_Ca	204_wt	99	3					
DON	8032_Ca	205_wt	99	1	6	0		0	
DON	8032_Ca	205_wt	99	2	8	0		0	
DON	8032_Ca	205_wt	99	3					
DON	448_Ubi	128_mut	100	1	8	0		0	
DON	448_Ubi	128_mut	100	2	12	0		0	
DON	448_Ubi	128_mut	100	3	13	0		0	
DON	448_Ubi	129_mut	100	1	8	0		0	
DON	448_Ubi	129_mut	100	2	11	0		0	
DON	448_Ubi	129_mut	100	3					
DON	448_Ubi	130_mut	100	1	7	0		0	
DON	448_Ubi	130_mut	100	2	12	0		0	
DON	448_Ubi	130_mut	100	3	13	0		0	
DON	448_Ubi	131_mut	100	1	8	0		0	
DON	448_Ubi	131_mut	100	2	12	0		0	
DON	448_Ubi	131_mut	100	3					
DON	48_Ubi_	313_mut	100	1	10	0		0	
DON	48_Ubi_	313_mut	100	2					
DON	48_Ubi_	313_mut	100	3					
DON	448_Ubi	132_mut	101	1	7	0		0	
DON	448_Ubi	132_mut	101	2	11	0		0	
DON	448_Ubi	132_mut	101	3	13	0		0	
DON	448_Ubi	133_mut	101	1					
DON	448_Ubi	133_mut	101	2					
DON	448_Ubi	133_mut	101	3					

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	448_Ubi	134_mut	101	1	6	0		0	
DON	448_Ubi	134_mut	101	2	11	0		0	
DON	448_Ubi	134_mut	101	3					
DON	448_Ubi	135_mut	101	1	7	0		0	
DON	448_Ubi	135_mut	101	2	8	0		0	
DON	448_Ubi	135_mut	101	3					
DON	641_Ubi	296_wt	102	1	11	0		0	
DON	641_Ubi	296_wt	102	2	13	0		0	
DON	641_Ubi	296_wt	102	3	15	0		0	
DON	641_Ubi	297_wt	102	1	8	0		0	
DON	641_Ubi	297_wt	102	2	11	0		0	
DON	641_Ubi	297_wt	102	3	11	0		0	
DON	641_Ubi	298_wt	102	1	7	0		0	
DON	641_Ubi	298_wt	102	2	11	0		0	
DON	641_Ubi	298_wt	102	3	11	0		0	
DON	6504_Ca	299_mut	103	1	8	0		0	
DON	6504_Ca	299_mut	103	2	11	0		0	
DON	6504_Ca	299_mut	103	3					
DON	6504_Ca	300_mut	103	1	8	0		0	
DON	6504_Ca	300_mut	103	2					
DON	6504_Ca	300_mut	103	3					
DON	6504_Ca	301_mut	103	1	7	0		0	
DON	6504_Ca	301_mut	103	2	11	0		0	
DON	6504_Ca	301_mut	103	3	14	0		0	
DON	6504_Ca	302_mut	103	1	8	0		0	
DON	6504_Ca	302_mut	103	2	11	0		0	
DON	6504_Ca	302_mut	103	3	13	0		0	
DON	6504_Ca	302_mut	103	4	13	0		0	
DON	6504_Ca	302_mut	103	5	14	0		0	
DON	1722_Ca	323_wt	104	1	7	0		0	
DON	1722_Ca	323_wt	104	2	11	0		0	
DON	1722_Ca	323_wt	104	3	13	0		0	
DON	1722_Ca	324_wt	104	1	8	0		0	
DON	1722_Ca	324_wt	104	2					
DON	1722_Ca	324_wt	104	3					
DON	1722_Ca	325_wt	104	1	11	0		0	
DON	1722_Ca	325_wt	104	2	11	0		0	
DON	1722_Ca	325_wt	104	3	14	0		0	
DON	1722_Ca	314_mut	105	1	8	0		0	
DON	1722_Ca	314_mut	105	2	16	0		0	
DON	1722_Ca	314_mut	105	3	16	0		0.5	
DON	1722_Ca	315_mut	105	1	8	0		0	
DON	1722_Ca	315_mut	105	2	16	0			
DON	1722_Ca	315_mut	105	3					
DON	1722_Ca	316_mut	105	1	8	0		0	
DON	1722_Ca	316_mut	105	2	14	0		0	
DON	1722_Ca	316_mut	105	3					
DON	6688_Ca	328_mut	106	1	7	0		0	
DON	6688_Ca	328_mut	106	2	13	0		0	
DON	6688_Ca	328_mut	106	3					
DON	6688_Ca	329_mut	106	1	7	0		0	
DON	6688_Ca	329_mut	106	2					
DON	6688_Ca	329_mut	106	3					
DON	6688_Ca	336_mut	106	1	7	0		0	
DON	6688_Ca	336_mut	106	2					
DON	6688_Ca	336_mut	106	3					
DON	6688_Ca	337_wt	107	1	7	0		0	
DON	6688_Ca	337_wt	107	2					
DON	6688_Ca	337_wt	107	3					
DON	6688_Ca	338_wt	107	1	7	0		0	
DON	6688_Ca	338_wt	107	2	14	0		0	
DON	6688_Ca	338_wt	107	3					
DON	6688_Ca	339_wt	107	1	6	0		0	
DON	6688_Ca	339_wt	107	2	13	0		0	
DON	6688_Ca	339_wt	107	3					
DON	6551_Ca	206_mut	108	1	10	0		0	

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Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	6551_Ca	206_mut	108	2	14	0		0	
DON	6551_Ca	206_mut	108	3	14	0		0	
DON	6551_Ca	208_mut	108	1					
DON	6551_Ca	208_mut	108	2					
DON	6551_Ca	208_mut	108	3					
DON	6551_Ca	209_mut	108	1	9	0		0	
DON	6551_Ca	209_mut	108	2	13	0		0	
DON	6551_Ca	209_mut	108	3	13	0		0	
DON	6551_Ca	209_mut	108	4	14	0		0	
DON	6046_Ca	231_wt	109	1	7	0		0	
DON	6046_Ca	231_wt	109	2	11	0		0	
DON	6046_Ca	231_wt	109	3	11	0		0	
DON	6046_Ca	232_wt	109	1	10	0		0	
DON	6046_Ca	232_wt	109	2	11	0		0	
DON	6046_Ca	232_wt	109	3					
DON	6046_Ca	233_wt	109	1	9	0		0	
DON	6046_Ca	233_wt	109	2	9	0		0	
DON	6046_Ca	233_wt	109	3	10	0		0	
DON	6046_Ca	234_wt	109	1	6	0		0	
DON	6046_Ca	234_wt	109	2	7	0		0	
DON	6046_Ca	234_wt	109	3	8	0		0	
DON	6046_Ca	234_wt	109	4	9	0		0	
DON	6046_Ca	234_wt	109	5	10	0		0	
DON	6046_Ca	235_wt	110	1	6	0		0	
DON	6046_Ca	235_wt	110	2	9	0		0	
DON	6046_Ca	235_wt	110	3	9	0		0	
DON	6046_Ca	235_wt	110	4	10	0		0	
DON	6046_Ca	236_wt	110	1	7	0		0	
DON	6046_Ca	236_wt	110	2	8	0		0	
DON	6046_Ca	236_wt	110	3	10	0		0	
DON	6046_Ca	236_wt	110	4	10	0		0	
DON	6046_Ca	237_wt	110	1	6	0		0	
DON	6046_Ca	237_wt	110	2	9	0		0	2?
DON	6046_Ca	237_wt	110	3	10	0		0	
DON	6046_Ca	237_wt	110	4	11	0		0	
DON	6046_Ca	226_mut	111	1	8	0		0	
DON	6046_Ca	226_mut	111	2	9	0		0	
DON	6046_Ca	226_mut	111	3	9	0		0	
DON	6046_Ca	227_mut	111	1	6	0		0	
DON	6046_Ca	227_mut	111	2	8	0		0	
DON	6046_Ca	227_mut	111	3	8	0		0	
DON	6046_Ca	227_mut	111	4	8	0		0	
DON	6046_Ca	228_mut	111	1	6	0		0	
DON	6046_Ca	228_mut	111	2	11	0		0?	
DON	6046_Ca	228_mut	111	3					
DON	6046_Ca	229_mut	112	1	7	0		0	
DON	6046_Ca	229_mut	112	2	9	0		0	
DON	6046_Ca	229_mut	112	3	14	0		0	
DON	6046_Ca	230_mut	112	1	6	0		0	
DON	6046_Ca	230_mut	112	2	8	0		0	
DON	6046_Ca	230_mut	112	3	10	0		0	
DON	6046_Ca	230_mut	112	4	11	0		0	
DON	6046_Ca	230_mut	112	5	14	0		0	
DON	8023_Ca	178_mut	113	1	7	0		0	
DON	8023_Ca	178_mut	113	2	9	0		0	
DON	8023_Ca	178_mut	113	3	10	0		0	
DON	8023_Ca	179_mut	113	1	6	0		0	
DON	8023_Ca	179_mut	113	2	8	0		0	
DON	8023_Ca	179_mut	113	2	9	0		0	
DON	8023_Ca	179_mut	113	3	9	0		0	
DON	8023_Ca	180_mut	113	1	8	0		0	
DON	8023_Ca	180_mut	113	2	9	0		0	
DON	8023_Ca	180_mut	113	3					
DON	8023_Ca	181_mut	114	1					
DON	8023_Ca	181_mut	114	2					
DON	8023_Ca	181_mut	114	3					

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Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	8023_Ca	182_mut	114	1	7	0		0	
DON	8023_Ca	182_mut	114	2	10	0		0	
DON	8023_Ca	182_mut	114	3	10	0		0	
DON	8023_Ca	182_mut	114	4	14	0		0	
DON	8023_Ca	182_mut	114	5	16	0		0	
DON	8023_Ca	183_mut	114	1					
DON	8023_Ca	183_mut	114	2					
DON	8023_Ca	183_mut	114	3					
DON	6504_Ca	306_wt	115	1	6	0		0	
DON	6504_Ca	306_wt	115	2	8	0		0	
DON	6504_Ca	306_wt	115	3					
DON	6504_Ca	307_wt	115	1	6	0		0	
DON	6504_Ca	307_wt	115	2	9	0		0	
DON	6504_Ca	307_wt	115	2	10	0		0	
DON	6504_Ca	307_wt	115	3	11	0		0	
DON	6504_Ca	308_wt	115	1	6	0		0	
DON	6504_Ca	308_wt	115	2	9	0		0	
DON	6504_Ca	308_wt	115	3	9	0		0	
DON	6504_Ca	309_wt	115	1	6	0		0	
DON	6504_Ca	309_wt	115	2	9	0		0	
DON	6504_Ca	309_wt	115	3	11	0		0	
DON	6504_Ca	310_wt	116	1	7	0		0	
DON	6504_Ca	310_wt	116	2	9	0		0	
DON	6504_Ca	310_wt	116	3	10	0		0	
DON	6504_Ca	310_wt	116	4	11	0		0	
DON	6504_Ca	311_wt	116	1	6	0		0	
DON	6504_Ca	311_wt	116	2	9	0		0	
DON	6504_Ca	311_wt	116	3	10	0		0	
DON	6504_Ca	311_wt	116	4	11	0		0	
DON	6504_Ca	312_wt	116	1	6	0		0	
DON	6504_Ca	312_wt	116	2	8	0		0	
DON	6504_Ca	312_wt	116	3					
DON	88_Gly	271_wt	117	1	6	0		0	
DON	88_Gly	271_wt	117	2	8	0		0	
DON	88_Gly	271_wt	117	3	8	0		0	
DON	88_Gly	271_wt	117	4	9	0		0	
DON	88_Gly	272_wt	117	1	6	0		0	
DON	88_Gly	272_wt	117	2	10	0		0	
DON	88_Gly	272_wt	117	3					
DON	88_Gly	273_wt	117	1	7	0		0	
DON	88_Gly	273_wt	117	2	9	0		0	
DON	88_Gly	273_wt	117	3	9	0		0	
DON	88_Gly	273_wt	117	4	10	0		0	
DON	88_Gly	275_wt	117	1	6	0		0	
DON	88_Gly	275_wt	117	2	9	0		0	
DON	88_Gly	275_wt	117	3					
DON	88_Gly	274_wt	118	1	7	0		0	
DON	88_Gly	274_wt	118	2	9	0		0	
DON	88_Gly	274_wt	118	3	10	0		0	
DON	88_Gly	276_wt	118	1					
DON	88_Gly	276_wt	118	2					
DON	88_Gly	276_wt	118	3					
DON	88_Gly	277_wt	118	1	7	0		0	
DON	88_Gly	277_wt	118	2	9	0		0	
DON	88_Gly	277_wt	118	3	10	0		0	
DON	88_Gly	277_wt	118	4	11	0		0	
DON	88_Gly	278_wt	118	1	7	0		0	
DON	88_Gly	278_wt	118	2	11	0		0	
DON	88_Gly	278_wt	118	3	12	0		0	
DON	88_Gly	264_mut	119	1	8	0		0	
DON	88_Gly	264_mut	119	2	10	0		0	
DON	88_Gly	264_mut	119	3					
DON	88_Gly	265_mut	119	1	8	0		0	
DON	88_Gly	265_mut	119	2	9	0		0	
DON	88_Gly	265_mut	119	3	11	0		0	
DON	88_Gly	265_mut	119	4	11	0		0	

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Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	88_Gly	266_mut	119	1	8	0		0	
DON	88_Gly	266_mut	119	2	10	0		0	
DON	88_Gly	266_mut	119	3	11	0		0	
DON	88_Gly	266_mut	119	4	11	0		0	
DON	88_Gly	260_mut	120	1	6	0		0	
DON	88_Gly	260_mut	120	2	8	0		0	
DON	88_Gly	260_mut	120	3	9	0		0	
DON	88_Gly	261_mut	120	1	7	0		0	
DON	88_Gly	261_mut	120	2	10	0		0	
DON	88_Gly	261_mut	120	3	10	0		0	
DON	88_Gly	262_mut	120	1	6	0		0	
DON	88_Gly	262_mut	120	2	10	0		0	
DON	88_Gly	262_mut	120	3					
DON	88_Gly	263_mut	120	1	8	0		0	
DON	88_Gly	263_mut	120	2	9	0		0	
DON	88_Gly	263_mut	120	3	9	0		0	
DON	3094_Gly	246_mut	121	1	8	0		0	
DON	3094_Gly	246_mut	121	2	10	0		0	
DON	3094_Gly	246_mut	121	3	11	0		0	
DON	3094_Gly	247_mut	121	1	8	0		0	
DON	3094_Gly	247_mut	121	2	11	0		0	
DON	3094_Gly	247_mut	121	3	11	0		0	
DON	3094_Gly	248_mut	121	1					
DON	3094_Gly	248_mut	121	2					
DON	3094_Gly	248_mut	121	3					
DON	3094_Gly	249_mut	121	1	7	0		0	
DON	3094_Gly	249_mut	121	2	11	2*0,5		2*0,5	
DON	3094_Gly	249_mut	121	3	11	0		0	
DON	6551_Ca	217_wt	122	1	7	0		0	
DON	6551_Ca	217_wt	122	2	10	0	2	0	2
DON	6551_Ca	217_wt	122	3	11	0		0	2
DON	6551_Ca	217_wt	122	4	11	0		0	2
DON	6551_Ca	219_wt	122	1	8	0		0	2
DON	6551_Ca	219_wt	122	2	11	0	2	2	
DON	6551_Ca	219_wt	122	3	11	0	2	2	
DON	6551_Ca	219_wt	122	4	12	0		2	
DON	6551_Ca	221_wt	122	1	7	0		0	
DON	6551_Ca	221_wt	122	2	10	0		0	
DON	6551_Ca	221_wt	122	3	10	0		0	
DON	6551_Ca	215_wt	123	1	9	0		0	
DON	6551_Ca	215_wt	123	2	11	0		0	
DON	6551_Ca	215_wt	123	3	12	0		0	
DON	6551_Ca	215_wt	123	4	12	0		0	
DON	6551_Ca	222_wt	123	1					
DON	6551_Ca	222_wt	123	2					
DON	6551_Ca	222_wt	123	3					
DON	6551_Ca	210_mut	124	1	9	0		0	
DON	6551_Ca	210_mut	124	2	12	0		0	
DON	6551_Ca	210_mut	124	3	14	0		0	
DON	6551_Ca	213_mut	124	1	11	0		0	
DON	6551_Ca	213_mut	124	2	11	0		0	
DON	6551_Ca	213_mut	124	3	11	0		0	
DON	6504_Ca	303_mut	125	1	9	0		0	
DON	6504_Ca	303_mut	125	2	10	0		0	
DON	6504_Ca	303_mut	125	3	11	0		0	
DON	6504_Ca	304_mut	125	1	8	0		0	
DON	6504_Ca	304_mut	125	2	10	0		0	
DON	6504_Ca	304_mut	125	3					
DON	6504_Ca	305_mut	125	1	6	0		0	
DON	6504_Ca	305_mut	125	2	10	0		0	
DON	6504_Ca	305_mut	125	3	11	0		0	
DON	641_Ubi	287_mut	126	1	6	0		0	
DON	641_Ubi	287_mut	126	2					
DON	641_Ubi	287_mut	126	3					
DON	641_Ubi	288_mut	126	1	7	0		0	
DON	641_Ubi	288_mut	126	2	12	0		0	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
DON	641_Ubi	288_mut	126	3	13	0		0	
DON	641_Ubi	289_mut	126	1	7	0		0	
DON	641_Ubi	289_mut	126	2	11	0		0	
DON	641_Ubi	289_mut	126	3	11	0		0	
DON	6688_Ca	330_mut	127	1	8	0		0	
DON	6688_Ca	330_mut	127	2	14	0		0	
DON	6688_Ca	330_mut	127	3					
DON	6688_Ca	331_mut	127	1	7	0		0	
DON	6688_Ca	331_mut	127	2	13	0		0	
DON	6688_Ca	331_mut	127	3	14	0		0	
DON	6688_Ca	332_mut	127	1	8	0		0	
DON	6688_Ca	332_mut	127	2	16	0		0	
DON	6688_Ca	332_mut	127	3					
DON	6688_Ca	340_wt	128	1	7	0		0	
DON	6688_Ca	340_wt	128	2					
DON	6688_Ca	340_wt	128	3					
DON	6688_Ca	341_wt	128	1	11	0		0	
DON	6688_Ca	341_wt	128	2	16	0		0	
DON	6688_Ca	341_wt	128	3					
DON	1722_Ca	319_mut	129	1	6	0		0	
DON	1722_Ca	319_mut	129	2	13	0		0	
DON	1722_Ca	319_mut	129	3	14	0		0	
DON	1722_Ca	320_mut	129	1	7	0		0	
DON	1722_Ca	320_mut	129	2	14	0		0	
DON	1722_Ca	320_mut	129	3					
DON	1722_Ca	326_wt	130	1	7	0		0	
DON	1722_Ca	326_wt	130	2	13	0		0	
DON	1722_Ca	326_wt	130	3	14	0		0	
DON	1722_Ca	327_wt	130	1	6	0		0	
DON	1722_Ca	327_wt	130	2	11	0		0	
DON	1722_Ca	327_wt	130	3	14	0		0	
Fus	1722_Ca	317_mut	255	1	7	2		2	
Fus	1722_Ca	317_mut	255	2	14	2		2	
Fus	1722_Ca	317_mut	255	3					
Fus	1722_Ca	318_mut	255	1	6	2		2	
Fus	1722_Ca	318_mut	255	2	12	1		2	
Fus	1722_Ca	318_mut	255	3	12	2		2	
Fus	88_Gly	267_wt	256	1	10	2		2	
Fus	88_Gly	267_wt	256	2	10	2		2	
Fus	88_Gly	267_wt	256	3					
Fus	88_Gly	268_wt	256	1	6	6		8	
Fus	88_Gly	268_wt	256	2	10	2		2	
Fus	88_Gly	268_wt	256	3					
Fus	88_Gly	269_wt	256	1	8	2		2	
Fus	88_Gly	269_wt	256	2	10	2		2	
Fus	88_Gly	269_wt	256	3	11	2		2	
Fus	88_Gly	270_wt	256	1	6	2		2	
Fus	88_Gly	270_wt	256	2	10	2		2	
Fus	88_Gly	270_wt	256	3	10	2		2	
Fus	88_Gly	270_wt	256	4	11	2		2	
Fus	8023_Ca	184_wt	257	1					
Fus	8023_Ca	184_wt	257	2					
Fus	8023_Ca	184_wt	257	3					
Fus	8023_Ca	185_wt	257	1	6	2		2	
Fus	8023_Ca	185_wt	257	2	9	2		2	
Fus	8023_Ca	185_wt	257	3	10	2		2	
Fus	8023_Ca	186_wt	257	1	6	2*0,5		2	
Fus	8023_Ca	186_wt	257	2	8	2		2	
Fus	8023_Ca	186_wt	257	3	10	2		2	
Fus	6551_Ca	214_wt	258	1	8	2		2	
Fus	6551_Ca	214_wt	258	2	10	2		2	
Fus	6551_Ca	214_wt	258	3	10	2		2	
Fus	6551_Ca	216_wt	258	1	9	2		2	
Fus	6551_Ca	216_wt	258	2					
Fus	6551_Ca	216_wt	258	3					
Fus	6551_Ca	218_wt	258	1	8	2		2	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
Fus	6551_Ca	218_wt	258	2	8	2		2	
Fus	6551_Ca	218_wt	258	3	9	2		2	5w
Fus	6551_Ca	218_wt	258	4	11	2		2	
Fus	6551_Ca	218_wt	258	5	11	2		2	
Fus	6551_Ca	207_mut	259	1	6	2		2	
Fus	6551_Ca	207_mut	259	2	8	2		2	
Fus	6551_Ca	207_mut	259	3	9	2		2	
Fus	6551_Ca	211_mut	259	1	8	2		2	
Fus	6551_Ca	211_mut	259	2	11	2		2	
Fus	6551_Ca	211_mut	259	3	11	2		2	
Fus	6551_Ca	212_mut	259	1	7	3		3	
Fus	6551_Ca	212_mut	258	2	9	2		2	
Fus	6551_Ca	212_mut	259	3	10	2		2	
Fus	6551_Ca	212_mut	259	4	10	2		2	
Fus	6046_Ca	223_mut	260	1					
Fus	6046_Ca	223_mut	260	2					
Fus	6046_Ca	223_mut	260	3					
Fus	6046_Ca	224_mut	260	1	6	1.5		2	
Fus	6046_Ca	224_mut	260	2	9	2		2	
Fus	6046_Ca	224_mut	260	3	10	2		2	
Fus	6046_Ca	225_mut	260	1	8	2		2	
Fus	6046_Ca	225_mut	260	2	9	2		2	
Fus	6046_Ca	225_mut	260	3	9	2		2	
Fus	6046_Ca	225_mut	260	4	9	2		2	
Fus	8023_Ca	175_mut	261	1					
Fus	8023_Ca	175_mut	261	2					
Fus	8023_Ca	175_mut	261	3					
Fus	8023_Ca	176_mut	261	1	7	2		2	
Fus	8023_Ca	176_mut	261	2					
Fus	8023_Ca	176_mut	261	3					
Fus	8023_Ca	177_mut	261	1	6	2		2	
Fus	8023_Ca	177_mut	261	2	9	2		2	
Fus	8023_Ca	177_mut	261	3	10	2		2	
Fus	1722_Ca	321_wt	262	1	6	2		2	
Fus	1722_Ca	321_wt	262	2	10	2		2	
Fus	1722_Ca	321_wt	262	3					
Fus	1722_Ca	322_wt	262	1	5	2		2	
Fus	1722_Ca	322_wt	262	2	9	2		2	
Fus	1722_Ca	322_wt	262	3					
Fus	448_Ubi	139_mut	276	1	10	6		6	
Fus	448_Ubi	139_mut	276	2	12	10		12	
Fus	448_Ubi	139_mut	276	3					
Fus	448_Ubi	140_mut	276	1	8	2		2	
Fus	448_Ubi	140_mut	276	2	11	1		2	
Fus	448_Ubi	140_mut	276	3					
Fus	448_Ubi	136_mut	277	1	6	2		2	
Fus	448_Ubi	136_mut	277	2	8	0.5		2	
Fus	448_Ubi	136_mut	277	3	10	2		2	
Fus	448_Ubi	137_mut	277	1	6	0.5		0.5	
Fus	448_Ubi	137_mut	277	2	10	2		2	
Fus	448_Ubi	137_mut	277	3	11	0.5		2	
Fus	448_Ubi	138_mut	277	1	8	1.5		2	
Fus	448_Ubi	138_mut	277	2	11	2		2	
Fus	448_Ubi	138_mut	277	3					
Fus	177_Ca	119_wt	278	1	6	2		2	
Fus	177_Ca	119_wt	278	2	9	2		2	
Fus	177_Ca	119_wt	278	3	9	2		2	
Fus	177_Ca	120_wt	278	1	7	2		2	
Fus	177_Ca	120_wt	278	2	9	2		2	
Fus	177_Ca	120_wt	278	3	9	2		2	
Fus	177_Ca	121_wt	278	1	7	2		2	
Fus	177_Ca	121_wt	278	2					
Fus	177_Ca	121_wt	278	3					
Fus	177_Ca	122_wt	278	1	9	2		2	
Fus	177_Ca	122_wt	278	2	9	2		2	
Fus	177_Ca	122_wt	278	3					

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Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
Fus	177_Ca	123_wt	278	1	9	2		2	
Fus	177_Ca	123_wt	278	2					
Fus	177_Ca	123_wt	278	3					
Fus	177_Ca	124_wt	279	1	10	2		2	
Fus	177_Ca	124_wt	279	2	10	2		2	
Fus	177_Ca	124_wt	279	3	11	2		2	
Fus	177_Ca	125_wt	279	1	8	2		2	
Fus	177_Ca	125_wt	279	2	11	2		2	
Fus	177_Ca	125_wt	279	3					
Fus	177_Ca	126_wt	279	1	7	2		2	
Fus	177_Ca	126_wt	279	2	9	2		2	
Fus	177_Ca	126_wt	279	3	10	2		2	
Fus	177_Ca	126_wt	279	4	11	2		2	
Fus	177_Ca	127_wt	279	1	8	2		2	
Fus	177_Ca	127_wt	279	2	9	2		2	
Fus	177_Ca	127_wt	279	3	11	2		2	
Fus	177_Ca	101_mut	280	1	6	20		20	
Fus	177_Ca	101_mut	280	2	9	8		18	
Fus	177_Ca	101_mut	280	3	10	8		9	
Fus	177_Ca	102_mut	280	1	8	2		2	
Fus	177_Ca	102_mut	280	2	9	8		16	
Fus	177_Ca	102_mut	280	3	10	2		2	
Fus	177_Ca	103_mut	280	1	6	16		16	
Fus	177_Ca	103_mut	280	2	8	12		20	
Fus	177_Ca	103_mut	280	3	9	8		20	
Fus	177_Ca	104_mut	280	1	7	2		3	
Fus	177_Ca	104_mut	280	2	10	10		10	
Fus	177_Ca	104_mut	280	3					
Fus	177_Ca	105_mut	281	1	7	2		2	
Fus	177_Ca	105_mut	281	2	7	20		20	
Fus	177_Ca	105_mut	281	3	10	2		2	
Fus	177_Ca	106_mut	281	1	8	18		18	
Fus	177_Ca	106_mut	281	2	9	20		20	
Fus	177_Ca	106_mut	281	3	10	10		12	
Fus	177_Ca	107_mut	281	1	8	18		18	
Fus	177_Ca	107_mut	281	2	10	8		10	
Fus	177_Ca	107_mut	281	3	10	14		20	
Fus	177_Ca	108_mut	281	1	9	12		20	
Fus	177_Ca	108_mut	281	2	10	18		18	
Fus	177_Ca	108_mut	281	3					
Fus	1399_Ca	86_wt	282	1	5	2		2	
Fus	1399_Ca	86_wt	282	2	7	2		2	
Fus	1399_Ca	86_wt	282	3	7	2		2	
Fus	1399_Ca	87_wt	282	1	6	2		2	
Fus	1399_Ca	87_wt	282	2	8	2		2	
Fus	1399_Ca	87_wt	282	3	8	2		2	
Fus	1399_Ca	88_wt	282	1	6	2		2	
Fus	1399_Ca	88_wt	282	2					
Fus	1399_Ca	88_wt	282	3					
Fus	1399_Ca	89_wt	282	1	6	2		2	
Fus	1399_Ca	89_wt	282	2	8	2		2	
Fus	1399_Ca	89_wt	282	3					
Fus	1399_Ca	90_wt	282	1	6	0.5		1.5	
Fus	1399_Ca	90_wt	282	2					
Fus	1399_Ca	90_wt	282	3					
Fus	2187_Ubi	162_wt	283	1	8	2		2	
Fus	2187_Ubi	162_wt	283	2	11	2		2	
Fus	2187_Ubi	162_wt	283	3					
Fus	2187_Ubi	163_wt	283	1	7	2		2	
Fus	2187_Ubi	163_wt	283	2	9	2		2	
Fus	2187_Ubi	163_wt	283	3	10	2		2	
Fus	2187_Ubi	164_wt	283	1	7	2		2	
Fus	2187_Ubi	164_wt	283	2	9	2		2	
Fus	2187_Ubi	164_wt	283	3					
Fus	2187_Ubi	165_wt	283	1	7	2		2	
Fus	2187_Ubi	165_wt	283	2	9	2		2	

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Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
Fus	2187_Ubi	165_wt	283	3	9	2		2	
Fus	2187_Ubi	153_mut	284	1	7	2		2	
Fus	2187_Ubi	153_mut	284	2	10	2		2	
Fus	2187_Ubi	153_mut	284	3	11	2		2	
Fus	2187_Ubi	154_mut	284	1	8	2		2	
Fus	2187_Ubi	154_mut	284	2	10	2		2	
Fus	2187_Ubi	154_mut	284	3	10	2		2	
Fus	2187_Ubi	155_mut	284	1	8	2		2	
Fus	2187_Ubi	155_mut	284	2	10	2		2	
Fus	2187_Ubi	155_mut	284	3	10	2		2	
Fus	8032_Ca	195_wt	285	1	4	3		3	
Fus	8032_Ca	195_wt	285	2	5	2		2	
Fus	8032_Ca	195_wt	285	3	5	1		0.5	
Fus	8032_Ca	196_wt	285	1	10	2		2	
Fus	8032_Ca	196_wt	285	2					
Fus	8032_Ca	196_wt	285	3					
Fus	8032_Ca	197_wt	285	1	8	2		3	
Fus	8032_Ca	197_wt	285	2	9	2		2	
Fus	8032_Ca	197_wt	285	3					
Fus	8032_Ca	198_wt	285	1	8	2		2	
Fus	8032_Ca	198_wt	285	2					
Fus	8032_Ca	198_wt	285	3					
Fus	8032_Ca	187_mut	286	1	3	2		2	
Fus	8032_Ca	187_mut	286	2	4	2		2	
Fus	8032_Ca	187_mut	286	3	4	2		2	
Fus	8032_Ca	187_mut	286	4	8	2		2	
Fus	8032_Ca	188_mut	286	1					
Fus	8032_Ca	188_mut	286	2					
Fus	8032_Ca	188_mut	286	3					
Fus	8032_Ca	189_mut	286	1	8	2		2	
Fus	8032_Ca	189_mut	286	2					
Fus	8032_Ca	189_mut	286	3					
Fus	256_Ubi	32_wt	287	1	10	2		2	
Fus	256_Ubi	32_wt	287	2					
Fus	256_Ubi	32_wt	287	3					
Fus	256_Ubi	33_wt	287	1	7	2		2	
Fus	256_Ubi	33_wt	287	2					
Fus	256_Ubi	33_wt	287	3					
Fus	256_Ubi	34_wt	287	1	7	2		2	
Fus	256_Ubi	34_wt	287	2	8	2		2	
Fus	256_Ubi	34_wt	287	3	9	2		2	
Fus	256_Ubi	35_wt	287	1	7	2		2	
Fus	256_Ubi	35_wt	287	2	9	2		2	
Fus	256_Ubi	35_wt	287	3	10	2		2	
Fus	256_Ubi	36_wt	287	1	6	2		2	
Fus	256_Ubi	36_wt	287	2					
Fus	256_Ubi	36_wt	287	3					
Fus	256_Ubi	37_wt	288	1	7	2		2	
Fus	256_Ubi	37_wt	288	2	10	2		2	
Fus	256_Ubi	37_wt	288	3					
Fus	256_Ubi	38_wt	288	1	7	2		2	
Fus	256_Ubi	38_wt	288	2	9	2		2	
Fus	256_Ubi	38_wt	288	3	10	2		2	
Fus	256_Ubi	39_wt	288	1	6	2		2	
Fus	256_Ubi	39_wt	288	2					
Fus	256_Ubi	39_wt	288	3					
Fus	256_Ubi	40_wt	288	1	9	2		2	
Fus	256_Ubi	40_wt	288	2	11	2		2	
Fus	256_Ubi	40_wt	288	3					
Fus	256_Ubi	41_wt	288	1	8	2		2	
Fus	256_Ubi	41_wt	288	2	8	2		2	
Fus	256_Ubi	41_wt	288	3	11	2		2	
Fus	256_Ubi	11_mut	289	1					
Fus	256_Ubi	11_mut	289	2					
Fus	256_Ubi	11_mut	289	3					
Fus	256_Ubi	12_mut	289	1	9	2		2	

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Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
Fus	256_Ubi	12_mut	289	2					
Fus	256_Ubi	12_mut	289	3					
Fus	256_Ubi	13_mut	289	1	6	8		12	
Fus	256_Ubi	13_mut	289	2	9	2		2	
Fus	256_Ubi	13_mut	289	3	10	2		2	
Fus	256_Ubi	14_mut	289	1	9	10		12	
Fus	256_Ubi	14_mut	289	2	9	2		2	
Fus	256_Ubi	14_mut	289	3					
Fus	256_Ubi	15_mut	289	1	7	2		6	
Fus	256_Ubi	15_mut	289	2	7	10		18	
Fus	256_Ubi	15_mut	289	3	8	10		16	
Fus	256_Ubi	15_mut	289	4	10	2		2	
Fus	256_Ubi	15_mut	289	5	9	10		14	
Fus	256_Ubi	16_mut	290	1	8	3		3	
Fus	256_Ubi	16_mut	290	2	10	6		6	
Fus	256_Ubi	16_mut	290	3					
Fus	256_Ubi	17_mut	290	1					
Fus	256_Ubi	17_mut	290	2					
Fus	256_Ubi	17_mut	290	3					
Fus	256_Ubi	18_mut	290	1	7	6		8	
Fus	256_Ubi	18_mut	290	2	10	4		5	
Fus	256_Ubi	18_mut	290	3	11	5		10	
Fus	256_Ubi	19_mut	290	1	7	10		20	
Fus	256_Ubi	19_mut	290	2	10	7		7	
Fus	256_Ubi	19_mut	290	3	11	5		10	
Fus	256_Ubi	20_mut	290	1	8	14		20	
Fus	256_Ubi	20_mut	290	2	11	4		12	
Fus	256_Ubi	20_mut	290	3					
Fus	1399_Ca	57_mut	291	1	6	12		20	
Fus	1399_Ca	57_mut	291	2	9	4		5	
Fus	1399_Ca	57_mut	291	3					
Fus	1399_Ca	58_mut	291	1					
Fus	1399_Ca	58_mut	291	2					
Fus	1399_Ca	58_mut	291	3					
Fus	1399_Ca	59_mut	291	1	6	2		2	
Fus	1399_Ca	59_mut	291	2	6	12		12	
Fus	1399_Ca	59_mut	291	3	9	8		10	
Fus	1399_Ca	60_mut	291	1	6	2		2	
Fus	1399_Ca	60_mut	291	2	8	12		18	
Fus	1399_Ca	60_mut	291	3	8	20		20	
Fus	1399_Ca	61_mut	291	1	8	2		5	
Fus	1399_Ca	61_mut	291	2	8	8		18	
Fus	1399_Ca	61_mut	291	3	8	10		18	
Fus	1399_Ca	62_mut	292	1	6	2		2	
Fus	1399_Ca	62_mut	292	2	8	7		7	w
Fus	1399_Ca	62_mut	292	3	9	2		2	
Fus	1399_Ca	63_mut	292	1	6	2		3	
Fus	1399_Ca	63_mut	292	2	8	3		3.5	
Fus	1399_Ca	63_mut	292	3	9	2		6	
Fus	1399_Ca	64_mut	292	1	5	2		2	
Fus	1399_Ca	64_mut	292	2	6	12		20	
Fus	1399_Ca	64_mut	292	3	7	10		14	
Fus	1399_Ca	64_mut	292	4	9	8		9	
Fus	1399_Ca	65_mut	292	1	7	2		2	
Fus	1399_Ca	65_mut	292	2					
Fus	1399_Ca	65_mut	292	3					
Fus	1399_Ca	66_mut	292	1	8	2		2	
Fus	1399_Ca	66_mut	292	2					
Fus	1399_Ca	66_mut	292	3					
Fus	1399_Ca	81_wt	293	1	7	2		2	
Fus	1399_Ca	81_wt	293	2					
Fus	1399_Ca	81_wt	293	3					
Fus	1399_Ca	82_wt	293	1	8	2		2	
Fus	1399_Ca	82_wt	293	2	9	2		2	
Fus	1399_Ca	82_wt	293	3					
Fus	1399_Ca	83_wt	293	1	7	2		2	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
Fus	1399_Ca	83_wt	293	2	8	2		2	
Fus	1399_Ca	83_wt	293	3	9	2		2	
Fus	1399_Ca	84_wt	293	1	6	1.5		2	
Fus	1399_Ca	84_wt	293	2	9	2		2	
Fus	1399_Ca	84_wt	293	3	9	2		2	
Fus	1399_Ca	85_wt	293	1	7	2		2	
Fus	1399_Ca	85_wt	293	2	9	2		2	
Fus	1399_Ca	85_wt	293	3	9	2		2	
Fus	1500_Gly	347_mut	294	1	6	2		2	
Fus	1500_Gly	347_mut	294	2	10	2		2	
Fus	1500_Gly	347_mut	294	3	11	2		2	
Fus	1500_Gly	348_mut	294	1	5	2		2	
Fus	1500_Gly	348_mut	294	2	8	2		2	
Fus	1500_Gly	348_mut	294	3	10	2		2	
Fus	1500_Gly	354_mut	294	1	5	2		2	
Fus	1500_Gly	354_mut	294	2	10	2		2	
Fus	1500_Gly	354_mut	294	3	10	2		2	
Fus	1500_Gly	360_wt	295	1	6	2		2	
Fus	1500_Gly	360_wt	295	2	6	2		2	
Fus	1500_Gly	360_wt	295	3	10	2		2	
Fus	1500_Gly	360_wt	295	4	10	2		2	
Fus	1500_Gly	360_wt	295	5	11	10		14	
Fus	1500_Gly	361_wt	295	1	10	2		2	
Fus	1500_Gly	361_wt	295	2	10	2		2	
Fus	1500_Gly	361_wt	295	3					
Fus	1500_Gly	362_wt	295	1	6	1.5		2	
Fus	1500_Gly	362_wt	295	2					
Fus	1500_Gly	362_wt	295	3					
Fus	1500_Gly	363_wt	296	1	6	2		2	
Fus	1500_Gly	363_wt	296	2	9	2		2	
Fus	1500_Gly	363_wt	296	3	10	2		2	
Fus	1500_Gly	364_wt	296	1	7	2		2	
Fus	1500_Gly	364_wt	296	2	10	2		2	
Fus	1500_Gly	364_wt	296	2	10	5		5	
Fus	1500_Gly	364_wt	296	3	11	2		2	
Fus	1500_Gly	365_wt	296	1	5	2		2	
Fus	1500_Gly	365_wt	296	2					
Fus	1500_Gly	365_wt	296	3					
Fus	448_Ubi	142_wt	297	1	7	2*0,5		-	
Fus	448_Ubi	142_wt	297	2	11	2		2	
Fus	448_Ubi	142_wt	297	3					
Fus	448_Ubi	143_wt	297	1	7	1.5		2	
Fus	448_Ubi	143_wt	297	2	9	2		2	
Fus	448_Ubi	143_wt	297	3					
Fus	448_Ubi	145_wt	297	1	6	1.5		2	
Fus	448_Ubi	145_wt	297	2	8	1.5		1.5	
Fus	448_Ubi	145_wt	297	3	11	2		2	
Fus	448_Ubi	146_wt	297	1					
Fus	448_Ubi	146_wt	297	2					
Fus	448_Ubi	146_wt	297	3					
Fus	6688_Ca	333_mut	298	1	12	2		2	
Fus	6688_Ca	333_mut	298	2	14	2		2	
Fus	6688_Ca	333_mut	298	3					
Fus	6688_Ca	334_mut	298	1	11	2		2	
Fus	6688_Ca	334_mut	298	2					
Fus	6688_Ca	334_mut	298	3					
Fus	6688_Ca	335_mut	298	1	7	1		2	
Fus	6688_Ca	335_mut	298	2	11	2		2	
Fus	6688_Ca	335_mut	298	3					
Fus	3094_Gly	242_mut	299	1	7	2		2	
Fus	3094_Gly	242_mut	299	2	10	8		10	
Fus	3094_Gly	242_mut	299	3					
Fus	3094_Gly	243_mut	299	1	8	2		2	
Fus	3094_Gly	243_mut	299	2					
Fus	3094_Gly	243_mut	299	3					
Fus	3094_Gly	244_mut	299	1	10	6		8	

Table continued on next page.

Table 48: Continued from previous page.

Treatment	Genotype	Wild-type or mutant-type	Pot	Head	Date	B1	weak B1	B2	weak B2
Fus	3094_Gly	244_mut	299	2	11	2		2	
Fus	3094_Gly	244_mut	299	3					
Fus	3094_Gly	245_mut	299	1	8	2		2	
Fus	3094_Gly	245_mut	299	2					
Fus	3094_Gly	245_mut	299	3					
Fus	641_Ubi	279_mut	300	1	6	2		2	
Fus	641_Ubi	279_mut	300	2	9	2		8	w
Fus	641_Ubi	279_mut	300	3	10	2		2	
Fus	641_Ubi	280_mut	300	1	6	14		14	
Fus	641_Ubi	280_mut	300	2	10	2		2	
Fus	641_Ubi	280_mut	300	3	11	7		7	
Fus	641_Ubi	281_mut	300	1	6	2		4	
Fus	641_Ubi	281_mut	300	2	10	2		2	
Fus	641_Ubi	281_mut	300	3					
Fus	641_Ubi	282_mut	300	1	6	2		2	
Fus	641_Ubi	282_mut	300	2	8	2	6w	8	w
Fus	641_Ubi	282_mut	300	3	10	2		2	
Fus	3094_Gly	238_mut	316	1	6	2		2	
Fus	3094_Gly	238_mut	316	2	9	2		2	
Fus	3094_Gly	238_mut	316	3	10	6		7	
Fus	3094_Gly	239_mut	316	1	7	2		2	
Fus	3094_Gly	239_mut	316	2	10	2		2	
Fus	3094_Gly	239_mut	316	3	10	8		10	
Fus	3094_Gly	240_mut	316	1	6	2		2	
Fus	3094_Gly	240_mut	316	2	9	2		2	
Fus	3094_Gly	240_mut	316	3	10	6		6	
Fus	3094_Gly	241_mut	316	1	6	2		2	
Fus	3094_Gly	241_mut	316	2	10	2		2	
Fus	3094_Gly	241_mut	316	3					
Fus	641_Ubi	290_wt	317	1	7	2		2	
Fus	641_Ubi	290_wt	317	2	11	2		2	
Fus	641_Ubi	290_wt	317	3					
Fus	641_Ubi	291_wt	317	1	7	2		2	
Fus	641_Ubi	291_wt	317	2	11	2		2	
Fus	641_Ubi	291_wt	317	3	11	2		2	
Fus	641_Ubi	292_wt	317	1	6	2		2	
Fus	641_Ubi		317	2	11	2		2	
Fus	641_Ubi	292_wt	317	3					
Fus	1500_Gly	349_mut	318	1	6	2		2	
Fus	1500_Gly	349_mut	318	2	10	2		2	
Fus	1500_Gly	349_mut	318	3	10	2		2	
Fus	1500_Gly	350_mut	318	1	5	2		2.5	
Fus	1500_Gly	350_mut	318	2	10	2		2	
Fus	1500_Gly	350_mut	318	3					
Fus	1500_Gly	351_mut	318	1	5	2		2	
Fus	1500_Gly	351_mut	318	2	10	2		2	
Fus	1500_Gly	351_mut	318	3					
Fus	1500_Gly	352_mut	318	1	6	2		2	
Fus	1500_Gly	352_mut	318	2	10	2		2	
Fus	1500_Gly	352_mut	318	3					

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