Genomic study on resistance to green rice leafhopper (Nephotettix cincticeps Uhler) of rice landraces (Oryza sativa L.) in Myanmar

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CONTENTS

GENERAL INTRODUCTION

The cultivated rice (*Oryza sativa*) is one of the important staple food for almost half of the world's population. Rice yield is affected by various abiotic or biotic stress; especially pests are one of the major factors. The green rice leafhopper (GRH, *Nephotettix cincticeps* (Uhler)) is a serious pest of cultivated rice (*Oryza sativa* L.) in the temperate regions of Asia (Ghauri 1971). GRH damages rice plants by sucking sap from the xylem and phloem of susceptible rice cultivars, thereby reducing yield (Pathak and Khan 1994). Additionally, GRH transmits viral diseases, such as rice dwarfs (Brar *et al*. 2009). Among various control measure, utilization of rice varieties with resistance gene is the most cost effective and efficient strategy to mitigate insect pest infestation.

To date, six genes and two quantitative trait loci (QTLs) conferring resistance to GRH have been identified in cultivated rice and its wild relatives (Table 1). However, with the emergence of new insect biotypes, the resistant cultivars have either lost or gradually reduced their resistance (Pathak and Heinrichs 1982). The GRH biotype 1, biotype 2 and biotype 3 were virulent against rice varieties with *GRH1, GRH2* and *GRH3* by rearing GRH on resistant rice cultivars (Hirae *et al*. 2007). Therefore, screening of large scale germplasm for GRH resistance, and exploration of resistance genes from these genetic resources play crucial role for development of durable GRH resistant varieties.

The cultivated rice has less sequence diversity than their wild relatives (Ikeda *et al*. 1993) due to favorable selection on agronomic traits for high yield. During domestication, only selected genes are passed to the next generation, thus narrowing the genetic diversity of cultivated rice and resulting in the loss of some insect resistance genes in cultivated rice (Chang 1985). However, most of the GRH resistance genes derived from *Oryza sativa*, the cultivated rice, especially Indica illustrating that *Oryza sativa* is the

valuable resource for genes conferring resistance to GRH. Therefore, we need to look back to *Oryza sativa* for exploitation of new GRH resistance genes.

The evolution of *Oryza sativa* primarily occurred along the foothills of Himalayan in South Asia, as well as in Southeast Asia and Southwest China (Chang 1976). However, the origins and domestication processes are still debated. A map of genomic variation constructed from 446 geographically diverse accessions of *Oryza rufipogon* and 1,083 *Oryza sativa* revealed that Japonica rice originated from a specific *Oryza rufipogon* population in southern China, while Indica rice later developed through crosses between Japonica rice and local wild rice as the initial cultivars spread into Southeast and South Asia (Huang *et al.* 2012). Regions like Nepal, northeast India, Upper Myanmar, Laos, Thailand, and southwest China are rich in rice genetic diversity based on isozyme distribution patterns (Nakagahra *et al*. 1975). Myanmar, located in a hotspot of rice genetic variation, has abundant rice genetic diversity. The diverse geographic zones, from the southern delta region to the northern mountainous areas, along with an appropriate agricultural system, also contribute to genetic diversity of rice. Studies consistently highlight the abundant genetic differences in Myanmar rice (Saw *et al.* 2006, Yamanaka *et al.* 2011, Wunna *et al.* 2016, Thant *et al.* 2021). A lot of rice landraces were preserved by indigenous people and many ethnic groups in Myanmar as on farm conservation (Yamanaka *et al*. 2011). Moreover, around 7,000 accessions are kept in the Seed Bank at the Department of Agricultural Research, Ministry of Agriculture, Livestock and Irrigation (MoALI), Yezin, Myanmar. Natural germplasm preserved through in situ and ex situ conservation is a valuable genetic reservoir for discovering new genes or alleles.

Advances in sequencing technologies have made it easier to study the genetic factors influencing the target traits. With the rapid development of genome sequencing

technologies, genome-wide association studies (GWAS) have emerged as a potent strategy for identifying QTLs in various crop germplasms using high-density single nucleotide variants and accurate phenotypic data (Zhao *et al*. 2011, Dhatt *et al*. 2021). GWAS has several advantages over bi-parental QTL mapping, including the ability to identify genes for specific traits in natural populations, and it offers higher resolution due to the many recombinations in natural populations. Over the past several years, GWAS have effectively identified QTLs associated with traits such as abiotic and biotic stress resistance in rice (Fujino *et al*. 2015, Dimkpa *et al*. 2016, Kang *et al*. 2016, Shi *et al*. 2021).

Although GWAS is a powerful approach for identifying QTLs for various traits of interest, it still remains some challenges such as occurrence of false associations due to missing genotyped data, low minor allele frequency and existence of population structure. A significant single nucleotide polymorphism (SNP) from a GWAS should not always be assumed as the causal variant and may require additional studies to map the precise location of the influential SNP (Bush and Moore 2012). Hence, the integration of linkage mapping and Genome-wide Association Studies (GWAS) serves as a complementary approach for identifying QTLs, addressing the limitations of each method. This combined strategy has been successful used in identifying QTLs for agronomic traits in maize (Liu *et al*. 2020) and husk morphology in maize (Cui *et al*. 2018).

Further, SNPs are one of the major limitation in GWAS because they usually provide only bi-allelic information at any single locus. An alternative approach to overcome this and increase allelic resolution is to perform haplotypes, a set of nearby genomic structural variations, such as polymorphic SNPs, with a strong linkage disequilibrium (LD) between them (Contreras-Soto *et al.* 2017). Haplotype blocks, which

combine two or more SNPs in strong LD, have higher heterozygosity due to their multi-allelic nature and are therefore more informative than single bi-allelic SNPs (Stephens *et al*. 2001, Lu *et al*. 2010). Recent applications of haplotype-based GWAS for various traits including yield, quality and stress tolerance in different plant species such as soybean (Contreras-Soto *et al*. 2017), wheat (Basile *et al*. 2019), rice (Wang *et al*. 2015) and maize (Yuan *et al*. 2019) revealed the success of haplotype analysis for identification of QTLs and crop improvement.

Plant-insect interactions are complex and multifaceted. In recent decades, there has been significant progress in understanding insect defense strategies, identifying insect resistance genes, and studying the molecular mechanisms of host-insect interactions across plant species. Plants defend against insect attacks with constitutive and induced strategies. Constitutive defenses include formation of physical barriers and production of chemical metabolites in plant to protect against insect attacks. Induced defenses involve the perception of insect effector molecules, leading to the production of specific chemicals, activation of signaling pathways, and changes in gene expression. Most insect resistance genes in plants encode receptors located on the plasma membrane or inside cells, highlighting the importance of induced defenses in plant immunity against insect attacks. Many insect pest resistance QTLs have been identified in major field crops over the past decades. However, only a limited number of actionable targets are known due to a lack of fine mapping and functional characterization. There is a growing need to clone and characterize candidate genes underlying the identified QTLs using fine mapping and map-based cloning approaches. These genes would help explain the molecular mechanisms of insect resistance in crop plants.

The first cloned resistance gene (R gene) was *Hm1* from maize (Johal and Briggs 1992) and the number of cloned R genes has steadily increase over years. Of the cloned R genes, sixty-one percent encode Nucleotide-binding domain and Leucine-rich Repeat (NLR) proteins (Kourelis and van der Hoorn 2018). NLR genes are a family of immune receptors that play a key role in plant defense against pathogens. They can recognize specific effector proteins produced by pathogen to suppress the host's immune response and trigger a robust immune response (Borrelli *et al*. 2018). Upon recognizing the effector, NLR proteins activate downstream signaling pathways including plant hormones, calcium ion signaling and transcription factor to induce the appropriate defense responses against insect infestation (Yan *et al*. 2023).

Rice plants defend against insects through various resistance mechanisms. Salicylic acid (SA), a key hormone, mediates the plant's response to brown planthopper (BPH) infestation (Li *et al*. 2021). SA not only plays a crucial role in the immediate response to BPH but also induces the production of other defensive compounds like phenolic, flavonoids those toxic and repellent to insects (Rashid *et al*. 2017). Rice plants respond to brown planthopper (BPH) attacks by upregulating specific genes associated with salicylic acid (SA) signaling, such as NPR1, WRKY, and PR genes leading to the activation of SA-related defense pathways and the subsequent synthesis of defensive compounds (Li *et al.* 2021). The role of physical barriers is one of the defense mechanism against phloem-feeding insects. Specifically, rice plants can modify their cell wall and cuticle to enhance resistance. Callose deposition at sieve plates and sieve-tube occlusion by proteins are common mechanism leading to phloem sealing and prevention of the insect from continuous feeding (Hao *et al.* 2008). Studying the molecular mechanisms underlying the cloned gene for the resistance shed light on the understanding of plant-

insect interaction and these insights are valuable for development of insect resistant rice varieties.

This study was conducted to explore the genetic basis of conferring resistance to GRH in Myanmar Indica landraces. The 224 accessions of Myanmar Indica diversity panel (MIDP) were evaluated for GRH resistance and associated genomic loci were identified through GWAS in Chapter I. In Chapter II, the causal gene of *GRH6*, which was detected in MIDP, was identified through gene cloning using the *Oryza nivara* bacterial artificial chromosome (BAC) clone IRGC105715-26E15, as *GRH6* had previously been mapped on *Oryza nivara* (Fujita *et al*. 2004). In Chapter III, haplotype variations in MIDP were assessed for three associated loci detected in the GWAS through haplotype analysis, and the accessions carrying the resistance haplotypes at these loci were estimated. In Chapter IV, the QTLs found in the GWAS were validated through bi-parental QTL mapping using F_2 populations derived from six MIDP donors, each possessing resistance haplotypes at the respective loci.

Table 1. Previously identified genes and QTLs conferring resistance to GRH

Genes/ QTLs	Chr.	Map position (Mbp)	Donor parent	Donor species	References
GRH1	5	6.7-9.7	IR24	*O. sativa*	Kadowaki *et al.* 2003
GRH1	5		Pe-bi-hun	*O. sativa*	Tamura *et al.* 1999
GRH1	5	8.1-8.8	Singwang	*O. sativa*	Park *et al.* 2013
GRH2	11L		Lepe dumai	*O. sativa*	Fukuta *et al.* 1998
GRH2	11L	23.3-24.7	DV85	*O. sativa*	Kadowaki *et al.* 2003
GRH3	6	15.1-19.7	Rantaj emas2	*O. sativa*	Saka *et al* . 2006
GRH3	6	20.0-20.4	Cheongnam	*O. sativa*	Hur *et al.* 2015
GRH4	3L		Lepe dumai	*O. sativa*	Fukuta *et al* . 1998
GRH4	3L	14.6-15.2	DV85	*O. sativa*	Kadowaki *et al.* 2003
GRH5	8L	26.5-27.6	W1962	*O. rufipogon*	Fujita *et al* . 2006
GRH6	4S	3.6-6.3	SML17	*O. sativa*	Tamura *et al.* 2004
GRH6	4S	4.4-4.7	IRGC105715	*O. nivira*	Fujita *et al.* 2004
qGRH4	4L	20.7-24.3	W1962	*O. rufipogon*	Fujita *et al.* 2010
qGRH9	9L	20.2-22.7	IRGC104038	*O. glaberrima*	Fujita *et al* . 2010

CHAPTER I

Genome-wide association study for green rice leafhopper (*Nephotettix cincticeps* (Uhler)) resistance of rice landraces (*O. sativa* L.) in Myanmar

Introduction

The green rice leafhopper (GRH), *Nephotettix cincticeps* (Uhler), is one of the major pest of cultivated rice in temperate Asia. One effective method to control this pest is by using rice varieties with resistance genes. To date, six genes and two quantitative trait loci (QTLs) conferring resistance to GRH have been identified in both cultivated and wild rice through genetic mapping using bi-parental segregation populations. However, the effectiveness of these resistance genes can be compromised by the emergence of new virulent insect biotypes. Consequently, it is recommended to utilize resistance genes derived from diverse germplasm to control the pest effectively and identifying these genes from diverse germplasm is crucial for plant breeders in developing sustainable pest-resistant rice varieties.

Genome-wide association study (GWAS) is an alternative approach of biparental QTL mapping for identifying the genomic regions associated with the target trait in diverse germplasms using genome-wide SNPs (Rafalski 2010). Over the past several years, GWAS have effectively identified quantitative trait loci (QTLs) associated with important traits in many crop species. In contrast to linkage mapping using bi-parental progeny, it is not necessary to develop the mapping population in GWAS because it identified the associated genomic region using the historic recombination events among the diverse germplasm. Moreover, once after genotyping the accessions, the genotypic data can be used for various traits in GWAS without further genotyping (Nakano *et al*.

2020). In linkage mapping, QTL with minor effects are difficult to be identified because of low resolution in bi-parental populations (Zhang *et al*. 2020). The rare alleles are also difficult to detected in segregating populations due to the lack of genetic diversity (Zhang *et al*. 2020). In contrast, GWAS detects the QTLs with high resolution based on the abundant genetic variations among diverse plant germplasms (Gao *et al*. 2019).

The efficiency of GWAS for identifying the true association is influenced by various factors such as the population size, population structure, and linkage disequilibrium (LD) and allele frequency. Increasing population size enhances the ability to identify the significant associations with larger effects that are more representative. A large population also overcome the challenges associations with rare genetic variants. (Alqudah *et al.* 2020). Population structure or genetic relatedness should be considered in genome association studies to avoid spurious association since the population contains related individuals (Sul *et al.* 2018). To solve the problem with population structure, the use of principal components (PC) to model relationships have been suggested (Price *et al.* 2006). High LD suggests that certain markers can efficiently capture the genetic information of nearby markers and a smaller number of genetic markers is sufficient to represent and cover the entire genome (Nakagahra and Hayashi 1977). Allele frequency is an important factor to avoid the mislead of GWAS output. Functional alleles that are present at a low frequency are difficult to be detected (Alqudah *et al*. 2020).

GWAS analysis with simple linear regression results in many false associations due to the existence of population structure and kinship among samples within the panel (Cortes *et al*. 2020). The mixed-model GWAS approach has been widely used to control false positives (type I errors) caused by the population structure and relatedness of the panel (Kang *et al*. 2008). The mixed model involved fixed effects of additive SNP effects

and population structure and random effects due to polygenic effects in the genetic background of lines defined as a kinship covariance matrix (Yu *et al*. 2006). The fixed and random effects were simultaneously estimated as the Best Linear Unbiased Estimates (BLUEs) and Best Linear Unbiased Predictions (BLUP) by solving a mixed-model equation (Henderson 1975). The genomic best linear unbiased prediction (GBLUP) uses a genomic relationship (GRM) matrix estimated from genome-wide SNP as an approximation of the kinship matrix (Hayes *et al.* 2009).

Recently, a Myanmar Indica diversity panel (MIDP) comprising of 250 Indica rice accessions, with genome-wide nucleotide variants identified through whole genome sequencing, was established to facilitate molecular genetic studies on Myanmar rice germplasm (Furuta *et al.* 2024). This panel is a precious genetic reservoir for exploiting the useful genes for various agronomically important traits and stress tolerance including GRH resistance. In this chapter, the 224 accessions of Myanmar Indica diversity panel (MIDP) were evaluated for GRH resistance and genome-wide association study was conducted using the phenotypic data and genome-wide single nucleotide polymorphism (SNPs) to identify the significant associations between the SNPs and GRH resistance.

Materials and method

Plant materials

In this study, the 224 accessions of the MIDP were evaluated for resistance to GRH (Table 2). The resistance aus cultivar from Bangladesh, DV85, which carries the resistance genes *GRH2* and *GRH4*, and the susceptible Japonica cultivar Taichung 65 (T65) were used as controls in the antibiosis test.

Table 2. Accessions of Myanmar Indica diversity panel (MIDP) used in genome-wide association study (GWAS) for GRH resistance

Sr. No.[a]	CC no.[b]	G no.[c]	Acc. no.[d]	Cultivar[e]	Region[f]
1	CC1	G1	000178	Paw San Hmwe	DR
2	CC10	G2	000621	Na Bya Sann	CDZ
3	CC11	G3	000660	Mine Taung	CDZ
4	CC14	G4	001351	Kauk Yin Phyu	NMR
5	CC26	G5	002288	Balu Swe	NMR
6	CC28	G6	002531	Phichenam	NMR
7	CC56	G7	003742	Cal Thau	WMR
8	CC82	G8	006123	Kauk Hnyin Nga Cheik	NMR
9	CC126	G10	007885	Ywat Oat Kauk Hnyin	ECR
10	CC163	G12	008805	Kauk Hnyin Phyu	CDZ
11	CC193	G13	000935	Khao Kan	EMR
12	CC206	G14	001228	Pa Din Thu Ma	DR
13	CC341	G15	000657	Thawt Gyi	CDZ
14	CC346	G16	000728	Kyaw Khaw	NMR
15	CC364	G17	001331	Pa Laung Kyar	CDZ
16	CC367	G18	001404	Sein Ti Saba	NMR
17	CC373	G20	001633	Ly Mee	ECR
18	CC376	G21	001768	Mya Wut Yee	EMR
19	CC379	G22	001781	Mauk Kon Gyi Zein	NMR
20	CC382	G23	002015	Kywe Phyu Saw	NMR
21	CC386	G24	002038	Ngwe Bya Gyi Kauk Hnyin Net	CDZ
22	CC401	G25	003023	Khauk Nan Swan	EMR
23	CC421	G27	005926	Khao Note Tit Own	DR
24	CC426	G28	006124	Ton Jan	NMR
25	CC442	G30	007836	Kauk Hnyin (white)	NMR
26	CC475	G31	010182	Naw Che	CDZ
27	CC491	G32	000581	Khaw Pae Ni	EMR
28	CC499	G33	000714	Chin Mee Shay	WMR
29	CC512	G34	007022	War Phyu	NMR
30	CC515	G35	000653	Gwa	DR
31	CC23	G36	002280	Ni Ka Re San Pyu	WCR
32	CC46	G38	003670	Kauk Hnyin Me	EMR
33	CC48	G39	003676	Khao Ann	EMR
34	CC57	G40	003751	Khao San Gyi	EMR
35	CC58	G41	003758	Khao Note Kin	EMR
36	CC59	G42	003764	Sa Ka Lay	EMR
37	CC65	G43	005808	Khao Pee Saing	EMR
38	CC66	G44	005841	Khao Khan Pu	EMR
39	CC67	G45	005850	Ma Kaw La	EMR
40	CC75	G46	006078	Kauk Hnyin Saba	EMR
41	CC76	G47	006080	Kauk Hnyin Saba	EMR
42	CC83	G48	006161	Khao Khin Phut	EMR
43	CC89	G49	006260	Bu Pa Yaung	EMR
44	CC92	G50	006273	Kauk Hnyin Me	EMR
45	CC99	G52	006429	Khao Sung Hung Yao	EMR
46	CC105	G53	006494	Khao Sai	EMR
47	CC116	G54	007443	Kauk Hnyin Net	EMR
48	CC147	G55	008027	Oat Kyai (B)	EMR

Table 2. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Acc. no.[d]	Cultivar[e]	Region[f]
49	CC164	G56	008807	Bul Mashal	NMR
50	CC166	G57	008859	Khao Pyi Seng	EMR
51	CC176	G58	010127	Muyinn Saba	CDZ
52	CC313	G59	009928	Saba Minn	CDZ
53	CC347	G62	000759	Khao Man Nain	EMR
54	CC365	G63	001368	Khao Khaw La	EMR
55	CC371	G64	001486	Yat Sauk	EMR
56	CC377	G65	001778	Ani Pyu Lar	WCR
57	CC400	G66	002842	Byat Kauk Hnyin	CDZ
58	CC439	G68	007444	Cheik Kauk Hnyin	EMR
59	CC445	G69	008377	Kauk Hnyin Nga Cheik	EMR
60	CC450	G70	002206	Khao Li Pon	EMR
61	CC493	G72	001594	Kauk Hnyin	CDZ
62	CC494	G73	001688	Kauk Hnyin Hnit	CDZ
63	CC7	G74	000563	Nga Bya Gyi	CDZ
64	CC9	G75	000586	Naw Phee	EMR
65	CC15	G76	001586	Maung Galay	CDZ
66	CC20	G77	002219	Mon Chee Sa Ba	CDZ
67	CC34	G78	003003	Nga Sein	CDZ
68	CC38	G79	003634	Nung Ding Thau	WMR
69	CC47	G80	003672	Kauk Hnyin	EMR
70	CC49	G81	003677	Yway	EMR
71	CC53	G82	003735	Biak Mang	WMR
72	CC61	G83	005731	Ywe	EMR
73	CC68	G85	005872	San Senn	WMR
74	CC71	G86	005940	Khao Lan Kan	EMR
75	CC73	G87	006015	Khao Swos	EMR
76	CC87	G89	006223	Saya Lay	EMR
77	CC94	G90	006287	Shwe War Yin	DR
78	CC96	G91	006418	Arshawarli	WCR
79	CC98	G92	006422	Kauk Chein	EMR
80	CC100	G93	006451	Boke Thwin Phyu	WCR
81	CC123	G97	007807	Hwon Yin	NMR
82	CC125	G98	007845	China 29	EMR
83	CC130	G99	007931	Kauk Hla Te Thi	EMR
84	CC131	G100	007933	Bu Ei Law	WMR
85	CC132	G101	007971	Mar Thu	EMR
86	CC139	G102	008570	Khauk Mwe Hnaung	ECR
87	CC142	G103	000024	Ku Taung Myo Tun	EMR
88	CC145	G105	007233	Ant paw	WMR
89	CC146	G106	008015	Sa Bong Thau	ECR
90	CC152	G107	008505	Pinto	WCR
91	CC158	G108	008740	Kari Let Yone	EMR
92	CC167	G109	008864	Khao Ho Mon(China 249)	CDZ
93	CC170	G110	008882	Shwe Att	WMR
94	CC171	G111	008885	Byit Thar	WCR
95	CC242	G113	002929	Kaoh Saing	WCR
96	CC436	G114	007048	Let To Ri	CDZ
97	CC469	G116	009152	Kyaung Ngo	EMR

Table 2. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Acc. no.[d]	Cultivar[e]	Region[f]
98	CC485	G118	010994	Khao Mit Nwe(Khao Per Won)	EMR
99	CC492	G119	001126	Taung Yoe Nga Cheik	EMR
100	CC12	G120	001003	Htaw Patt Sein	CDZ
101	CC17	G121	009022	Sae Ti Eine	DR
102	CC79	G122	006098	Hay Wun Dar	WCR
103	CC106	G123	007034	Japan Ni	WCR
104	CC143	G124	000770	Shwe Dinga	DR
105	CC198	G125	001089	Innma Ye Baw	DR
106	CC259	G127	002649	Khao Pin Lain	CDZ
107	CC316	G129	010017	Kyauk Yway (Thekone)	DR
108	CC318	G130	010123	Sin Ku	DR
109	CC326	G131	010936	Innma Kywe Kyae	DR
110	CC402	G132	003080	San Hmwe	CDZ
111	CC24	G133	002286	Mai Saw	CDZ
112	CC36	G134	003616	Duh Thim	WMR
113	CC153	G136	008506	Du Htung	WMR
114	CC184	G138	000008	Oat Pho-3	DR
115	CC192	G139	000607	Maung Pha Lo	DR
116	CC202	G140	001141	Ban Gauk Nga Sein	DR
117	CC208	G141	001400	Ma Kauk Hmwe	CDZ
118	CC217	G142	001537	Pegu Gyi	DR
119	CC224	G143	001939	Ya Gyaw	DR
120	CC235	G144	002516	Myaung Mya	DR
121	CC240	G145	002760	Shwe Thwe Tun	DR
122	CC257	G148	008369	Kauk Hnyin Ah Phyu	ECR
123	CC275	G149	008650	Kywat Shay	WCR
124	CC306	G150	009033	Ayeyarmin	CDZ
125	CC312	G151	009922	Kauk	CDZ
126	CC314	G152	009977	Shwe Ta Sote Yoe Ni	WCR
127	CC320	G153	010163	Kauk Hnyin	ECR
128	CC333	G155	000083	Rakhaing Pho	DR
129	CC342	G156	000663	Phi Zin	ECR
130	CC351	G157	000863	Shut Mee	EMR
131	CC354	G158	000973	Kywet Thwa	DR
132	CC360	G159	001092	Nga Shink Thway	DR
133	CC374	G160	001715	Ywet Thay Gyi	CDZ
134	CC403	G161	003099	Khauk Hauk	EMR
135	CC417	G162	005862	Ck Khwa	ECR
136	CC422	G163	005958	Kyun Shwe War	CDZ
137	CC423	G164	005964	Lone Pu	EMR
138	CC427	G165	006155	Hay Wun Dar	WCR
139	CC434	G166	006416	Phyine Zin Phyu	WCR
140	CC435	G167	006417	Phyine Zin Ni	WCR
141	CC458	G168	008712	Kyun Shwe War	ECR
142	CC461	G169	008837	King Bwee (A Thay)	WMR
143	CC463	G170	008894	Ma Ma Lay Saba	ECR
144	CC509	G171	005960	Eka Ye Saba	DR
145	CC513	G172	007829	Kauk Yin	CDZ
146	CC3	G174	000464	Ant Baw	WCR

Table 2. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Acc. no.[d]	Cultivar[e]	Region[f]
147	CC30	G175	002798	Thia Rall	WMR
148	CC151	G176	008504	Lai Fang	WMR
149	CC212	G178	001445	Shwe Kyi Tauk	EMR
150	CC239	G180	002687	Kyar Si Kauk Hnyin	ECR
151	CC273	G183	008625	Pauk War (Kauk Hnyin)	WCR
152	CC274	G184	008628	Myo Raw (Wan Me)	WCR
153	CC280	G186	008703	Kauk Hnyin	CDZ
154	CC286	G187	008745	Shan Ma Yin	CDZ
155	CC375	G188	001718	Bamaw Pyan	NMR
156	CC411	G190	003737	Gesma	WMR
157	CC418	G191	005870	Ye Pyar Nga Cheik	CDZ
158	CC444	G192	008365	Kauk Hnyin Me	ECR
159	CC460	G193	008829	Kauk Hnyin Yin (Myaungmya)	DR
160	CC477	G194	010197	Matho	WMR
161	CC484	G195	010990	Hauk Pee Sai	NMR
162	CC523	G198	003310	Shwe War Kauk Hnyin	DR
163	CC529	G199	007921	Nga Kyein Thee	WCR
164	CC13	G200	001063	Sit Pwa	DR
165	CC189	G201	000418	Thon Hnan Pwa	DR
166	CC195	G202	000982	Kywe Kye Kare	ECR
167	CC197	G203	001088	Pho Kaw Gyi	DR
168	CC211	G205	001411	Kyathaung Hmwe	DR
169	CC215	G207	001499	Balu Gyun Nga Sein	DR
170	CC221	G208	001663	Shwe War Tun	DR
171	CC223	G210	001928	Kauk Hnyin	DR
172	CC230	G212	002452	Thet Lat Nga Kyauk	DR
173	CC231	G213	002486	Wet Si Pyu	CDZ
174	CC236	G214	002582	Ya Gyaw Taung Pyan	DR
175	CC262	G215	001838	Tha Zin Tan	DR
176	CC270	G216	008476	Ma Kye Kye	ECR
177	CC271	G217	008493	Thone Hnan Saba	ECR
178	CC287	G219	008756	Palae Thwe	ECR
179	CC288	G220	008772	Shwe Yaung Htun	ECR
180	CC289	G221	008784	Mang Nuai Thau	WMR
181	CC292	G222	008801	Mee Tauk	DR
182	CC300	G223	008939	Tan Kang	WMR
183	CC304	G224	009013	Chan Nyeing	CDZ
184	CC307	G225	009142	Du Ya Boe	CDZ
185	CC308	G226	009143	Nga Pyar Gyi	CDZ
186	CC319	G227	010137	Kauk Hynin	CDZ
187	CC331	G228	010959	Duyabo	CDZ
188	CC352	G229	000921	Padan Nga Sein	DR
189	CC355	G230	001005	Yoe Sein	DR
190	CC361	G231	001107	Ka Lei	DR
191	CC395	G232	002386	Det Kwa	WCR
192	CC396	G233	002524	Khao Ma Phoot	EMR
193	CC406	G234	003223	Rakhaing Thu Ma	WCR
194	CC415	G237	005746	Kayin Gyi	ECR
195	CC459	G240	008822	Soke Phwa	DR

Table 2. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Acc. no.[d]	Cultivar[e]	Region[f]
196	CC490	G241	000439	Kyar Ama Gyi	EMR
197	CC500	G242	002293	Thet Yin Ka La Gyi	CDZ
198	CC505	G243	002666	Kywe Lan	CDZ
199	CC508	G244	005956	Taung Htate Pan	CDZ
200	CC517	G245	001960	Law Thaw Gyi	ECR
201	CC525	G246	005879	Mya Sein	WCR
202	CC528	G247	007019	Kywet Chay Sa Ba	WCR
203	CC6	G248	000533	Kauk Hnyin War	CDZ
204	CC8	G249	000569	Nar Tha Pu	CDZ
205	CC19	G250	002214	Chi Nyo	CDZ
206	CC22	G251	002273	Khao Phar	EMR
207	CC33	G252	002982	Naung Htu Phyu	NMR
208	CC35	G253	003182	Sein Wine Yin	CDZ
209	CC172	G254	008886	Taung O Nu (Ashay)	WMR
210	CC199	G255	001093	Yan Gon Nga Cheik	DR
211	CC232	G256	002495	Hmawbi Nga Kywe	DR
212	CC244	G257	002988	Moe Thee Myo	CDZ
213	CC248	G258	003161	In Gyin (A)	CDZ
214	CC265	G259	007299	Taung Hteik Pan	CDZ
215	CC268	G261	007382	Kaing Saba	EMR
216	CC291	G262	008800	Hlae Oo Khwe	DR
217	CC310	G263	009172	Kauk Hnyin Hmwe	CDZ
218	CC324	G264	010535	Byat	NMR
219	CC336	G265	000236	Chin Kauk Hnyin	CDZ
220	CC368	G266	001419	Kauk Hnyin Nga Cheik	CDZ
221	CC431	G268	006294	Sabanet	CDZ
222	CC468	G269	009141	Kauk Hnyin	NMR
223	CC483	G270	010983	Nagayar	ECR
224	CC516	G272	001514	Kauk Thwe Mee Shay	CDZ
225				Taichung 65 (Suceptible check)	
226				DV85 (Resistant check)	

[a] Serial number.

[b] Core collections of rice conserved in Seedbank of Department of Agricultural Research, Myanmar.

[c] Number of Myanmar Indica diversity panel designated as "G" where the accessions were whole genome sequenced.

[d] Accesion number lsited based on Seedbank of Department of Agricultural Research, Myanmar.

[e] Cultivar name given by local farmer.

[f] NMR, EMR, ECR, CDZ, DR, WMR, and WCR represent the northern mountain region, east mountain region, east costal region, central dry zone, delta region, west mountain region, and west costal region, respectively.

Antibiosis test for GRH resistance

The GRH strain used in this study was collected in Fukuoka, Japan, in 1991 and has been maintained by continuously rearing on seedlings of the susceptible Japonica cultivar Nipponbare under conditions of 25 ºC with 16 h of light and 8 h of darkness. This species is distributed in East Asia and is not found in South and Southeast Asia, including Myanmar. An antibiosis test was conducted to evaluate the resistance to GRH. Ten-day-old rice seedlings were infested with ten first-instar GRH nymphs in test tubes with five replicates per accession for Myanmar landraces, resistant and susceptible checks. Nymph mortality (%) for each accession was determined at 3, 5, and 7 days after infestation (DAI). Accessions exhibiting nymph mortality rates of less than 30 %, 30–70 %, and greater than 70% at 7DAI were categorized as susceptible (S), moderately resistant (MR), and highly resistant (HR) classes, respectively.

Genotyping

Whole genomic sequences of the diversity panel were determined with DNA nanoball-sequencing (DNB-seq) technologies. Cutting off of the bases with the low-quality score were conducted with the Trimmomatic software (Bolger *et al*. 2014). The qualified pair-end reads were mapped on 'Nipponbare' reference sequence (Os-Nipponbare-Reference-IRGSP-1.0, Kawahara *et al*. 2013) using the bwa-mem software (Li 2013). The variant call procedure follows the best practice workflow of the germline short variant discovery of the Genome analysis tool kit version 4 (GATK4) software (Poplin *et al*. 2017). The obtained variants were selected by hard filtering conditions; "QD < 2.0" --filter-name "QD2" -filter "QUAL < 30" --filter-name "QUAL30" -filter "SOR > 3.0" --filter-name "SOR3" -filter "FS > 60.0" --filter-name "FS60" -filter "MQ < 40.0" --filter-name

"MQ40" -filter "MQRankSum < -12.5" --filter-name "MQRankSum-12.5" -filter "ReadPosRankSum < -8.0" --filter-name "ReadPosRankSum-8". Using the vcftools software (Danecek *et al*. 2011), the variants were filtered with a minor allele frequency (maf) of more than 0.05 and genotype missing frequency of less than 0.1.

Genome-wide association study

GWAS using the genomic-best linear unbiased predication (GBLUP) was conducted using the R package rrBLUP (Endelman 2011). The genomic relationship matrix was computed from the 256,347 SNP loci randomly selected across the 12 chromosomes using the A.mat() function. A $-\log_{10}(p)$ value exceeding five was deemed significant for associations. Marker-trait association (MTA) was defined as the peak SNP with highest $-\log_{10}(p)$ value within the genomic region significantly associated with the phenotype. The linkage disequilibrium (LD) block containing significant MTAs was discerned using LD heat maps constructed with the R/LD heat map software (Shin *et al*. 2006). To identify candidate genes within the associated genomic region, the annotated genes with specific functions related to disease and insect resistance in the *Oryza sativa* reference sequence (Os-Nipponbare-Reference-IRGSP-1.0; RAP database) situated within the LD block were considered.

Results

Resistance in Myanmar rice cultivars to GRH

In this study, 224 accessions from the Myanmar Indica Diversity Panel (MIDP) were evaluated for resistance to GRH. Nymph mortality was assessed at 3 days after infestation (DAI), 5DAI, and 7DAI. The susceptible control, T65, displayed low GRH nymph

mortality (NM) of 1.1 % at 3DAI, 1.1 % at 5DAI, and 3.4 % at 7DAI. In contrast, the resistant control DV85 exhibited high nymph mortality rates of 99.5 % at 3DAI, 100 % at 5DAI, and 100 % at 7DAI (Figure 1, Table 3) Continuous phenotypic variations in nymph mortality, ranging from 0 to 100 %, were observed across the three evaluation times. Accessions showing MR and HR phenotypes increased from 3DAI to 7DAI (Figure 1, Table 3). At 3DAI, 76, 29, and 119 accessions belonged to the HR, MR, and S classes, respectively. However, the 83 accessions classified as the S class at 3DAI exhibited moderate to high resistance at 5DAI and 7DAI (Table 3). Of the 224 accessions, 36 exhibited S, 61 displayed MR, and 127 demonstrated HR at 7DAI (Table 3).

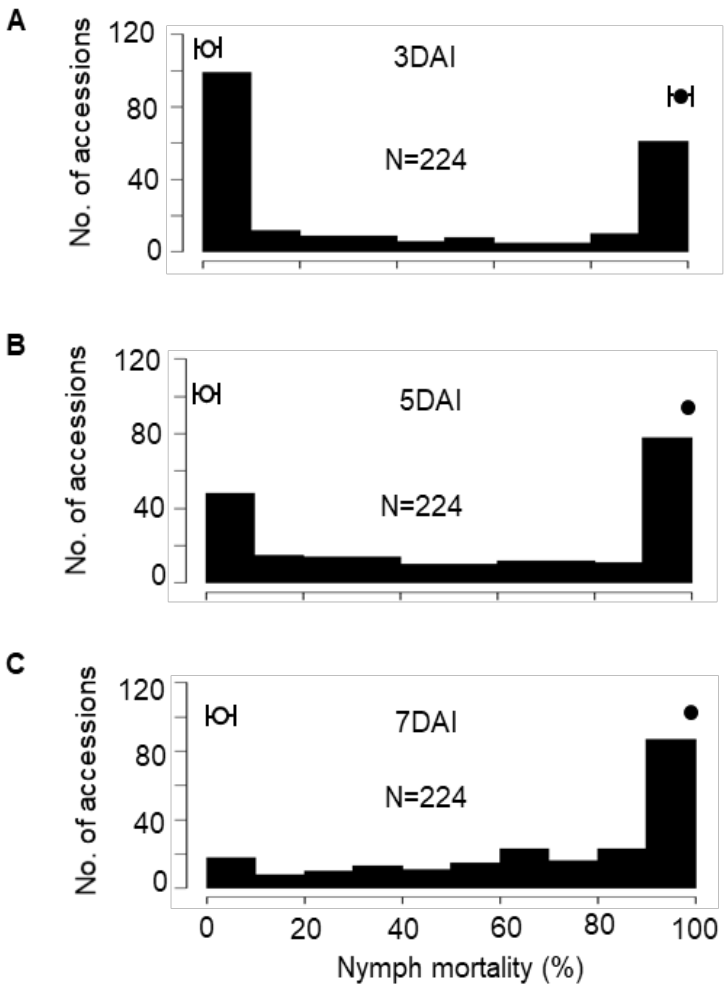

Fig. 1. Frequency distribution of nymph mortality of GRH in Myanmar Indica rice landraces at (A) 3 days after infestation (DAI), (B) 5DAI and (C) 7DAI. White and black circles represent phenotypic scores of Taichung65 (T65) for susceptible and DV85 for resistant controls.

Table 3. Nymph mortality (%) of green rice leafhopper in Myanmar Indica diversity panel (MIDP) (n=224) and classification for resistance to GRH

Sr. No.[a]	CC no.[b]	G no.[c]	Nymph mortality (%)[d]						Classification[e]
			3DAI		5DAI		7DAI		
			Mean	SE	Mean	SE	Mean	SE	
1	CC20	G77	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
2	CC12	G120	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
3	CC17	G121	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
4	CC79	G122	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
5	CC316	G129	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
6	CC318	G130	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
7	CC240	G145	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
8	CC314	G152	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
9	CC461	G169	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
10	CC418	G191	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
11	CC477	G194	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
12	CC13	G200	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
13	CC189	G201	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
14	CC197	G203	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
15	CC223	G210	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
16	CC270	G216	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
17	CC289	G221	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
18	CC307	G225	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
19	CC308	G226	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
20	CC361	G231	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
21	CC459	G240	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
22	CC525	G246	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
23	CC310	G263	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
24	CC368	G266	100.0 ±	0.0	100.0 ±	0.0	100.0 ±	0.0	HR
25	CC123	G97	98.0 ±	2.0	100.0 ±	0.0	100.0 ±	0.0	HR
26	CC198	G125	98.0 ±	2.0	100.0 ±	0.0	100.0 ±	0.0	HR
27	CC423	G164	98.0 ±	2.0	100.0 ±	0.0	100.0 ±	0.0	HR
28	CC505	G243	98.0 ±	2.0	98.0 ±	2.0	98.0 ±	2.0	HR
29	CC106	G123	97.8 ±	2.2	97.8 ±	2.2	97.8 ±	2.2	HR
30	CC326	G131	97.8 ±	2.2	100.0 ±	0.0	100.0 ±	0.0	HR
31	CC402	G132	97.8 ±	2.2	100.0 ±	0.0	100.0 ±	0.0	HR
32	CC36	G134	97.8 ±	2.2	97.8 ±	2.2	97.8 ±	2.2	HR
33	CC463	G170	97.8 ±	2.2	97.8 ±	2.2	100.0 ±	0.0	HR
34	CC331	G228	97.8 ±	2.2	97.8 ±	2.2	97.8 ±	2.2	HR
35	CC352	G229	97.8 ±	2.2	100.0 ±	0.0	100.0 ±	0.0	HR
36	CC395	G232	97.8 ±	2.2	97.8 ±	2.2	100.0 ±	0.0	HR
37	CC508	G244	97.8 ±	2.2	100.0 ±	0.0	100.0 ±	0.0	HR
38	CC517	G245	97.8 ±	2.2	100.0 ±	0.0	100.0 ±	0.0	HR
39	CC528	G247	97.8 ±	2.2	100.0 ±	0.0	100.0 ±	0.0	HR
40	CC450	G70	97.5 ±	2.5	100.0 ±	0.0	100.0 ±	0.0	HR
41	CC7	G74	97.5 ±	2.5	100.0 ±	0.0	100.0 ±	0.0	HR
42	CC34	G78	97.5 ±	2.5	97.5 ±	2.5	97.5 ±	2.5	HR
43	CC87	G89	97.5 ±	2.5	100.0 ±	0.0	100.0 ±	0.0	HR
44	CC152	G107	97.5 ±	2.5	100.0 ±	0.0	100.0 ±	0.0	HR
45	CC288	G220	97.5 ±	2.5	100.0 ±	0.0	100.0 ±	0.0	HR
46	CC396	G233	97.5 ±	2.5	100.0 ±	0.0	100.0 ±	0.0	HR
47	CC406	G234	97.5 ±	2.5	100.0 ±	0.0	100.0 ±	0.0	HR
48	CC257	G148	97.1 ±	2.9	100.0 ±	0.0	100.0 ±	0.0	HR

Table 3. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Nymph mortality (%)[d]						Classification[e]
			3DAI		5DAI		7DAI		
			Mean	SE	Mean	SE	Mean	SE	
49	CC439	G68	95.8 ±	2.6	98.0 ±	2.0	100.0 ±	0.0	HR
50	CC417	G162	95.8 ±	2.6	100.0 ±	0.0	100.0 ±	0.0	HR
51	CC98	G92	95.6 ±	4.4	97.8 ±	2.2	100.0 ±	0.0	HR
52	CC354	G158	95.6 ±	4.4	100.0 ±	0.0	100.0 ±	0.0	HR
53	CC434	G166	95.5 ±	2.8	97.5 ±	2.5	97.5 ±	2.5	HR
54	CC184	G138	94.9 ±	3.1	100.0 ±	0.0	100.0 ±	0.0	HR
55	CC304	G224	94.9 ±	3.1	97.1 ±	2.9	100.0 ±	0.0	HR
56	CC153	G136	93.8 ±	4.1	100.0 ±	0.0	100.0 ±	0.0	HR
57	CC485	G118	93.3 ±	2.7	100.0 ±	0.0	100.0 ±	0.0	HR
58	CC280	G186	93.3 ±	6.7	97.8 ±	2.2	100.0 ±	0.0	HR
59	CC292	G222	93.1 ±	4.5	95.3 ±	2.9	95.3 ±	2.9	HR
60	CC58	G41	92.8 ±	4.9	100.0 ±	0.0	100.0 ±	0.0	HR
61	CC427	G165	91.6 ±	3.8	100.0 ±	0.0	100.0 ±	0.0	HR
62	CC143	G124	90.0 ±	6.1	95.0 ±	5.0	100.0 ±	0.0	HR
63	CC158	G108	89.3 ±	6.9	95.6 ±	4.4	97.8 ±	2.2	HR
64	CC306	G150	88.9 ±	8.6	95.6 ±	2.7	100.0 ±	0.0	HR
65	CC444	G192	87.5 ±	3.9	97.5 ±	2.5	100.0 ±	0.0	HR
66	CC217	G142	87.0 ±	8.6	94.9 ±	3.1	100.0 ±	0.0	HR
67	CC96	G91	85.6 ±	5.0	92.7 ±	3.0	97.8 ±	2.2	HR
68	CC268	G261	85.3 ±	3.8	98.0 ±	2.0	100.0 ±	0.0	HR
69	CC212	G178	84.3 ±	4.5	95.3 ±	2.9	97.8 ±	2.2	HR
70	CC142	G103	81.9 ±	5.9	90.6 ±	5.8	93.1 ±	4.5	HR
71	CC15	G76	81.1 ±	6.7	97.8 ±	2.2	97.8 ±	2.2	HR
72	CC492	G119	80.0 ±	20.0	80.0 ±	20.0	95.6 ±	4.4	HR
73	CC202	G140	78.1 ±	5.3	84.3 ±	4.5	86.3 ±	4.5	HR
74	CC68	G85	74.0 ±	6.9	94.0 ±	6.0	98.0 ±	2.0	HR
75	CC342	G156	71.5 ±	8.9	93.5 ±	2.7	96.0 ±	2.4	HR
76	CC379	G22	71.4 ±	16.8	92.8 ±	4.9	97.5 ±	2.5	HR
77	CC139	G102	69.2 ±	7.7	88.9 ±	8.6	93.3 ±	4.4	HR
78	CC242	G113	66.1 ±	9.6	87.8 ±	0.3	95.0 ±	3.1	HR
79	CC26	G5	66.1 ±	3.5	91.8 ±	5.8	96.0 ±	4.0	HR
80	CC286	G187	64.4 ±	7.1	85.2 ±	1.9	92.7 ±	4.6	HR
81	CC491	G32	63.3 ±	7.6	80.9 ±	3.8	91.3 ±	2.2	HR
82	CC274	G184	59.0 ±	12.5	78.0 ±	13.4	80.5 ±	14.1	HR
83	CC523	G198	58.3 ±	6.0	82.8 ±	7.9	82.8 ±	7.9	HR
84	CC131	G100	56.4 ±	14.3	90.2 ±	2.5	95.3 ±	2.9	HR
85	CC10	G2	55.3 ±	10.0	91.1 ±	6.5	100.0 ±	0.0	HR
86	CC271	G217	54.7 ±	5.3	59.2 ±	4.0	68.2 ±	3.9	MR
87	CC125	G98	54.6 ±	5.4	79.8 ±	7.0	92.8 ±	4.9	HR
88	CC73	G87	51.6 ±	9.7	77.7 ±	5.6	85.8 ±	2.6	HR
89	CC236	G214	50.1 ±	11.9	65.7 ±	13.0	72.4 ±	11.4	HR
90	CC244	G257	48.3 ±	8.7	63.5 ±	3.2	74.1 ±	8.9	HR
91	CC259	G127	45.4 ±	13.4	64.0 ±	14.6	80.1 ±	6.0	HR
92	CC291	G262	44.5 ±	6.8	62.8 ±	11.2	73.9 ±	6.9	HR
93	CC239	G180	43.6 ±	9.0	62.2 ±	5.3	71.7 ±	4.4	HR
94	CC65	G43	41.9 ±	14.0	87.1 ±	4.8	89.3 ±	5.5	HR
95	CC147	G55	40.3 ±	13.7	64.7 ±	14.4	64.7 ±	14.4	MR
96	CC76	G47	37.5 ±	12.5	66.7 ±	11.5	87.5 ±	5.6	HR
97	CC94	G90	37.5 ±	8.3	53.8 ±	12.3	85.2 ±	4.4	HR

Table 3. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Nymph mortality (%)[d]						Classification[e]
			3DAI		5DAI		7DAI		
			Mean	SE	Mean	SE	Mean	SE	
98	CC512	G34	36.0 ±	12.1	84.0 ±	4.0	84.0 ±	4.0	HR
99	CC132	G101	34.9 ±	5.1	61.2 ±	11.0	70.2 ±	11.0	HR
100	CC8	G249	34.7 ±	13.4	73.8 ±	9.2	76.3 ±	8.7	HR
101	CC386	G24	34.4 ±	7.5	75.8 ±	5.2	83.1 ±	7.4	HR
102	CC529	G199	34.0 ±	3.9	78.7 ±	9.3	89.3 ±	4.7	HR
103	CC57	G40	32.9 ±	17.1	89.0 ±	5.1	100.0 ±	0.0	HR
104	CC490	G241	32.7 ±	11.8	75.1 ±	8.8	77.3 ±	9.8	HR
105	CC163	G12	17.8 ±	6.7	91.1 ±	2.3	97.5 ±	2.5	HR
106	CC313	G59	0.0 ±	0.0	79.6 ±	8.4	95.8 ±	2.6	HR
107	CC89	G49	2.5 ±	2.5	69.9 ±	15.3	95.0 ±	5.0	HR
108	CC23	G36	28.8 ±	8.8	82.7 ±	9.2	89.8 ±	7.7	HR
109	CC347	G62	14.4 ±	6.2	80.4 ±	7.8	89.1 ±	4.7	HR
110	CC426	G28	17.8 ±	8.3	78.4 ±	6.8	87.1 ±	6.3	HR
111	CC500	G242	28.3 ±	2.6	74.5 ±	9.0	86.2 ±	4.9	HR
112	CC382	G23	7.5 ±	6.0	45.6 ±	13.0	85.6 ±	17.6	HR
113	CC360	G159	0.0 ±	0.0	32.0 ±	11.5	85.1 ±	3.4	HR
114	CC499	G33	23.9 ±	10.6	52.2 ±	19.7	82.9 ±	11.1	HR
115	CC371	G64	0.0 ±	0.0	54.7 ±	7.0	82.3 ±	3.7	HR
116	CC376	G21	11.7 ±	7.3	55.8 ±	16.4	81.4 ±	7.6	HR
117	CC509	G171	15.7 ±	10.2	63.9 ±	6.8	81.1 ±	6.7	HR
118	CC208	G141	0.0 ±	0.0	30.6 ±	13.0	81.0 ±	6.5	HR
119	CC6	G248	4.0 ±	4.0	56.0 ±	14.4	80.5 ±	8.6	HR
120	CC374	G160	4.0 ±	2.4	44.9 ±	13.2	80.0 ±	20.0	HR
121	CC364	G17	28.4 ±	12.0	77.1 ±	7.1	79.6 ±	7.3	HR
122	CC83	G48	10.7 ±	5.5	66.8 ±	11.9	78.0 ±	8.8	HR
123	CC401	G25	4.7 ±	2.9	48.7 ±	9.4	77.2 ±	9.5	HR
124	CC421	G27	8.0 ±	8.0	43.2 ±	19.5	76.6 ±	12.7	HR
125	CC100	G93	21.0 ±	9.7	50.2 ±	7.9	74.9 ±	6.7	HR
126	CC403	G161	2.2 ±	2.2	35.7 ±	13.6	72.8 ±	5.3	HR
127	CC164	G56	6.7 ±	6.7	45.9 ±	10.5	71.1 ±	14.8	HR
128	CC435	G167	4.4 ±	2.7	17.8 ±	15.1	70.2 ±	8.2	HR
129	CC442	G30	25.4 ±	7.7	51.9 ±	4.7	68.8 ±	6.7	MR
130	CC215	G207	3.3 ±	3.3	13.1 ±	4.1	13.1 ±	4.1	MR
131	CC14	G4	0.0 ±	0.0	35.6 ±	9.5	68.5 ±	7.1	MR
132	CC71	G86	0.0 ±	0.0	14.5 ±	11.6	68.2 ±	9.8	MR
133	CC145	G105	23.0 ±	3.8	52.5 ±	12.0	68.1 ±	4.0	MR
134	CC82	G8	9.9 ±	6.1	31.8 ±	8.6	68.1 ±	6.1	MR
135	CC151	G176	2.0 ±	2.0	27.3 ±	10.5	68.0 ±	12.8	MR
136	CC146	G106	5.3 ±	3.4	39.6 ±	14.6	67.3 ±	18.2	MR
137	CC28	G6	0.0 ±	0.0	35.3 ±	13.4	67.3 ±	9.3	MR
138	CC324	G264	0.0 ±	0.0	6.4 ±	4.4	67.2 ±	7.6	MR
139	CC484	G195	2.5 ±	2.5	23.6 ±	3.3	66.7 ±	8.9	MR
140	CC47	G80	0.0 ±	0.0	18.4 ±	7.6	66.2 ±	9.1	MR
141	CC38	G79	11.1 ±	11.1	45.1 ±	9.5	66.0 ±	6.8	MR
142	CC346	G16	5.0 ±	5.0	61.8 ±	6.9	64.1 ±	8.3	MR
143	CC336	G265	0.0 ±	0.0	5.0 ±	5.0	63.8 ±	13.5	MR
144	CC422	G163	4.2 ±	2.6	9.2 ±	4.6	63.5 ±	7.8	MR
145	CC48	G39	22.9 ±	10.6	42.3 ±	14.3	62.8 ±	10.6	MR
146	CC99	G52	0.0 ±	0.0	45.0 ±	14.7	62.0 ±	16.9	MR

Table 3. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Nymph mortality (%)[d]						Classification[e]
			3DAI		5DAI		7DAI		
			Mean	SE	Mean	SE	Mean	SE	
147	CC67	G45	0.0 ±	0.0	54.4 ±	10.6	60.9 ±	12.4	MR
148	CC170	G110	16.0 ±	16.0	20.5 ±	15.1	60.9 ±	10.1	MR
149	CC33	G252	8.4 ±	5.2	35.1 ±	14.6	60.5 ±	17.6	MR
150	CC468	G269	0.0 ±	0.0	16.4 ±	9.3	59.4 ±	12.3	MR
151	CC341	G15	0.0 ±	0.0	16.1 ±	4.7	58.6 ±	5.7	MR
152	CC431	G268	0.0 ±	0.0	21.4 ±	8.9	58.6 ±	13.1	MR
153	CC172	G254	2.5 ±	2.5	14.2 ±	6.7	58.5 ±	9.7	MR
154	CC333	G155	2.2 ±	2.2	8.9 ±	6.5	58.1 ±	17.3	MR
155	CC56	G7	9.3 ±	2.4	31.5 ±	8.7	57.5 ±	6.6	MR
156	CC211	G205	14.4 ±	9.2	49.7 ±	18.3	57.0 ±	16.1	MR
157	CC11	G3	0.0 ±	0.0	31.3 ±	10.9	56.8 ±	9.2	MR
158	CC232	G256	5.0 ±	5.0	9.0 ±	5.6	55.3 ±	3.4	MR
159	CC365	G63	9.4 ±	5.8	49.2 ±	14.0	54.0 ±	13.0	MR
160	CC367	G18	2.2 ±	2.2	4.7 ±	2.9	53.6 ±	15.3	MR
161	CC320	G153	4.0 ±	4.0	27.2 ±	10.8	52.3 ±	8.0	MR
162	CC351	G157	0.0 ±	0.0	2.0 ±	2.0	51.4 ±	3.5	MR
163	CC515	G35	0.0 ±	0.0	31.7 ±	18.3	51.4 ±	14.5	MR
164	CC126	G10	0.0 ±	0.0	25.4 ±	12.1	50.5 ±	15.4	MR
165	CC373	G20	0.0 ±	0.0	9.7 ±	7.3	49.1 ±	14.4	MR
166	CC9	G75	8.3 ±	8.3	31.0 ±	9.8	47.6 ±	8.5	MR
167	CC265	G259	8.0 ±	4.9	14.2 ±	7.4	47.1 ±	16.4	MR
168	CC469	G116	0.0 ±	0.0	10.0 ±	10.0	47.0 ±	18.2	MR
169	CC193	G13	4.0 ±	2.4	26.2 ±	7.4	46.4 ±	12.5	MR
170	CC176	G58	0.0 ±	0.0	28.6 ±	18.2	45.3 ±	19.7	MR
171	CC300	G223	4.4 ±	2.7	13.3 ±	5.4	44.4 ±	9.3	MR
172	CC199	G255	0.0 ±	0.0	13.0 ±	7.3	44.2 ±	14.6	MR
173	CC24	G133	2.2 ±	2.2	20.2 ±	12.2	44.0 ±	9.2	MR
174	CC458	G168	0.0 ±	0.0	23.0 ±	10.4	40.6 ±	8.0	MR
175	CC411	G190	6.4 ±	4.4	25.6 ±	12.2	40.5 ±	9.0	MR
176	CC513	G172	0.0 ±	0.0	2.0 ±	2.0	39.6 ±	11.9	MR
177	CC516	G272	11.1 ±	6.1	16.8 ±	6.1	38.5 ±	9.9	MR
178	CC460	G193	2.0 ±	2.0	12.0 ±	9.7	38.2 ±	11.5	MR
179	CC167	G109	2.5 ±	2.5	4.3 ±	2.7	36.9 ±	14.4	MR
180	CC445	G69	12.2 ±	9.7	17.2 ±	9.4	35.8 ±	14.9	MR
181	CC248	G258	0.0 ±	0.0	2.5 ±	2.5	35.8 ±	12.6	MR
182	CC436	G114	6.4 ±	4.4	10.4 ±	4.7	35.1 ±	7.0	MR
183	CC493	G72	22.2 ±	13.0	34.2 ±	12.0	34.2 ±	12.0	MR
184	CC273	G183	4.2 ±	2.6	4.2 ±	2.6	31.9 ±	11.4	MR
185	CC92	G50	9.8 ±	6.1	19.2 ±	10.7	31.7 ±	14.7	MR
186	CC3	G174	0.0 ±	0.0	0.0 ±	0.0	31.4 ±	7.4	MR
187	CC1	G1	2.2 ±	2.2	20.0 ±	10.8	31.1 ±	15.4	MR
188	CC206	G14	0.0 ±	0.0	21.4 ±	10.8	30.6 ±	9.5	MR
189	CC46	G38	2.0 ±	2.0	6.5 ±	2.7	29.4 ±	8.5	S
190	CC230	G212	61.0 ±	19.9	70.5 ±	17.7	80.0 ±	20.4	HR
191	CC53	G82	0.0 ±	0.0	4.0 ±	2.4	28.2 ±	13.8	S
192	CC30	G175	0.0 ±	0.0	3.6 ±	3.6	28.1 ±	14.6	S
193	CC192	G139	17.4 ±	7.5	25.1 ±	10.8	27.9 ±	9.5	S
194	CC75	G46	2.5 ±	2.5	7.5 ±	3.1	26.4 ±	9.4	S
195	CC415	G237	2.0 ±	2.0	2.0 ±	2.0	23.6 ±	9.3	S

Table 3. Continued

Sr. No.[a]	CC no.[b]	G no.[c]	Nymph mortality (%)[d]						Classification[e]
			3DAI		5DAI		7DAI		
			Mean	SE	Mean	SE	Mean	SE	
196	CC105	G53	0.0 ±	0.0	5.0 ±	3.1	22.5 ±	10.0	S
197	CC221	G208	2.5 ±	2.5	5.0 ±	3.1	21.4 ±	4.2	S
198	CC319	G227	2.2 ±	2.2	4.2 ±	2.6	20.9 ±	9.5	S
199	CC375	G188	0.0 ±	0.0	2.2 ±	2.2	15.6 ±	6.7	S
200	CC275	G149	0.0 ±	0.0	4.4 ±	4.4	14.9 ±	7.0	S
201	CC231	G213	2.0 ±	2.0	9.0 ±	3.9	13.5 ±	6.2	S
202	CC312	G151	4.4 ±	2.7	4.4 ±	2.7	13.3 ±	8.2	S
203	CC262	G215	2.2 ±	2.2	2.2 ±	2.2	12.6 ±	4.0	S
204	CC19	G250	0.0 ±	0.0	0.0 ±	0.0	11.1 ±	5.0	S
205	CC130	G99	0.0 ±	0.0	0.0 ±	0.0	11.0 ±	5.5	S
206	CC166	G57	2.2 ±	2.2	4.2 ±	2.6	10.9 ±	8.6	S
207	CC171	G111	0.0 ±	0.0	0.0 ±	0.0	9.4 ±	4.6	S
208	CC287	G219	1.8 ±	1.8	4.0 ±	2.5	8.8 ±	4.2	S
209	CC377	G65	0.0 ±	0.0	0.0 ±	0.0	8.6 ±	8.6	S
210	CC195	G202	0.0 ±	0.0	0.0 ±	0.0	8.0 ±	5.8	S
211	CC66	G44	0.0 ±	0.0	0.0 ±	0.0	7.3 ±	4.6	S
212	CC22	G251	4.4 ±	4.4	6.9 ±	4.5	6.9 ±	4.5	S
213	CC475	G31	0.0 ±	0.0	6.7 ±	4.4	6.7 ±	4.4	S
214	CC400	G66	2.2 ±	2.2	2.2 ±	2.2	6.7 ±	4.4	S
215	CC235	G144	2.2 ±	2.2	2.2 ±	2.2	6.4 ±	4.4	S
216	CC116	G54	0.0 ±	0.0	2.0 ±	2.0	6.2 ±	4.1	S
217	CC224	G143	0.0 ±	0.0	2.5 ±	2.5	5.0 ±	5.0	S
218	CC355	G230	2.5 ±	2.5	4.7 ±	2.9	4.7 ±	2.9	S
219	CC61	G83	2.5 ±	2.5	2.5 ±	2.5	4.7 ±	2.9	S
220	CC483	G270	0.0 ±	0.0	0.0 ±	0.0	2.9 ±	2.9	S
221	CC49	G81	0.0 ±	0.0	2.5 ±	2.5	2.5 ±	2.5	S
222	CC494	G73	0.0 ±	0.0	0.0 ±	0.0	2.5 ±	2.5	S
223	CC59	G42	0.0 ±	0.0	2.0 ±	2.0	2.0 ±	2.0	S
224	CC35	G253	0.0 ±	0.0	0.0 ±	0.0	0.0 ±	0.0	S
225	T65		1.1 ±	0.8	1.1 ±	0.8	3.4 ±	1.5	S
226	DV85		99.5 ±	0.5	100.0 ±	0.0	100.0 ±	0.0	HR

[a] Serial number

[b] Core collections of rice conserved in Seedbank of Department of Agricultural Research, Myanmar

[c] Number of Myanmar Indica diversity panel designated as "G" where the accessions were whole genome sequenced

[d] Nymph mortality (%) of GRH evaluated at 3 days after infestation (DAI), 5DAI and 7DAI.

[e] S, MR and HR represent susceptible, moderately resistance, and highly resistance to GRH, respectively.

Genome-wide association study

GWAS using GBLUP for Myanmar Indica rice landraces was conducted to identify the genomic regions significantly associated with the resistance to GRH. On chromosome 5, the marker trait associations MTA5_3DAI, MTA5_5DAI, MTA5_7DAI were detected with highest -$\log_{10}(p)$ values of 21.3, 13.4, and 9.5, respectively (Fig. 2A-C, Table 4) within the apparent LD block spanning from 7.97 to 9.76 Mbp of the Nipponbare reference genome in the panel (Fig. 4A-B). Since this LD block contained the previously reported resistance gene, *GRH1* (Tamura *et al*. 1999, Kadowaki *et al*. 2003), which was delimited within a 670 kb region (Park *et al*. 2013), it was speculated that MTA5 corresponds to *GRH1* and predominantly control the resistance to GRH in MIDP (Fig. 4A-B).

On chromosome 4, the marker trait associations: MTA4_3DAI, MTA4_5DAI and MTA4_7DAI were detected with -$\log_{10}(p)$ values of 9.7, 8.2, and 5.6, respectively (Fig. 2A-C, Table 4). The genomic region with SNP markers having -$\log_{10}(p)$ values above suggestive threshold lines spanned between 4.3 and 5.1 Mbp regions of the Nipponbare reference genome in the panel (Fig. 5A). The previously reported gene, *GRH6* (Tamura *et al*. 2004, Fujita *et al*. 2004) delimited within 31.2 kb of the genomic region between the DNA markers c60k and 7L16f, corresponding to 4,460,301 – 4,491,541 bp in the reference sequence of Nipponbare (Phi *et al*. 2019) was located in this genomic region (Fig. 5A). However, the candidate region of *GRH6* and the peak SNPs of MTA4 at 3DAI, 5DAI, and 7DAI were included in the weak LD block (Fig. 5A-B) and approximately 400kb far from each other. Therefore, it remained need to confirm whether *GRH6* was candidate gene for this genomic region or not.

On chromosome 11, the marker trait associations on chromosome 11 (MTA11_5DAI and MTA11_7DAI) were detected at the later stages of infestation at

5DAI and 7DAI (Fig. 2B-C, Table 4,). The LD block located closely to MTA11_5DAI and MTA11_7DAI ranged from 27.11 to 27.12 Mbp of the Nipponbare reference genome in the panel (Fig. 6A-B). No previously identified genes or QTLs for GRH resistance were found within this region, suggesting that MTA11 was likely to be a novel QTL for GRH resistance.

Quantile-quantile (QQ) plots of -$\log_{10}(p)$ at 3DAI, 5DAI, and 7DAI show inflated -$\log_{10}(p)$ values from the expected distribution under the null hypothesis, where there are no QTL. However, as the SNPs with inflated -$\log_{10}(p)$ values were derived from the MTA4, MTA5, and MTA11 regions, these high -$\log_{10}(p)$ values were considered to be derived from significant QTLs (Fig. 3).

GWAS of GRH resistance in MIDP after excluding the predominance effect of GRH1

To examine more precise locations of the detected MTAs on chromosomes 4 and 11, haplotypes at the most predominantly affected locus *GRH1* for the GRH resistance in MDIP was defined, and GWAS was performed again using the MIDP accessions without the accessions carrying the resistant haplotype at *GRH1* (*refer haplotype analysis section in Chapter III*).

Next, the GWAS using the 184 accessions carrying the susceptible haplotype at *GRH1* (Hap*GRH1*B) was conducted. The marker-trait associations on chromosome 4, MTA4*_3DAI, MTA4*_5DAI, and MTA4*_7DAI, were detected at 4,791,920 bp and 4,404,524 bp with the -log10(*p*) values of 11.2, 7.8 and 5.9 respectively (Fig. 7, Table 5). The second highest peak SNP detected at 3DAI was located at 4,481,803 bp within the high-resolution mapping region of *GRH6* (Fig. 8A-B). Other significantly associated SNPs at 5DAI and 7DAI were observed around the high-resolution mapping region of

GRH6 (Fig. 8A-B). These results suggested that removing the resistant accessions harboring the resistant haplotype at *GRH1* (Hap*GRH1*A) allow to more accurately finding marker-trait associations around the *GRH6* region. Similarly, MTA11*_5DAI and MTA11*_7DAI were identified at 27,121,054 bp with the -log10(*p*) values of 7.0 and 7.3 respectively (Fig. 9A-B, Table 5). These positions were similar with the result of previous GWAS (Fig. 6A-B).

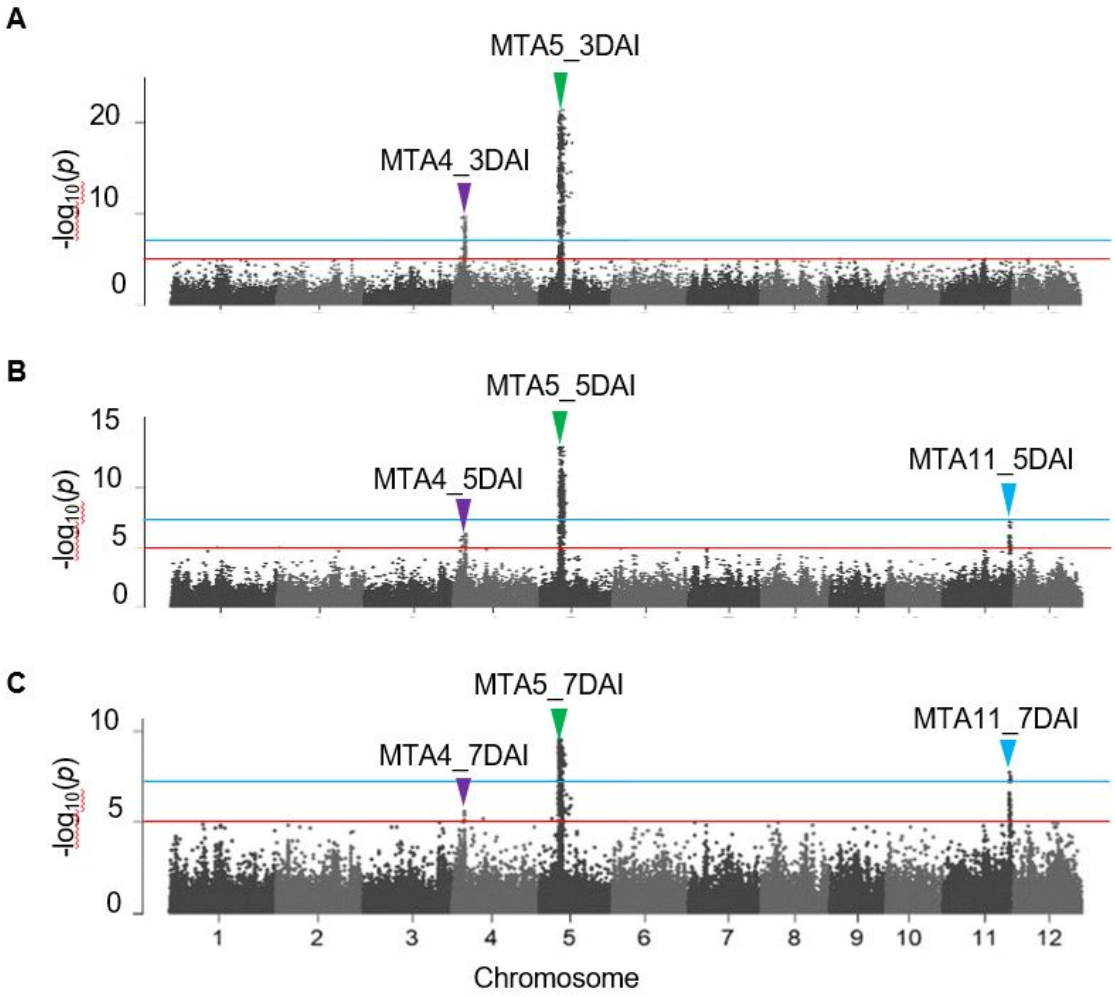

Fig. 2. Manhattan plots for genome-wide associations of nymph mortality (%) of green rice leafhopper infested to the accessions of MIDP (n=224) at (A) 3DAI, (B) 5DAI and (C) 7DAI. The red and blue horizontal lines represent suggestive and significant threshold levels at α= 1e-5 and α = 5e-8, respectively indicating the possible locus conferring resistance to GRH. The purple, green and blue arrowheads represent peak -log$_{10}$(p) positions at the marker-trait associations on chromosome 4 (MTA4), chromosome 5 (MTA5), and chromosome 11 (MTA11), respectively.

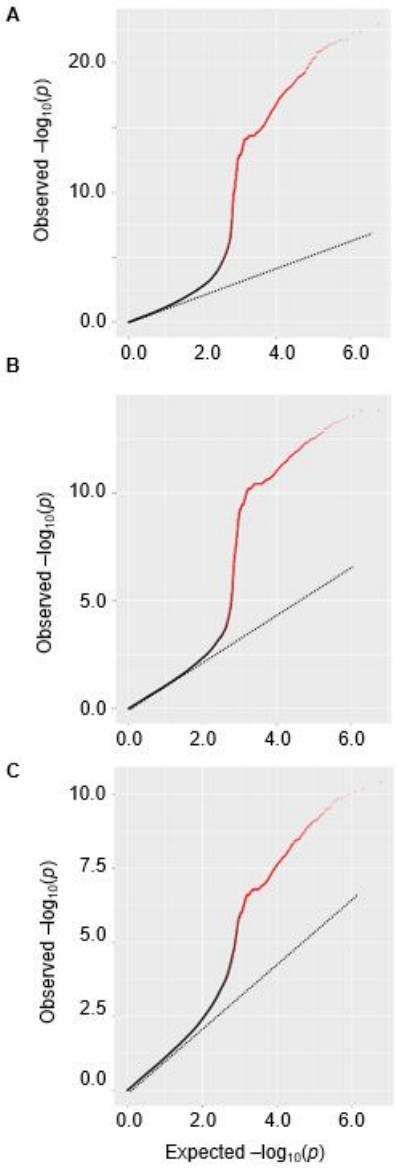

Fig. 3. Quantile-quantile plots of the obtained *p* distribution in GWAS for nymph mortality with GRH resistance in Myanmar Indica rice landraces (n=224) at (A) three days after infestation (DAI), (B) 5DAI, and (C) 7DAI. SNP markers in the MTA4, MTA5, and MTA11 regions are shown in red. The diagonal dotted lines represent the expected distribution under the null hypothesis, where there are no QTL.

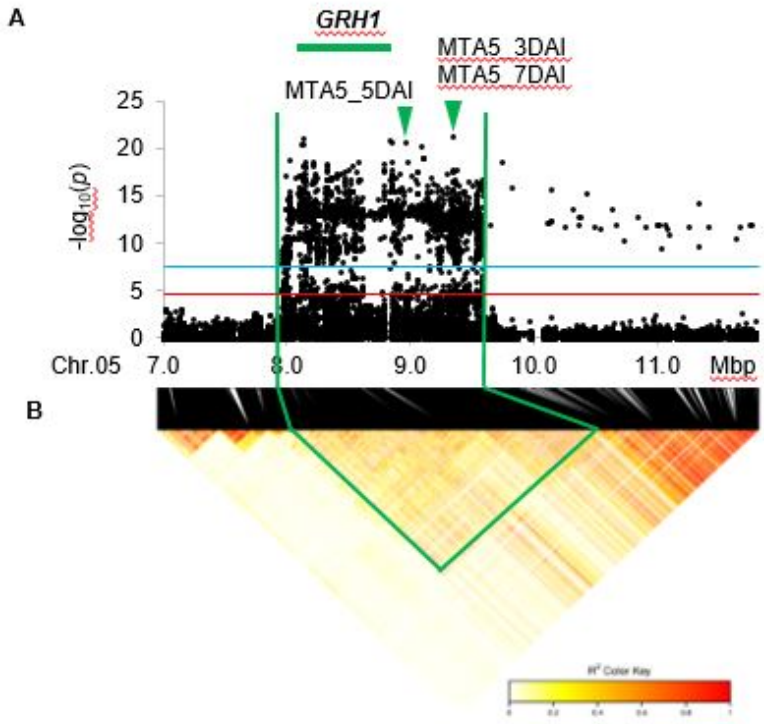

Fig. 4. Significant associations between SNP markers and nymph mortality (%) of green rice leafhopper (GRH) in MIDP (N=224) around the region of marker-trait associations on chromosome 5 (MTA5) at 3 days after infestation of GRH. (A) Local Manhattan plot around MTA5 region. The red and blue horizontal lines represent suggestive and significant threshold levels at α= 1e-5 and α = 5e-8, respectively. The green arrowheads represent peak -$\log_{10}(p)$ positions at the 3DAI, 5DAI and 7DAI. Green rectangle indicates the fine mapping region of previously reported gene: *GRH1* (Park *et al.* 2013) (B) Linkage disequilibrium (LD) heat map around MTA5 region. Green lines represent the region containing the SNP markers with high LD values.

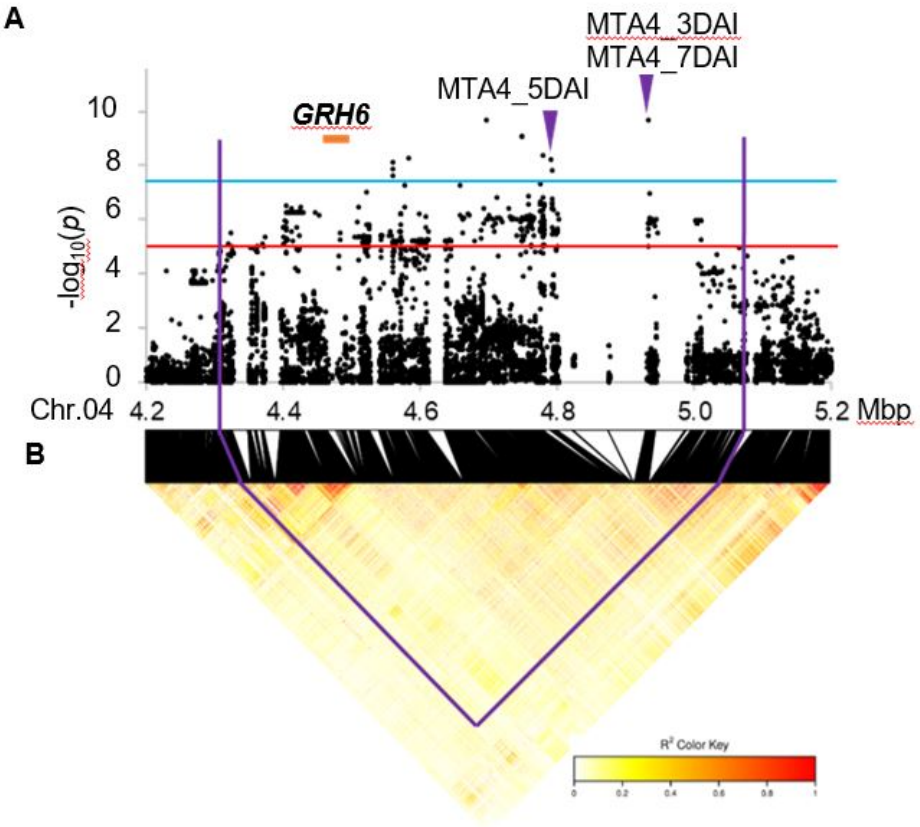

Fig. 5. Significant associations between SNP markers and nymph mortality (%) of green rice leafhopper (GRH) in MIDP (N=224) around the region of marker-trait associations on chromosome 4 (MTA4) at 3 days after infestation of GRH. (A) Local Manhattan plot around MTA4 region. The red and blue horizontal lines represent suggestive and significant threshold levels at α= 1e-5 and α = 5e-8, respectively. The purple arrowheads represent peak -$\log_{10}(p)$ positions at the 3DAI, 5DAI and 7DAI. Orange rectangle indicates the high resolution mapping region of previously reported gene: *GRH6* (Phi *et al.* 2013) (B) Linkage disequilibrium (LD) heat map around MTA4 region. Purple lines represent the region containing the SNP markers with high LD values.

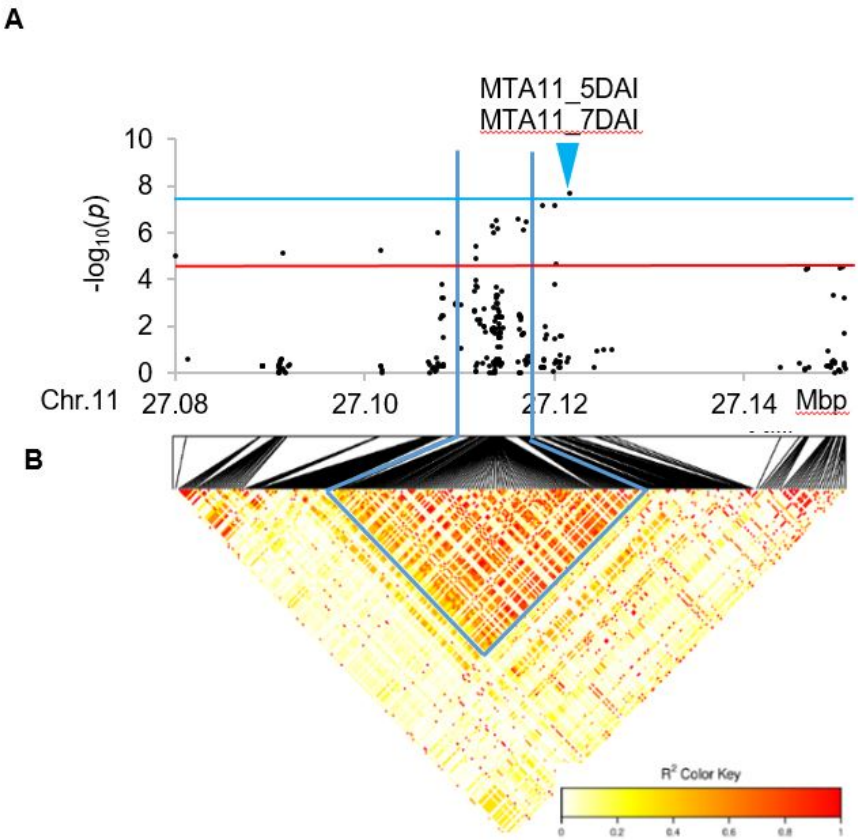

Fig. 6. Significant associations between SNP markers and nymph mortality (%) of green rice leafhopper (GRH) in MIDP (N=224) around the region of marker-trait associations on chromosome 11 (MTA11) at 7 days after infestation of GRH. (A) Local Manhattan plot around MTA11 region. The red and blue horizontal lines represent suggestive and significant threshold levels at α= 1e-5 and α = 5e-8, respectively. The blue arrowheads represent peak -$\log_{10}(p)$ positions at the 5DAI and 7DAI. (B) Linkage disequilibrium (LD) heat map around MTA11 region. Blue lines represent the region containing the SNP markers with high LD values

Table 4. Marker traits associations conferring resistance to GRH based on genome-wide association study in Myanmar Indica rice landraces (n=224)

Marker trait association (MTA)	Chr.	Position of MTAs (bp)	-log$_{10}$(p)	SNP allele (Allele 1/ Allele 2)	NM (%) [a]		Reported gene flanking to MTA
					Allele 1 homozygous	Allele 2 homozygous	
MTA4_3DAI	4	4,932,132	9.7	A/G	35.0	97.2	*GRH6*
MTA5_3DAI	5	9,349,253	21.3	G/A	28.3	97.8	*GRH1*
MTA4_5DAI	4	4,791,920	8.2	A/G	50.2	96.1	*GRH6*
MTA5_5DAI	5	8,952,557	13.4	G/T	29.1	98.1	*GRH1*
MTA11_5DAI	11	27,121,054	7.1	C/A	52.5	75.7	
MTA4_7DAI	4	4,932,132	5.6	A/G	66.1	99.7	*GRH6*
MTA5_7DAI	5	9,349,253	9.5	G/A	62.3	99.6	*GRH1*
MTA11_7DAI	11	27,121,054	7.7	C/A	66.0	86.5	

[a] NM represent nymph mortality (%) of GRH.

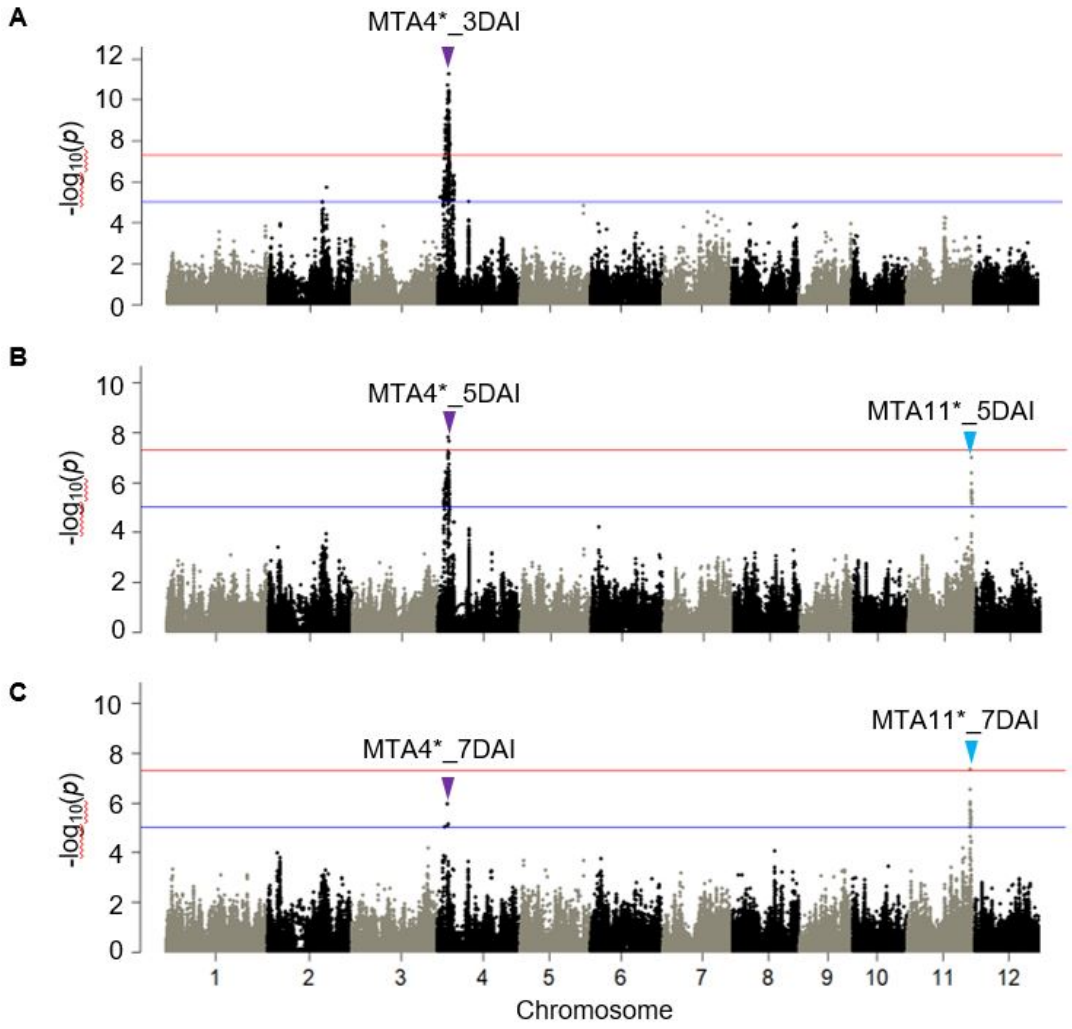

Fig. 7. Manhattan plots for genome-wide associations of nymph mortality (%) of green rice leafhopper infested to the accessions of MIDP (n=184) after selecting the accessions carrying susceptible haplotype(Hap*GRH1*B) at *GRH1* (*Refer to haplotype analysis in Chapter III*) at (A) 3DAI, (B) 5DAI and (C) 7DAI. The blue and red horizontal lines represent suggestive and significant threshold levels at α= 1e-5 and α = 5e-8, respectively indicating the possible locus conferring resistance to GRH. The purple and blue arrowheads represent peak -log$_{10}$(*p*) positions at the marker-trait associations on chromosome 4 (MTA4) and chromosome 11 (MTA11), respectively.

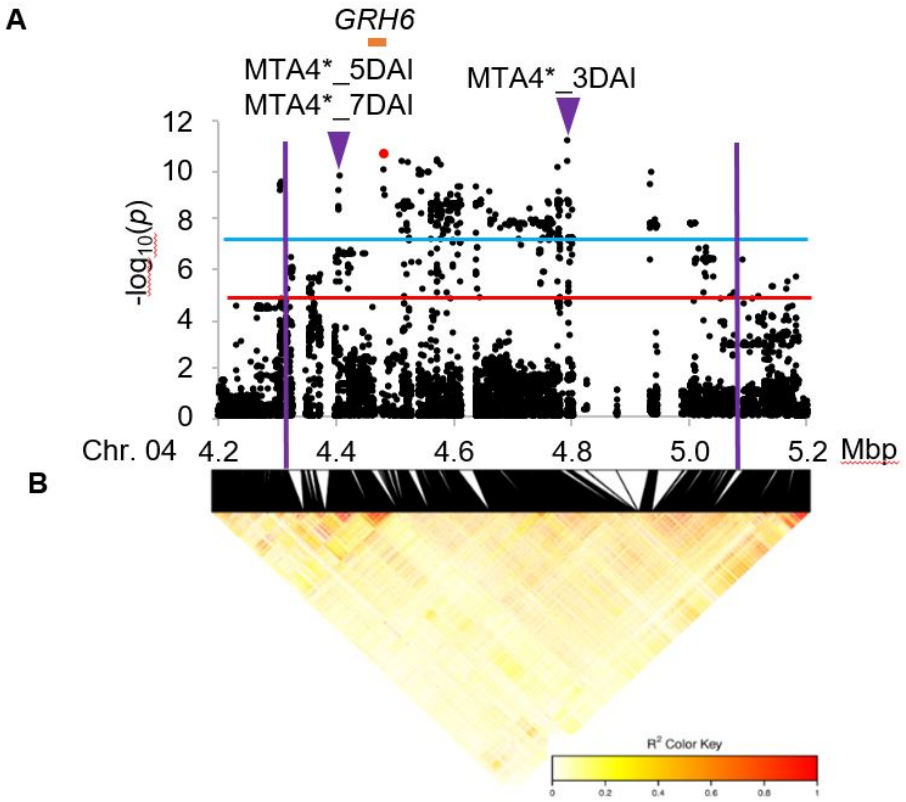

Fig. 8. Significant associations with GRH resistance on chromosome 4 in Myanmar Indica rice landraces (n=184) at 3DAI after selecting the accessions carrying susceptible haplotype(Hap*GRH1*B) at *GRH1* (*Refer to haplotype analysis in Chapter III*). (A) Local Manhattan plot around MTA4 region. The red and blue horizontal lines represent suggestive and significant threshold levels at α= 1e-5 and α = 5e-8, respectively. Purple lines represent the genomic region significantly associated with resistance to GRH. The purple arrowheads represent SNP markers with the highest -$\log_{10}(p)$ values at 3DAI, 5DAI, and 7DAI. Orange rectangle indicates the high resolution mapping region of previously reported gene: *GRH6* (Phi *et al.* 2019). Red dot represents the position of second highest peak maker of MTA4 region. (B) Linkage disequilibrium (LD) heat map around MTA4 region.

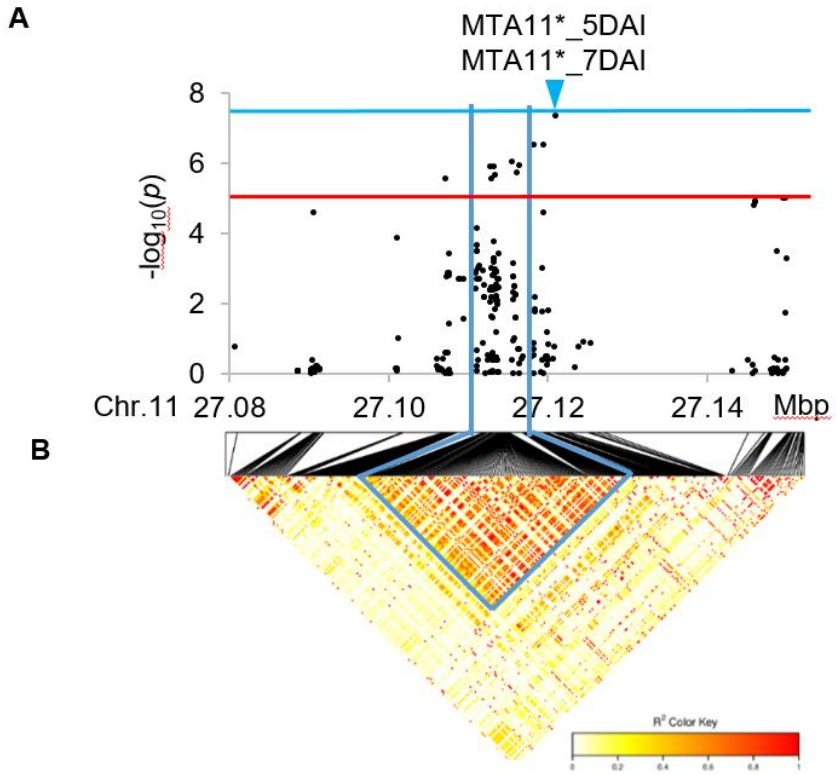

Fig. 9. Significant associations with GRH resistance on chromosome 11 in Myanmar Indica rice landraces (n=184) at 7DAI after selecting the accessions carrying susceptible haplotype(Hap*GRH1*B) at *GRH1* (*Refer to haplotype analysis in Chapter III*). (A) Local Manhattan plots for MTA11 at 7DAI. The red and blue horizontal lines represent suggestive and significant threshold levels at α= 1e-5 and α = 5e-8, respectively. The blue arrowheads indicate the marker-trait associations detected at 5 and 7DAI.

Table 5. Marker-trait associations conferring resistance to GRH based on genome-wide association study in MIDP using the accessions carrying susceptible haplotype at *GRH1*, Hap*GRH1*B (n=184)

Marker trait association (MTA)	Chr.	Position of MTAs (bp)	-log$_{10}$(p)	SNP allele (Allele 1/ Allele 2)	NM(%)[a]		Reported gene flanking to MTA
					Allele1 homozygous	Allele 2 homozygous	
MTA4*_3DAI	4	4,791,920	11.2	A/G	22.4	88.0	*GRH6*
MTA4*_5DAI	4	4,404,524	7.8	G/A	39.8	90.8	*GRH6*
MTA11*_5DAI	11	27,121,054	7.0	C/A	41.5	75.1	
MTA4*_7DAI	4	4,404,524	5.9	G/A	57.4	95.8	*GRH6*
MTA11*_7DAI	11	27,121,054	7.3	C/A	58.1	86.2	

[a] NM represent nymph mortality (%) of GRH.

Discussion

QTL governing delayed resistant effect

In the present study, we identified 188 accessions with MR and HR at 7DAI in the panel. Among the 188 accessions, 105 reached MR and HR levels within 3DAI. Conversely, the subsequent 83 accessions showed delayed expression of resistance, achieving MR to HR levels at 5DAI or 7DAI (Fig. 1A-C, Table 3). GWAS detected three genomic regions conferring resistance to GRH on chromosomes 4 (MTA4), 5 (MTA5), and 11 (MTA11): MTA4 and MTA5 were identified in all evaluation periods with high -$\log_{10}(p)$ values. However, MTA11 was found at 5DAI and 7DAI with lower -$\log_{10}(p)$ values, suggesting that MTA4 and MTA5 were major resistance genes, and that MTA11 may be a major QTL with delayed resistance (Fig. 2A-C, Table 4).

Identity of MTA with previously reported loci

On chromosome 5, *GRH1* corresponded to resistance in this panel, as *GRH1* and MTA5_3DAI, MTA5_5DAI and MTA5_7DAI were present within the same apparent LD block (Fig. 4A-B). The peak SNPs of MTA5 region detected at different evaluated periods: MTA5_3DAI, MTA5_5DAI and MTA5_7DAI were not stable in position even though these SNPs belonged in the same LD block containing *GRH1* (Fig. 4A-B). It is possible that suggested positions of QTL may shift between different time points or trials because of the high phenotypic variance and dynamic nature of plant-insect interaction. Similar trend was observed between *GRH6* and peak SNPs of MTA4: MTA4_3DAI, MTA4_5DAI and MTA4_7DAI on chromosome 4 (Fig. 5A). One of the previously reported genes, *GRH2* was found on the long arm of chromosome 11 between the makers RM5349 and G2-10 (23.3 to 24.7 Mbp) (Kadowaki *et al*. 2003). In GRH*2*-NIL, 75%

nymph mortality was observed at 3DAI (Fujita *et al.* 2010) suggesting *GRH2* expressed resistance at early stage. However, MTA11 was detected at later stage in GWAS (5 DAI and 7 DAI) (Fig. 2B-C), and located in a different region approximately 2.4 Mbp far from *GRH2* speculating that MTA11 is likely a novel locus independently from *GRH2*,

On chromosome 4, the highest peak SNP of MTA4 region at 4,932,132 bp (MTA4_3DAI) and the other high significantly associated SNPs were far from *GRH6*, and they were located within the weak LD block (Fig. 5A-B). After excluding the predominance effect of major resistance gene in this population, *GRH1*, marker-trait associations around *GRH6* region were more accurately observed, suggesting that *GRH6* contributed to GRH resistance in this diversity panel (Fig. 8A).

CHAPTER II

Gene cloning of *GRH6*

Introduction

Phloem-feeding insects such as planthoppers, leafhoppers, aphids, and whiteflies pose significant threats to rice crops. Enhancing crop resistance to these pests is vital for future food security. Plants have developed complex defense mechanisms to combat insect pests. This diverse defense system includes protective strategies that range from physical barriers to chemical secondary metabolites and inducible or adaptive defenses. To defend against various types of insects, plants have developed physical barriers like trichomes, waxes, cuticle layers, and spines to prevent insect attachment, feeding, and oviposition. Plants also make their leaves and roots tougher to deter herbivores. Hardening the plant's outer parts discourages insects from feeding. Additionally, plants strengthen their cell walls with compounds like silica, suberin, callose, and cellulose, making it harder for insects to feed (Satyabrata *et al.* 2021). Plants produce a variety of bioactive compounds and secondary metabolites that fight against insects. These chemicals not only reduce insect attacks but also limit herbivory damage and trigger other defenses (Erb and Reymond 2019). Many of these compounds act as toxins, affecting the insects' digestive and nervous systems, leading to stunted growth or death. They also influence the taste, smell, and color of the plant (Kessler *et al*. 2006). Alkaloids, which are found in almost all plants, play a significant role in defending against insects.

In addition to structural and chemical defenses, plants use physiological processes like recognizing insect effectors and activating signaling pathways to respond to herbivory (Erb *et al*. 2012). These signaling events trigger the production of

phytohormones and the activation of defense response genes. The recognition of insect feeding usually starts at the attack site but can spread to nearby cells and throughout the plant, initiating systemic defenses (Satyabrata *et al.* 2021). When insects feed or lay eggs on plants, they release oral secretions and oviposition fluids, which are crucial in plant-insect interactions (Miles 1999). Insect saliva, which is essential for feeding, contains compounds that can trigger or suppress plant immune responses (Miles 1999). Identified elicitors in insect oral secretions, such as ß-glucosidase, fatty acid-amino acid conjugates (FACs), volicitin, and caeliferins, activate the phytohormone signaling pathway, leading to defense responses against insects (Mattiacci *et al*. 1995).

Transcription factors (TFs) that operate downstream of hormonal signaling pathways are crucial regulators of plant defense and essential for insect resistance (Yang *et al*. 2016). Specifically, WRKY TFs play a key role in general insect resistance in rice. The transcription factors WRKY and MYB are vital for orchestrating rice defense responses by activating the transcription of genes responsible for the biosynthesis and signaling of these phytohormones (Eulgem *et al*. 2000, Chen and Padi 2022). OsWRKY89 enhances resistance to white-backed planthopper (WBPH) by increasing leaf wax deposition, stem lignification, and salicylic acid (SA) accumulation (Wang *et al.* 2007). Similarly, OsWRKY45 boosts brown planthopper (BPH) resistance by increasing hydrogen peroxide (H_2O_2) and ethylene (ET) production (Huangfu *et al*. 2016). Recently, OsbHLH61 and OsbHLH96 were shown to significantly induce defense genes, leading to BPH resistance in rice (Wang *et al*. 2019). These studies highlight the role of multiple novel TFs in plant defense against insect attacks.

Most of the previously cloned resistance genes (R genes) in plants encode proteins with the nucleotide-binding site leucine-rich repeat (NBS-LRR) domains. These proteins

recognize pathogen effectors and trigger defense responses (Lee and Yeom 2015). Genes encoding NBS-LRR proteins are prevalent in plants; for example, there are 149 and 500 NBS-LRR family genes in the genomes of Arabidopsis and rice, respectively (Wang *et al.* 2023). Effector-triggered immunity in plants activates a series of defense-related signaling pathways, which leads to the accumulation of substances, such as callose, in sieve tubes. These changes, including cell-wall thickening, hinder BPH feeding, growth, and reproduction (Yan *et al*. 2023).

Many resistance genes are clustered in the plant genome, likely due to tandem duplications. They also exhibit high levels of inter- and intraspecific variation in copy number, owing to mechanisms such as unequal crossing-over, sequence exchange, and gene conversion (Kuang *et al*. 2004). Four notable gene clusters contained several brown planthopper (BPH) resistance genes: clusters A, B, C, and D on chromosome 12, the short arm of chromosome 4, chromosome 6, and the long arm of chromosome 4. Genes resistant to sucking insect pests of rice including *Glh14, Grh6-nivara, Bph15, Bph17, Bph20* and *Bph12* (Sebastian *et al*. 1996, Fujita *et al*. 2004, Yang *et al*. 2004, Sun *et al*. 2005, Rahman *et al*. 2009, Qiu *et al.* 2012) are found within cluster B on the short arm of chromosome 4. Resistance genes containing NBS-LRR domains are more abundant in Asian cultivated rice (*O. sativa* L.) than in their wild ancestors because of the tandem duplication of chromosomal segments during domestication and repeated cultivation of specific genotypes (Mizuno *et al*. 2020).

To date, several QTLs for resistance to phloem-feeding insects have been identified in rice, with some successfully cloned using positional cloning methods. Cloning and characterizing these resistance genes provide a deeper understanding of the molecular mechanisms underlying plant-insect interactions and play a crucial role in

breeding resistant rice varieties. This knowledge not only enhances our comprehension of how plants defend themselves against pests but also aids in the development of rice varieties with improved resistance to phloem-feeding insects, ensuring more sustainable and effective pest management strategies.

In Chapter I, *GRH6* was identified on chromosome 4 in Myanmar Indica Diversity Panel (MIDP) through GWAS. *GRH6* had previously been mapped on the short arm of chromosome 4 in the Surinam cultivar "SML17" (Tamura *et al*. 2004) and in a wild accession of *Oryza nivara*, IRGC105715 (WK56) (Fujita *et al*. 2004). Further, *GRH6* region had been delimited to 32.2 kbp size of the Nipponbare genome (4460301 to 4491541 bp) and four predicted genes putatively encoding a cysteine synthase (*Os04g0165700*), a hypothetical protein (*OS04g0165801*) and two conserved hypothetical proteins containing a leucine-rich repeat (LRR) domain (*Os04g0165900* and *Os04g0166000*) were detected at this region (Phi *et al*. 2019). However, the causal gene of *GRH6* had not been detected yet and the molecular mechanism underlying this gene is still unknown. In this chapter, the candidate locus of *GRH6* was initially estimated by comparing the nucleotide sequence between the Nipponbare and *O. nivara* accession, WK56, which was the donor of *GRH6*. Then, the causal locus of *GRH6* was isolated by gene cloning and complementation test was performed to confirm that the *O. nivara* (WK56) allele at candidate gene of *GRH6* correspond to GRH resistance. Furthermore, the expression of *GRH6* was investigated in the resistant line, *GRH6*-*WK56*-NIL, and studied on the effect of resistance mechanism of *GRH6* on the feeding activity of GRH through honeydew test.

Materials and method

Plant materials

The *O. nivara* bacterial artificial chromosome (BAC) clone IRGC105715-26E15 was used as donor of *GRH6* and the susceptible Asian cultivated rice T65 (*Oryza sativa* L. ssp. japonica cv. Taichung 65) was used as recipient plant in the transformation experiment for gene cloning. A near-isogenic line harboring *GRH6* (*GRH6-WK56* NIL) and T65 were used as resistant plant carrying *GRH6* and susceptible plant carrying no resistance gene respectively in the expression analysis and honeydew test. *GRH6-WK56* NIL was developed from *Oryza nivara* accession WK56 (IRGC105715) by continuous backcrossing to the recurrent parent, T65 (Fujita *et al*. 2010). The WK56 accession was isolated at Kyushu University, Fukuoka, Japan, through successive self-pollination using the accession IRGC105715 obtained from the International Rice Research Institute, Los Baños, Philippines.

Gene cloning of GRH6

Firstly, genomic sequence of WK56 and Nipponbare was analyzed to identify the causal locus of *GRH6*, and then it was also validated by PCR amplification using three primer pairs (F2-R1, F1-R2, F1-R1). The primers information was listed in Appendix 1. The PCR was conducted using KOD plus DNA polymerase for the product size more than 1 kbp and the reaction comprised an initial denaturation at 95 ºC for 0.1 s, followed by 30 cycles at 98 ºC for 10 s, 60 ºC for 5 s per kbp, 68 ºC for 5 s, and final extension was 68 ºC for 5 s. The Go Taq DNA polymerase was used in PCR for the product size less than 1kbp and the reaction consisted of 5min at 95 ºC, then 40 cycles of 95 ºC for 30seconds, 55 ºC for 30s and 72 ºC for 30s. A 12.6 kbp genomic DNA fragment containing

Os04g0166000 was restricted from *Oryza nivara* bacterial artificial chromosome (BAC) clone IRGC105715-26E15 with the restriction enzyme *Avr*II and inserted into the Ti plasmid binary vector pPZP2H-*lac* (Fuse *et al*. 2001) which was digested with *Xba*I. The obtained constructs were incorporated into the bacteria host cell, *Escherichia coli* (*E. coli*, HST08) by heat-shock transformation method at 42 ºC for 45 seconds. The positive colonies containing the transgene were determined by colony PCR using the primer GRH6oligo2. The primer used in gene cloning was mention in Appendix 1. The plasmid DNA was isolated by Nucleospin Plamid Quick Pure protocol and introduced into the susceptible Japonica cultivar, T65 by *Agrobacterium*-mediated transformation (Toki 1997). The callus induction, infection of *Agrobacterium*, selection of transformants, calli regeneration and the components of various medium used were described in (Appendix 2). the transgenic plants were grown in the temperature and humidity controlled transgenic green house.

Complementation test

To confirm the WK56 allele at *Os04g0166000* correspond to GRH resistance, complementation test was conducted using the transgenic plants carrying *Avr*II fragment containing transgene and those carrying empty vector. The plants with transgene was selected by the primer pair *35S_F/HPT_R*. The primer used in complementation test were listed in Appendix 3. Thirteen T_0 plants containing *Avr*II fragment and five T_0 plants containing empty vector were evaluated for GRH resistance using antibiosis as described in Chapter 1. Nymph mortality (%) was evaluated at 3DAI, 5DAI and 7DAI.

Expression analysis

Roughly 3 cm of leaf blade from 12-day-old seedlings of infested and non-infested T65 and *GHR6-WK56* NIL was collected at 0, 1, 2 and 3DAI using three replications. Day zero means time point just before GRH infestation. The samples were instantly frozen in liquid nitrogen and stored at -60 °C prior to extraction. The frozen samples were pulverized using a bead shocker (Yasui Kikai, Osaka, Japan). Total RNA was extracted using an RNeasy Plant Mini Kit (Qiagen, Hilden, Germany). First-strand cDNA synthesis ensued from 4.5 µl of total RNA using ReverTra Ace reverse transcriptase (Toyobo) with an Oligo(dT)15 primer. qRT-PCR was performed using the GRH6RT1 primer, which were design potentially to amplify from the T65 allele at *Os04g0165900* and both alleles at *Os04g0166000* (*GRH6*). Rice Ub-CEP52-1 (*Os03g0234200*), which encodes the ubiquitin-fused 60S ribosomal protein L40-1, was amplified using ubiquitin primers as an internal control for qRT-PCR on a MX3000P real-time PCR system (Agilent Technologies, Santa Clara, CA, USA) using the KAPA SYBR FAST qPCR Master Mix Universal Kit (Kapa Biosystems, MA, USA). Primers used in expression analysis were listed in Appendix 1. The expression level in the samples was quantified relative to the 0-day post-infestation T65 sample. GRH nymph mortality (%) in infested *GRH6-WK56* NIL and T65 was assessed from 0 to 3 days post-infestation.

Honeydew test

The feeding ability of GRH on the T65 and *GRH6-WK56* NIL was explored using the honeydew test. The Whatman No.1 filter paper was immersed in a bromocresol green solution (2 mg/ml in ethanol) for 5 min and left to air-dry for an hour. A plastic dish was positioned beneath the seedlings, and bromocresol green-treated filter paper was placed

on the dish. An overturned plastic cup was then placed on the filter paper. Ten adult female GRH insects of preoviposition stage, starved for 2 h, were introduced into each plant and allowed to feed for 24 h. The alkaline pH of the phloem sap caused the honeydew of the insects to form blue-colored spots from the reaction with bromocresol green on the filter paper (Bharathi *et al*. 2018). The honeydew area was analyzed using ImageJ software.

Results

Gene cloning of GRH6

The candidate locus of *GRH6* was identified by comparing the sequences between the susceptible cultivar Nipponbare and WK56, the donor of *GRH6*. The genomic region of *GRH6* in Nipponbare contained four predicted genes putatively encoding a cysteine synthase (*Os04g0165700*), a hypothetical protein (*Os04g0165801*), and two conserved hypothetical proteins containing a leucine-rich repeat (LRR) domain (*Os04g0165900* and *Os04g0166000*) (Phi *et al*. 2019, Fig. 10A). The predicted genes *Os04g0165900* and *Os04g0166000* were located in the tandemly duplicated genomic regions, tandem1 (14,993 bp) and tandem2 (17,557 bp) respectively, in the reference sequence of Nipponbare (Fig. 10A). In contrast, tandem 1 genomic region was deleted in WK56 and it had only one gene (*Os04g0166000*) encoding LRR protein within the genomic region of *GRH6*. To confirm the non-tandem duplication in the WK56, PCR was conducted using the genomic DNA of Nipponbare, T65 and *GRH6-WK56* NILs with three primer pairs including F2-R1, F1-R2, F1-R1 (Fig. 10A-B). PCR amplification were successful in Nipponbare, T65, and *GRH6-WK56* NILs for the primer pairs F2-R1 and F1-R2, suggesting that these primers were functional for the annealing and amplification of the

PCR products of three cultivars (Fig. 10B). In contrast, no amplification was observed from *GRH6-WK56* NIL, whereas PCR amplifications were obtained from Nipponbare and T65 using primers F1 and R1 owing to their tandem duplication (Fig. 10B). The results revealed that the tandem1 genomic region was deleted in WK56 and it had only one copy of the gene (*Os04g0166000*) at *GRH6* region. The genomic sequence is deposited in the DNA Data Bank of Japan (Accession No. LC781942). Using the full-length Nipponbare cDNA, the deduced coding sequence of *Os04g0166000*/*GRH6-WK56* was 1,018 aa in length, encoding two LRR domains (Fig. 10C). Differences in coding region of WK56 at *Os04g0166000* compared with susceptible cultivar, Nipponbare were observed, resulting had two insertions, three deletions, and 65 amino acid substitutions in Nipponbare (Fig. 11). These sequences discrepancies between WK56 and Nipponbare may affect the structure and function of the encoded protein. Additionally, *Os04g0165900,* on the tandem1 segment of Nipponbare lacked the N-terminal region, first codon, transcription initiation site, and promoter region (Fig. 10A, black box). On the basis of these observations, we assumed that *Os04g0166000* was likely to be candidate of *GRH6* and selected for complementation test.

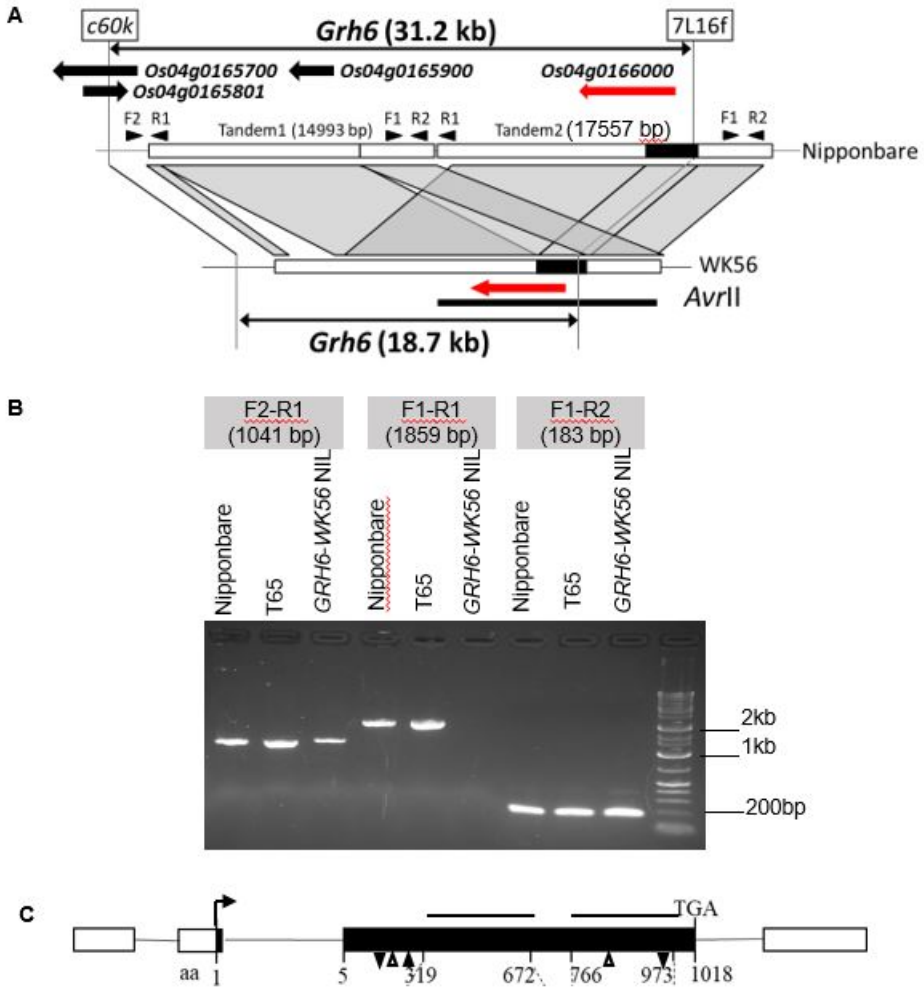

Fig. 10. Map-based cloning of *GRH6.* (A) Genetic and physical position of *GRH6*. Genes models annotated in the Rice Genome Annotation Project Database (RAP-DB) were shown as black arrows. Primers for confirmation of tandemly duplicated regions (Tandem 1 and Tandem 2) were shown in arrowheads. The black bar represents the *Avr*II fragment containing *Os04g0166000* (B) Confirmation of non-tandem duplication in WK56 at *GRH6* genomic region. PCR amplification from Nipponbare, T65, and *GRH6* NILs was successful using the primer pairs F2-R1 and F1-R2, suggesting that these primers were functional for the annealing and amplification of the PCR products. Nevertheless, no amplification was obtained from *GRH6* NIL, whereas PCR amplifications were obtained from

Nipponbare and T65 using primers F1 and R1 owing to their tandem duplication. The results revealed that the tandem1 genomic region was deleted in WK56 and WK56 had only one copy of the gene. (C) Structure of *GRH6-WK56*. White boxes represent the 5' and 3' untranslated regions (UTRs), and black boxes represent the coding sequence. The black bars represent the deduced LRR domains. Black and white arrowheads represent insertion and deletion of amino acid respectively. Bent arrow indicates ATG start codons and TGA indicates stop codons.

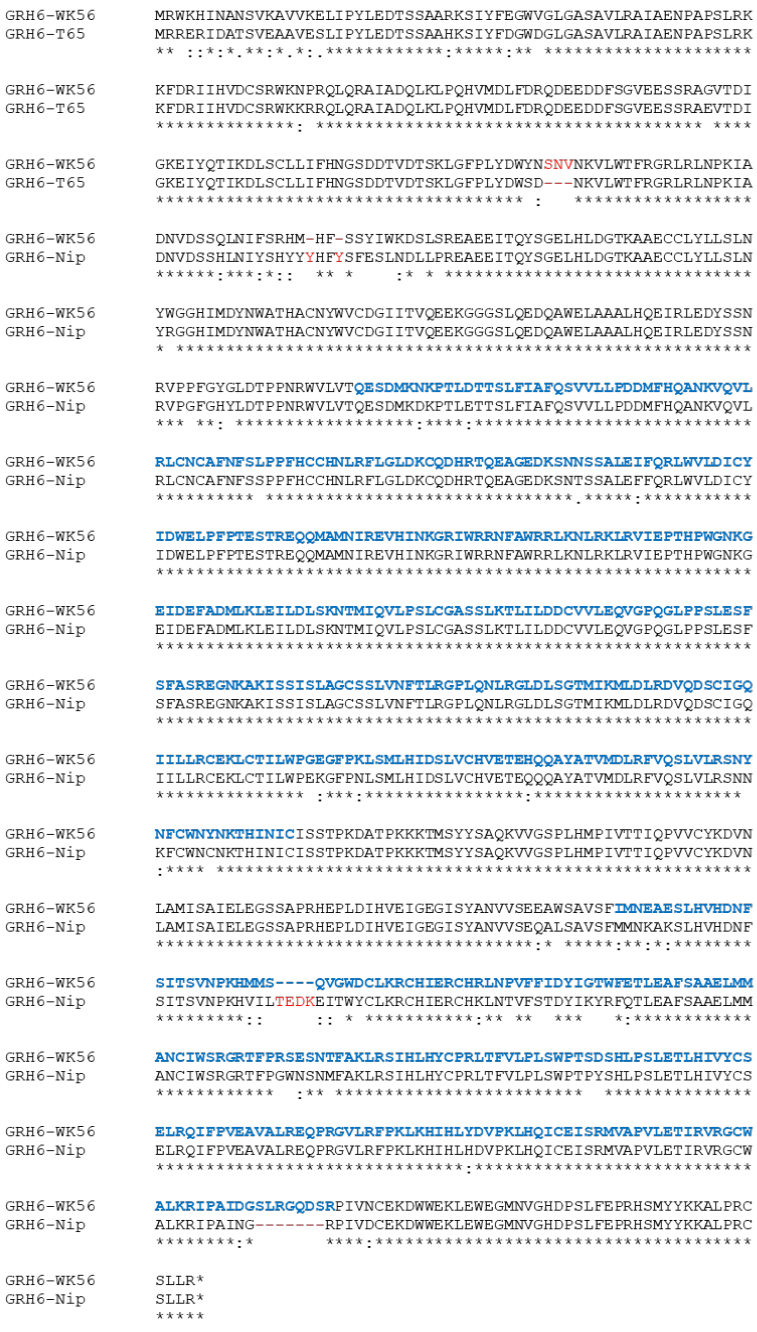

Fig. 11. Amino acid sequence comparison of *Os04g0166000* in IRGC105715 (WK56) and Nipponbare. Blue letters represent the LRR domain. Red letters represent insertion and deletion of amino acid. The asterisk (*) represents where amino acids in two sequences are identical. The colon (:) and black dot (.) indicate conserved substitution and semi-conserved of amino acid, respectively.

Complementation test

A total of 12.6 kbp genomic DNA fragment containing *Os04g0166000* and 4.7 kb length of 5' region of the gene was restricted from *Oryza nivara* bacterial artificial chromosome (BAC) clone IRGC105715-26E15 with the restriction enzyme *Avr*II and inserted into the Ti plasmid binary vector pPZP2H-*lac* (Fuse *et al*. 2001) which was digested with *Xba*I. The obtained constructs were transformed into the susceptible genetic background T65 by *Agrobacterium*-mediated transformation. The obtained T_0 plants were selected for transgene using the primer pair *35S_F/HPT_R*, and T_0 plants with transgene were evaluated for GRH resistance using antibiosis test. The nymph mortality of T_0 plants containing the *Avr*II fragment gradually increased from 0DAI to 7DAI, and all T_0 plants showed a moderate to high resistance to GRH at 7DAI, whereas T_0 plants harboring the empty vector showed susceptible to GRH (Fig. 12). This result revealed that the WK56 allele at *Os04g0166000* contributed to GRH resistance and designated as *GRH6*.

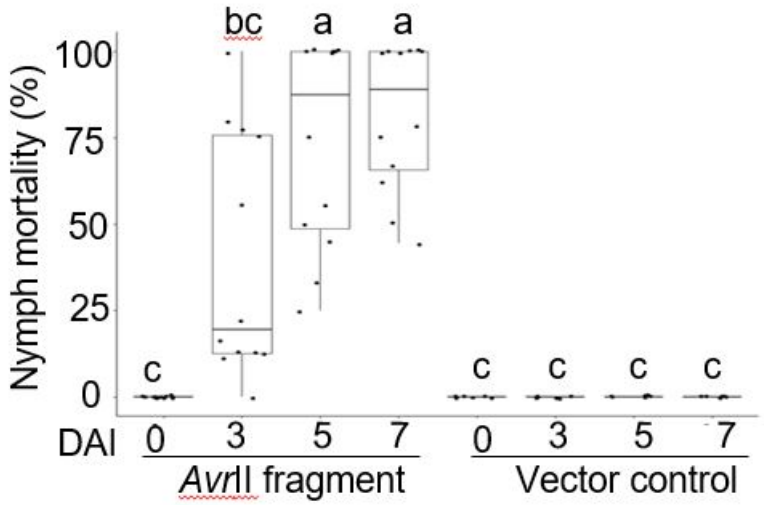

Fig. 12. Complementation test for confirmation of IRGC105715(WK56) allele at *Os04g0166000* correspond to GRH resistance. Nymph mortality of GRH in T_0 transgenic plants carrying *Avr*II fragment containing the transgene (*Os04g0166000*) using thirteen replications and vector control using six replications at 0 days after infestation (DAI), 3DAI, 5DAI and 7DAI, respectively. Different alphabets show that mean values from each days after infestation was significantly different each other.

Expression analysis

The expression of *GRH6* in leaf blades from infested and non-infested T65 and *GRH6-WK56* NIL was investigated using quantitative real-time PCR (qRT-PCR). qRT-PCR was conducted using the primers which were design potentially to amplify from the T65 allele at *Os04g0165900* and both alleles at *Os04g0166000* (*GRH6*). The expression level of *Os04g0166000* on the leaf blade of the GRH infested *GRH6-WK56* NIL at 1DAI was unchanged to 0DAI but was significantly up-regulated at 2DAI and 3DAI (Fig. 13). But the expression level of *Os04g0165900* and *Os04g0166000* was unchanged in infested T65 leaf (Fig. 13). In contrast, the expression was not up-regulated in non-infested leaves of both *GRH6-WK56* NIL and T65 (Fig. 13). These results demonstrated that the expression of only *GRH6-WK56* was up-regulated by GRH infestation. The nymph mortality of GRH significantly increased at 2DAI and 3DAI in the *GRH6-WK56* NIL (Fig. 14). In contrast, GRH nymph on T65 showed no mortality, suggesting that *GRH6* sufficiently induced nymph mortality at 2DAI and 3DAI and lead to insect death (Fig. 14). Since the high-resolution mapping of *GRH6* revealed that the insertion-deletion polymorphism marker, 7L16f, delimited the candidate region of *GRH6* in downstream of 7L16f, it was postulated that 16 of single nucleotide or insertion-deletion polymorphisms between the T65 and WK56 might determine the transcriptional differences between the two alleles at *GRH6* (Fig. 15).

Honeydew test

The feeding ability of GRH on *GRH6-WK56* NIL and T65 was investigated by observing and analyzing the honeydew deposit from ten adult female GRH feeding 24 hours on each plant. The honeydew derived from the phloem feeding were found as blue rimmed spots

on the bromocresol treated filter paper. A large blue-rimmed spot area was observed in T65 due to honeydew deposits derived from the phloem 24 h after infestation (Fig. 16A-B). However, no blue spots were observed in *GRH6-WK56* NIL, suggesting that *GRH6* confers resistance to GRH by inhibiting its sucking from the phloem.

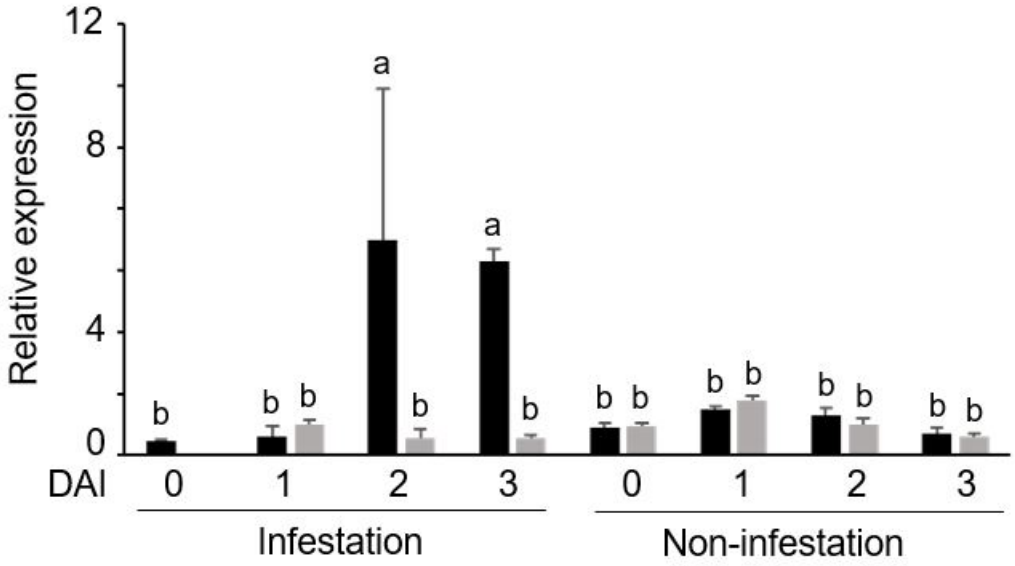

Fig. 13. Time course expression of *Os04g0166000* in the leaf blades of infested and non-infested of *GRH6-WK56* NIL and T65 using three replicates. The black and grey bars represent *GRH6-WK56* NIL and T65 respectively. Zero-day after infestation (0 DAI) is the time point before GRH infestation. The expression level in the samples was quantified relative to the 0-day post-infestation of T65 sample. Bars with the different letters are significantly different at Tukey-Kramer multiple range test.

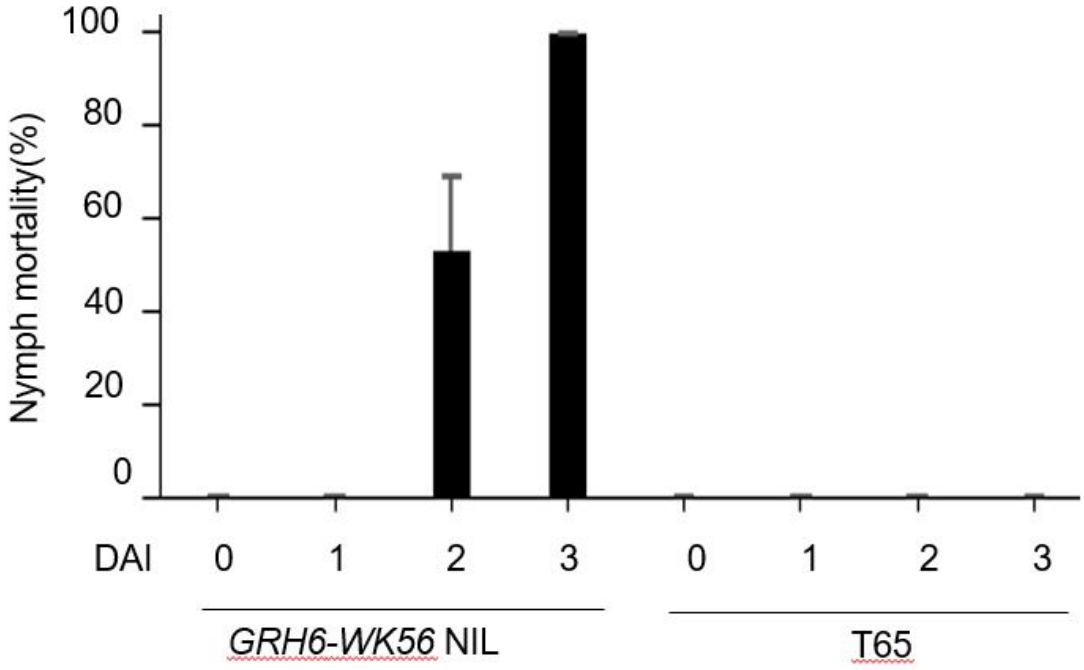

Fig. 14. Nymph mortality (%) of GRH in *GRH6-WK56* NIL and T65 evaluated at 0 days after infestation (DAI), 1DAI, 2DAI and 3DAI, respectively. The mean value was determined based on the averages of three replicates.

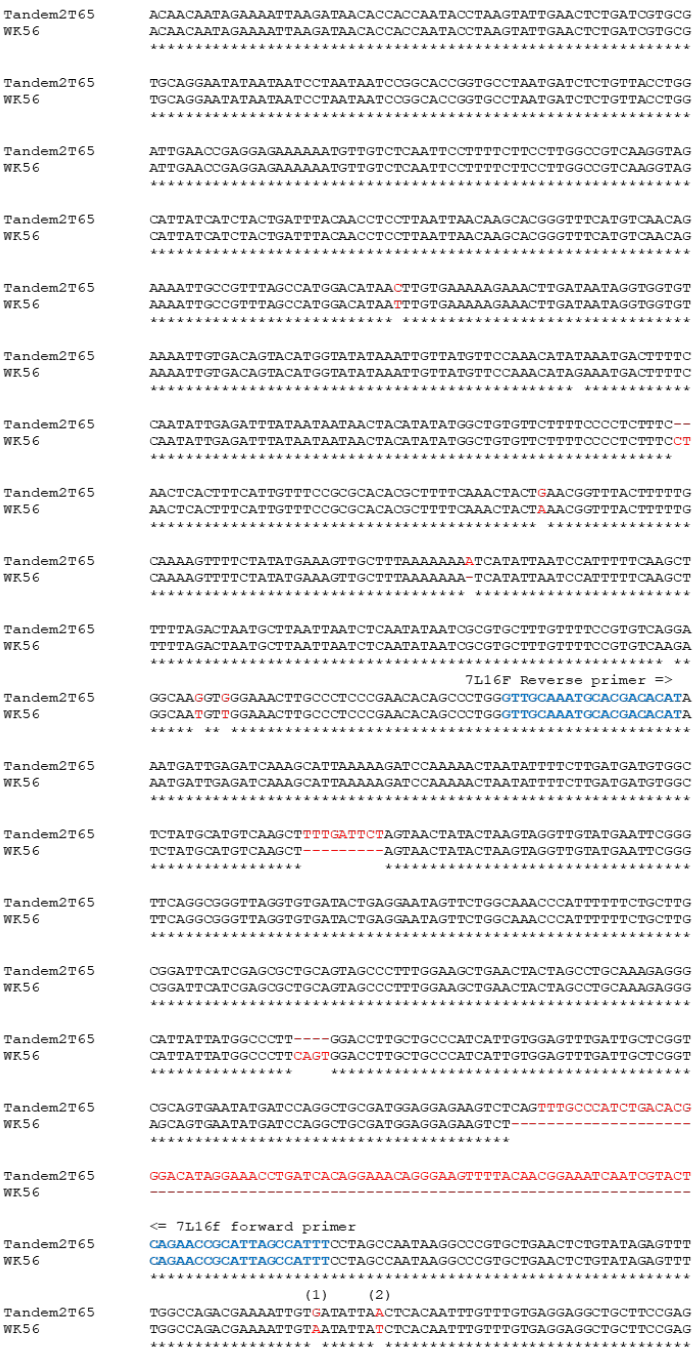

Fig. 15. The promoter sequences of *GRH6* were derived from WK56 and a tandem 2 copy of T65. The location of the insertion/deletion marker, 7L16f, used for the fine mapping of *GRH6* (Phi *et al.* 2019) is shown in blue. Sixteen possible causative polymorphisms, which may involve low or no expression of the T65 alleles, are marked in parentheses. This figure continues on the following pages.

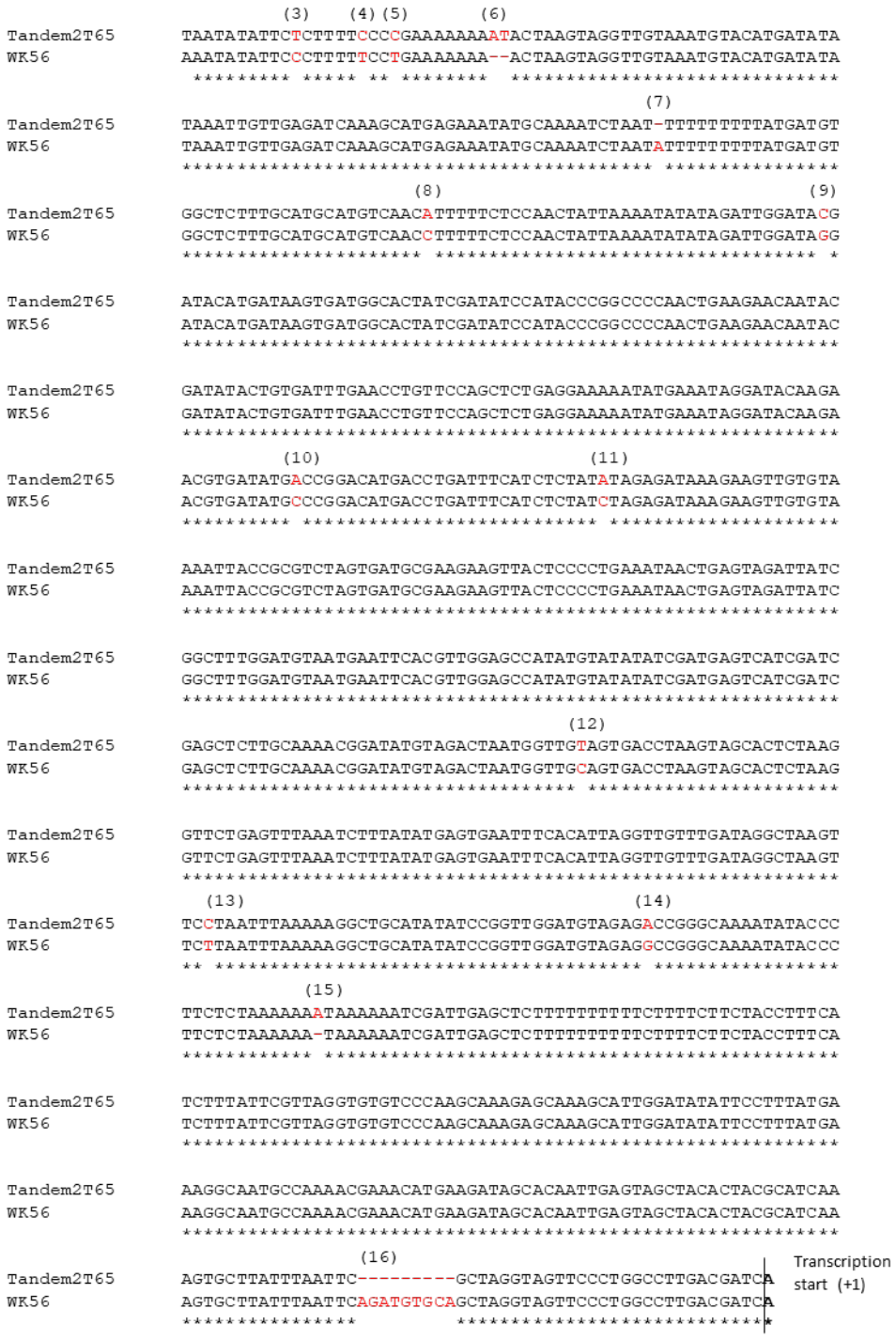

Fig. 15. Continued

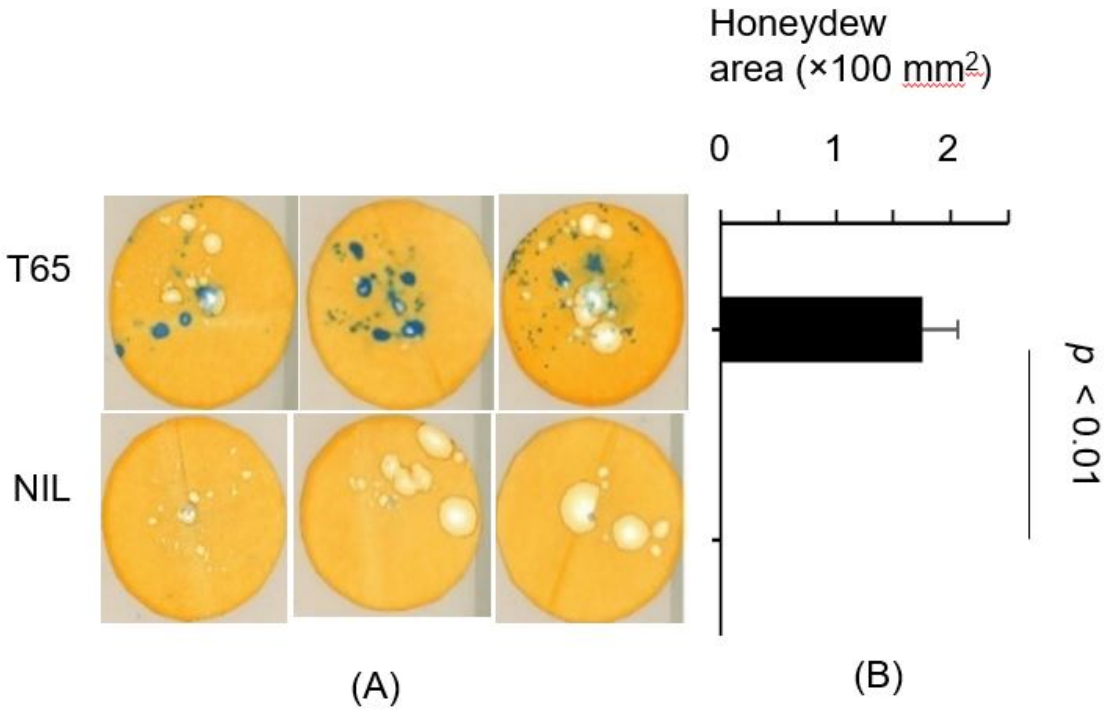

Fig. 16. Honeydew assay for GRH after 24 hour feeding on twelve days old plants for *GRH6* NIL and T65 with three replicates. (A) Images of phloem-derived honeydew of GRH (Blue rimmed spots) on the filter paper immersed in a bromocresol green solution. The rice seedlings were grown in plastic caps. The bromocresol treated filter paper was placed on the plastic dish which is positioned at the base of rice seedling. The rice seedling and the filter paper were completely covered by a transparent plastic cap. Ten female GRH (pre-oviposition stage) starved for 2 hours were introduced into each plant and allowed to feed for 24 hours. The high pH of phloem sap caused the honeydew of GRH to form blue colored spots after reacting with bromocresol green on filter paper. (B) Area of phloem-derived honeydew (mm^2) of GRH feeding on *GRH6-WK56* NIL and T65 and analyzed by ImageJ software.

Discussion

Genetic differences of resistant and susceptible alleles

Expression analysis revealed that the expression of *GRH6-WK56* NIL was induced by GRH infection (Fig. 13). Transformation of *Avr*II fragment induced GRH resistance to the T65, suggesting that a reduced 5' region (promoter region) of *GRH6* in WK56 might control the expression and induced GRH resistance. The gene promoter region plays a critical role in controlling gene expression by binding transcription factors to various *cis*-regulatory elements in the promoter (Chang *et al*. 2023). In this study, insertions, deletions, and nucleotide polymorphisms were found in the promoter region of *GRH6* between *WK56* and T65 (Fig. 15), suggesting that polymorphisms in this promoter region enhance gene expression in *GRH6-WK56* NIL. Therefore, we hypothesized that the promoter region of *GRH6* in WK56 might contain specific sequences or motifs recognized by transcription factors activated in response to insect infestation, leading to increased gene expression in *GRH6-WK56* NIL. Additionally, insertion, deletion and polymorphism of amino acid in the coding region of *GRH6* especially the LRR domains, the recognition site for effector, were observed between WK56 and Nipponbare (Fig. 11), assuming that amino acid polymorphism may affect the structure and function of the recognition of effector. Further experiments including evaluation of over expressor of the susceptible Nipponbare or T65 allele at *GRH6* or swapping promoter and CDS between the resistant and susceptible alleles play an important role to identify the functional polymorphism between the alleles.

Function of GRH6

GRH6-WK56 encodes the protein with two LRR domains (Fig. 10C). In rice, most cloned R genes encode CC-NBS-LRR domain-containing proteins, for example (*BPH14, BPH26, BPH9, BPH18, BPH30*) (Du *et al*. 2009, Tamura *et al*. 2014, Zhao *et al.* 2016, Ji *et al*. 2016, Shi *et al*. 2021). NBS-LRR proteins recognize pathogen effectors when the pathogens secrete molecules (effectors) into plant cells to suppress the immune response of the plant and trigger potent innate immune responses (McHale *et al*. 2006). *BPH9*, which encodes CC-NBS-NBS-LRR, activates salicylic acid and jasmonic acid signaling pathways in rice plants upon BPH feeding, and confers resistance to BPH (Zhao *et al*. 2016). *BPH14* encoding CC-NBS-LRR induced callose deposition in sieve tubes, reducing BPH feeding, development, and survival (Du *et al*. 2009). Both *BPH30* (allelic to *Os04g0115650*) and its homolog *BPH40* (allelic to *Os04g0166000*) encode the protein with two LRR domains enhanced cellulose and hemicellulose synthesis, making the cell walls stiffer and sclerenchyma thicker; these were the physical barriers preventing BPH from feeding on the phloem (Shi *et al.* 2021). Since *GRH6* is the same locus as *BPH40*, there is a possibility that the function of *GRH6* was likely to be similar that of *BPH40* which is related to cell wall fortification in the sclerenchyma and inhibition of the feeding of insects to the phloem. The honeydew assay revealed *GRH6* prevent the feeding of GRH to the phloem in *GRH6-WK56* NIL. The detailed molecular functions of *GRH6* should be investigated in future studies.

CHAPTER III

Haplotype analysis for green rice leaf hopper (*Nephotettix cincticeps* (Uhler)) resistance of rice landraces (*O. sativa* L.) in Myanmar

Introduction

Recent advances in sequencing technology have allowed the application of genome-wide single nucleotide polymorphism (SNP) data for detecting associations between alleles and trait of interest. However, GWAS has some limitations. In GWAS, individual SNP markers are bi-allelic and are poorly informative (Wurschum *et al*. 2013). Significant SNPs do not often represent the causal molecular variants (Zargar *et al*. 2015, Bhat *et al*. 2016). To overcome the limitations of GWAS and increase the resolution of candidate genomic regions, haplotype analysis is considered as an effective approach in association studies (Qian *et al*. 2017).

A haplotype consists of closely linked genomic structural variations, such as polymorphic SNPs, with strong linkage disequilibrium (LD) among them (Maldonado *et al*. 2019). LD is a property of SNPs in genomic sequence that explain the degree to which an allele of one SNP is inherited or correlated with an allele of another SNP within a population (Bush and Moore 2012). When SNPs have strong LD, the alleles of a few SNPs in a specific haplotype can effectively predict the alleles of other SNPs in the same LD block. Therefore, a small number of common SNPs selected from the group of SNPs within LD are sufficient to define the relevant haplotype and these SNPs can capture the genetic information associate with the trait. (Takeuchi *et al*. 2005).

The multi-allelic nature of haplotype blocks is more informative than bi-allelic nature of individual SNP marker due to the abundance of haplotype variants that indicate

recombination and mutation events within genes (Stephens *et al*. 2001). Compared with individual SNP markers, haplotypes provide more accurate predictions of genomic regions associated with low-heritable quantitative traits (Calus *et al*. 2009). Recent applications of haplotype-based GWAS for various traits including yield, quality and stress tolerance in different plant species such as soybean (Contreras-Soto *et al*. 2017), wheat (Basile *et al*. 2019), rice (Wang *et al*. 2017) and maize (Yuan *et al*. 2019) revealed the success of haplotype analysis for trait discovery and crop improvement.

Even though the haplotype-based GWAS provide the accurate identification of genomic region associated with the trait, the precise implementation of the variant calling step in genome-wide sequencing still remains as the pivotal for the success of association studies. The repetitive regions of the genome are the biggest challenges in variant calling. The inaccurate reannealing of DNA strands in these repeat regions can result in the expansion or contraction of the repeated sequences. The allelic variants containing such repeats, often surpass the typical read in short read sequencing and leads to inaccurate identification of the genetic variations. Consequently, the inaccurate variant calling generates false associations in genome-wide sequencing analysis (Koboldt 2020).

In Chapter I, GWAS identified three marker-trait associations including MTA4, MTA5, and MTA11 in MIDP, and estimated that *GRH6*, *GRH1*, and one novel QTL on chromosome 11 were candidate genes corresponding to GRH resistance. In Chapter II, the causal gene of *GRH6* was identified through gene cloning. In this chapter, haplotype variations for *GRH6*, *GRH1*, and MTA11 were investigated by haplotype analysis, identifying accessions with resistant haplotypes. Additionally, the genetic composition underlying GRH resistance in the Myanmar population is elucidated by examining haplotype combinations at the three loci and their geographical distribution.

Materials and method

Plant materials

The 224 accessions of MIDP collected from the seven geographic region of Myanmar including the northern mountainous region (NMR), eastern mountainous region (EMR), western mountainous region (WMR), central dry zone (CDZ), western coastal region (WCR), delta region (DR), and eastern coastal region (ECR) were used for haplotype analysis (Table 2).

Evaluation of GRH resistance

Evaluation of GRH resistance in 224 accessions of MIDP at 3DAI, 5DAI and 7DAI had been conducted in Chapter I.

SNP markers used for haplotype analysis

A genomic haplotype is characterized by a combination of nucleotide variations at multiple SNPs with highest $-\log_{10}(p)$ value within the fine mapping region of QTL or genomic region associated with the GRH resistance. The highest associated six SNP markers at 8117089 bp, 8132152 bp, 8144446 bp, 8208059 bp, 832bp5226 bp, 8571734 bp within the fine mapping regions of *GRH1* (Fig. 17), and three SNPs at 27118206 bp, 27119453 bp, 27121054 bp within the genomic regions of MTA11 (Fig. 18) were used for the haplotype analysis. For haplotype analysis in causal gene of *GRH6* (*Os04g0166000*) that was cloned in Chapter II, the unfiltered genotyped data was considered to access all variants of the SNPs and InDel calls. After filtering the SNPs and InDels with a minor allele frequency (maf) of more than 0.05 and genotype missing frequency of less than 0.1, the high quality SNPs and InDels in coding sequence (CDS),

intron and untranslated regions (UTR) were used for haplotype variation analysis. The 15 SNPs and 4 InDels in coding sequence region (CDS), 4 SNPs in untranslated region, 12SNPs and 2 InDels in intron region were used (Fig. 19). Haplotypes with frequencies exceeding 0.05 were considered major haplotype variants and the remaining ones were regarded as unknown haplotypes. Turkey- Kramer test was performed to test the statistical significance difference of each haplotype.

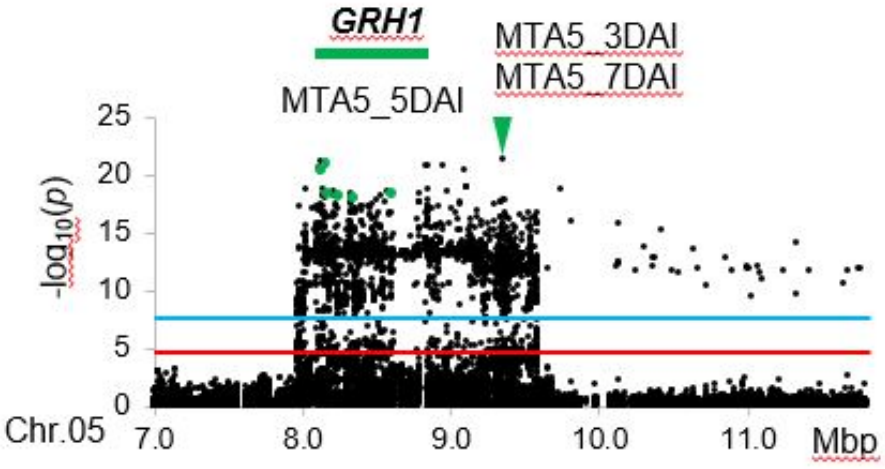

Fig. 17. Chromosomal location of SNP markers used for haplotype analysis associated with resistance to GRH at the genomic region of *GRH1*. The green rectangle represents the position of fine mapping region of *GRH1* in the Nipponbare genome. The green dots represent the highest associated SNP markers with resistance to GRH within the genomic region of *GRH1*, and they are used in haplotype analysis.

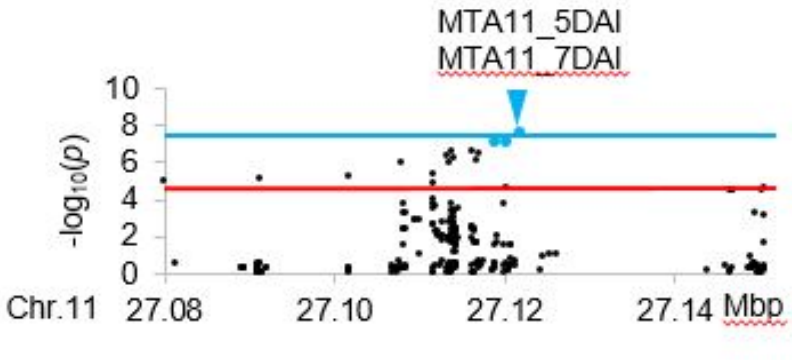

Fig. 18. Chromosomal location of SNP markers used for haplotype analysis associated with resistance to GRH at the genomic region of MTA11. The blue dots represent the highest associated SNP markers with resistance to GRH within the genomic region of MTA11, and they are used in haplotype analysis.

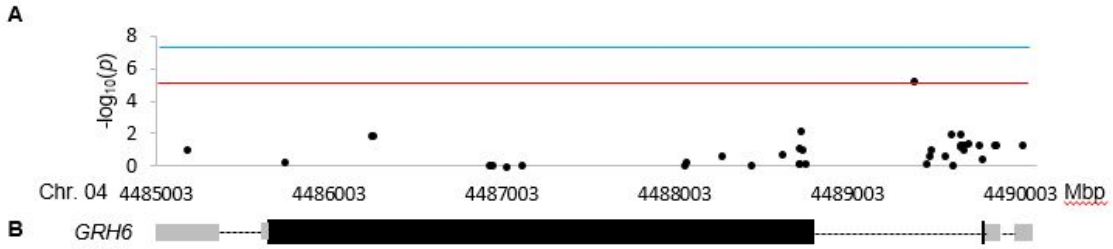

Fig. 19. (A) Chromosomal location of SNP and Indel markers used for haplotype analysis associated with resistance to GRH at *GRH6*. (B) Gene structure of *GRH6* where black and grey boxes represent the exon and UTR region, and black line represents the intron.

Results

Haplotype analysis for GRH resistance in MIDP at GRH1 and MTA11

Haplotype analysis at the *GRH1* region were performed using the six SNPs from the highest -log$_{10}$(p) values in the candidate region of fine-mapping of *GRH1* at 8,117,089 bp, 8,132,152 bp, 8,144,446 bp, 8,208,059bp, 8,325,226 bp and 8,571,734 bp (Fig. 17). The analysis showed that the two genomic haplotypes, Hap*GRH1*A and Hap*GRH1*B, were found associated to resistant and susceptible nymph mortality, respectively (Fig. 20A-B, Table 6). Thirty-two accessions possessing the Hap*GRH1*A haplotype showed significantly higher nymph mortality at 3DAI, whereas accessions carrying the haplotype Hap*GRH1*B showed a wide range of variation in nymph mortality (Fig. 20A-B, Table 6).

Two major genomic haplotypes (HapMTA11A and HapMTA11B), estimated by the three strongest associated SNPs at 27,118,206, 27,119,453 and 27,121,054 bp with highest -log$_{10}$(p) values within the genomic region of MTA11 were observed (Fig. 21A-B, Table7). Thirty-three accessions harboring the HapMTA11A haplotype exhibited moderate to high nymph mortality at 7DAI (Fig. 21A-B, Table7) but not at 3DAI and 5DAI, demonstrating that HapMTA11A contributed to GRH resistance with a delayed resistance effect, whereas the accessions harboring the haplotype HapMTA11B showed high variation in nymph mortality (Fig. 21A-B, Table7).

Haplotype estimation for causal gene of GRH6 in MIDP

Haplotype analysis for the causal gene of *GRH6* (*Os04g0166000*) was performed using the 15 SNPs and 4 InDels in coding sequence region (CDS), 4 SNPs in untranslated region, 12 SNPs and 2 InDels in intron region (Fig. 22A-B). Of the identified seven major haplotypes (Hap*GRH6*A, Hap*GRH6*B, Hap*GRH6*C, Hap*GRH6*D, Hap*GRH6*E,

Hap*GRH6*F, Hap*GRH6*G), all the accessions in Hap*GRH6*A showed highly resistance to GRH at 3DAI whereas the average nymph mortality of the other haplotypes (Hap*GRH6*B-G) showed susceptible resistance with less than 35% nymph mortality without significant difference among them (Fig. 22C, Table 8). These results suggested that Hap*GRH6*A correspond to GRH resistance. A wide range of nymph mortality (%) of GRH from 0 to 100% was observed in the other six haplotypes (Fig. 22C, Table 8), suggesting that the phenotypic variation might be due to background QTL. However, the resistance allele donor at *GRH6* of wild species, WK56, and T65 did not possess the resistant haplotype (*GRH6*HapA) (Data not shown). Expanding haplotype estimation constructed in Myanmar cultivated rice to the wild rice may not be straightforward.

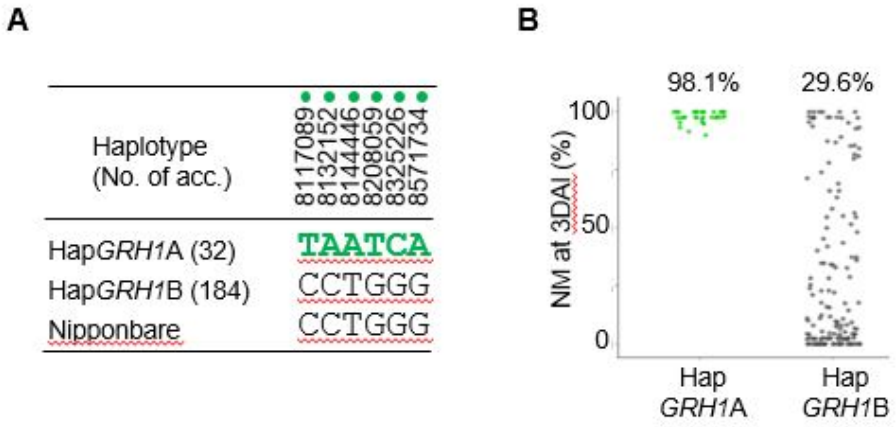

Fig. 20. Resistance to green rice leafhopper controlled by QTL located closely linked to *GRH1* in MIDP (n=224). (A) Haplotype variations around genomic region of *GRH1* in MIDP and their SNP allele. Haplotypes with frequencies exceeding 0.05 were considered major haplotype variants. (B) Nymph motility (%) of GRH by the major haplotypes around genomic region of *GRH1* at 3DAI. The number on the scatter plot is mean value of the nymph mortality (%) of each haplotype.

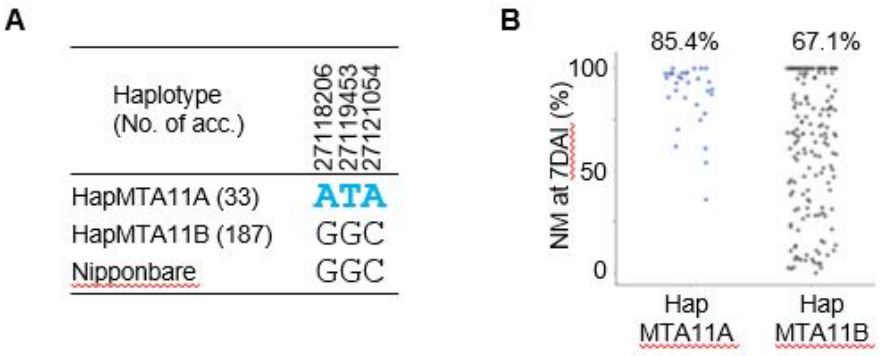

Fig. 21. Resistance to green rice leafhopper controlled by QTL located closely linked to marker-trait association on chromosome 11 (MTA11) in MIDP (n=224). (A) Haplotype variations around MTA11 in MIDP and their SNP allele. Haplotypes with frequencies exceeding 0.05 were considered major haplotype variants. (B) Nymph motility (%) of GRH by the major haplotypes of MTA11 at 7DAI. The number on the scatter plot is mean value of the nymph mortality of each haplotype.

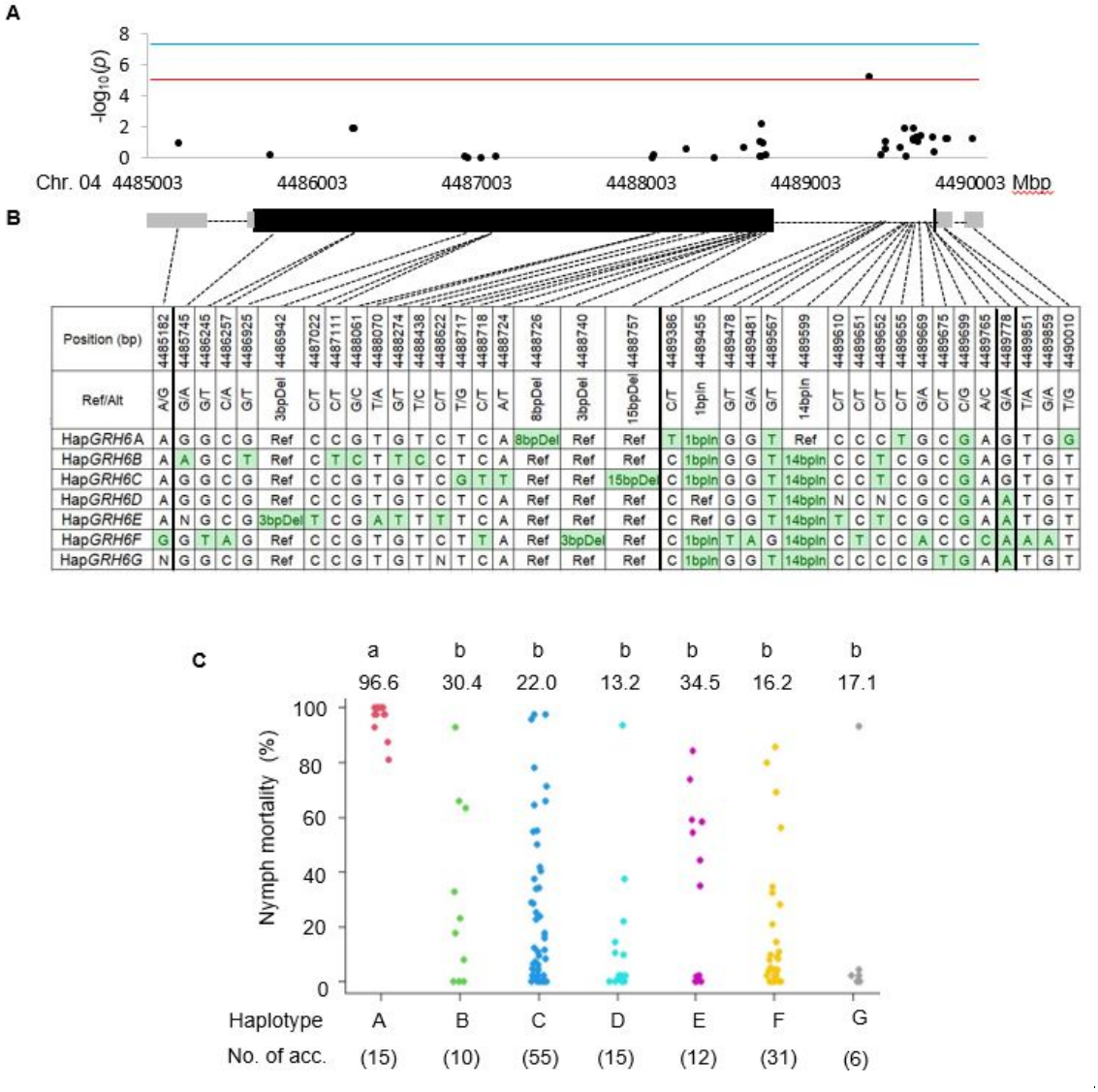

Fig. 22. Polymorphism at causal gene of *GRH6* (*Os04g0166000*) in MIDP (A) Associations between SNPs, InDel marker and nymph mortality of GRH at *Os04g0166000* (B) Gene structure of *GRH6* and haplotype variations in MIDP at *Os04g0166000* based on 15 SNPs and 4 InDels in CDS, 4 SNPs in UTR, 12 SNPs and 2 InDels in intron. Black and grey boxes represent the exon and UTR region, and black line represents the intron. Haplotypes with frequencies

exceeding 0.05 were considered major haplotype variants. (C) Nymph motility (%) of GRH in MIDP containing in seven major haplotypes of *Os04g0166000* at 3DAI. The number on the scatter plot is mean value of the nymph mortality (%) of each haplotype. Different alphabets indicate that mean values of each haplotype was significantly different each other.

Table 6. Haplotype variations of MIDP at the genomic region of *GRH1*

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH1*[d]						Haplotype[e]	Nymph mortality (%)[f]		
			chr05_8117089	chr05_8132152	chr05_8144446	chr05_8208059	chr05_8325226	chr05_8571734		3DAI	5DAI	7DAI
1	CC1	G1	C	C	T	G	G	G	Hap*GRH1* B	2.2	20.0	31.1
2	CC3	G174	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	31.4
3	CC6	G248	C	C	T	G	G	G	Hap*GRH1* B	4.0	56.0	80.5
4	CC7	G74	C	C	T	G	G	G	Hap*GRH1* B	97.5	100.0	100.0
5	CC8	G249	C	C	T	G	G	G	Hap*GRH1* B	34.7	73.8	76.3
6	CC9	G75	C	C	T	G	G	G	Hap*GRH1* B	8.3	31.0	47.6
7	CC10	G2	C	C	T	G	G	G	Hap*GRH1* B	55.3	91.1	100.0
8	CC11	G3	C	C	T	G	G	G	Hap*GRH1* B	0.0	31.3	56.8
9	CC12	G120	N	A	A	T	C	N	Unknown	100.0	100.0	100.0
10	CC13	G200	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
11	CC14	G4	C	C	T	G	G	G	Hap*GRH1* B	0.0	35.6	68.5
12	CC15	G76	C	C	T	G	G	G	Hap*GRH1* B	81.1	97.8	97.8
13	CC17	G121	T	A	A	N	C	A	Hap*GRH1* A	100.0	100.0	100.0
14	CC19	G250	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	11.1
15	CC20	G77	T	A	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
16	CC22	G251	C	C	T	G	G	G	Hap*GRH1* B	4.4	6.9	6.9
17	CC23	G36	C	C	T	G	G	G	Hap*GRH1* B	28.8	82.7	89.8
18	CC24	G133	C	C	T	G	G	G	Hap*GRH1* B	2.2	20.2	44.0
19	CC26	G5	C	C	T	G	G	G	Hap*GRH1* B	66.1	91.8	96.0
20	CC28	G6	C	C	T	G	G	G	Hap*GRH1* B	0.0	35.3	67.3
21	CC30	G175	C	C	T	G	G	G	Hap*GRH1* B	0.0	3.6	28.1
22	CC33	G252	C	C	T	G	G	N	Hap*GRH1* B	8.4	35.1	60.5
23	CC34	G78	T	A	A	T	C	A	Hap*GRH1* A	97.5	97.5	97.5
24	CC35	G253	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	0.0
25	CC36	G134	C	C	T	G	G	G	Hap*GRH1* B	97.8	97.8	97.8
26	CC38	G79	C	C	T	G	G	G	Hap*GRH1* B	11.1	45.1	66.0
27	CC46	G38	C	C	T	G	G	G	Hap*GRH1* B	2.0	6.5	29.4
28	CC47	G80	C	C	T	G	G	G	Hap*GRH1* B	0.0	18.4	66.2
29	CC48	G39	C	C	T	G	G	G	Hap*GRH1* B	22.9	42.3	62.8
30	CC49	G81	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.5	2.5
31	CC53	G82	C	C	T	G	G	G	Hap*GRH1* B	0.0	4.0	28.2
32	CC56	G7	C	C	T	G	G	G	Hap*GRH1* B	9.3	31.5	57.5
33	CC57	G40	C	C	T	G	G	G	Hap*GRH1* B	32.9	89.0	100.0
34	CC58	G41	C	C	T	G	G	G	Hap*GRH1* B	92.8	100.0	100.0
35	CC59	G42	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.0	2.0
36	CC61	G83	C	C	T	G	G	G	Hap*GRH1* B	2.5	2.5	4.7
37	CC65	G43	C	C	T	G	G	G	Hap*GRH1* B	41.9	87.1	89.3
38	CC66	G44	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	7.3
39	CC67	G45	C	C	T	G	G	G	Hap*GRH1* B	0.0	54.4	60.9
40	CC68	G85	C	C	T	G	G	G	Hap*GRH1* B	74.0	94.0	98.0
41	CC71	G86	C	C	T	G	G	G	Hap*GRH1* B	0.0	14.5	68.2
42	CC73	G87	C	C	T	G	G	G	Hap*GRH1* B	51.6	77.7	85.8
43	CC75	G46	C	C	T	G	G	G	Hap*GRH1* B	2.5	7.5	26.4
44	CC76	G47	C	C	T	G	G	G	Hap*GRH1* B	37.5	66.7	87.5
45	CC79	G122	T	A	A	T	C	N	Hap*GRH1* A	100.0	100.0	100.0
46	CC82	G8	C	C	T	G	G	G	Hap*GRH1* B	9.9	31.8	68.1

Table 6. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH1*[d]						Haplotype[e]	Nymph mortality (%)[f]		
			chr05_8117089	chr05_8132152	chr05_8144446	chr05_8208059	chr05_8325226	chr05_8571734		3DAI	5DAI	7DAI
47	CC83	G48	C	C	T	G	G	G	Hap*GRH1* B	10.7	66.8	78.0
48	CC87	G89	C	C	T	G	G	G	Hap*GRH1* B	97.5	100.0	100.0
49	CC89	G49	C	C	T	G	G	G	Hap*GRH1* B	2.5	69.9	95.0
50	CC92	G50	C	C	T	G	G	G	Hap*GRH1* B	9.8	19.2	31.7
51	CC94	G90	C	C	T	G	G	G	Hap*GRH1* B	37.5	53.8	85.2
52	CC96	G91	C	C	T	G	G	G	Hap*GRH1* B	85.6	92.7	97.8
53	CC98	G92	T	A	A	T	C	A	Hap*GRH1* A	95.6	97.8	100.0
54	CC99	G52	C	C	T	G	G	G	Hap*GRH1* B	0.0	45.0	62.0
55	CC100	G93	C	C	T	G	G	G	Hap*GRH1* B	21.0	50.2	74.9
56	CC105	G53	C	C	T	G	G	G	Hap*GRH1* B	0.0	5.0	22.5
57	CC106	G123	T	A	A	N	C	A	Hap*GRH1* A	97.8	97.8	97.8
58	CC116	G54	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.0	6.2
59	CC123	G97	T	A	N	N	C	A	Unknown	98.0	100.0	100.0
60	CC125	G98	C	C	T	G	N	G	Hap*GRH1* B	54.6	79.8	92.8
61	CC126	G10	C	C	T	G	G	G	Hap*GRH1* B	0.0	25.4	50.5
62	CC130	G99	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	11.0
63	CC131	G100	C	C	T	G	G	G	Hap*GRH1* B	56.4	90.2	95.3
64	CC132	G101	C	C	T	G	G	G	Hap*GRH1* B	34.9	61.2	70.2
65	CC139	G102	C	C	T	G	G	G	Hap*GRH1* B	69.2	88.9	93.3
66	CC142	G103	C	C	T	G	G	G	Hap*GRH1* B	81.9	90.6	93.1
67	CC143	G124	T	N	N	T	N	A	Unknown	90.0	95.0	100.0
68	CC145	G105	C	C	T	G	G	G	Hap*GRH1* B	23.0	52.5	68.1
69	CC146	G106	C	C	T	G	G	G	Hap*GRH1* B	5.3	39.6	67.3
70	CC147	G55	C	C	T	G	G	G	Hap*GRH1* B	40.3	64.7	64.7
71	CC151	G176	C	C	T	G	G	G	Hap*GRH1* B	2.0	27.3	68.0
72	CC152	G107	T	A	A	T	C	A	Hap*GRH1* A	97.5	100.0	100.0
73	CC153	G136	C	C	T	G	G	G	Hap*GRH1* B	93.8	100.0	100.0
74	CC158	G108	C	C	T	G	G	G	Hap*GRH1* B	89.3	95.6	97.8
75	CC163	G12	C	C	T	G	G	G	Hap*GRH1* B	17.8	91.1	97.5
76	CC164	G56	C	C	T	G	G	G	Hap*GRH1* B	6.7	45.9	71.1
77	CC166	G57	C	C	T	G	G	G	Hap*GRH1* B	2.2	4.2	10.9
78	CC167	G109	N	C	T	G	G	N	Hap*GRH1* B	2.5	4.3	36.9
79	CC170	G110	C	C	T	G	G	G	Hap*GRH1* B	16.0	20.5	60.9
80	CC171	G111	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	9.4
81	CC172	G254	C	C	T	G	G	G	Hap*GRH1* B	2.5	14.2	58.5
82	CC176	G58	C	C	T	G	G	G	Hap*GRH1* B	0.0	28.6	45.3
83	CC184	G138	T	A	A	T	C	A	Hap*GRH1* A	94.9	100.0	100.0
84	CC189	G201	T	A	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
85	CC192	G139	C	C	T	G	G	G	Hap*GRH1* B	17.4	25.1	27.9
86	CC193	G13	C	C	T	G	G	G	Hap*GRH1* B	4.0	26.2	46.4
87	CC195	G202	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	8.0
88	CC197	G203	T	A	N	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
89	CC198	G125	T	N	A	T	C	A	Hap*GRH1* A	98.0	100.0	100.0
90	CC199	G255	C	C	T	G	G	G	Hap*GRH1* B	0.0	13.0	44.2
91	CC202	G140	C	C	T	G	G	G	Hap*GRH1* B	78.1	84.3	86.3
92	CC206	G14	C	C	T	G	G	G	Hap*GRH1* B	0.0	21.4	30.6

Table 6. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH1*[d]						Haplotype[e]	Nymph mortality (%)[f]		
			chr05_8117089	chr05_8132152	chr05_8144446	chr05_8208059	chr05_8325226	chr05_8571734		3DAI	5DAI	7DAI
93	CC208	G141	C	C	T	G	G	G	Hap*GRH1* B	0.0	30.6	81.0
94	CC211	G205	C	C	T	G	G	G	Hap*GRH1* B	14.4	49.7	57.0
95	CC212	G178	C	C	T	G	G	G	Hap*GRH1* B	84.3	95.3	97.8
96	CC215	G207	C	C	T	G	G	G	Hap*GRH1* B	3.3	13.1	13.1
97	CC217	G142	C	C	T	G	G	G	Hap*GRH1* B	87.0	94.9	100.0
98	CC221	G208	C	C	T	G	G	G	Hap*GRH1* B	2.5	5.0	21.4
99	CC223	G210	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
100	CC224	G143	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.5	5.0
101	CC230	G212	N	N	T	N	N	N	Unknown	61.0	70.5	80.0
102	CC231	G213	C	C	T	G	G	G	Hap*GRH1* B	2.0	9.0	13.5
103	CC232	G256	C	C	T	G	G	G	Hap*GRH1* B	5.0	9.0	55.3
104	CC235	G144	C	C	T	G	G	G	Hap*GRH1* B	2.2	2.2	6.4
105	CC236	G214	C	C	T	G	G	G	Hap*GRH1* B	50.1	65.7	72.4
106	CC239	G180	C	C	T	G	G	G	Hap*GRH1* B	43.6	62.2	71.7
107	CC240	G145	T	N	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
108	CC242	G113	C	C	T	G	G	G	Hap*GRH1* B	66.1	87.8	95.0
109	CC244	G257	C	C	T	G	G	G	Hap*GRH1* B	48.3	63.5	74.1
110	CC248	G258	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.5	35.8
111	CC257	G148	T	A	A	T	C	A	Hap*GRH1* A	97.1	100.0	100.0
112	CC259	G127	C	C	T	G	G	G	Hap*GRH1* B	45.4	64.0	80.1
113	CC262	G215	C	C	T	G	G	G	Hap*GRH1* B	2.2	2.2	12.6
114	CC265	G259	C	C	T	G	G	G	Hap*GRH1* B	8.0	14.2	47.1
115	CC268	G261	C	C	T	G	G	G	Hap*GRH1* B	85.3	98.0	100.0
116	CC270	G216	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
117	CC271	G217	C	C	T	G	G	G	Hap*GRH1* B	54.7	59.2	68.2
118	CC273	G183	C	C	T	G	G	G	Hap*GRH1* B	4.2	4.2	31.9
119	CC274	G184	C	C	T	G	G	G	Hap*GRH1* B	59.0	78.0	80.5
120	CC275	G149	C	C	T	G	G	G	Hap*GRH1* B	0.0	4.4	14.9
121	CC280	G186	N	A	A	T	C	A	Hap*GRH1* A	93.3	97.8	100.0
122	CC286	G187	C	C	T	G	G	G	Hap*GRH1* B	64.4	85.2	92.7
123	CC287	G219	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
124	CC288	G220	T	N	A	T	C	A	Hap*GRH1* A	97.5	100.0	100.0
125	CC289	G221	T	A	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
126	CC291	G262	N	C	T	G	G	G	Hap*GRH1* B	44.5	62.8	73.9
127	CC292	G222	C	C	T	G	G	G	Hap*GRH1* B	93.1	95.3	95.3
128	CC300	G223	C	C	T	G	G	G	Hap*GRH1* B	4.4	13.3	44.4
129	CC304	G224	T	A	A	T	C	A	Hap*GRH1* A	94.9	97.1	100.0
130	CC306	G150	C	C	T	G	G	G	Hap*GRH1* B	88.9	95.6	100.0
131	CC307	G225	N	N	N	T	C	A	Unknown	100.0	100.0	100.0
132	CC308	G226	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
133	CC310	G263	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
134	CC312	G151	C	C	T	G	G	G	Hap*GRH1* B	4.4	4.4	13.3
135	CC313	G59	C	C	T	G	G	G	Hap*GRH1* B	0.0	79.6	95.8
136	CC314	G152	T	A	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
137	CC316	G129	T	A	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
138	CC318	G130	T	A	A	T	C	N	Hap*GRH1* A	100.0	100.0	100.0

Table 6. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH1*[d]						Haplotype[e]	Nymph mortality (%)[f]		
			chr05_8117089	chr05_8132152	chr05_8144446	chr05_8208059	chr05_8325226	chr05_8571734		3DAI	5DAI	7DAI
139	CC319	G227	C	C	T	G	G	G	Hap*GRH1* B	2.2	4.2	20.9
140	CC320	G153	C	C	T	G	G	G	Hap*GRH1* B	4.0	27.2	52.3
141	CC324	G264	C	C	T	G	G	G	Hap*GRH1* B	0.0	6.4	67.2
142	CC326	G131	N	A	A	N	C	A	Unknown	97.8	100.0	100.0
143	CC331	G228	N	A	A	T	C	A	Hap*GRH1* A	97.8	97.8	97.8
144	CC333	G155	C	C	T	G	G	G	Hap*GRH1* B	2.2	8.9	58.1
145	CC336	G265	C	C	T	G	G	G	Hap*GRH1* B	0.0	5.0	63.8
146	CC341	G15	C	C	T	G	G	G	Hap*GRH1* B	0.0	16.1	58.6
147	CC342	G156	C	C	T	G	G	G	Hap*GRH1* B	71.5	93.5	96.0
148	CC346	G16	C	C	T	G	G	G	Hap*GRH1* B	5.0	61.8	64.1
149	CC347	G62	C	C	T	G	G	G	Hap*GRH1* B	14.4	80.4	89.1
150	CC351	G157	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.0	51.4
151	CC352	G229	T	A	A	T	C	A	Hap*GRH1* A	97.8	100.0	100.0
152	CC354	G158	T	A	A	N	N	A	Unknown	95.6	100.0	100.0
153	CC355	G230	C	C	T	G	G	G	Hap*GRH1* B	2.5	4.7	4.7
154	CC360	G159	C	C	T	G	G	G	Hap*GRH1* B	0.0	32.0	85.1
155	CC361	G231	T	N	N	T	C	N	Unknown	100.0	100.0	100.0
156	CC364	G17	C	C	T	G	G	G	Hap*GRH1* B	28.4	77.1	79.6
157	CC365	G63	C	C	T	G	G	G	Hap*GRH1* B	9.4	49.2	54.0
158	CC367	G18	C	C	T	G	G	G	Hap*GRH1* B	2.2	4.7	53.6
159	CC368	G266	T	A	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
160	CC371	G64	C	C	T	G	G	G	Hap*GRH1* B	0.0	54.7	82.3
161	CC373	G20	C	C	T	G	G	G	Hap*GRH1* B	0.0	9.7	49.1
162	CC374	G160	C	C	T	G	G	G	Hap*GRH1* B	4.0	44.9	80.0
163	CC375	G188	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.2	15.6
164	CC376	G21	C	C	T	G	G	G	Hap*GRH1* B	11.7	55.8	81.4
165	CC377	G65	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	8.6
166	CC379	G22	C	C	T	G	G	G	Hap*GRH1* B	71.4	92.8	97.5
167	CC382	G23	C	C	T	G	G	G	Hap*GRH1* B	7.5	45.6	85.6
168	CC386	G24	C	C	T	G	G	G	Hap*GRH1* B	34.4	75.8	83.1
169	CC395	G232	C	C	T	G	G	G	Hap*GRH1* B	97.8	97.8	100.0
170	CC396	G233	T	A	A	T	C	A	Hap*GRH1* A	97.5	100.0	100.0
171	CC400	G66	C	C	T	G	G	N	Hap*GRH1* B	2.2	2.2	6.7
172	CC401	G25	C	C	T	G	G	G	Hap*GRH1* B	4.7	48.7	77.2
173	CC402	G132	N	A	A	T	C	A	Hap*GRH1* A	97.8	100.0	100.0
174	CC403	G161	C	C	T	G	G	G	Hap*GRH1* B	2.2	35.7	72.8
175	CC406	G234	C	C	T	G	G	G	Hap*GRH1* B	97.5	100.0	100.0
176	CC411	G190	C	C	T	G	G	G	Hap*GRH1* B	6.4	25.6	40.5
177	CC415	G237	C	C	T	G	G	G	Hap*GRH1* B	2.0	2.0	23.6
178	CC417	G162	T	A	A	T	C	A	Hap*GRH1* A	95.8	100.0	100.0
179	CC418	G191	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
180	CC421	G27	C	C	T	G	G	G	Hap*GRH1* B	8.0	43.2	76.6
181	CC422	G163	C	C	T	G	G	G	Hap*GRH1* B	4.2	9.2	63.5
182	CC423	G164	C	C	T	G	N	G	Hap*GRH1* B	98.0	100.0	100.0
183	CC426	G28	C	C	T	G	G	G	Hap*GRH1* B	17.8	78.4	87.1
184	CC427	G165	T	N	A	T	C	A	Hap*GRH1* A	91.6	100.0	100.0

Table 6. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH1*[d]						Haplotype[e]	Nymph mortality (%)[f]		
			chr05_8117089	chr05_8132152	chr05_8144446	chr05_8208059	chr05_8325226	chr05_8571734		3DAI	5DAI	7DAI
185	CC431	G268	C	C	T	G	G	G	Hap*GRH1* B	0.0	21.4	58.6
186	CC434	G166	N	C	T	G	G	G	Hap*GRH1* B	95.5	97.5	97.5
187	CC435	G167	C	C	T	G	G	G	Hap*GRH1* B	4.4	17.8	70.2
188	CC436	G114	C	C	T	G	G	G	Hap*GRH1* B	6.4	10.4	35.1
189	CC439	G68	C	C	T	G	G	G	Hap*GRH1* B	95.8	98.0	100.0
190	CC442	G30	C	C	T	G	G	G	Hap*GRH1* B	25.4	51.9	68.8
191	CC444	G192	C	C	T	G	G	G	Hap*GRH1* B	87.5	97.5	100.0
192	CC445	G69	C	C	T	G	G	G	Hap*GRH1* B	12.2	17.2	35.8
193	CC450	G70	C	C	T	G	G	G	Hap*GRH1* B	97.5	100.0	100.0
194	CC458	G168	C	C	T	G	G	G	Hap*GRH1* B	0.0	23.0	40.6
195	CC459	G240	C	C	T	G	G	G	Hap*GRH1* B	100.0	100.0	100.0
196	CC460	G193	C	C	T	G	G	N	Hap*GRH1* B	2.0	12.0	38.2
197	CC461	G169	T	A	A	T	C	N	Hap*GRH1* A	100.0	100.0	100.0
198	CC463	G170	T	A	A	T	C	A	Hap*GRH1* A	97.8	97.8	100.0
199	CC468	G269	C	C	T	G	G	G	Hap*GRH1* B	0.0	16.4	59.4
200	CC469	G116	C	C	T	G	G	G	Hap*GRH1* B	0.0	10.0	47.0
201	CC475	G31	C	C	T	G	G	G	Hap*GRH1* B	0.0	6.7	6.7
202	CC477	G194	T	A	A	T	C	A	Hap*GRH1* A	100.0	100.0	100.0
203	CC483	G270	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	2.9
204	CC484	G195	C	C	T	G	G	G	Hap*GRH1* B	2.5	23.6	66.7
205	CC485	G118	C	C	T	G	G	G	Hap*GRH1* B	93.3	100.0	100.0
206	CC490	G241	C	C	T	G	G	G	Hap*GRH1* B	32.7	75.1	77.3
207	CC491	G32	C	C	T	G	G	G	Hap*GRH1* B	63.3	80.9	91.3
208	CC492	G119	N	C	T	G	G	G	Hap*GRH1* B	80.0	80.0	95.6
209	CC493	G72	C	C	T	G	G	G	Hap*GRH1* B	22.2	34.2	34.2
210	CC494	G73	C	C	T	G	G	G	Hap*GRH1* B	0.0	0.0	2.5
211	CC499	G33	C	C	T	G	G	G	Hap*GRH1* B	23.9	52.2	82.9
212	CC500	G242	C	C	T	G	G	G	Hap*GRH1* B	28.3	74.5	86.2
213	CC505	G243	T	A	A	T	C	A	Hap*GRH1* A	98.0	98.0	98.0
214	CC508	G244	C	C	T	N	G	G	Hap*GRH1* B	97.8	100.0	100.0
215	CC509	G171	C	C	T	G	G	G	Hap*GRH1* B	15.7	63.9	81.1
216	CC512	G34	C	C	T	G	G	G	Hap*GRH1* B	36.0	84.0	84.0
217	CC513	G172	C	C	T	G	G	G	Hap*GRH1* B	0.0	2.0	39.6
218	CC515	G35	C	C	T	G	G	G	Hap*GRH1* B	0.0	31.7	51.4
219	CC516	G272	C	C	T	G	G	G	Hap*GRH1* B	11.1	16.8	38.5
220	CC517	G245	T	A	A	T	C	A	Hap*GRH1* A	97.8	100.0	100.0
221	CC523	G198	C	C	T	G	G	G	Hap*GRH1* B	58.3	82.8	82.8
222	CC525	G246	C	C	T	G	N	G	Hap*GRH1* B	100.0	100.0	100.0
223	CC528	G247	C	C	T	G	G	G	Hap*GRH1* B	97.8	100.0	100.0
224	CC529	G199	C	C	T	G	G	G	Hap*GRH1* B	34.0	78.7	89.3

[a] Serial number

[b] Core collections of rice conserved in Seedbank of Department of Agricultural Research, Myanmar

[c] Number of Myanmar Indica diversity panel designated as "G" where the accessions were whole genome sequenced

[d] SNP markers located in the genomic region of GRH1 and used in haplotype analysis

[e] Haplotype variation at the genomic region of GRH1
[f] Nymph mortality (%) of GRH evaluated at 3 days after infestation (DAI), 5DAI and 3DAI

Table 7. Haplotype variations of MIDP at the genomic region of MTA11

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at MTA11[d]			Haplotype[e]	Nymph mortality (%)[f]		
			chr11_27118206	chr11_27119453	chr11_27121054		3DAI	5DAI	7DAI
1	CC1	G1	G	G	C	HapMTA11B	2.2	20.0	31.1
2	CC3	G174	G	G	C	HapMTA11B	0.0	0.0	31.4
3	CC6	G248	G	G	C	HapMTA11B	4.0	56.0	80.5
4	CC7	G74	G	G	N	HapMTA11B	97.5	100.0	100.0
5	CC8	G249	G	G	C	HapMTA11B	34.7	73.8	76.3
6	CC9	G75	G	G	C	HapMTA11B	8.3	31.0	47.6
7	CC10	G2	G	G	C	HapMTA11B	55.3	91.1	100.0
8	CC11	G3	G	G	C	HapMTA11B	0.0	31.3	56.8
9	CC12	G120	G	G	C	HapMTA11B	100.0	100.0	100.0
10	CC13	G200	G	G	C	HapMTA11B	100.0	100.0	100.0
11	CC14	G4	G	G	C	HapMTA11B	0.0	35.6	68.5
12	CC15	G76	G	G	C	HapMTA11B	81.1	97.8	97.8
13	CC17	G121	G	G	C	HapMTA11B	100.0	100.0	100.0
14	CC19	G250	G	G	C	HapMTA11B	0.0	0.0	11.1
15	CC20	G77	G	G	C	HapMTA11B	100.0	100.0	100.0
16	CC22	G251	G	G	C	HapMTA11B	4.4	6.9	6.9
17	CC23	G36	A	T	A	HapMTA11A	28.8	82.7	89.8
18	CC24	G133	G	G	C	HapMTA11B	2.2	20.2	44.0
19	CC26	G5	A	T	A	HapMTA11A	66.1	91.8	96.0
20	CC28	G6	G	G	C	HapMTA11B	0.0	35.3	67.3
21	CC30	G175	G	G	C	HapMTA11B	0.0	3.6	28.1
22	CC33	G252	G	G	C	HapMTA11B	8.4	35.1	60.5
23	CC34	G78	A	T	A	HapMTA11A	97.5	97.5	97.5
24	CC35	G253	G	G	C	HapMTA11B	0.0	0.0	0.0
25	CC36	G134	A	T	A	HapMTA11A	97.8	97.8	97.8
26	CC38	G79	G	G	C	HapMTA11B	11.1	45.1	66.0
27	CC46	G38	G	G	C	HapMTA11B	2.0	6.5	29.4
28	CC47	G80	A	T	A	HapMTA11A	0.0	18.4	66.2
29	CC48	G39	G	G	C	HapMTA11B	22.9	42.3	62.8
30	CC49	G81	G	G	C	HapMTA11B	0.0	2.5	2.5
31	CC53	G82	G	G	C	HapMTA11B	0.0	4.0	28.2
32	CC56	G7	G	G	C	HapMTA11B	9.3	31.5	57.5
33	CC57	G40	A	T	A	HapMTA11A	32.9	89.0	100.0
34	CC58	G41	A	T	A	HapMTA11A	92.8	100.0	100.0
35	CC59	G42	G	G	C	HapMTA11B	0.0	2.0	2.0
36	CC61	G83	G	G	C	HapMTA11B	2.5	2.5	4.7
37	CC65	G43	A	T	A	HapMTA11A	41.9	87.1	89.3
38	CC66	G44	G	G	C	Unknown	0.0	0.0	7.3
39	CC67	G45	A	T	A	HapMTA11A	0.0	54.4	60.9
40	CC68	G85	A	T	A	HapMTA11A	74.0	94.0	98.0
41	CC71	G86	G	G	C	HapMTA11B	0.0	14.5	68.2
42	CC73	G87	A	T	A	HapMTA11A	51.6	77.7	85.8
43	CC75	G46	G	G	C	HapMTA11B	2.5	7.5	26.4
44	CC76	G47	A	T	A	HapMTA11A	37.5	66.7	87.5
45	CC79	G122	G	G	C	HapMTA11B	100.0	100.0	100.0
46	CC82	G8	G	G	C	HapMTA11B	9.9	31.8	68.1

Table 7. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at MTA11[d]			Haplotype[e]	Nymph mortality (%)[f]		
			chr11_27118206	chr11_27119453	chr11_27121054		3DAI	5DAI	7DAI
47	CC83	G48	A	T	A	HapMTA11A	10.7	66.8	78.0
48	CC87	G89	G	G	C	HapMTA11B	97.5	100.0	100.0
49	CC89	G49	A	T	A	HapMTA11A	2.5	69.9	95.0
50	CC92	G50	G	G	C	HapMTA11B	9.8	19.2	31.7
51	CC94	G90	G	G	C	HapMTA11B	37.5	53.8	85.2
52	CC96	G91	A	T	A	HapMTA11A	85.6	92.7	97.8
53	CC98	G92	G	G	C	HapMTA11B	95.6	97.8	100.0
54	CC99	G52	A	T	A	HapMTA11A	0.0	45.0	62.0
55	CC100	G93	A	T	A	HapMTA11A	21.0	50.2	74.9
56	CC105	G53	G	G	C	HapMTA11B	0.0	5.0	22.5
57	CC106	G123	G	G	C	HapMTA11B	97.8	97.8	97.8
58	CC116	G54	G	G	C	HapMTA11B	0.0	2.0	6.2
59	CC123	G97	G	G	C	HapMTA11B	98.0	100.0	100.0
60	CC125	G98	A	T	A	HapMTA11A	54.6	79.8	92.8
61	CC126	G10	G	G	C	HapMTA11B	0.0	25.4	50.5
62	CC130	G99	G	G	C	HapMTA11B	0.0	0.0	11.0
63	CC131	G100	A	T	A	HapMTA11A	56.4	90.2	95.3
64	CC132	G101	A	T	A	HapMTA11A	34.9	61.2	70.2
65	CC139	G102	A	T	A	HapMTA11A	69.2	88.9	93.3
66	CC142	G103	A	T	A	HapMTA11A	81.9	90.6	93.1
67	CC143	G124	G	G	C	HapMTA11B	90.0	95.0	100.0
68	CC145	G105	G	G	C	HapMTA11B	23.0	52.5	68.1
69	CC146	G106	G	G	N	HapMTA11B	5.3	39.6	67.3
70	CC147	G55	A	T	A	Unknown	40.3	64.7	64.7
71	CC151	G176	G	G	C	HapMTA11B	2.0	27.3	68.0
72	CC152	G107	G	G	C	HapMTA11B	97.5	100.0	100.0
73	CC153	G136	A	T	A	HapMTA11A	93.8	100.0	100.0
74	CC158	G108	A	T	A	HapMTA11A	89.3	95.6	97.8
75	CC163	G12	A	T	A	HapMTA11A	17.8	91.1	97.5
76	CC164	G56	G	G	C	HapMTA11B	6.7	45.9	71.1
77	CC166	G57	G	G	C	HapMTA11B	2.2	4.2	10.9
78	CC167	G109	G	G	C	HapMTA11B	2.5	4.3	36.9
79	CC170	G110	G	G	C	HapMTA11B	16.0	20.5	60.9
80	CC171	G111	G	G	N	HapMTA11B	0.0	0.0	9.4
81	CC172	G254	G	G	C	HapMTA11B	2.5	14.2	58.5
82	CC176	G58	G	G	C	HapMTA11B	0.0	28.6	45.3
83	CC184	G138	G	G	C	HapMTA11B	94.9	100.0	100.0
84	CC189	G201	G	G	C	HapMTA11B	100.0	100.0	100.0
85	CC192	G139	G	G	C	HapMTA11B	17.4	25.1	27.9
86	CC193	G13	A	T	A	HapMTA11A	4.0	26.2	46.4
87	CC195	G202	G	G	C	HapMTA11B	0.0	0.0	8.0
88	CC197	G203	G	G	C	HapMTA11B	100.0	100.0	100.0
89	CC198	G125	G	G	C	HapMTA11B	98.0	100.0	100.0
90	CC199	G255	G	G	C	HapMTA11B	0.0	13.0	44.2
91	CC202	G140	G	G	C	HapMTA11B	78.1	84.3	86.3
92	CC206	G14	G	G	C	HapMTA11B	0.0	21.4	30.6

Table 7. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at MTA11[d]			Haplotype[e]	Nymph mortality (%)[f]		
			chr11_27118206	chr11_27119453	chr11_27121054		3DAI	5DAI	7DAI
93	CC208	G141	G	G	C	HapMTA11B	0.0	30.6	81.0
94	CC211	G205	G	G	C	HapMTA11B	14.4	49.7	57.0
95	CC212	G178	G	G	C	HapMTA11B	84.3	95.3	97.8
96	CC215	G207	G	G	C	HapMTA11B	3.3	13.1	13.1
97	CC217	G142	G	G	C	HapMTA11B	87.0	94.9	100.0
98	CC221	G208	G	G	C	HapMTA11B	2.5	5.0	21.4
99	CC223	G210	G	G	C	HapMTA11B	100.0	100.0	100.0
100	CC224	G143	G	G	C	HapMTA11B	0.0	2.5	5.0
101	CC230	G212	G	G	C	HapMTA11B	61.0	70.5	80.0
102	CC231	G213	G	G	C	HapMTA11B	2.0	9.0	13.5
103	CC232	G256	G	G	C	HapMTA11B	5.0	9.0	55.3
104	CC235	G144	G	G	C	HapMTA11B	2.2	2.2	6.4
105	CC236	G214	G	G	C	HapMTA11B	50.1	65.7	72.4
106	CC239	G180	G	G	C	HapMTA11B	43.6	62.2	71.7
107	CC240	G145	G	G	C	HapMTA11B	100.0	100.0	100.0
108	CC242	G113	G	G	C	HapMTA11B	66.1	87.8	95.0
109	CC244	G257	G	G	C	HapMTA11B	48.3	63.5	74.1
110	CC248	G258	G	G	C	HapMTA11B	0.0	2.5	35.8
111	CC257	G148	G	G	C	HapMTA11B	97.1	100.0	100.0
112	CC259	G127	G	G	C	HapMTA11B	45.4	64.0	80.1
113	CC262	G215	G	G	C	HapMTA11B	2.2	2.2	12.6
114	CC265	G259	G	G	C	HapMTA11B	8.0	14.2	47.1
115	CC268	G261	G	G	C	HapMTA11B	85.3	98.0	100.0
116	CC270	G216	G	G	C	HapMTA11B	100.0	100.0	100.0
117	CC271	G217	G	G	C	HapMTA11B	54.7	59.2	68.2
118	CC273	G183	G	G	C	HapMTA11B	4.2	4.2	31.9
119	CC274	G184	G	G	C	HapMTA11B	59.0	78.0	80.5
120	CC275	G149	G	G	C	HapMTA11B	0.0	4.4	14.9
121	CC280	G186	G	G	C	HapMTA11B	93.3	97.8	100.0
122	CC286	G187	G	G	C	HapMTA11B	64.4	85.2	92.7
123	CC287	G219	G	G	C	HapMTA11B	100.0	100.0	100.0
124	CC288	G220	G	G	C	HapMTA11B	97.5	100.0	100.0
125	CC289	G221	G	G	C	HapMTA11B	100.0	100.0	100.0
126	CC291	G262	G	G	C	HapMTA11B	44.5	62.8	73.9
127	CC292	G222	G	G	C	HapMTA11B	93.1	95.3	95.3
128	CC300	G223	G	G	C	HapMTA11B	4.4	13.3	44.4
129	CC304	G224	G	G	C	HapMTA11B	94.9	97.1	100.0
130	CC306	G150	G	G	C	HapMTA11B	88.9	95.6	100.0
131	CC307	G225	G	G	C	HapMTA11B	100.0	100.0	100.0
132	CC308	G226	G	G	C	HapMTA11B	100.0	100.0	100.0
133	CC310	G263	G	G	C	HapMTA11B	100.0	100.0	100.0
134	CC312	G151	G	G	N	HapMTA11B	4.4	4.4	13.3
135	CC313	G59	G	G	A	HapMTA11B	0.0	79.6	95.8
136	CC314	G152	G	G	C	HapMTA11B	100.0	100.0	100.0
137	CC316	G129	G	G	C	HapMTA11B	100.0	100.0	100.0
138	CC318	G130	G	G	C	HapMTA11B	100.0	100.0	100.0

Table 7. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at MTA11[d]			Haplotype[e]	Nymph mortality (%)[f]		
			chr11_27118206	chr11_27119453	chr11_27121054		3DAI	5DAI	7DAI
139	CC319	G227	G	G	C	HapMTA11B	2.2	4.2	20.9
140	CC320	G153	G	G	C	HapMTA11B	4.0	27.2	52.3
141	CC324	G264	G	G	C	HapMTA11B	0.0	6.4	67.2
142	CC326	G131	G	G	C	HapMTA11B	97.8	100.0	100.0
143	CC331	G228	G	G	C	HapMTA11B	97.8	97.8	97.8
144	CC333	G155	G	G	C	HapMTA11B	2.2	8.9	58.1
145	CC336	G265	G	G	C	HapMTA11B	0.0	5.0	63.8
146	CC341	G15	G	G	C	HapMTA11B	0.0	16.1	58.6
147	CC342	G156	G	G	C	HapMTA11B	71.5	93.5	96.0
148	CC346	G16	G	G	C	HapMTA11B	5.0	61.8	64.1
149	CC347	G62	A	T	A	HapMTA11A	14.4	80.4	89.1
150	CC351	G157	G	G	C	HapMTA11B	0.0	2.0	51.4
151	CC352	G229	G	G	C	HapMTA11B	97.8	100.0	100.0
152	CC354	G158	G	G	C	HapMTA11B	95.6	100.0	100.0
153	CC355	G230	G	G	C	HapMTA11B	2.5	4.7	4.7
154	CC360	G159	G	G	C	HapMTA11B	0.0	32.0	85.1
155	CC361	G231	G	G	C	HapMTA11B	100.0	100.0	100.0
156	CC364	G17	G	G	C	HapMTA11B	28.4	77.1	79.6
157	CC365	G63	A	T	A	HapMTA11A	9.4	49.2	54.0
158	CC367	G18	N	N	N	Unknown	2.2	4.7	53.6
159	CC368	G266	G	G	C	HapMTA11B	100.0	100.0	100.0
160	CC371	G64	A	T	A	HapMTA11A	0.0	54.7	82.3
161	CC373	G20	G	G	C	HapMTA11B	0.0	9.7	49.1
162	CC374	G160	G	G	C	HapMTA11B	4.0	44.9	80.0
163	CC375	G188	G	G	C	HapMTA11B	0.0	2.2	15.6
164	CC376	G21	G	G	C	HapMTA11B	11.7	55.8	81.4
165	CC377	G65	G	G	C	HapMTA11B	0.0	0.0	8.6
166	CC379	G22	A	T	A	HapMTA11A	71.4	92.8	97.5
167	CC382	G23	A	T	A	HapMTA11A	7.5	45.6	85.6
168	CC386	G24	G	G	C	HapMTA11B	34.4	75.8	83.1
169	CC395	G232	G	G	C	HapMTA11B	97.8	97.8	100.0
170	CC396	G233	G	G	C	HapMTA11B	97.5	100.0	100.0
171	CC400	G66	G	G	C	HapMTA11B	2.2	2.2	6.7
172	CC401	G25	G	G	C	HapMTA11B	4.7	48.7	77.2
173	CC402	G132	G	G	C	HapMTA11B	97.8	100.0	100.0
174	CC403	G161	G	G	C	HapMTA11B	2.2	35.7	72.8
175	CC406	G234	G	G	C	HapMTA11B	97.5	100.0	100.0
176	CC411	G190	G	G	C	HapMTA11B	6.4	25.6	40.5
177	CC415	G237	G	G	C	HapMTA11B	2.0	2.0	23.6
178	CC417	G162	G	G	C	HapMTA11B	95.8	100.0	100.0
179	CC418	G191	G	G	C	HapMTA11B	100.0	100.0	100.0
180	CC421	G27	G	G	C	HapMTA11B	8.0	43.2	76.6
181	CC422	G163	G	G	C	HapMTA11B	4.2	9.2	63.5
182	CC423	G164	G	G	C	HapMTA11B	98.0	100.0	100.0
183	CC426	G28	G	G	C	HapMTA11B	17.8	78.4	87.1
184	CC427	G165	G	G	C	HapMTA11B	91.6	100.0	100.0

Table 7. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at MTA11[d]			Haplotype[e]	Nymph mortality (%)[f]		
			chr11_27118206	chr11_27119453	chr11_27121054		3DAI	5DAI	7DAI
185	CC431	G268	G	G	N	HapMTA11B	0.0	21.4	58.6
186	CC434	G166	G	G	C	HapMTA11B	95.5	97.5	97.5
187	CC435	G167	G	G	C	HapMTA11B	4.4	17.8	70.2
188	CC436	G114	G	G	C	HapMTA11B	6.4	10.4	35.1
189	CC439	G68	A	T	A	HapMTA11A	95.8	98.0	100.0
190	CC442	G30	G	G	C	HapMTA11B	25.4	51.9	68.8
191	CC444	G192	G	G	C	HapMTA11B	87.5	97.5	100.0
192	CC445	G69	G	G	C	Unknown	12.2	17.2	35.8
193	CC450	G70	G	G	C	HapMTA11B	97.5	100.0	100.0
194	CC458	G168	G	G	C	HapMTA11B	0.0	23.0	40.6
195	CC459	G240	G	G	C	HapMTA11B	100.0	100.0	100.0
196	CC460	G193	G	G	C	HapMTA11B	2.0	12.0	38.2
197	CC461	G169	G	G	C	HapMTA11B	100.0	100.0	100.0
198	CC463	G170	G	G	C	HapMTA11B	97.8	97.8	100.0
199	CC468	G269	G	G	C	HapMTA11B	0.0	16.4	59.4
200	CC469	G116	G	G	C	HapMTA11B	0.0	10.0	47.0
201	CC475	G31	G	G	C	HapMTA11B	0.0	6.7	6.7
202	CC477	G194	G	G	C	HapMTA11B	100.0	100.0	100.0
203	CC483	G270	G	G	C	HapMTA11B	0.0	0.0	2.9
204	CC484	G195	G	G	C	HapMTA11B	2.5	23.6	66.7
205	CC485	G118	G	G	C	HapMTA11B	93.3	100.0	100.0
206	CC490	G241	G	G	C	HapMTA11B	32.7	75.1	77.3
207	CC491	G32	G	G	C	HapMTA11B	63.3	80.9	91.3
208	CC492	G119	A	T	A	HapMTA11A	80.0	80.0	95.6
209	CC493	G72	G	G	C	HapMTA11B	22.2	34.2	34.2
210	CC494	G73	G	G	C	HapMTA11B	0.0	0.0	2.5
211	CC499	G33	G	G	C	HapMTA11B	23.9	52.2	82.9
212	CC500	G242	G	G	C	HapMTA11B	28.3	74.5	86.2
213	CC505	G243	G	G	C	HapMTA11B	98.0	98.0	98.0
214	CC508	G244	G	G	C	HapMTA11B	97.8	100.0	100.0
215	CC509	G171	G	G	C	HapMTA11B	15.7	63.9	81.1
216	CC512	G34	G	G	C	HapMTA11B	36.0	84.0	84.0
217	CC513	G172	G	G	C	HapMTA11B	0.0	2.0	39.6
218	CC515	G35	G	G	C	HapMTA11B	0.0	31.7	51.4
219	CC516	G272	G	G	C	HapMTA11B	11.1	16.8	38.5
220	CC517	G245	G	G	C	HapMTA11B	97.8	100.0	100.0
221	CC523	G198	G	G	C	HapMTA11B	58.3	82.8	82.8
222	CC525	G246	G	G	C	HapMTA11B	100.0	100.0	100.0
223	CC528	G247	G	G	C	HapMTA11B	97.8	100.0	100.0
224	CC529	G199	G	G	C	HapMTA11B	34.0	78.7	89.3

[a] Serial number

[b] Core collections of rice conserved in Seedbank of Department of Agricultural Research, Myanmar

[c] Number of Myanmar Indica diversity panel designated as "G" where the accessions were whole genome sequenced

[d] SNP markers located in the genomic region of MTA11 and used in haplotype analysis

[e] Haplotype variation at the genomic region of MTA11

[f] Nymph mortality (%) of GRH evaluated at 3 days after infestation (DAI), 5DAI and 3DAI

Table 8. Haplotype variations of MIDP at the candidate gene of *GRH6* (*Os04g0166000*)

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH6*[d]																																					Haplotype[e]	Nymph mortality (%)[f]		
			chr04_4485182	chr04_4485745	chr04_4486245	chr04_4486257	chr04_4486925	chr04_4486942	chr04_4487022	chr04_4487111	chr04_4488061	chr04_4488070	chr04_4488274	chr04_4488438	chr04_4488622	chr04_4488717	chr04_4488718	chr04_4488724	chr04_4488726	chr04_4488740	chr04_4488757	chr04_4489386	chr04_4489455	chr04_4489478	chr04_4489481	chr04_4489567	chr04_4489599	chr04_4489610	chr04_4489651	chr04_4489652	chr04_4489655	chr04_4489669	chr04_4489675	chr04_4489699	chr04_4489765	chr04_4489778	chr04_4489851	chr04_4489859	chr04_4490010		3DAI	5DAI	7DAI
1	CC1	G1	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	2.2	20.0	31.1
2	CC3	G174	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	0.0	0.0	31.4
3	CC6	G248	A	G	G	C	G	Ref	C	C	G	T	G	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	G	Unknown	4.0	56.0	80.5
4	CC7	G74	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Hap*GRH6* A	97.5	100.0	100.0
5	CC8	G249	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	34.7	73.8	76.3
6	CC9	G75	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	8.3	31.0	47.6
7	CC10	G2	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	55.3	91.1	100.0
8	CC11	G3	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	0.0	31.3	56.8
9	CC12	G120	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Unknown	100.0	100.0	100.0
10	CC13	G200	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	100.0	100.0	100.0
11	CC14	G4	A	G	G	C	G	Ref	C	C	G	T	T	T	C	T	C	A	Ref	Ref	Ref	N	N	N	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	T	Unknown	0.0	35.6	68.5
12	CC15	G76	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Hap*GRH6* A	81.1	97.8	97.8
13	CC17	G121	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	100.0	100.0	100.0
14	CC19	G250	A	G	G	C	G	Ref	C	C	G	T	G	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	G	Unknown	0.0	0.0	11.1
15	CC20	G77	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	100.0	100.0	100.0
16	CC22	G251	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	4.4	6.9	6.9
17	CC23	G36	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	28.8	82.7	89.8
18	CC24	G133	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	2.2	20.2	44.0
19	CC26	G5	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	66.1	91.8	96.0
20	CC28	G6	A	G	G	C	G	Ref	C	C	G	T	G	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	G	Unknown	0.0	35.3	67.3
21	CC30	G175	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	0.0	3.6	28.1
22	CC33	G252	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	8.4	35.1	60.5
23	CC34	G78	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	97.5	97.5	97.5
24	CC35	G253	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	0.0	0.0	0.0
25	CC36	G134	N	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	N	N	N	T	14bpIn	C	C	C	C	G	C	G	A	N	N	N	T	Unknown	97.8	97.8	97.8
26	CC38	G79	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	11.1	45.1	66.0
27	CC46	G38	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	N	N	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	2.0	6.5	29.4
28	CC47	G80	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	N	N	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	0.0	18.4	66.2
29	CC48	G39	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	22.9	42.3	62.8
30	CC49	G81	N	G	G	C	G	Ref	C	C	G	T	G	T	C	N	N	N	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	0.0	2.5	2.5
31	CC53	G82	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* B	0.0	4.0	28.2
32	CC56	G7	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	9.3	31.5	57.5
33	CC57	G40	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* B	32.9	89.0	100.0
34	CC58	G41	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* B	92.8	100.0	100.0
35	CC59	G42	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* B	0.0	2.0	2.0

Table 8. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH6*[d] chr04_4485182	chr04_4485745	chr04_4486245	chr04_4486257	chr04_4486925	chr04_4486942	chr04_4487022	chr04_4487111	chr04_4488061	chr04_4488070	chr04_4488274	chr04_4488438	chr04_4488622	chr04_4488717	chr04_4488718	chr04_4488724	chr04_4488726	S04_4488740	chr04_4488757	chr04_4489386	chr04_4489455	chr04_4489478	chr04_4489481	chr04_4489567	chr04_4489599	chr04_4489610	chr04_4489651	chr04_4489652	chr04_4489655	chr04_4489669	chr04_4489675	chr04_4489699	chr04_4489765	chr04_4489778	chr04_4489851	chr04_4489859	chr04_4490010	Haplotype[e]	Nymph mortality (%)[f] 3DAI	5DAI	7DAI
36	CC61	G83	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	2.5	2.5	4.7
37	CC65	G43	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	41.9	87.1	89.3
38	CC66	G44	A	N	G	C	N	Ref	C	N	N	T	N	N	C	N	N	N	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Unknown	0.0	0.0	7.3
39	CC67	G45	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	0.0	54.4	60.9
40	CC68	G85	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	74.0	94.0	98.0
41	CC71	G86	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	0.0	14.5	68.2
42	CC73	G87	A	G	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Unknown	51.6	77.7	85.8
43	CC75	G46	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	2.5	7.5	26.4
44	CC76	G47	A	G	G	C	G	Ref	C	C	G	T	G	T	C	N	C	N	Ref	N	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	37.5	66.7	87.5
45	CC79	G122	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	100.0	100.0	100.0
46	CC82	G8	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	9.9	31.8	68.1
47	CC83	G48	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	N	N	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	10.7	66.8	78.0
48	CC87	G89	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	97.5	100.0	100.0
49	CC89	G49	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	2.5	69.9	95.0
50	CC92	G50	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	N	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	9.8	19.2	31.7
51	CC94	G90	A	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	N	C	C	C	N	N	N	G	A	G	T	G	N	Hap*GRH6* D	37.5	53.8	85.2
52	CC96	G91	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	85.6	92.7	97.8
53	CC98	G92	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	95.6	97.8	100.0
54	CC99	G52	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	0.0	45.0	62.0
55	CC100	G93	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	21.0	50.2	74.9
56	CC105	G53	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	0.0	5.0	22.5
57	CC106	G123	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	97.8	97.8	97.8
58	CC116	G54	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	0.0	2.0	6.2
59	CC123	G97	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	98.0	100.0	100.0
60	CC125	G98	A	N	G	C	G	3bpDel	T	C	G	N	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	54.6	79.8	92.8
61	CC126	G10	N	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Unknown	0.0	25.4	50.5
62	CC130	G99	A	N	G	C	G	3bpDel	T	C	G	N	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	0.0	0.0	11.0
63	CC131	G100	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	56.4	90.2	95.3
64	CC132	G101	A	N	G	C	G	3bpDel	T	C	N	N	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	34.9	61.2	70.2
65	CC139	G102	G	G	T	A	G	Ref	C	N	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	69.2	88.9	93.3
66	CC142	G103	A	G	N	N	G	Ref	C	C	G	T	G	T	T	T	C	A	Ref	Ref	Ref	C	N	G	G	T	14bpIn	C	C	C	C	G	C	G	A	A	A	A	T	Unknown	81.9	90.6	93.1
67	CC143	G124	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Unknown	90.0	95.0	100.0
68	CC145	G105	A	A	G	C	N	N	N	N	N	T	C	C	T	C	A	Ref	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* B	23.0	52.5	68.1
69	CC146	G106	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	5.3	39.6	67.3
70	CC147	G55	A	G	G	C	G	Ref	C	C	G	N	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	40.3	64.7	64.7

Table 8. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH6*[d]																																				Haplotype[e]	Nymph mortality (%)[f]		
			chr04_4485182	chr04_4485745	chr04_4486245	chr04_4486257	chr04_4486925	chr04_4486942	chr04_4487022	chr04_4487111	chr04_4488061	chr04_4488070	chr04_4488274	chr04_4488438	chr04_4488622	chr04_4488717	chr04_4488718	chr04_4488724	chr04_4488726	S04_4488740	chr04_4488757	chr04_4489386	chr04_4489455	chr04_4489478	chr04_4489481	chr04_4489567	chr04_4489599	chr04_4489610	chr04_4489651	chr04_4489652	chr04_4489655	chr04_4489669	chr04_4489675	chr04_4489699	chr04_4489765	chr04_4489778	chr04_4489851	chr04_4489859	chr04_4490010		3DAI	5DAI	7DAI
71	CC151	G176	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	2.0	27.3	68.0
72	CC152	G107	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	HapGRH6 B	97.5	100.0	100.0
73	CC153	G136	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	HapGRH6 D	93.8	100.0	100.0
74	CC158	G108	A	G	N	N	G	Ref	C	C	G	T	N	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	A	A	A	T	Unknown	89.3	95.6	97.8
75	CC163	G12	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 B	17.8	91.1	97.5
76	CC164	G56	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	6.7	45.9	71.1
77	CC166	G57	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	2.2	4.2	10.9
78	CC167	G109	N	G	N	N	G	Ref	C	N	N	N	N	N	N	T	C	A	Ref	Ref	Ref	N	1bpIn	G	G	T	14bpIn	C	C	C	C	N	N	N	A	A	T	G	T	HapGRH6 G	2.5	4.3	36.9
79	CC170	G110	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	16.0	20.5	60.9
80	CC171	G111	G	G	T	A	G	Ref	C	C	N	N	T	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	0.0	0.0	9.4
81	CC172	G254	A	N	G	C	N	N	N	N	N	N	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	HapGRH6 E	2.5	14.2	58.5
82	CC176	G58	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	N	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	0.0	28.6	45.3
83	CC184	G138	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	94.9	100.0	100.0
84	CC189	G201	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	HapGRH6 A	100.0	100.0	100.0
85	CC192	G139	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	17.4	25.1	27.9
86	CC193	G13	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	4.0	26.2	46.4
87	CC195	G202	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 B	0.0	0.0	8.0
88	CC197	G203	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Unknown	100.0	100.0	100.0
89	CC198	G125	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	98.0	100.0	100.0
90	CC199	G255	A	G	G	C	G	Ref	N	N	N	N	G	T	C	N	N	N	N	N	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Unknown	0.0	13.0	44.2
91	CC202	G140	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	78.1	84.3	86.3
92	CC206	G14	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	0.0	21.4	30.6
93	CC208	G141	A	G	G	C	G	Ref	C	C	G	T	G	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	G	Unknown	0.0	30.6	81.0
94	CC211	G205	G	G	T	A	G	Ref	C	N	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	14.4	49.7	57.0
95	CC212	G178	A	N	G	C	G	3bpDel	T	C	G	N	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	HapGRH6 E	84.3	95.3	97.8
96	CC215	G207	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Unknown	3.3	13.1	13.1
97	CC217	G142	N	G	G	C	G	Ref	C	C	G	T	G	T	N	N	N	N	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	N	C	G	C	G	A	N	N	N	T	Unknown	87.0	94.9	100.0
98	CC221	G208	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	N	N	N	N	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	2.5	5.0	21.4
99	CC223	G210	A	G	G	C	G	Ref	C	C	N	N	N	T	C	T	C	A	Ref	Ref	Ref	N	N	N	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	T	Unknown	100.0	100.0	100.0
100	CC224	G143	A	G	G	C	G	Ref	C	C	G	T	T	T	C	T	C	A	Ref	Ref	Ref	N	N	N	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	T	Unknown	0.0	2.5	5.0
101	CC230	G212	A	G	N	N	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	HapGRH6 A	61.0	70.5	80.0
102	CC231	G213	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	HapGRH6 E	2.0	9.0	13.5
103	CC232	G256	A	G	G	C	G	Ref	C	C	G	T	T	T	C	T	C	A	Ref	Ref	Ref	N	N	N	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	T	Unknown	5.0	9.0	55.3
104	CC235	G144	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	2.2	2.2	6.4
105	CC236	G214	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	50.1	65.7	72.4

Table 8. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH6*[d]																																		Haplotype[e]	Nymph mortality (%)[f]		
			chr04_4485182	chr04_4485745	chr04_4486245	chr04_4486257	chr04_4486925	chr04_4486942	chr04_4487022	chr04_4487111	chr04_4488061	chr04_4488070	chr04_4488274	chr04_4488438	chr04_4488622	chr04_4488717	chr04_4488718	chr04_4488724	chr04_4488726	S04_4488740	chr04_4488757	chr04_4489386	chr04_4489455	chr04_4489478	chr04_4489481	chr04_4489567	chr04_4489599	chr04_4489610	chr04_4489651	chr04_4489652	chr04_4489655	chr04_4489669	chr04_4489675	chr04_4489699	chr04_4489765	chr04_4489778	chr04_4489851	chr04_4489859	chr04_4490010		3DAI	5DAI	7DAI
106	CC239	G180	A	G	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Unknown	43.6	62.2	71.7
107	CC240	G145	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	100.0	100.0	100.0
108	CC242	G113	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	N	C	G	A	A	T	G	T	Hap*GRH6* B	66.1	87.8	95.0
109	CC244	G257	A	G	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Unknown	48.3	63.5	74.1
110	CC248	G258	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	0.0	2.5	35.8
111	CC257	G148	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	97.1	100.0	100.0
112	CC259	G127	A	G	G	C	G	Ref	C	C	N	N	N	T	C	T	C	A	Ref	Ref	Ref	N	N	N	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	T	Unknown	45.4	64.0	80.1
113	CC262	G215	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Unknown	2.2	2.2	12.6
114	CC265	G259	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* B	8.0	14.2	47.1
115	CC268	G261	G	G	G	C	G	Ref	C	C	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	T	G	G	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Unknown	85.3	98.0	100.0
116	CC270	G216	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	N	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Hap*GRH6* A	100.0	100.0	100.0
117	CC271	G217	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	54.7	59.2	68.2
118	CC273	G183	A	G	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Unknown	4.2	4.2	31.9
119	CC274	G184	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	59.0	78.0	80.5
120	CC275	G149	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	0.0	4.4	14.9
121	CC280	G186	A	N	G	C	N	N	N	N	N	N	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	93.3	97.8	100.0
122	CC286	G187	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	64.4	85.2	92.7
123	CC287	G219	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Hap*GRH6* A	100.0	100.0	100.0
124	CC288	G220	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Hap*GRH6* A	97.5	100.0	100.0
125	CC289	G221	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	100.0	100.0	100.0
126	CC291	G262	A	N	N	N	N	N	N	N	N	N	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	N	N	T	G	T	Hap*GRH6* E	44.5	62.8	73.9
127	CC292	G222	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	93.1	95.3	95.3
128	CC300	G223	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	4.4	13.3	44.4
129	CC304	G224	N	N	N	N	N	Ref	C	C	N	T	N	N	C	T	N	A	N	N	Ref	C	1bpIn	N	N	N	N	C	N	C	N	N	C	N	N	N	N	N	N	Unknown	94.9	97.1	100.0
130	CC306	G150	N	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	N	N	N	T	14bpIn	C	C	C	C	G	C	G	A	N	N	N	T	Unknown	88.9	95.6	100.0
131	CC307	G225	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	100.0	100.0	100.0
132	CC308	G226	A	G	G	C	G	Ref	C	C	N	T	N	T	C	T	C	A	Ref	Ref	Ref	N	1bpIn	G	G	N	14bpIn	C	C	C	C	G	C	G	A	A	T	G	T	Unknown	100.0	100.0	100.0
133	CC310	G263	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Hap*GRH6* A	100.0	100.0	100.0
134	CC312	G151	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	4.4	4.4	13.3
135	CC313	G59	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	0.0	79.6	95.8
136	CC314	G152	N	G	G	C	G	Ref	C	C	G	T	G	T	N	N	N	N	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	N	C	G	C	G	A	N	N	N	T	Unknown	100.0	100.0	100.0
137	CC316	G129	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	100.0	100.0	100.0
138	CC318	G130	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	100.0	100.0	100.0
139	CC319	G227	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	N	N	1bpIn	G	N	T	14bpIn	C	C	C	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	2.2	4.2	20.9
140	CC320	G153	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	N	N	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	4.0	27.2	52.3

Table 8. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH6*[d]																																			Haplotype[e]	Nymph mortality (%)[f]		
			chr04_4485182	chr04_4485745	chr04_4486245	chr04_4486257	chr04_4486925	chr04_4486942	chr04_4487022	chr04_4487111	chr04_4488061	chr04_4488070	chr04_4488274	chr04_4488438	chr04_4488622	chr04_4488717	chr04_4488718	chr04_4488724	chr04_4488726	S04_4488740	chr04_4488757	chr04_4489386	chr04_4489455	chr04_4489478	chr04_4489481	chr04_4489567	chr04_4489599	chr04_4489610	chr04_4489651	chr04_4489652	chr04_4489655	chr04_4489669	chr04_4489675	chr04_4489699	chr04_4489765	chr04_4489778	chr04_4489851	chr04_4489859	chr04_4490010		3DAI	5DAI	7DAI
141	CC324	G264	A	G	G	C	G	Ref	C	C	G	T	G	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	G	Unknown	0.0	6.4	67.2
142	CC326	G131	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	97.8	100.0	100.0
143	CC331	G228	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	HapGRH6 A	97.8	97.8	97.8
144	CC333	G155	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	2.2	8.9	58.1
145	CC336	G265	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	0.0	5.0	63.8
146	CC341	G15	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	0.0	16.1	58.6
147	CC342	G156	N	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	N	N	N	T	14bpIn	C	C	C	C	G	C	G	A	N	N	N	T	Unknown	71.5	93.5	96.0
148	CC346	G16	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	N	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	5.0	61.8	64.1
149	CC347	G62	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	N	N	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	HapGRH6 D	14.4	80.4	89.1
150	CC351	G157	A	G	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Unknown	0.0	2.0	51.4
151	CC352	G229	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Unknown	97.8	100.0	100.0
152	CC354	G158	N	G	G	C	G	Ref	C	C	G	T	G	T	N	N	N	N	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	N	C	G	C	G	A	N	N	N	T	Unknown	95.6	100.0	100.0
153	CC355	G230	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	N	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	A	T	G	T	HapGRH6 D	2.5	4.7	4.7
154	CC360	G159	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	0.0	32.0	85.1
155	CC361	G231	A	A	G	C	T	Ref	C	T	N	T	T	C	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Unknown	100.0	100.0	100.0
156	CC364	G17	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	28.4	77.1	79.6
157	CC365	G63	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	9.4	49.2	54.0
158	CC367	G18	A	G	G	C	G	Ref	C	C	G	T	N	T	C	N	N	N	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	N	C	G	C	G	A	G	T	G	T	Unknown	2.2	4.7	53.6
159	CC368	G266	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	HapGRH6 E	100.0	100.0	100.0
160	CC371	G64	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	N	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	HapGRH6 D	0.0	54.7	82.3
161	CC373	G20	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	HapGRH6 E	0.0	9.7	49.1
162	CC374	G160	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	4.0	44.9	80.0
163	CC375	G188	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	0.0	2.2	15.6
164	CC376	G21	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	11.7	55.8	81.4
165	CC377	G65	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	0.0	0.0	8.6
166	CC379	G22	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	71.4	92.8	97.5
167	CC382	G23	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	7.5	45.6	85.6
168	CC386	G24	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	34.4	75.8	83.1
169	CC395	G232	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	HapGRH6 A	97.8	97.8	100.0
170	CC396	G233	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	HapGRH6 A	97.5	100.0	100.0
171	CC400	G66	G	G	G	C	G	Ref	C	N	N	N	G	T	T	T	C	A	Ref	Ref	Ref	T	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	G	T	G	T	Unknown	2.2	2.2	6.7
172	CC401	G25	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	HapGRH6 C	4.7	48.7	77.2
173	CC402	G132	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	HapGRH6 F	97.8	100.0	100.0
174	CC403	G161	N	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	N	N	N	T	Unknown	2.2	35.7	72.8
175	CC406	G234	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	HapGRH6 A	97.5	100.0	100.0

Table 8. Continued

			SNP marker at *GRH6*[d]																																						Nymph mortality (%)[f]		
Sr. no.[a]	CC no.[b]	G no.[c]	chr04_4485182	chr04_4485745	chr04_4486245	chr04_4486257	chr04_4486925	chr04_4486942	chr04_4487022	chr04_4487111	chr04_4488061	chr04_4488070	chr04_4488274	chr04_4488438	chr04_4488622	chr04_4488717	chr04_4488718	chr04_4488724	chr04_4488726	S04_4488740	chr04_4488757	chr04_4489386	chr04_4489455	chr04_4489478	chr04_4489481	chr04_4489567	chr04_4489599	chr04_4489610	chr04_4489651	chr04_4489652	chr04_4489655	chr04_4489669	chr04_4489675	chr04_4489699	chr04_4489765	chr04_4489778	chr04_4489851	chr04_4489859	chr04_4490010	Haplotype[e]	3DAI	5DAI	7DAI
176	CC411	G190	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	6.4	25.6	40.5
177	CC415	G237	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	2.0	2.0	23.6
178	CC417	G162	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	95.8	100.0	100.0
179	CC418	G191	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	100.0	100.0	100.0
180	CC421	G27	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	8.0	43.2	76.6
181	CC422	G163	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	4.2	9.2	63.5
182	CC423	G164	A	G	N	N	N	N	N	N	N	N	G	T	C	T	C	A	N	N	N	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Unknown	98.0	100.0	100.0
183	CC426	G28	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	17.8	78.4	87.1
184	CC427	G165	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	91.6	100.0	100.0
185	CC431	G268	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	0.0	21.4	58.6
186	CC434	G166	N	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	N	N	N	T	14bpIn	C	C	C	C	G	C	G	A	N	N	N	T	Unknown	95.5	97.5	97.5
187	CC435	G167	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	4.4	17.8	70.2
188	CC436	G114	A	G	N	N	G	Ref	C	C	G	T	G	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	C	G	A	A	A	A	T	Unknown	6.4	10.4	35.1
189	CC439	G68	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	95.8	98.0	100.0
190	CC442	G30	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	25.4	51.9	68.8
191	CC444	G192	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	87.5	97.5	100.0
192	CC445	G69	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	12.2	17.2	35.8
193	CC450	G70	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	97.5	100.0	100.0
194	CC458	G168	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	0.0	23.0	40.6
195	CC459	G240	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	100.0	100.0	100.0
196	CC460	G193	A	G	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Unknown	2.0	12.0	38.2
197	CC461	G169	N	G	G	C	G	Ref	C	C	G	T	G	T	N	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	100.0	100.0	100.0
198	CC463	G170	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	97.8	97.8	100.0
199	CC468	G269	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	0.0	16.4	59.4
200	CC469	G116	A	N	N	N	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	T	Unknown	0.0	10.0	47.0
201	CC475	G31	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	G	14bpIn	C	C	C	C	G	C	C	A	A	T	G	T	Unknown	0.0	6.7	6.7
202	CC477	G194	N	G	N	N	G	Ref	C	C	G	T	G	T	C	N	T	N	Ref	N	N	C	1bpIn	N	N	N	14bpIn	C	N	N	C	N	C	N	N	N	N	N	T	Unknown	100.0	100.0	100.0
203	CC483	G270	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	0.0	0.0	2.9
204	CC484	G195	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	2.5	23.6	66.7
205	CC485	G118	N	N	G	C	N	Ref	C	N	N	T	N	N	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	C	C	G	T	G	A	A	T	G	T	Hap*GRH6* G	93.3	100.0	100.0
206	CC490	G241	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	32.7	75.1	77.3
207	CC491	G32	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	Ref	Ref	Ref	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* B	63.3	80.9	91.3
208	CC492	G119	G	G	T	A	G	Ref	C	N	N	N	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	80.0	80.0	95.6
209	CC493	G72	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	N	C	N	C	G	C	G	A	A	T	G	T	Hap*GRH6* D	22.2	34.2	34.2
210	CC494	G73	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	0.0	0.0	2.5

Table 8. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	SNP marker at *GRH6*[d]																																			Haplotype[e]	Nymph mortality (%)[f]		
			chr04_4485182	chr04_4485745	chr04_4486245	chr04_4486257	chr04_4486925	chr04_4486942	chr04_4487022	chr04_4487111	chr04_4488061	chr04_4488070	chr04_4488274	chr04_4488438	chr04_4488622	chr04_4488717	chr04_4488718	chr04_4488724	chr04_4488726	S04_4488740	chr04_4488757	chr04_4489386	chr04_4489455	chr04_4489478	chr04_4489481	chr04_4489567	chr04_4489599	chr04_4489610	chr04_4489651	chr04_4489652	chr04_4489655	chr04_4489669	chr04_4489675	chr04_4489699	chr04_4489765	chr04_4489778	chr04_4489851	chr04_4489859	chr04_4490010		3DAI	5DAI	7DAI
211	CC499	G33	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	23.9	52.2	82.9
212	CC500	G242	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	28.3	74.5	86.2
213	CC505	G243	A	A	G	C	T	Ref	C	T	C	T	T	C	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Unknown	98.0	98.0	98.0
214	CC508	G244	A	G	G	C	N	N	N	N	N	N	G	T	C	T	C	A	N	N	N	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	97.8	100.0	100.0
215	CC509	G171	N	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Unknown	15.7	63.9	81.1
216	CC512	G34	A	N	G	C	G	Ref	C	N	N	N	G	T	C	N	N	N	N	N	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Unknown	36.0	84.0	84.0
217	CC513	G172	G	G	T	A	G	Ref	C	N	N	N	G	T	C	T	T	N	N	N	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	0.0	2.0	39.6
218	CC515	G35	A	G	G	C	G	Ref	N	N	N	N	G	T	C	N	N	N	N	N	N	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Unknown	0.0	31.7	51.4
219	CC516	G272	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	11.1	16.8	38.5
220	CC517	G245	G	G	T	A	G	Ref	C	C	G	T	G	T	C	T	T	A	Ref	3bpDel	Ref	C	1bpIn	T	A	G	14bpIn	C	T	C	C	A	C	C	C	A	A	A	T	Hap*GRH6* F	97.8	100.0	100.0
221	CC523	G198	A	N	G	C	G	3bpDel	T	C	G	A	T	T	T	T	C	A	Ref	Ref	Ref	C	Ref	G	G	T	14bpIn	T	C	T	C	G	C	G	A	A	T	G	T	Hap*GRH6* E	58.3	82.8	82.8
222	CC525	G246	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	8bpDel	Ref	Ref	T	1bpIn	G	G	T	Ref	C	C	C	T	G	C	G	A	G	T	G	G	Hap*GRH6* A	100.0	100.0	100.0
223	CC528	G247	A	G	G	C	G	Ref	C	C	G	T	G	T	C	T	C	A	N	Ref	Ref	T	1bpIn	G	G	T	N	C	C	C	N	G	N	G	A	G	T	G	N	Hap*GRH6* A	97.8	100.0	100.0
224	CC529	G199	A	G	G	C	G	Ref	C	C	G	T	G	T	C	G	T	T	Ref	Ref	15bpDel	C	1bpIn	G	G	T	14bpIn	C	C	T	C	G	C	G	A	G	T	G	T	Hap*GRH6* C	34.0	78.7	89.3

[a] Serial number

[b] Core collections of rice conserved in Seedbank of Department of Agricultural Research, Myanmar

[c] Number of Myanmar Indica diversity panel designated as "G" where the accessions were whole genome sequenced

[d] SNP markers located in the genomic region of *GRH6* and used in haplotype analysis

[e] Haplotype variation at the genomic region of *GRH6*

[f] Nymph mortality (%) of GRH evaluated at 3 days after infestation (DAI), 5DAI and 3DAI

Distribution of combined resistant haplotypes in Myanmar landraces

Combinations of resistant haplotypes in MIDP were also investigated using 168 accessions after excluding the accessions with unknown haplotypes (Fig. 23). No accessions carrying of the three resistant haplotypes were identified. Forty-one accessions that carried at least one or more resistant haplotypes at major resistance genes including *GRH1* and *GRH6* (Hap*GRH6*A/Hap*GRH1*A/HapMTA11B, Hap*GRH6*B-G/Hap*GRH1*A/HapMTA11A, Hap*GRH6*A/Hap*GRH1*B/HapMTA11B, Hap*GRH6*B-G/Hap*GRH1*A/HapMTA11B) exhibited high levels of resistance (Fig. 23, Table 9). Twenty-eight accessions belonging to the resistant haplotype at MTA11 (Hap*GRH6*B-G/Hap*GRH1*B/HapMTA11A) exhibited low to high nymph mortality at 3DAI and showed moderate to high nymph mortality at 7DAI (Fig. 23, Table 9) assuming that this is likely to be explained by the delay resistance effect of HapMTA11A. Nevertheless, the remaining accessions carrying no resistance haplotype at the loci detected in this study (Hap*GRH6*B-G/Hap*GRH1*B/ HapMTA11B) showed a wide phenotypic variation in NM ranging from 0 to 100 % (Fig. 23, Table 9). This suggests that the resistance may be due to other unidentified QTLs in this study. Cultivars with unknown alleles of GRH resistance are potential donors for exploring novel alleles that confer GRH resistance.

The geographical distribution of the combination of resistant haplotypes was further investigated. Accessions were collected from seven geographical regions: the northern mountainous region (NMR), eastern mountainous region (EMR), western mountainous region 21 (WMR), central dry zone (CDZ), western coastal region (WCR), delta region (DR), and eastern coastal region (ECR) (Fig. 24A). Accessions harboring major resistant haplotypes at major resistance genes including *GRH6* and *GRH1* (Hap*GRH6*A, and Hap*GRH1*A) were mainly observed in the CDZ and southern region

of Myanmar including WCR, DR, and ECR, and their frequencies were higher than those in mountainous regions, including NMR, EMR, WMR (Fig. 24B, Table 10). In contrast, accessions carrying resistant haplotype at MTA11 HapMTA11A) was mainly distributed in EMR and partly in NMR, WMR and WCR (Fig. 24B, Table 10) speculating that MTA11 is likely the main resistance QTL or gene for accessions from mountainous regions.

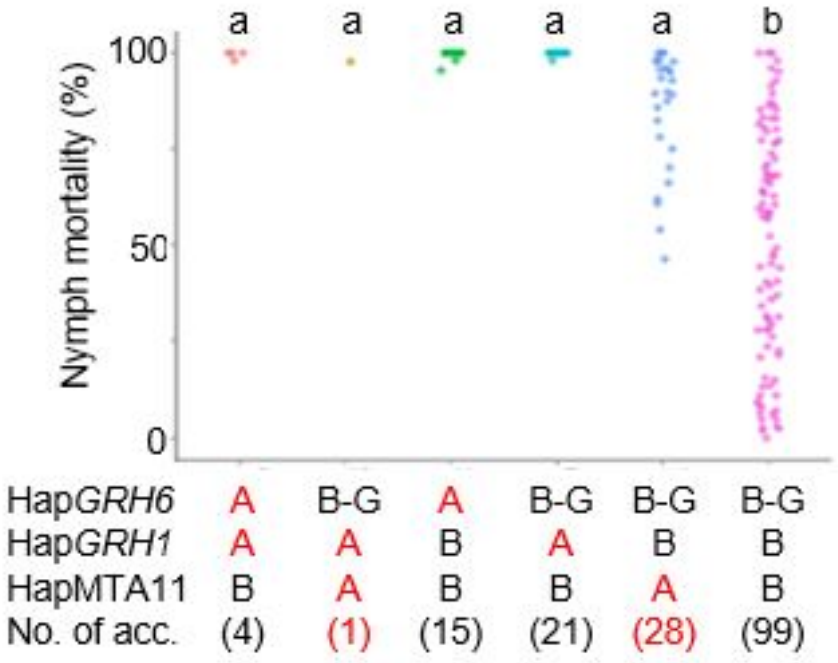

Fig. 23. Resistance to GRH in MIDP controlled by the different combinations (n=168) of haplotypes at three QTLs including *GRH6*, *GRH1* and MTA11 at 7DAI. The number in the parenthesis represents the number of accessions in each haplotype combination. Red letter "A" represents the resistant haplotype and the black letter "B-G" represents the susceptible haplotype at *GRH1*, *GRH6* and MTA11.

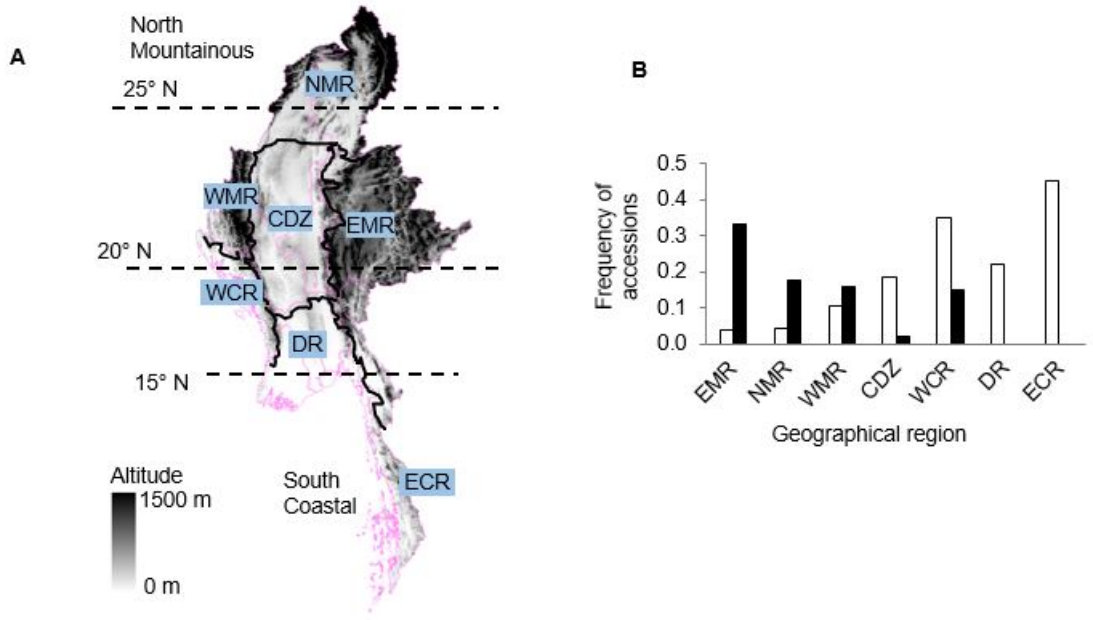

Fig. 24. Geographical distribution of the germplasm in MIDP (n=168) conferring resistance to GRH defined by resistant haplotypes. (A) The seven geographical regions of Myanmar. NMR, EMR, WMR, CDZ, WCR, DR, and ECR represent the northern mountainous region, eastern mountainous region, western mountainous region, central dry zone, western coastal region, delta region, and eastern coastal region, respectively. The color changing from grey to black represent low to high altitude. (B) Frequency of accessions carrying different haplotype combinations distributed in seven geographical regions of Myanmar. White bars represent the frequency of accessions carrying Hap*GRH6*A or Hap*GRH1*A or both of them. Black bars represent the frequency of accessions carrying HapMTA11A. Detailed numbers of accessions by the geographical regions were shown in the Table 10.

Table 9. Haplotype variations of the accessions in MIDP at the three loci detected in GWAS

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
1	CC1	G1	Paw San Hmwe	DR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	2.2	20.0	31.1
2	CC3	G174	Ant Baw	WCR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	0.0	31.4
3	CC6	G248	Kauk Hnyin War	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	4.0	56.0	80.5
4	CC7	G74	Nga Bya Gyi	NMR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	97.5	100.0	100.0
5	CC8	G249	Nar Tha Pu	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	34.7	73.8	76.3
6	CC9	G75	Naw Phee	EMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	8.3	31.0	47.6
7	CC10	G2	Na Bya Sann	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	55.3	91.1	100.0
8	CC11	G3	Mine Taung	CDZ	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	0.0	31.3	56.8
9	CC12	G120	Htaw Patt Sein	CDZ	Unknown	Unknown	HapMTA11B	100.0	100.0	100.0
10	CC13	G200	Sit Pwa	DR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
11	CC14	G4	Kauk Yin Phyu	NMR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	35.6	68.5
12	CC15	G76	Maung Galay	CDZ	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	81.1	97.8	97.8
13	CC17	G121	Sae Ti Eine	WCR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
14	CC19	G250	Chi Nyo	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	0.0	0.0	11.1
15	CC20	G77	Mon Chee Sa Ba	CDZ	Hap*GRH6* C	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
16	CC22	G251	Khao Phar	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	4.4	6.9	6.9
17	CC23	G36	Ni Ka Re San Pyu	WCR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	28.8	82.7	89.8
18	CC24	G133	Mai Saw	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	2.2	20.2	44.0
19	CC26	G5	Balu Swe	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	66.1	91.8	96.0
20	CC28	G6	Phichenam	NMR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	35.3	67.3
21	CC30	G175	Thia Rall	WMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	3.6	28.1
22	CC33	G252	Naung Htu Phyu	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	8.4	35.1	60.5
23	CC34	G78	Nga Sein	CDZ	Hap*GRH6* G	Hap*GRH1* A	HapMTA11A	97.5	97.5	97.5
24	CC35	G253	Sein Wine Yin	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	0.0	0.0
25	CC36	G134	Duh Thim	WMR	Unknown	Hap*GRH1* B	HapMTA11A	97.8	97.8	97.8
26	CC38	G79	Nung Ding Thau	WMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	11.1	45.1	66.0
27	CC46	G38	Kauk Hnyin Me	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	2.0	6.5	29.4
28	CC47	G80	Kauk Hnyin	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11A	0.0	18.4	66.2
29	CC48	G39	Khao Ann	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	22.9	42.3	62.8
30	CC49	G81	Yway	EMR	Hap*GRH6* G	Hap*GRH1* B	HapMTA11B	0.0	2.5	2.5

Table 9. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
31	CC53	G82	Biak Mang	WMR	Hap*GRH6* B	Hap*GRH1* B	HapMTA11B	0.0	4.0	28.2
32	CC56	G7	Cal Thau	WMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	9.3	31.5	57.5
33	CC57	G40	Khao San Gyi	EMR	Hap*GRH6* B	Hap*GRH1* B	HapMTA11A	32.9	89.0	100.0
34	CC58	G41	Khao Note Kin	EMR	Hap*GRH6* B	Hap*GRH1* B	HapMTA11A	92.8	100.0	100.0
35	CC59	G42	Sa Ka Lay	EMR	Hap*GRH6* B	Hap*GRH1* B	HapMTA11B	0.0	2.0	2.0
36	CC61	G83	Ywe	EMR	Hap*GRH6* G	Hap*GRH1* B	HapMTA11B	2.5	2.5	4.7
37	CC65	G43	Khao Pee Saing	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	41.9	87.1	89.3
38	CC66	G44	Khao Khan Pu	EMR	Unknown	Hap*GRH1* B	Unknown	0.0	0.0	7.3
39	CC67	G45	Ma Kaw La	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11A	0.0	54.4	60.9
40	CC68	G85	San Senn	WMR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11A	74.0	94.0	98.0
41	CC71	G86	Khao Lan Kan	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	14.5	68.2
42	CC73	G87	Khao Swos	EMR	Unknown	Hap*GRH1* B	HapMTA11A	51.6	77.7	85.8
43	CC75	G46	Kauk Hnyin Saba	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	2.5	7.5	26.4
44	CC76	G47	Kauk Hnyin Saba	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	37.5	66.7	87.5
45	CC79	G122	Hay Wun Dar	WCR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
46	CC82	G8	Kauk Hnyin Nga Cheik	NMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	9.9	31.8	68.1
47	CC83	G48	Khao Khin Phut	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11A	10.7	66.8	78.0
48	CC87	G89	Saya Lay	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	97.5	100.0	100.0
49	CC89	G49	Bu Pa Yaung	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	2.5	69.9	95.0
50	CC92	G50	Kauk Hnyin Me	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	9.8	19.2	31.7
51	CC94	G90	Shwe War Yin	DR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	37.5	53.8	85.2
52	CC96	G91	Arshawarli	WCR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11A	85.6	92.7	97.8
53	CC98	G92	Kauk Chein	EMR	Hap*GRH6* C	Hap*GRH1* A	HapMTA11B	95.6	97.8	100.0
54	CC99	G52	Khao Sung Hung Yao	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	0.0	45.0	62.0
55	CC100	G93	Boke Thwin Phyu	WCR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11A	21.0	50.2	74.9
56	CC105	G53	Khao Sai	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	0.0	5.0	22.5
57	CC106	G123	Japan Ni	WCR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	97.8	97.8	97.8
58	CC116	G54	Kauk Hnyin Net	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	0.0	2.0	6.2
59	CC123	G97	Hwon Yin	WMR	Hap*GRH6* G	Unknown	HapMTA11B	98.0	100.0	100.0
60	CC125	G98	China 29	NMR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11A	54.6	79.8	92.8

Table 9. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
61	CC126	G10	Ywat Oat Kauk Hnyin	ECR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	25.4	50.5
62	CC130	G99	Kauk Hla Te Thi	EMR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	0.0	0.0	11.0
63	CC131	G100	Bu Ei Law	EMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11A	56.4	90.2	95.3
64	CC132	G101	Mar Thu	WMR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11A	34.9	61.2	70.2
65	CC139	G102	Khauk Mwe Hnaung	EMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11A	69.2	88.9	93.3
66	CC142	G103	Ku Taung Myo Tun	ECR	Unknown	Hap*GRH1* B	HapMTA11A	81.9	90.6	93.1
67	CC143	G124	Shwe Dinga	DR	Unknown	Unknown	HapMTA11B	90.0	95.0	100.0
68	CC145	G105	Ant paw	CDZ	Hap*GRH6* B	Hap*GRH1* B	HapMTA11B	23.0	52.5	68.1
69	CC146	G106	Sa Bong Thau	WMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	5.3	39.6	67.3
70	CC147	G55	Oat Kyai (B)	EMR	Hap*GRH6* C	Hap*GRH1* B	Unknown	40.3	64.7	64.7
71	CC151	G176	Lai Fang	WMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	2.0	27.3	68.0
72	CC152	G107	Pinto	ECR	Hap*GRH6* B	Hap*GRH1* A	HapMTA11B	97.5	100.0	100.0
73	CC153	G136	Du Htung	WMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11A	93.8	100.0	100.0
74	CC158	G108	Kari Let Yone	WCR	Unknown	Hap*GRH1* B	HapMTA11A	89.3	95.6	97.8
75	CC163	G12	Kauk Hnyin Phyu	CDZ	Hap*GRH6* B	Hap*GRH1* B	HapMTA11A	17.8	91.1	97.5
76	CC164	G56	Bul Mashal	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	6.7	45.9	71.1
77	CC166	G57	Khao Pyi Seng	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	2.2	4.2	10.9
78	CC167	G109	Khao Ho Mon(China 249)	EMR	Hap*GRH6* G	Hap*GRH1* B	HapMTA11B	2.5	4.3	36.9
79	CC170	G110	Shwe Att	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	16.0	20.5	60.9
80	CC171	G111	Byit Thar	WMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	0.0	9.4
81	CC172	G254	Taung O Nu (Ashay)	WMR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	2.5	14.2	58.5
82	CC176	G58	Muyinn Saba	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	28.6	45.3
83	CC184	G138	Oat Pho-3	DR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	94.9	100.0	100.0
84	CC189	G201	Thon Hnan Pwa	DR	Hap*GRH6* A	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
85	CC192	G139	Maung Pha Lo	DR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	17.4	25.1	27.9
86	CC193	G13	Khao Kan	EMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11A	4.0	26.2	46.4
87	CC195	G202	Kywe Kye Kare	ECR	Hap*GRH6* B	Hap*GRH1* B	HapMTA11B	0.0	0.0	8.0
88	CC197	G203	Pho Kaw Gyi	DR	Unknown	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
89	CC198	G125	Innma Ye Baw	DR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	98.0	100.0	100.0
90	CC199	G255	Yan Gon Nga Cheik	DR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	13.0	44.2

Table 9. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
91	CC202	G140	Ban Gauk Nga Sein	DR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	78.1	84.3	86.3
92	CC206	G14	Pa Din Thu Ma	DR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	21.4	30.6
93	CC208	G141	Ma Kauk Hmwe	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	0.0	30.6	81.0
94	CC211	G205	Kyathaung Hmwe	DR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	14.4	49.7	57.0
95	CC212	G178	Shwe Kyi Tauk	EMR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	84.3	95.3	97.8
96	CC215	G179	Balu Gyun Nga Sein	DR	Unknown	Hap*GRH1* B	HapMTA11B	3.3	13.1	13.1
97	CC217	G180	Pegu Gyi	DR	Unknown	Hap*GRH1* B	HapMTA11B	87.0	94.9	100.0
98	CC221	G181	Shwe War Tun	DR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	2.5	5.0	21.4
99	CC223	G182	Kauk Hnyin	DR	Unknown	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
100	CC224	G183	Ya Gyaw	DR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	2.5	5.0
101	CC230	G184	Thet Lat Nga Kyauk	DR	Hap*GRH6* A	Unknown	HapMTA11B	61.0	70.5	80.0
102	CC231	G185	Wet Si Pyu	CDZ	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	2.0	9.0	13.5
103	CC232	G186	Hmawbi Nga Kywe	DR	Unknown	Hap*GRH1* B	HapMTA11B	5.0	9.0	55.3
104	CC235	G187	Myaung Mya	DR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	2.2	2.2	6.4
105	CC236	G188	Ya Gyaw Taung Pyan	DR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	50.1	65.7	72.4
106	CC239	G189	Kyar Si Kauk Hnyin	ECR	Unknown	Hap*GRH1* B	HapMTA11B	43.6	62.2	71.7
107	CC240	G190	Shwe Thwe Tun	DR	Hap*GRH6* G	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
108	CC242	G191	Kaoh Saing	EMR	Hap*GRH6* B	Hap*GRH1* B	HapMTA11B	66.1	87.8	95.0
109	CC244	G192	Moe Thee Myo	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	48.3	63.5	74.1
110	CC248	G193	In Gyin (A)	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	2.5	35.8
111	CC257	G194	Kauk Hnyin Ah Phyu	ECR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	97.1	100.0	100.0
112	CC259	G195	Khao Pin Lain	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	45.4	64.0	80.1
113	CC262	G196	Tha Zin Tan	DR	Unknown	Hap*GRH1* B	HapMTA11B	2.2	2.2	12.6
114	CC265	G197	Taung Hteik Pan	CDZ	Hap*GRH6* B	Hap*GRH1* B	HapMTA11B	8.0	14.2	47.1
115	CC268	G198	Kaing Saba	EMR	Unknown	Hap*GRH1* B	HapMTA11B	85.3	98.0	100.0
116	CC270	G199	Ma Kye Kye	ECR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
117	CC271	G200	Thone Hnan Saba	ECR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	54.7	59.2	68.2
118	CC273	G201	Pauk War (Kauk Hnyin)	WCR	Unknown	Hap*GRH1* B	HapMTA11B	4.2	4.2	31.9
119	CC274	G202	Myo Raw (Wan Me)	WCR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	59.0	78.0	80.5
120	CC275	G203	Kywat Shay	WCR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	4.4	14.9

Table 9. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
121	CC280	G186	Kauk Hnyin	CDZ	Hap*GRH6* E	Hap*GRH1* A	HapMTA11B	93.3	97.8	100.0
122	CC286	G187	Shan Ma Yin	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	64.4	85.2	92.7
123	CC287	G219	Palae Thwe	ECR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
124	CC288	G220	Shwe Yaung Htun	ECR	Hap*GRH6* A	Hap*GRH1* A	HapMTA11B	97.5	100.0	100.0
125	CC289	G221	Mang Nuai Thau	WMR	Hap*GRH6* G	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
126	CC291	G262	Hlae Oo Khwe	DR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	44.5	62.8	73.9
127	CC292	G222	Mee Tauk	DR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	93.1	95.3	95.3
128	CC300	G223	Tan Kang	WMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	4.4	13.3	44.4
129	CC304	G224	Chan Nyeing	CDZ	Unknown	Hap*GRH1* A	HapMTA11B	94.9	97.1	100.0
130	CC306	G150	Ayeyarmin	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	88.9	95.6	100.0
131	CC307	G225	Du Ya Boe	CDZ	Hap*GRH6* A	Unknown	HapMTA11B	100.0	100.0	100.0
132	CC308	G226	Nga Pyar Gyi	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
133	CC310	G263	Kauk Hnyin Hmwe	CDZ	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
134	CC312	G151	Kauk	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	4.4	4.4	13.3
135	CC313	G59	Saba Minn	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	79.6	95.8
136	CC314	G152	Shwe Ta Sote Yoe Ni	CDZ	Unknown	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
137	CC316	G129	Kyauk Yway (Thekone)	DR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
138	CC318	G130	Sin Ku	DR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
139	CC319	G227	Kauk Hynin	CDZ	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	2.2	4.2	20.9
140	CC320	G153	Kauk Hnyin	ECR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	4.0	27.2	52.3
141	CC324	G264	Byat	NMR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	6.4	67.2
142	CC326	G131	Innma Kywe Kyae	DR	Hap*GRH6* F	Unknown	HapMTA11B	97.8	100.0	100.0
143	CC331	G228	Duyabo	CDZ	Hap*GRH6* A	Hap*GRH1* A	HapMTA11B	97.8	97.8	97.8
144	CC333	G155	Rakhaing Pho	DR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	2.2	8.9	58.1
145	CC336	G265	Chin Kauk Hnyin	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	5.0	63.8
146	CC341	G15	Thawt Gyi	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	16.1	58.6
147	CC342	G156	Phi Zin	ECR	Unknown	Hap*GRH1* B	HapMTA11B	71.5	93.5	96.0
148	CC346	G16	Kyaw Khaw	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	5.0	61.8	64.1
149	CC347	G62	Khao Man Nain	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11A	14.4	80.4	89.1
150	CC351	G157	Shut Mee	EMR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	2.0	51.4

Table 9. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
151	CC352	G229	Padan Nga Sein	DR	Unknown	Hap*GRH1* A	HapMTA11B	97.8	100.0	100.0
152	CC354	G158	Kywet Thwa	DR	Unknown	Unknown	HapMTA11B	95.6	100.0	100.0
153	CC355	G230	Yoe Sein	DR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	2.5	4.7	4.7
154	CC360	G159	Nga Shink Thway	DR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	32.0	85.1
155	CC361	G231	Ka Lei	DR	Unknown	Unknown	HapMTA11B	100.0	100.0	100.0
156	CC364	G17	Pa Laung Kyar	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	28.4	77.1	79.6
157	CC365	G63	Khao Khaw La	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	9.4	49.2	54.0
158	CC367	G18	Sein Ti Saba	NMR	Unknown	Hap*GRH1* B	Unknown	2.2	4.7	53.6
159	CC368	G266	Kauk Hnyin Nga Cheik	CDZ	Hap*GRH6* E	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
160	CC371	G64	Yat Sauk	EMR	Hap*GRH6* D	Hap*GRH1* B	HapMTA11A	0.0	54.7	82.3
161	CC373	G20	Ly Mee	ECR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	0.0	9.7	49.1
162	CC374	G160	Ywet Thay Gyi	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	4.0	44.9	80.0
163	CC375	G188	Bamaw Pyan	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	2.2	15.6
164	CC376	G21	Mya Wut Yee	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	11.7	55.8	81.4
165	CC377	G65	Ani Pyu Lar	WCR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	0.0	8.6
166	CC379	G22	Mauk Kon Gyi Zein	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	71.4	92.8	97.5
167	CC382	G23	Kywe Phyu Saw	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	7.5	45.6	85.6
168	CC386	G24	Ngwe Bya Gyi Kauk Hnyin Net	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	34.4	75.8	83.1
169	CC395	G232	Det Kwa	WCR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	97.8	97.8	100.0
170	CC396	G233	Khao Ma Phoot	EMR	Hap*GRH6* A	Hap*GRH1* A	HapMTA11B	97.5	100.0	100.0
171	CC400	G66	Byat Kauk Hnyin	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	2.2	2.2	6.7
172	CC401	G25	Khauk Nan Swan	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	4.7	48.7	77.2
173	CC402	G132	San Hmwe	CDZ	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	97.8	100.0	100.0
174	CC403	G161	Khauk Hauk	EMR	Unknown	Hap*GRH1* B	HapMTA11B	2.2	35.7	72.8
175	CC406	G234	Rakhaing Thu Ma	WCR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	97.5	100.0	100.0
176	CC411	G190	Gesma	WMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	6.4	25.6	40.5
177	CC415	G237	Kayin Gyi	ECR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	2.0	2.0	23.6
178	CC417	G162	Ck Khwa	ECR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	95.8	100.0	100.0
179	CC418	G191	Ye Pyar Nga Cheik	CDZ	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
180	CC421	G27	Khao Note Tit Own	DR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	8.0	43.2	76.6

Table 9. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
181	CC422	G163	Kyun Shwe War	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	4.2	9.2	63.5
182	CC423	G164	Lone Pu	EMR	Unknown	Hap*GRH1* B	HapMTA11B	98.0	100.0	100.0
183	CC426	G28	Ton Jan	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	17.8	78.4	87.1
184	CC427	G165	Hay Wun Dar	WCR	Hap*GRH6* G	Hap*GRH1* A	HapMTA11B	91.6	100.0	100.0
185	CC431	G268	Sabanet	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	21.4	58.6
186	CC434	G166	Phyine Zin Phyu	WCR	Unknown	Hap*GRH1* B	HapMTA11B	95.5	97.5	97.5
187	CC435	G167	Phyine Zin Ni	WCR	Hap*GRH6* G	Hap*GRH1* B	HapMTA11B	4.4	17.8	70.2
188	CC436	G114	Let To Ri	WCR	Unknown	Hap*GRH1* B	HapMTA11B	6.4	10.4	35.1
189	CC439	G68	Cheik Kauk Hnyin	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11A	95.8	98.0	100.0
190	CC442	G30	Kauk Hnyin (white)	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	25.4	51.9	68.8
191	CC444	G192	Kauk Hnyin Me	ECR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	87.5	97.5	100.0
192	CC445	G69	Kauk Hnyin Nga Cheik	EMR	Hap*GRH6* C	Hap*GRH1* B	Unknown	12.2	17.2	35.8
193	CC450	G70	Khao Li Pon	EMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	97.5	100.0	100.0
194	CC458	G168	Kyun Shwe War	ECR	Hap*GRH6* G	Hap*GRH1* B	HapMTA11B	0.0	23.0	40.6
195	CC459	G240	Soke Phwa	DR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
196	CC460	G193	Kauk Hnyin Yin (Myaungmya)	DR	Unknown	Hap*GRH1* B	HapMTA11B	2.0	12.0	38.2
197	CC461	G169	King Bwee (A Thay)	WMR	Hap*GRH6* G	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
198	CC463	G170	Ma Ma Lay Saba	ECR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	97.8	97.8	100.0
199	CC468	G269	Kauk Hnyin	NMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	16.4	59.4
200	CC469	G116	Kyaung Ngo	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	0.0	10.0	47.0
201	CC475	G31	Naw Che	CDZ	Unknown	Hap*GRH1* B	HapMTA11B	0.0	6.7	6.7
202	CC477	G194	Matho	WMR	Unknown	Hap*GRH1* A	HapMTA11B	100.0	100.0	100.0
203	CC483	G270	Nagayar	ECR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	0.0	2.9
204	CC484	G195	Hauk Pee Sai	NMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	2.5	23.6	66.7
205	CC485	G118	Khao Mit Nwe(Khao Per Won)	EMR	Hap*GRH6* G	Hap*GRH1* B	HapMTA11B	93.3	100.0	100.0
206	CC490	G241	Kyar Ama Gyi	EMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	32.7	75.1	77.3
207	CC491	G32	Khaw Pae Ni	EMR	Hap*GRH6* B	Hap*GRH1* B	HapMTA11B	63.3	80.9	91.3
208	CC492	G119	Taung Yoe Nga Cheik	EMR	Hap*GRH6* F	Hap*GRH1* B	HapMTA11A	80.0	80.0	95.6
209	CC493	G72	Kauk Hnyin	CDZ	Hap*GRH6* D	Hap*GRH1* B	HapMTA11B	22.2	34.2	34.2
210	CC494	G73	Kauk Hnyin Hnit	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	0.0	0.0	2.5

Table 9. Continued

Sr. no.[a]	CC no.[b]	G no.[c]	Cultivar name[d]	Region[e]	Haplotype at[f]			Nymph mortality (%)[g]		
					GRH6	*GRH1*	MTA11	3DAI	5DAI	7DAI
211	CC499	G33	Chin Mee Shay	WMR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	23.9	52.2	82.9
212	CC500	G242	Thet Yin Ka La Gyi	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	28.3	74.5	86.2
213	CC505	G243	Kywe Lan	NMR	Unknown	Hap*GRH1* A	HapMTA11B	98.0	98.0	98.0
214	CC508	G244	Taung Htate Pan	CDZ	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	97.8	100.0	100.0
215	CC509	G171	Eka Ye Saba	DR	Unknown	Hap*GRH1* B	HapMTA11B	15.7	63.9	81.1
216	CC512	G34	War Phyu	NMR	Unknown	Hap*GRH1* B	HapMTA11B	36.0	84.0	84.0
217	CC513	G172	Kauk Yin	CDZ	Hap*GRH6* F	Hap*GRH1* B	HapMTA11B	0.0	2.0	39.6
218	CC515	G35	Gwa	DR	Unknown	Hap*GRH1* B	HapMTA11B	0.0	31.7	51.4
219	CC516	G272	Kauk Thwe Mee Shay	CDZ	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	11.1	16.8	38.5
220	CC517	G245	Law Thaw Gyi	ECR	Hap*GRH6* F	Hap*GRH1* A	HapMTA11B	97.8	100.0	100.0
221	CC523	G198	Shwe War Kauk Hnyin	DR	Hap*GRH6* E	Hap*GRH1* B	HapMTA11B	58.3	82.8	82.8
222	CC525	G246	Mya Sein	NA	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	100.0	100.0	100.0
223	CC528	G247	Kywet Chay Sa Ba	WCR	Hap*GRH6* A	Hap*GRH1* B	HapMTA11B	97.8	100.0	100.0
224	CC529	G199	Nga Kyein Thee	WCR	Hap*GRH6* C	Hap*GRH1* B	HapMTA11B	34.0	78.7	89.3

[a] Serial number

[b] Core collections of rice conserved in Seedbank of Department of Agricultural Research, Myanmar

[c] Number of Myanmar Indica diversity panel designated as "G" where the accessions were whole genome sequenced

[d] Cultivar name given by local farmer

[e] NMR, EMR, ECR, CDZ, DR, WMR, and WCR represent the northern mountain region, east mountain region, east costal region, central dry zone, delta region, west mountain region, and west costal region, respectively.

[f] Haplotype variation at *GRH6, GRH1* and MTA11

[g] Nymph mortality (%) of GRH evaluated at 3 days after infestation (DAI), 5DAI and 3DAI

Table 10. Frequency of accessions carrying different haplotype combinations distributed in seven geographical regions of Myanmar

Haplotype combination[a]	Geographical region [b]						
	EMR	NMR	WMR	CDZ	WCR	DR	ECR
Hap*GRH6* A/Hap*GRH1* A/HapMTA11B	1	0	0	1	0	1	1
Hap*GRH6* G/Hap*GRH1* A/HapMTA11A	0	0	0	1	0	0	0
Hap*GRH6* A/Hap*GRH1B* /HapMTA11B	1	1	0	4	3	3	3
Hap*GRH6* F/ Hap*GRH1* A/HapMTA11B	0	0	0	1	4	4	4
Hap*GRH6G* /Hap*GRH1* A/HapMTA11B	0	0	3	1	0	2	0
Hap*GRH6E* /Hap*GRH1* A/HapMTA11B	1	0	0	1	0	0	0
Hap*GRH6B* /Hap*GRH1B* /HapMTA11A	5	0	1	0	0	0	0
Hap*GRH6C* /Hap*GRH1B* /HapMTA11A	2	0	0	1	0	0	0
Hap*GRH6D* /Hap*GRH1B* /HapMTA11A	1	1	2	0	0	0	0
Hap*GRH6E* /Hap*GRH1B* /HapMTA11A	5	3	0	0	1	0	0
Hap*GRH6F* /Hap*GRH1B* /HapMTA11A	4	0	0	0	2	0	0
Others	19	12	10	22	6	16	8
Total	39	17	16	32	16	26	16

[a] Combination of haplotypes at *GRH6*, *GRH1* and MTA11.

[b] NMR, EMR, ECR, CDZ, DR, WMR, and WCR represent the northern mountain region, east mountain region, east costal region, central dry zone, delta region, west mountain region, and west costal region, respectively.

Discussion

Estimation of resistant haplotypes

GWAS identified three genomic regions including *GRH1*, *GRH6* and MTA11 associated with GRH resistance in MIDP. Using the highest associated SNPs within the genomic region of *GRH1* and MTA11, the accessions carrying resistant and susceptible haplotype were estimated (Fig. 20B, Fig. 21B), and all the accessions carrying the resistant haplotype at *GRH1* (Hap*GRH1*A) and MTA11 (HapMTA11A) exhibited moderate to high resistance to GRH (Fig. 20B, Fig. 21B, Table 6, Table 7). The haplotype analysis performed for the causal gene of *GRH6* which was identified by gene cloning in Chapter II revealed that Hap*GRH6*A correspond to GRH resistance in this panel (Fig. 22C, Table 8). However, some functional SNPs and InDels within the *GRH6* gene region seemed to be excluded after filtering with maximum missing frequency of less than 0.1 and minor allele frequency more than 0.05. As *GRH6* is located in the tandemly duplicate region on chromosome 4, the functional SNPs at *GRH6* may not be detected well due to incorrect or missed SNP calling resulted from the complexity of short read mapping on the duplicate genomic region (Ko *et al*. 2022).

Haplotype combination

Analysis of haplotype combinations showed that 69 accessions possessed at least one resistant haplotype at the detected loci and were resistant to GRH (Fig. 23, Table 9). Conversely, 71 accessions lacking a resistant haplotype at any detected locus still demonstrated moderate-to-high resistance to GRH (Fig. 23, Table 9). This implies that these accessions are invaluable donors of novel QTLs that confer GRH resistance. GWAS and haplotype analysis accurately identified three known genes and three novel genes

significantly linked to the heading date, and uncovered functional and nonfunctional alleles of each detected gene in North Korean rice populations (Jadamba *et al*. 2022). These findings suggest that haplotype analysis can identify genomic regions related to traits more accurately than a single SNP marker.

Geographical distribution of resistant haplotype explained region specific QTLs

Accessions carrying one or more major resistant haplotypes at the major reported genes: *GRH6* and *GRH1* (Hap*GRH6*A, and Hap*GRH1*A) were mainly distributed in the southern area of Myanmar, including WCR, DR and ECR (Fig. 24B and Table 10). Population structure analysis of the MIDP showed that while geographical factors might influence genetic variations in Myanmar's rice landraces, they do not significantly drive the genetic differentiation within the subpopulation (Furuta *et al.* 2024). GRH is an endemic species distributed in East Asia and not in Myanmar. Therefore, the resistant haplotype distributions at the GRH resistant locus are likely to be resulted from the genetic drift from the ancestral populations or hitchhiking by selection to the other traits without intentionality. Another possibility is that constant exposure of host plants to insect pests was occurred in southern Myanmar because of year-round intensive rice growing, and a warm and humid climate favorable for the proliferation of insect pests in these areas. Such kind of condition presumably enrichment to the major resistance genes (*GRH6* and *GRH1*) in southern regions. While GRH was not found in Myanmar, one of the related species, green leafhopper: GLH (*Nephotettix virescens*) was mainly distributed in South East Asia including Myanmar (Wilson and Claridge 1985). Some GRH resistance genes also resistance to GLH, for example, *GRH2* and *GRH4* in DV85 rice cultivar (Yasui and Yshimura, 1999). Since *GRH6* was found within the region of *Glh14*, we speculated that

GRH6 locus might have undergone indirect selection to GLH resistance in Southern parts of Myanmar. Same phenomenon was likely occurred in indirect selection for BPH resistance since *GRH6* located at the same position of *BPH40* (Shi *et al.* 2021). The current resistant haplotype distribution is likely to resulted from the selection of resistance to BPH and GLH.

CHAPTER IV

Genetic mapping for QTLs conferring resistance to green rice leafhopper (*Nephotettix cincticeps* (Uhler)) of rice landraces (*Oryza sativa* L.) in Myanmar

Introduction

Genetic mapping for QTL is the process of identifying specific genomic regions associated with variation in quantitative traits by correlating genetic markers with trait measurements. It involves two main methods such as linkage mapping and association mapping**.** Linkage Mapping, known as family-based mapping, is a conventional method based on genetic recombination within bi-parental mapping populations including F_2, double-haploid (DH), backcross, recombinant inbred lines (RIL), and near isogenic lines (NIL) (Xu *et al.* 2017). The development of DNA markers in the 1980s was a significant advancement, allowing for more effective QTL mapping. Linkage mapping depends on the co-segregation of marker loci and QTLs due to their linkage. Microsatellites or SSR markers are widely used in QTL mapping due to their co-dominance, locus sensitivity, and multi-allelic nature, making them ideal for building genetic linkage maps and analyzing QTLs (Jasim Aljumaili *et al.* 2018).

With the advance of next-generation sequencing (NGS), high-throughput sequencing identifies single-nucleotide polymorphisms (SNPs), which serve as markers linking genotypes to phenotypes. Genotyping-by-sequencing (GBS) is a technique that discovers genetic variants and rapidly genotypes the samples. By using restriction enzymes, GBS reduces genome complexity, fragmenting the genome and sequencing the ends with short-read platforms. GBS can provide higher quantities of informative data. Despite its cost-effectiveness, GBS produces considerable missing data and needs

sophisticated bioinformatics analysis (Wickland *et al.* 2017). Over the past two decades, linkage mapping has been widely used to identify QTL in many plant species. However, it has several drawbacks, including low resolution, limited allele diversity, and slow progress. Additionally, the process requires genotyping a population of plants from a bi-parental cross, which is labor-intensive, time-consuming, and costly (Xu *et al*. 2017).

In contrast, association mapping, also known as natural population-based mapping, leverages historical recombination events across many generations to achieve high resolution. Genome-wide association studies (GWAS) are commonly used in this context, capturing extensive genetic variation and detecting small effect sizes in large populations. Association mapping offers several advantages over linkage mapping: (i) it reduces the time and cost associated with developing segregating populations by utilizing existing ones, (ii) it can identify multiple allelic variations and favorable alleles in a single analysis, and (iii) it provides high-resolution fine mapping of QTLs. However, GWAS can be affected by genotyping errors and population structure issues, leading to false positives or negatives (Emebiri *et al*. 2019). Significant SNP associations may not always indicate causal variants, necessitating additional studies for accurate mapping.

Combining linkage and association mapping methods addresses their individual limitations. Linkage mapping is highly effective in tracing inheritance through generations and identifying genes associated with Mendelian inheritance and rare variants, particularly in species with low genetic variation. This integrated approach provides a robust strategy for identifying QTLs, as demonstrated in studies of agronomic traits and husk morphology in maize (Liu *et al.* 2020, Cui *et al*. 2018).

In Chapter I, a large germplasm of the Myanmar Indica diversity panel (MIDP) was evaluated for GRH resistance, with 188 accessions showing moderate to high

resistance. GWAS identified two known genes: *GRH6* on chromosome 4 and *GRH1* on chromosome 5, as well as one novel locus on chromosome 11 (MTA11). In Chapter III, haplotype analysis estimated the accessions carrying resistant haplotypes at *GRH6*, *GRH1*, and MTA11. Additionally, several moderate to high resistant accessions did not carry resistant haplotypes at these three loci. The resistance in these accessions likely derived from unknown QTLs, making them valuable candidate donors for identifying novel QTLs conferring resistance to GRH.

The objectives of this chapter were to validate the QTLs detected in GWAS and to identify unknown QTLs not detected in GWAS. Genetic mapping for QTLs resistance to GRH was conducted in bi-parental segregating populations (F_2) using PCR-based markers and genotyping-by-sequencing (GBS). Several F_2 populations were developed from crosses between resistant accessions from MIDP and Taichung65 (T65), a susceptible Japonica cultivar. To avoid F_2 sterility in the crosses between Indica and Japonica, the resistant MIDP donors were also crossed with Indica cultivars, including IR24 (a resistant Indica variety carrying *GRH1*), Kangdang18 (KD18, a resistant Indica variety with an unknown resistance gene), and susceptible accessions from MIDP, generating additional F_2 populations. The resistant donors were selected based on their haplotypes at the three detected loci: *GRH6*, *GRH1*, and MTA11.

Four populations derived from crosses between T65 and four resistant MIDP accessions, along with one population from a cross between IR24 and a resistant Myanmar accession, were selected for QTL analysis using PCR-based markers to validate QTLs detected in GWAS. Each donor carried a resistant haplotype at one of *GRH6*, *GRH1*, or MTA11. Additionally, a population from a cross between IR24 and a resistant MIDP

accession without resistant haplotypes at any GWAS-detected loci was used for genetic mapping to identify unknown QTLs not detected in GWAS.

Materials and methods

Development of populations

The resistant donors from the MIDP were selected based on their haplotypes at identified loci to develop the populations. The donors included six accessions with the resistant haplotype at *GRH6*, fourteen at *GRH1*, nine at MTA11, two at both *GRH6* and *GRH1*, one at both *GRH1* and MTA11, and ten resistant accessions without resistant haplotypes at any detected loci. These donors were randomly crossed with T65 (a susceptible Japonica cultivar), IR24 (a resistant Indica variety carrying *GRH1*), KD18 (a resistant Indica variety with an unknown resistance gene), and susceptible accessions from MIDP to generate F_2 populations. The resulting F_2 populations from various cross combinations are listed in Appendix 3. These populations are valuable genetic resources for QTL mapping, developing pyramiding lines, and studying gene interactions. Some populations were selected for QTL analysis to validate those detected in GWAS and to identify previously unidentified QTLs.

Plant materials used in genetic mapping

Four donors were selected based on their haplotypes at detected loci: CC528 and CC310 with resistant haplotype at *GRH6*, CC98 carrying resistant haplotype at *GRH1*, and CC125 harboring resistant haplotype at MTA11. Each donor was crossed with T65, and the resulting F_1 plants were self-pollinated to generate F_2 populations (Fig. 25A-D). These four F_2 populations were evaluated for GRH resistance at the seedling stage and subjected

to QTL analysis using Indel markers. Additionally, an F_2 population from the cross between IR24 and CC96 processing the resistance haplotype at MTA11, was analyzed using SSR markers to validate MTA11 through QTL analysis (Fig. 25E). Another F_2 population from the cross between IR24 and CC426, which did not carry resistance haplotypes at any detected loci, was used for genetic mapping of QTLs conferring resistance to GRH through genotyping-by-sequencing (GBS) technology to identify novel or unknown QTLs (Fig. 25F).

GRH strain

The GRH strain used in this study was collected in Fukuoka, Japan, in 1990 and has been maintained by continuously rearing on seedlings of the susceptible Japonica cultivar Nipponbare under conditions of 25 ºC with 16 h of light and 8 h of darkness.

Evaluation for GRH resistance

An antibiosis test was conducted to evaluate the resistance to GRH. Ten-day-old rice seedlings were infested with ten first-instar GRH nymphs per test tubes. Nymph mortality (%) for each plant was determined at 3, 5, and 7 days after infestation (DAI).

Genotyping by PCR markers

Total genomic DNA of each F_2 plants was extracted by potassium acetate method. To develop agarose-gel based InDel markers around the genomic region significantly associated with GRH resistance detected by GWAS, InDel variants of CC528, CC310, CC98, CC125 and T65 were called with GATK4 GenotypeGVCFs. Indel variants were screened using SelectVariants with the '-select-type INDEL' option. A total of five, two

and six InDel markers covering the regions of MTA4, MTA5 and MTA11 respectively were used for the QTL confirmation (Appendix 4). The genotypes of marker loci were determined by PCR amplification in a PCR system-9700 (Applied Biosystems, Foster City, CA, USA). The PCR reaction contained an amount of approximately 10 ng of genomic DNA, 0.2 μM primer, and 7.5 μl of GoTaq Master mix (Promega, Fitchburg, WI, USA). The thermal profile included an initial denaturation at 95 ºC for 5 min, 35 cycles of 95 ºC for 30 s, 55 ºC for 30 s, and 72 ºC for 30 s, and a final elongation step at 72 ºC for 7 mins. The amplified products were visualized by a 4 % agarose gel electrophoresis in 0.5x TBE buffer. The gels were stained by ethidium bromide and photographed under ultraviolet light.

Genotyping-by-sequencing and genotype call

For the genotyping of F_2 populations, genomic DNA was extracted from fresh leaf samples using potassium acetate method. Library preparation adapted to the Illumina dual index system was conducted according to Poland *et al.* (2012) with minor modifications. Briefly, genomic DNA samples were digested with *Pst*I and *Msp*I restriction enzymes at 37ºC for 2 hours, followed by an inactivation step at 65ºC for 15 minutes. An oligonucleotide pair of the adaptor compatible with the *Pst*I sticky end, 5'-CACGACGCTCTTCCGATCT (barcode sequence) TGCA-3' and 5'-(complementary barcode sequence) AGATCGGAAGAGCGTCGTG-3' and another pair compatible with the MspI end, 5'-CGAGATCGGAAGAGCACACTCTTTCCCTACACGAC-3' and 5'-GTGACTGGAGTTCAGACGTGTGCTCTTCCGATCT-3', were annealed in AB buffer. After an adaptor ligation reaction containing a set of 96 unique barcodes and pooling samples, the obtained product was purified by Qiagen QIAquick PCR Purification Kit

(Qiagen, Venlo, Netherlands) and amplified using NEBNext i5 and i7 primers (New England Biolabs, Ipswich, MA, USA) compatible with the Illumina dual index system using KAPA HiFi HotStart ReadyMix (Kapa Biosystems, Wilmington, MA, USA). DNA fragments ranging from 200 to 700 bp were obtained using 0.85-0.56 × SPRIselect beads (Beckman Coulter, Brea, CA, USA). Following library quantitation by quantitative PCR, prepared libraries were sequenced using Illumina NovaSeq 150 PE platform (Azenta, Tokyo, Japan). The obtained fastq files were analyzed using the TASSEL GBS V2 pipeline (Glaubitz *et al*. 2014). The LB-Impute software was used to impute missing genotypes (Fragoso *et al*. 2016). Between the parents, the polymorphic SNP genotypes were selected and converted to ABH genotypes in the custom script (https://github.com/qikushu/gbs).

QTL analysis

QTL analysis was performed using R/qtl (Broman and Sen 2009). The marker regression was used to estimate genetic effect and logarithm of odds (LOD) score. The LOD threshold more than 1% level of significant were defined as significant QTL. LOD threshold was calculated from 1000 permutation test.

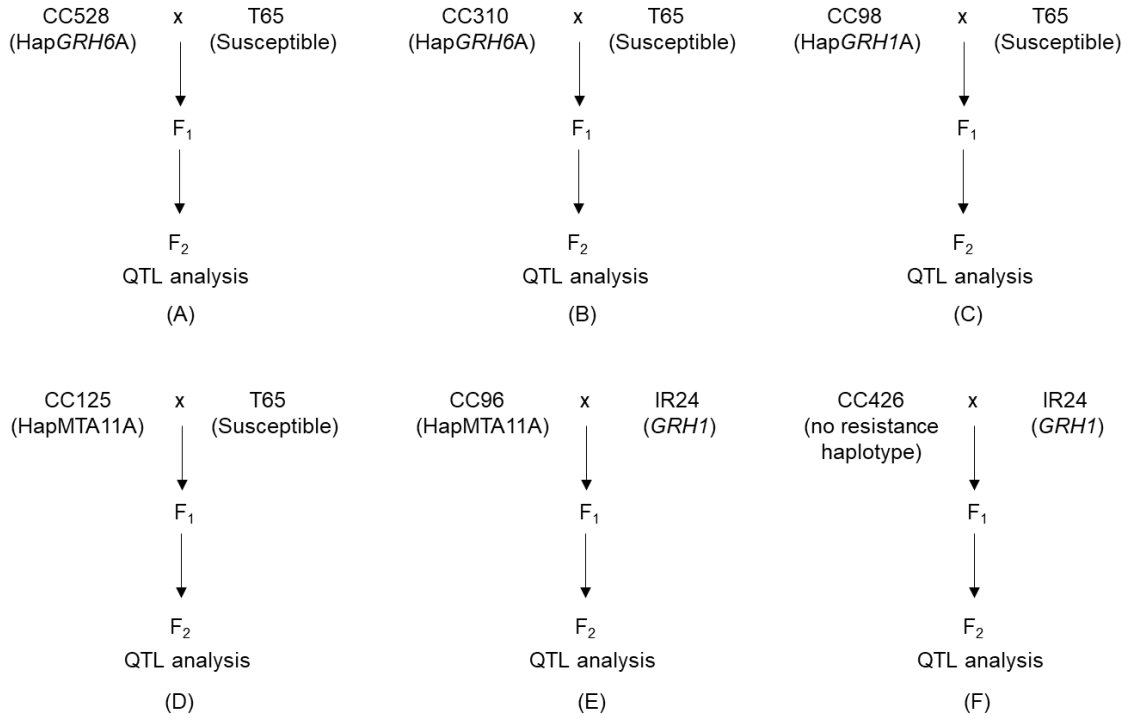

Fig. 25. The breeding schemes for the development of populations used in genetic mapping of QTLs conferring resistance to GRH

Results

Validation of QTLs conferring resistance to GRH derived from CC528

For validation of MTA4 detected in GWAS, a total of 62 F_2 individuals derived from the cross between GRH susceptible Japonica cultivar, T65 and CC528 carrying resistant haplotype at *GRH6* (Hap*GRH6*A) were evaluated for GRH resistance at the seedling stage using antibiosis test. T65 showed no nymph mortality whereas CC528 showed high nymph mortality of 90.0% at 3DAI (Fig. 26). The F_2 population showed continuous segregation of nymph mortality (%) at 3DAI (Fig. 26). Five InDel markers around MTA4 were used for QTL analysis (Appendix 4). A significant QTL, *qGRH4*, was identified on chromosome 4 with a peak at 4,521,451 bp, an LOD score of 20.6, and explaining 77.7% of the phenotypic variance (Table 11). The genomic region of *qGRH4* overlapped with the MTA4 region detected in GWAS (Fig. 27). Additionally, the previously reported gene *GRH6* was found within this region (Fig. 27).

Validation of QTLs conferring resistance to GRH derived from CC310

For further validation of MTA4 detected in GWAS, another F_2 population derived from the cross between T65 and CC310 carrying resistance haplotype at *GRH6* (Hap*GRH6*A) was used for QTL analysis. A total of 80 F_2 individuals were evaluated for GRH resistance at the seedling stage using antibiosis test. T65 showed no nymph mortality whereas CC310 showed complete nymph mortality at 3DAI (Fig. 28). The F_2 population showed discrete segregation of nymph mortality at 3DAI (Fig. 28). Five InDel markers around MTA4 were developed to validate the QTL (Appendix 4). This study detected a significant QTL, *qGRH4*, on chromosome 4 with the peak at 4,440,537 bp, showing an LOD score of 31.5 and explaining 83.7% of the phenotypic variance (Table 12). The

qGRH4 genomic region matched the MTA4 region identified in GWAS (Fig. 29), and the *GRH6* was present within this area (Fig. 29).

Validation of QTLs conferring resistance to GRH derived from CC98

To confirm MTA5 detected in the GWAS, a total of 75 F_2 individuals, originating from a cross between T65 and CC98 with the resistance haplotype at *GRH1* (Hap*GRH1*A), were assessed for GRH resistance at the seedling stage using an antibiosis test. T65 showed no nymph mortality whereas CC98 showed high nymph mortality of 81.9% at 3DAI (Fig. 30). The F_2 population showed discrete segregation of nymph mortality (%) (Fig. 30). Two InDel markers around MTA5 were developed to validate the QTL detected in GWAS (Appendix 4). A significant QTL, designated as *qGRH5*, was found on chromosome 5 with the highest LOD score of 33.6 at position 8,057,416 bp, explaining 87.3% of the phenotypic variance (Table 13). The *qGRH5* genomic region coincided with the MTA5 region identified in the GWAS (Fig. 31). Moreover, the previously reported gene *GRH1* was located within this region (Fig. 31).

Validation of QTLs conferring resistance to GRH derived from CC125

To validate MTA11 as a QTL, QTL analysis was conducted in the bi-parental F_2 population derived from the cross between CC125, the Myanmar accession carrying the resistance haplotype at MTA11 (HapMTA11A) and the susceptible japonica cultivar T65 using the six InDel markers around MTA11 region (Appendix 4). T65 showed no nymph mortality whereas CC125 showed high nymph mortality of 92.0% at 7DAI (Fig. 32). F_2 individuals showed continuous segregation in nymph mortality of GRH (Fig. 32). A significant QTL, *qGRH11*, was found on chromosome 11 with the peak LOD score of 9.7,

explaining 43.7 % of the phenotypic variation (Table 14). The QTL peak was represented by the InDel marker MTA11Id1 at 27,177,381 bp around the MTA11 region, and QTL region covered from MTA11Id8 (26,265,353 bp) to MTA11Id9 (28,967,652 bp) overlapping with the MTA11 region (27,107,110 bp to 27,121,054 bp) (Fig. 33). While the previously reported gene, *GRH2*, was located near the *qGRH11* region, the LOD value of *GRH2* region was lower than that of *qGRH11* speculating that the QTL closely linked to MTA11 was likely a novel GRH resistance locus on chromosome 11 (Fig. 33).

Validation of QTLs conferring resistance to GRH derived from CC96

For further validation of MTA11, another F_2 population derived from a cross between the Indica cultivar IR24 and CC96, which carries the resistance haplotype at MTA11 (HapMTA11A), was used. However, IR24 carries the previously reported gene *GRH1*. Initially, a total of 400 individuals from this F_2 population were evaluated for GRH resistance using an antibiosis test. Most of the individuals showed high resistance to GRH at 3DAI (data not shown). To exclude the effect of *GRH1* in the F_2 individuals, 91 individuals with nymph mortalities lower than 90% were selected. Among these, accessions homozygous for the IR24 allele and heterozygous at the *GRH1* region were excluded using SSR marker RM18180. The remaining 78 individuals with homozygous for CC96 allele at *GRH1* region were used in QTL analysis (Appendix 5). The F_2 population showed continuous segregation of nymph mortality (Fig. 34). The SSR marker RM1233 was used for QTL analysis to confirm MTA11 (Appendix 5). A significant QTL, *qGRH11*, was found at 27,005,005 bp on chromosome 11 with an LOD score of 11.8, explaining 48.9% of the phenotypic variation (Table 15). The position of *qGRH11* was near the MTA11 region (Fig. 35).

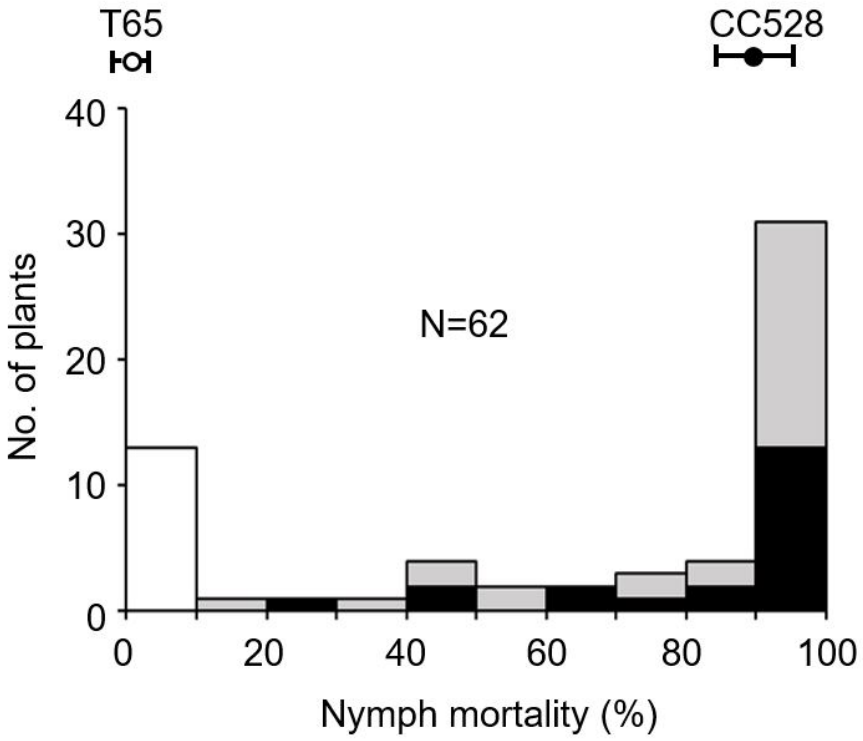

Fig. 26. Frequency distribution of nymph mortality of green rice leafhopper in F_2 population derived from the cross between CC528 (resistance) and T65 (susceptible) at 3 days after infestation (DAI). Black, grey and white boxes represented the homozygous for CC528, heterozygous, and homozygous for T65 plants at the Grh6Id4 Indel marker, respectively.

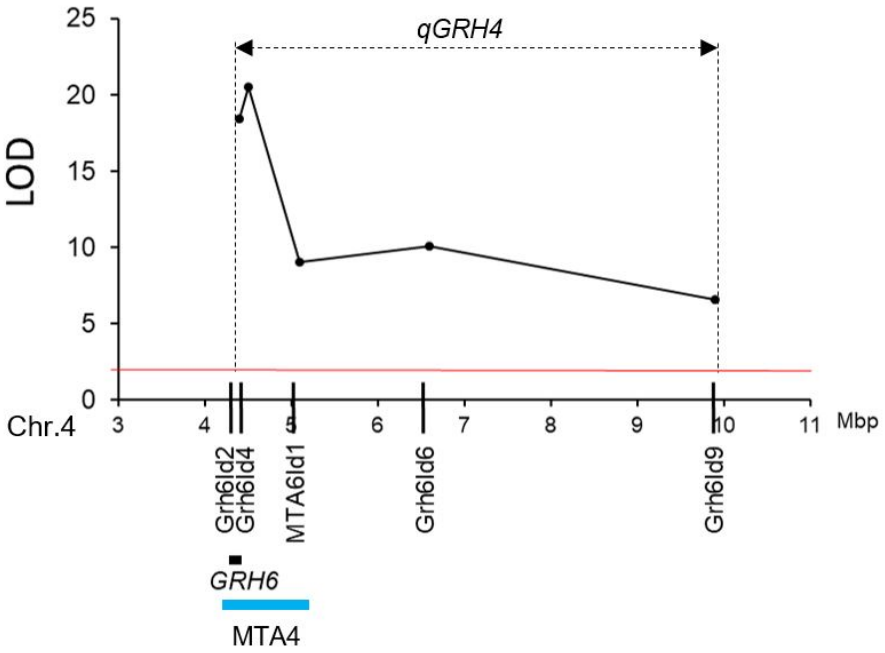

Fig. 27. Location of QTL conferring resistance to GRH on chromosome 4 identified using five InDel markers in the F_2 population derived from the cross between CC528 (resistance) and T65 (susceptible) at 3 days after infestation (DAI). The red horizontal line represented 1% significant threshold level in 1000 times permutation test. Horizontal black arrow represented the genomic region of QTL. Horizontal black and blue bars indicated the position of previously reported QTL and marker-trait association (MTA) detected in genome-wide association study (GWAS).

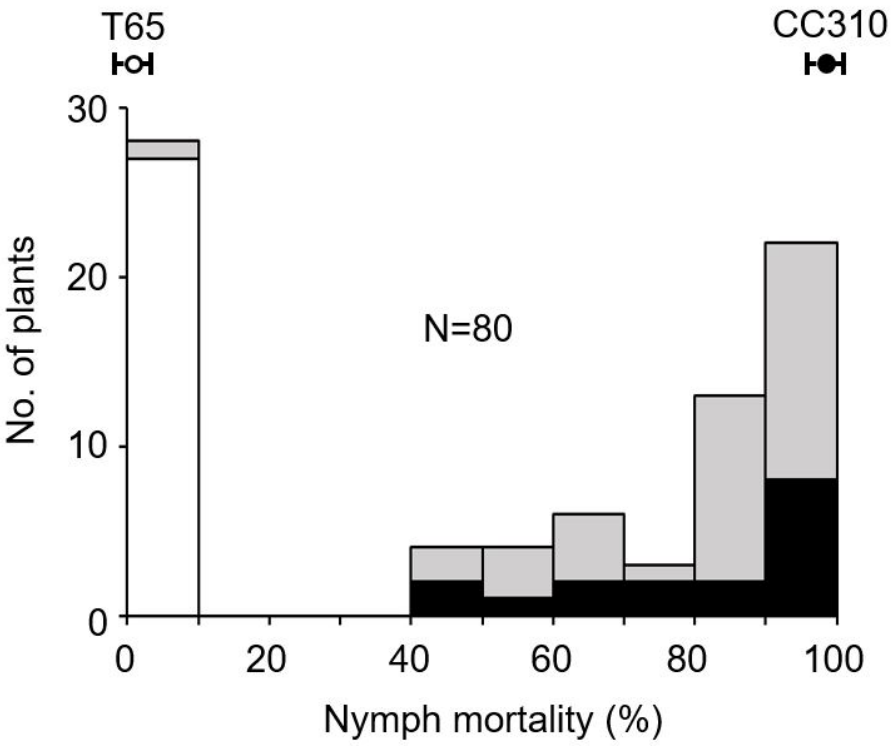

Fig. 28. Frequency distribution of nymph mortality of green rice leafhopper in F_2 population derived from the cross between CC310 (resistance) and T65 (susceptible) at 3 days after infestation (DAI). Black, grey and white boxes represented the homozygous for CC310, heterozygous, and homozygous for T65 plants at the Grh6Id2 Indel marker, respectively.

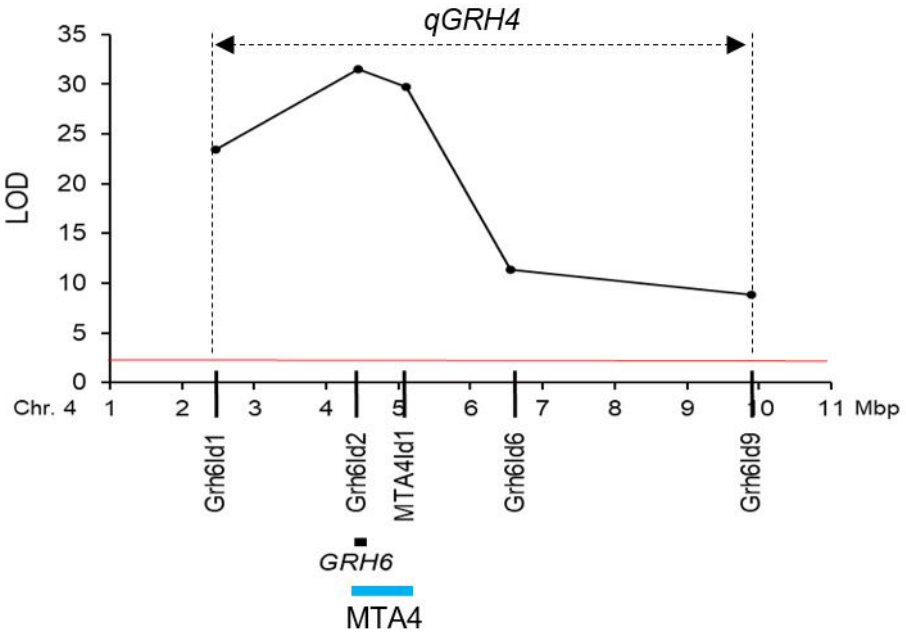

Fig. 29. Location of QTL conferring resistance to GRH on chromosome 4 identified using five InDel markers in the F_2 population derived from the cross between CC310 (resistance) and T65 (susceptible) at 3 days after infestation (DAI). The red horizontal line represented 1% significant threshold level in 1000 times permutation test. Horizontal black arrow represented the genomic region of QTL. Horizontal black and blue bars indicated the position of previously reported QTL and marker-trait association (MTA) detected in genome-wide association study (GWAS).

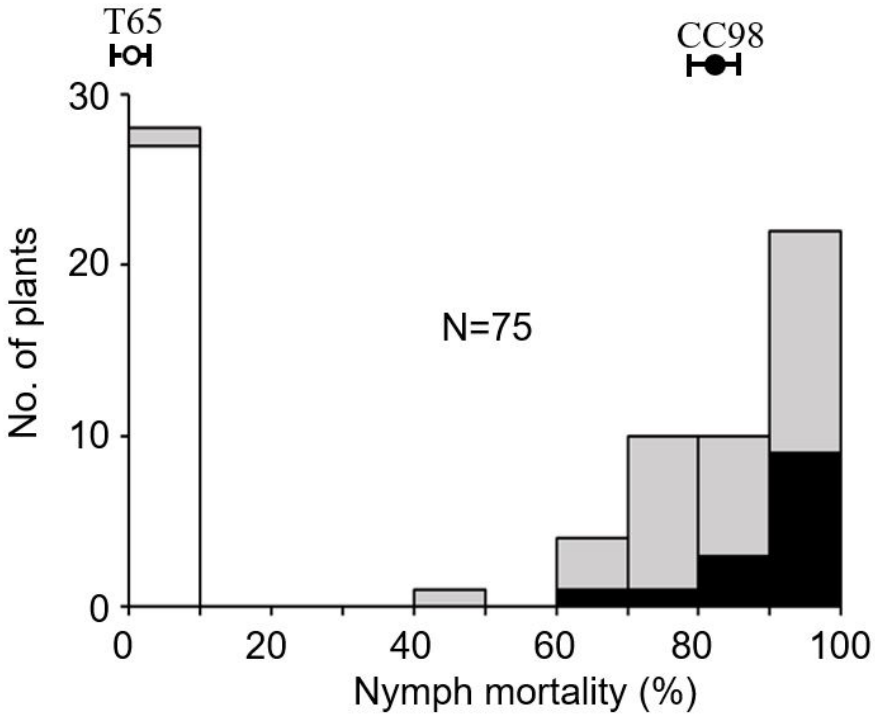

Fig. 30. Frequency distribution of nymph mortality of green rice leafhopper in F_2 population derived from the cross between CC98 (resistance) and T65(susceptible) at 3 days after infestation (DAI). Black, grey and white boxes represented the homozygous for CC98, heterozygous, and homozygous for T65 plants at the Grh1Id2 Indel marker, respectively.

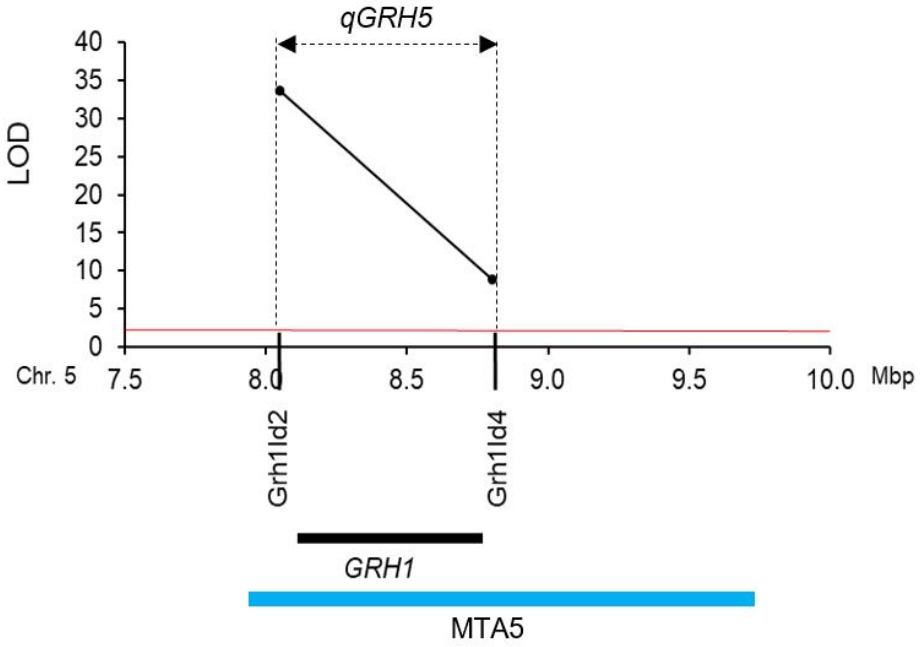

Fig. 31. Location of QTL conferring resistance to GRH on chromosome 5 identified using two InDel markers in the F_2 population derived from the cross between CC98 (resistance) and T65(susceptible) at 3 days after infestation (DAI). The red horizontal line represented 1% significant threshold level in 1000 times permutation test. Horizontal black arrow represented the genomic region of QTL. Horizontal black and blue bars indicated the position of previously reported QTL and marker-trait association (MTA) detected in genome-wide association study (GWAS).

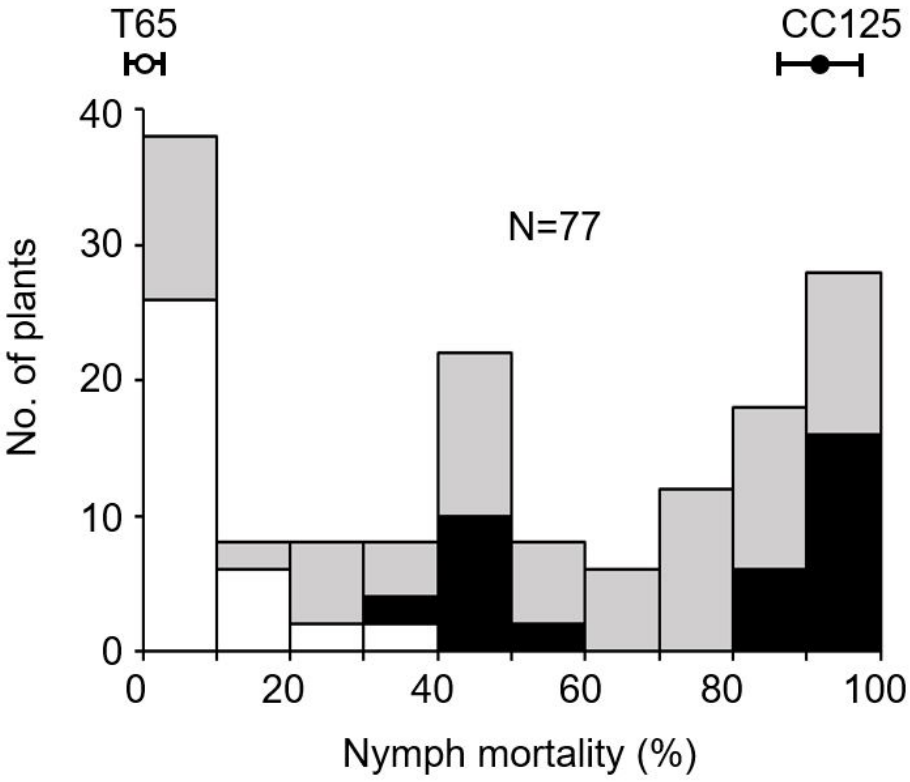

Fig. 32. Frequency distribution of nymph mortality of green rice leafhopper in F_2 population derived from the cross between CC125 (resistance) and T65 (susceptible) at 7 days after infestation (DAI). Black, grey and white boxes represented the homozygous for CC125, heterozygous, and homozygous for T65 plants at the MTA11Id1 Indel marker, respectively.

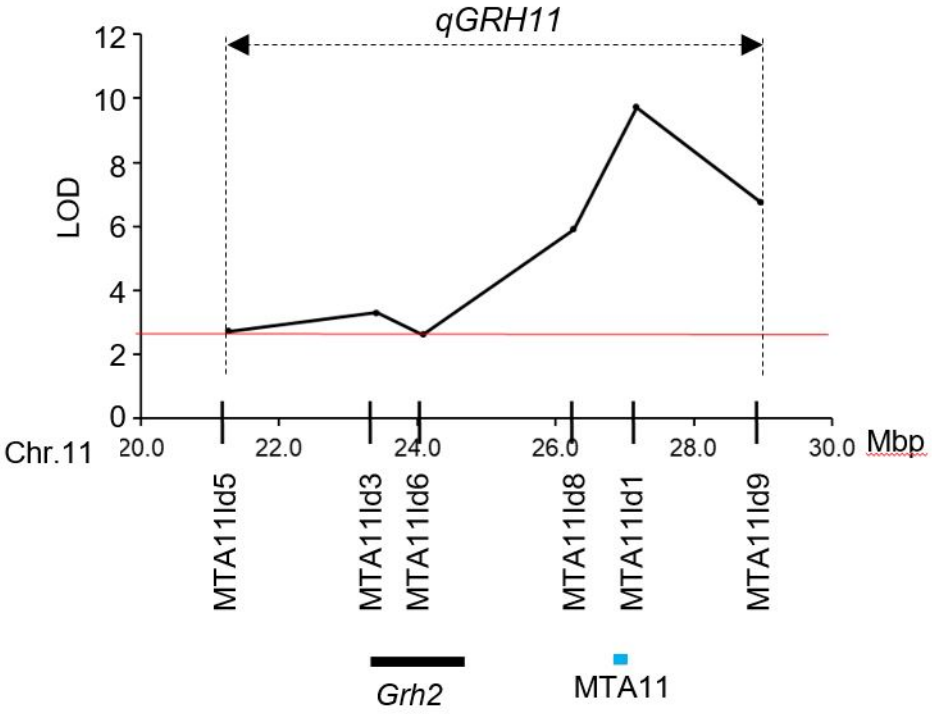

Fig. 33. Location of QTL conferring resistance to GRH on chromosome 11 identified using six InDel markers in the F_2 population derived from the cross between CC125 (resistance) and T65 (susceptible) at 7 days after infestation (DAI). The red horizontal line represented 1% significant threshold level in 1000 times permutation test. Horizontal black arrow represented the genomic region of QTL. Horizontal black and blue bars indicated the position of previously reported QTL and marker-trait association (MTA) detected in genome-wide association study (GWAS).

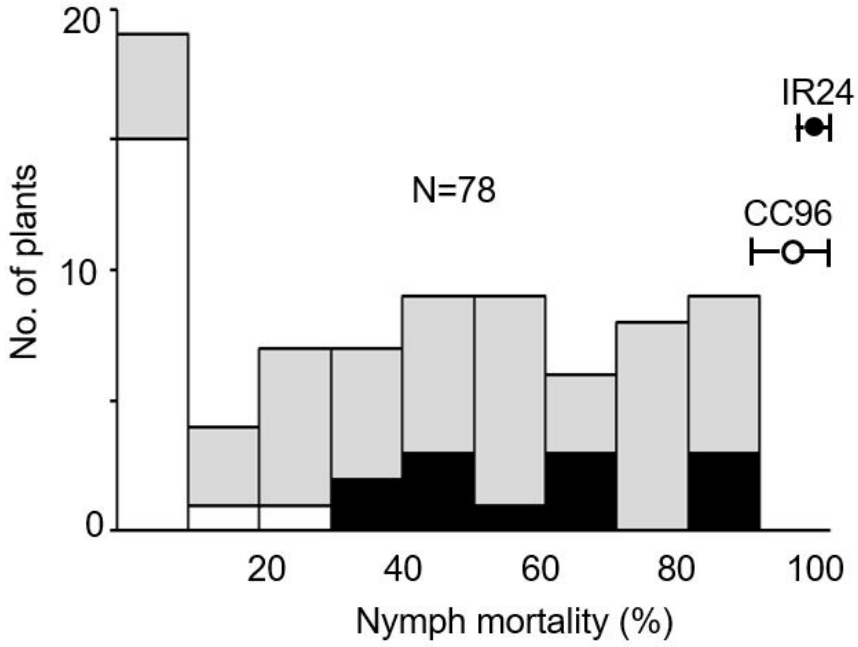

Fig. 34. Frequency distribution of nymph mortality of green rice leafhopper in F_2 population derived from the cross between CC96 (resistance) and IR24 (resistance) at 3DAI. Black, grey and white boxes represented the homozygous for CC96, heterozygous, and homozygous for IR24 plants at the RM1233 SSR marker, respectively.

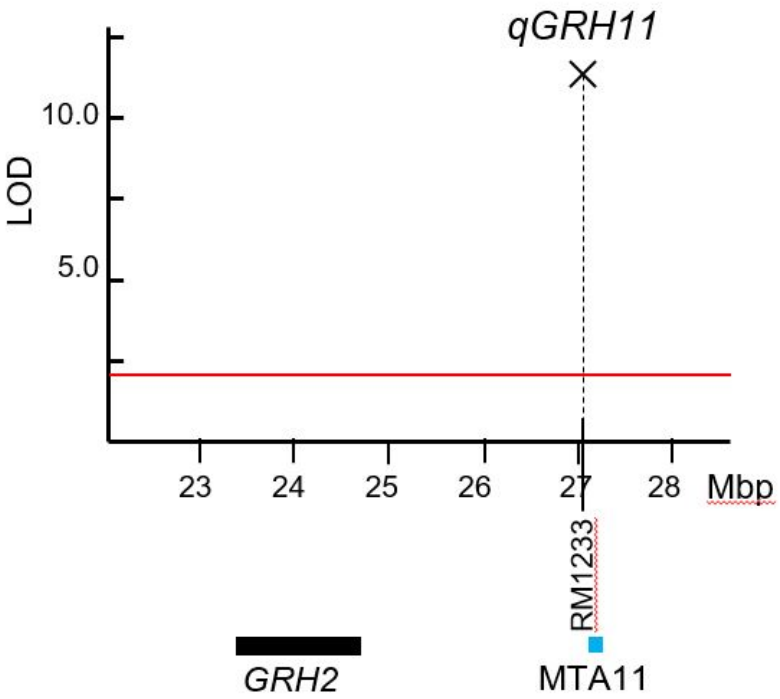

Fig. 35. Location of QTL conferring resistance to GRH on chromosome 11 identified using RM1233 SSR marker in the F_2 population derived from the cross between CC96 (resistance) and IR24 (resistance) at 3 days after infestation (DAI). The red horizontal line represented 1% significant threshold level in 1000 times permutation test. Horizontal black and blue bars indicated the position of previously reported QTL and marker-trait association (MTA) detected in genome-wide association study (GWAS).

Table 11. QTL detected in F_2 population derived from the cross between CC528 (resistance) and T65 (susceptible)

QTL	Marker	Chr.	Position of peak marker (bp)	Peak LOD	Additive effect [a]	Dominance effect [b]	PVE [c] (%)	LOD threshold [d]	Reported gene	MTA in GWAS
qGRH4	Grh6Id4	4	4,521,451	20.6**	43.0	42.2	77.7	2.6	*GRH6*	MTA4

[a] A LOD threshold of 2.6 at an experiment-wise level of 1% was used.

[b] Calculated as (X-Y)/2 where X and Y are trait values of CC528 homozygous and T65 homozygous plants, respectively.

[c] Calculated as Z-(X+Y)/2, where Z is the trait value of heterozygous plants.

[d] Percentage of explained phenotypic variation.

Table 12. QTL detected in F_2 population derived from the cross between CC310 (resistance) and T65 (susceptible)

QTL	Marker	Chr.	Position of peak marker (bp)	Peak LOD	Additive effect[a]	Dominance effect[b]	PVE[c] (%)	LOD threshold[d]	Reported gene	MTA in GWAS
qGRH4	Grh6Id2	4	4,440,537	31.5	41.4	40.9	83.7	2.8	*GRH6*	MTA4

[a] A LOD threshold of 2.8 at an experiment-wise level of 1% was used.

[b] Calculated as (X-Y)/2 where X and Y are trait values of CC310 homozygous and T65 homozygous plants, respectively.

[c] Calculated as Z-(X+Y)/2, where Z is the trait value of heterozygous plants.

[d] Percentage of explained phenotypic variation.

Table 13. QTL detected in F_2 population derived from the cross between CC98 (resistance) and T65 (susceptible)

QTL	Marker	Chr.	Position of peak marker (bp)	Peak LOD	Additive effect [a]	Dominance effect [b]	PVE [c] (%)	LOD threshold [d]	Reported gene	MTA in GWAS
qGRH5	Grh1Id2	5	8,057,416	33.6	45.7	35.0	87.3	2.2	*GRH1*	MTA5

[a] A LOD threshold of 2.2 at an experiment-wise level of 1% was used.

[b] Calculated as (X-Y)/2 where X and Y are trait values of CC98 homozygous and T65 homozygous plants, respectively.

[c] Calculated as Z-(X+Y)/2, where Z is the trait value of heterozygous plants.

[d] Percentage of explained phenotypic variation.

Table 14. QTL detected in F_2 population derived from the cross between CC125 (resistance) and T65 (susceptible)

QTL	Marker	Chr.	Position of peak marker (bp)	Peak LOD	Additive effect [a]	Dominance effect [b]	PVE [c] (%)	LOD threshold [d]	Reported gene	MTA in GWAS
qGRH11	MTA11Id1	11	27,177,381	9.7	33.5	18.7	43.7	2.6		MTA11

[a] A LOD threshold of 2.6 at an experiment-wise level of 1% was used.

[b] Calculated as (X-Y)/2 where X and Y are trait values of CC125 homozygous and T65 homozygous plants, respectively.

[c] Calculated as Z-(X+Y)/2, where Z is the trait value of heterozygous plants.

[d] Percentage of explained phenotypic variation.

Table 15. QTL detected in F_2 population derived from the cross between CC96 (resistance) and IR24 (resistance)

QTL	Marker	Chr.	Position of peak marker (bp)	Peak LOD	Additive effect [a]	Dominance effect [b]	PVE [c] (%)	LOD threshold [d]	Reported gene	MTA in GWAS
qGRH11	RM1233	11	27,005,000	11.8	28.7	18.1	48.9	2.1		MTA11

[a] A LOD threshold of 2.1 at an experiment-wise level of 1% was used.

[b] Calculated as (X-Y)/2 where X and Y are trait values of CC96 homozygous and IR24 homozygous plants, respectively.

[c] Calculated as Z-(X+Y)/2, where Z is the trait value of heterozygous plants.

[d] Percentage of explained phenotypic variation.

Identification of QTLs conferring resistance to GRH derived from CC426

To identify unknown QTLs those undetected in GWAS, an F_2 population derived from the cross between IR24 and CC426, which lacked resistant haplotypes at *GRH6*, *GRH1*, and MTA11, was used for QTL mapping via the GBS approach. The 88 individuals were evaluated for GRH resistance using an antibiosis test. The F_2 population showed continuous segregation in nymph mortality from 0% to 90% at 3DAI (Fig. 36). A total of 3,539 polymorphic SNPs covering twelve rice chromosomes were used for marker regression. Two significant QTLs, *qGRH5.1* and *qGRH5.2*, were identified on chromosome 5 (Fig. 37, Table 16). The *qGRH5.1*, ranging from 6,239,590 bp to 9,473,797 bp, had peak LOD scores of 12.2 and explained 47.1% of the phenotypic variance (Table 16). This region included the previously reported *GRH1* (Fig. 38). The *qGRH5.1* had a negative additive effect of 30.2, indicating that the IR24 allele increased nymph mortality (Table 16). The *qGRH5.2*, ranging from 14,978,395 bp to 18,503,289 bp, had peak LOD scores of 10.6 and explained 42.5% of the phenotypic variance (Table 16). This QTL also had a negative additive effect of 31.3, suggesting the IR24 allele increased nymph mortality (Table 16). No previously mapped gene was found in this region (Fig. 38). No additional QTLs derived from the Myanmar accession CC 426 were detected in this population (Fig. 37).

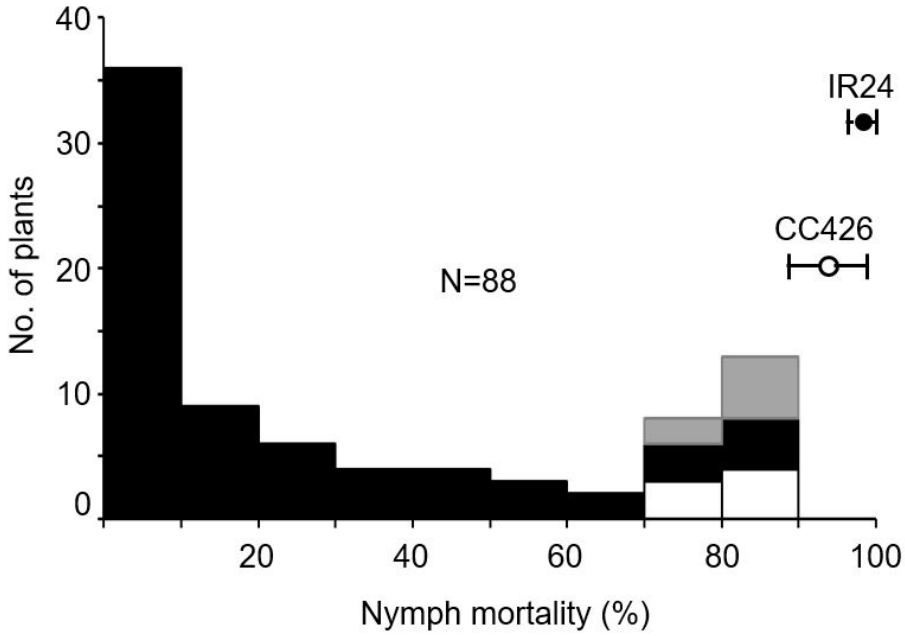

Fig. 36. Frequency distribution of nymph mortality of green rice leafhopper in F_2 population derived from the cross between CC426 (resistance) and IR24 (resistance) at 3 days after infestation (DAI). Black, grey and white boxes represented the homozygous for CC426, heterozygous, and homozygous for IR24 plants at the peak SNP marker S05_9344843 respectively.

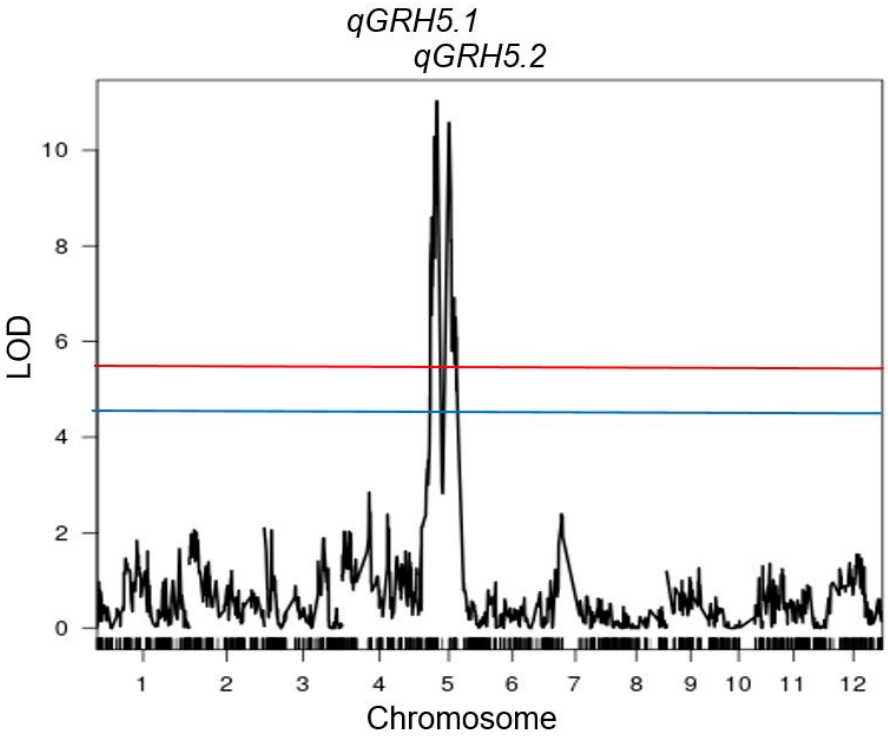

Fig. 37. Genome-wide distribution of logarithm of odds (LOD) obtained by marker regression analysis in F_2 cross between CC426 (resistance) and IR24 (resistance). The red and blue lines indicated thresholds of significance level of 1% and 5%, respectively.

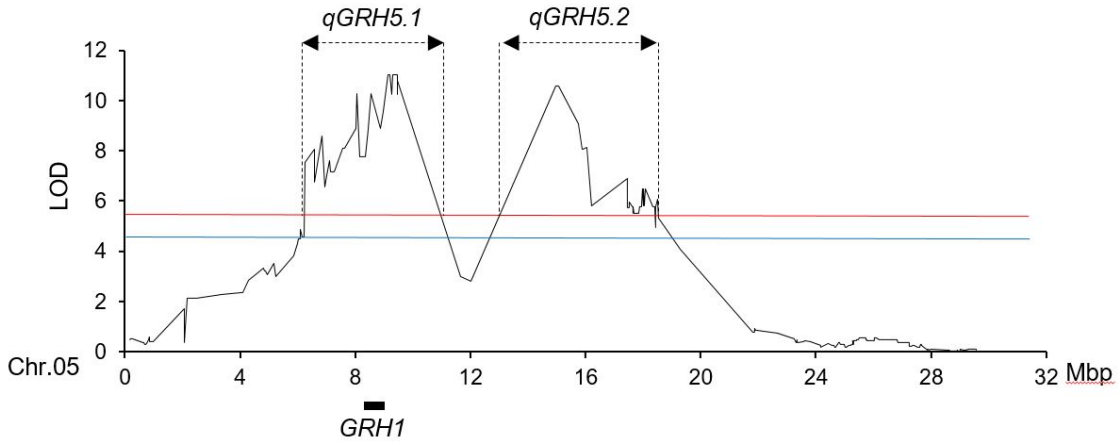

Fig. 38. Location of QTLs conferring resistance to GRH on chromosome 5 identified by GBS approach in the F_2 population derived from the cross between CC426 (resistance) and IR24 (resistance) at 3 days after infestation (DAI). The red and blue horizontal lines represented 1% and 5% significant threshold level respectively in 1000 times permutation test. Horizontal black arrow represented the genomic region of QTL. Horizontal black bar indicated the position of previously reported QTL.

Table 16. QTL detected in F_2 population derived from the cross between CC426 (resistance) and IR24 (resistance)

QTL	QTL region		Chr.	Position of peak marker (bp)	Peak LOD	Additive effect [a]	Dominance effect [b]	PVE [c] (%)	LOD threshold [d]	Reported gene
	From	To								
qGRH5.1	6,239,590	9,473,797	5	9,344,843	12.2	-30.2	31.7	47.1	5.6	*GRH1*
qGRH5.2	14,978,395	18,503,289	5	14,948,395	10.6	-31.3	13.3	42.5	5.6	

[a] A LOD threshold of 5.6 at an experiment-wise level of 1% was used.

[b] Calculated as (X-Y)/2 where X and Y are trait values of CC426 homozygous and IR24 homozygous plants, respectively.

[c] Calculated as Z-(X+Y)/2, where Z is the trait value of heterozygous plants.

[d] Percentage of explained phenotypic variation.

Discussion

In this study, a total of three QTLs were identified across six bi-parental populations derived from six different donors of MIDP. QTL analysis revealed that *qGRH4*, derived from CC528 and CC310, overlapped with *GRH6* (Tables 11 and Table 12), and *qGRH5*, derived from CC98, coincided with *GRH1* (Table 13). In Chapter II and III, GWAS and haplotype analysis indicated that CC528 and CC310 carried the resistant haplotype at *GRH6*, while CC98 carried the resistant haplotype at *GRH1* (Table 9). These findings confirm that the QTLs identified through GWAS were validated by QTL mapping using bi-parental populations, demonstrating the effectiveness of haplotype-based donor selection for QTL mapping.

Additionally, *qGRH11*, derived from CC96 and CC125, was identified on chromosome 11 (Tables 14 and Table 15). Although the previously reported gene *GRH2* is also located on the long arm of chromosome 11 (Kadowaki *et al*. 2003), *qGRH11* was mapped to a different region, approximately 2.4 Mb away from *GRH2* (Fig. 33). This suggests that *qGRH11* is likely a novel locus independent of *GRH2*. The highest peak of *qGRH11* is near the genomic region of MTA11, detected in GWAS (Fig. 33), further validating MTA11 as a novel QTL identified by the bi-parental population. However, distinguishing between the regions of *GRH2* and *qGRH11* was challenging due to the limited number of markers and the low-resolution F_2 mapping population used in this study. Therefore, further confirmation of the candidate region for *qGRH11* is needed using high-resolution mapping populations such as chromosome segment substitution lines (CSSLs) and recombinant inbred lines (RILs).

Using the GBS approach, genome-wide QTL mapping in an F_2 population derived from the cross between IR24 and CC426 identified two QTLs, *qGRH5.1* and *qGRH5.2*,

spanning regions from 6.2 to 9.5 Mb and 14.7 to 18.5 Mb, respectively (Table 16). However, only two SNPs below the significance threshold were detected within the region between 9.5 and 14.7 Mb (Fig. 38), raising the possibility of either two distinct QTLs or one QTL with broader interval ranging from 6.2 to 18.5 Mb with a low-significance region. This low-significance region might be due to genotyping inaccuracies. The poor precision of QTL location could be attributed to limited marker density in the genomic region between 9.5 and 14.7 Mb. Larger gaps between markers can cause the QTL region to be less precisely mapped, resulting in broader QTL intervals where the exact location of the QTL cannot be pinpointed. Increasing marker density improves the precision of QTL localization and the accuracy of effect estimates for small and medium-sized QTLs (Stange *et al.* 2013). To clarify this, further studies are needed to increase marker density in this region and perform fine mapping using advanced mapping populations.

The region of *qGRH5.1* overlapped with the previously reported gene *GRH1* (Fig. 38). The IR24 allele at this region increased nymph mortality (Table 16), suggesting that *qGRH5.1* was derived from IR24, which carries *GRH1* (Kadowaki *et al*. 2003). No additional QTLs were detected from CC426, leading to the speculation that resistance in the donor CC426 might be controlled by minor resistance genes. In the population with the genetic background of IR24 carrying *GRH1*, the effect of minor resistance genes might be completely masked by the major resistance gene *GRH1*. Similarly, in near isogenic lines of *O. rufipogon*, while a minor resistance gene on chromosome 4 provides moderate resistance in the *grh5/grh5* background, its effect is masked by the major resistance gene *Grh5* in the *Grh5/Grh5* background (Fujita *et al*. 2006). Therefore, further investigation is required to identify minor resistance QTLs in CC426. This can be

achieved by using advanced mapping populations and increasing marker density. Additionally, using an independent mapping population with a different genetic background is another strategy. Populations derived from the resistance accessions of MIDP that lack the resistance haplotypes at *GRH6*, *GRH1*, and MTA11 but have a susceptible T65 background are candidates for identifying novel QTLs conferring resistance to GRH (Appendix 3). By employing these strategies, minor resistance QTLs that might be masked in other genetic backgrounds can be better detected.

SUMMARY AND CONCLUSION

Green rice leafhopper (GRH, *Nephotettix cincticeps* (Uhler)) is a serious insect pest of rice in the temperate regions of Asia. Utilization of host plant resistance is one of the most effective strategy to mitigate insect pest infestation. The resistance of varieties carrying single resistance gene is easy to break down due to evolution of new biotypes. Therefore, it is still need to explore new resistance genes and integrate them into the rice cultivars to combat the virulent GRH populations. The rice landraces in Myanmar is one of the precious genetic reservoir for rice improvement for the agronomically important genes such as abiotic and biotic stress resistance. The objective of this thesis is to study the genetic basis of conferring resistance to GRH in landraces of Myanmar rice.

First, 224 rice accessions from the Myanmar Indica Rice Diversity Panel (MIDP) were evaluated for GRH resistance using an antibiosis test at the seedling stage. Of these, 188 accessions exhibited moderate to high resistance to GRH at 7 days after infestation (DAI). GWAS identified three significant genomic regions associated with GRH resistance: MTA4, MTA5, and MTA11, located on chromosomes 4, 5, and 11, respectively. MTA4 and MTA5 were consistently significant across all evaluation periods (3DAI, 5DAI, and 7DAI), suggesting they are major QTLs, while MTA11 appeared at 5DAI and 7DAI, indicating it may be a delay response QTL. Linkage disequilibrium (LD) analysis revealed that the peak SNP in the MTA5 region was near the previously reported gene *GRH1*, suggesting *GRH1* as a candidate gene for MTA5. Given MTA5's consistent significance, *GRH1* is likely a major genetic factor for GRH resistance in MIDP. The MTA11 region with no previously reported QTLs in the same LD block, is proposed as a novel QTL. Although *GRH6* was located near MTA4, the peak SNP of MTA4 was about 400 kb away from *GRH6*, within a weak LD block, speculating that the effect of major

resistance gene *GRH1* might interfere the detection of functional SNPs. To address this, GWAS was repeated excluding the accessions with the resistance haplotype at *GRH1*. Several highly associated SNPs were found at *GRH6* region and the second highest peak of MTA4 region was located within the high resolution mapping region of *GRH6*, speculating that *GRH6* was likely the candidate gene of MTA4 region. In this chapter, GWAS allowed substantial achievement for identifying two known genes: *GRH6* and *GRH1* and one novel QTL (MTA11) conferring resistance to GRH in Myanmar Indica landraces.

In Chapter II, the causal gene for *GRH6* was identified through gene cloning using the *Oryza nivara* BAC clone IRGC105715-26E15. *GRH6*, previously mapped on chromosome 4 in both the Surinam cultivar "SML17" and the wild accession *Oryza nivara,* IRGC105715 (WK56), was delimited to a 32.2 kbp region in the Nipponbare genome. This region contained four predicted genes, including *Os04g0165900* and *Os04g0166000*, which encodes protein with leucine-rich repeat (LRR) domains. However, the causal gene for *GRH6* had not been detected, and its molecular mechanism was still unknown. In this study, genomic sequence analysis and PCR confirmation showed that *Oryza nivara,* WK56 has a single copy of gene (*Os04g0166000*) at the genomic region of *GRH6* due to the absence of tandem duplication. This gene was cloned and introduced into the susceptible cultivar T65 via *Agrobacterium*-mediated transformation. The resulting transgenic plants (T_0) exhibited moderate to high GRH resistance, confirming that the WK56 allele at *Os04g0166000* is associated with GRH resistance, and designated as *GRH6-WK56*. *GRH6-WK56* encodes a protein with two LRR domains, with several insertions, deletion, and amino acid polymorphisms between WK56 and Nipponbare potentially affecting protein structure and function. Quantitative real-time PCR (qRT-

PCR) analysis showed that *GRH6* expression was significantly upregulated in the GRH6-NIL plants at 2 and 3 days after GRH infestation, leading to insect death by inhibiting feeding. Differences in the promoter region of *GRH6* between WK56 and T65, including insertions, deletions, and nucleotide polymorphisms, may regulate gene expression. This chapter advances the understanding of *GRH6* by identifying the causal gene and elucidating its role in GRH resistance.

In Chapter III, haplotype variations for *GRH6*, *GRH1*, and MTA11 were investigated by haplotype analysis and the accessions with resistance haplotypes were estimated. Two major haplotypes (HapA and HapB) were observed for the genomic region of *GRH1* and MTA11. Hap*GRH1*A and HapMTA11A corresponded to GRH resistance for each respective locus. Seven major haplotypes were found for *GRH6* (Hap*GRH6*A to Hap*GRH6*G), with Hap*GRH6*A being associated with GRH resistance. Haplotype analysis revealed that 41 accessions with one or more resistance haplotypes at major reported genes (Hap*GRH6*A and Hap*GRH1*A) showed high resistance to GRH, while 28 accessions with the resistance haplotype only at MTA11 (HapMTA11A) exhibited moderate to high resistance. Several resistant accessions lacked known resistance haplotypes, suggesting their resistance might be due to unknown QTLs. This analysis explained the haplotype-based genetic architecture of Myanmar landraces, identifying them as potential candidates for further breeding programs. Their haplotype information is crucial for developing rice varieties with enhanced GRH resistance. The geographical distribution of resistance haplotypes was also investigated. Accessions with resistance haplotypes at major genes, including *GRH6* and *GRH1*, were mainly found in the central dry zone and southern regions of Myanmar, while MTA11 was predominantly

distributed in mountainous regions. This distribution highlighted region-specific QTLs in the MIDP panel.

In Chapter IV, to validate QTLs detected by GWAS and identify unknown QTLs not found in GWAS, genetic mapping for GRH resistance was conducted using six bi-parental F_2 populations derived from six independent MIDP donors. These donors were chosen based on their haplotypes. For QTL validation, five donors carrying resistance haplotypes at either *GRH6*, *GRH1*, or MTA11 were analyzed using PCR-based markers and genotyping-by-sequencing (GBS). This analysis identified three QTLs: *qGRH4*, which overlapped with *GRH6*; *qGRH5*, which coincided with *GRH1*; and *qGRH11*, which mapped to a genomic region overlapped with MTA11 and distinct from *GRH2*. To identify unknown QTLs undetected by GWAS, genetic mapping was conducted using GBS in the F_2 population from the cross between IR24 and CC426 which did not carry resistance haplotypes at *GRH6*, *GRH1*, or MTA11. The detected *qGRH5.1* and *qGRH5.2* overlapped with *GRH1* and were derived from IR24, which carries *GRH1*. No QTLs derived from CC426 were identified in this population. This suggests that GRH resistance in CC426 might be controlled by minor QTLs, with the effect of *GRH1* potentially masking these minor QTLs. Consequently, this chapter focused on validating the three loci detected by GWAS including *GRH6*, *GRH1*, and MTA11 through QTL analysis using bi-parental mapping populations. The successful validation of GWAS-detected QTLs through bi-parental mapping demonstrates the effectiveness of haplotype-based donor selection for QTL mapping. Unknown QTLs not detected by GWAS could not be identified.

This dissertation provides a comprehensive understanding of the genetic factors affecting resistance to GRH in Myanmar landraces. By using GWAS, two major reported

genes and one novel QTL associated with GRH resistance were identified. The causal gene for *GRH6* was identified through gene cloning. Haplotype analysis estimated the accessions carrying resistance haplotypes at detected loci. The haplotype information of donor parents was crucial for validating QTLs using bi-parental populations. QTL analysis confirmed the three QTLs detected by GWAS. These findings enhance our understanding of the molecular mechanisms behind GRH resistance and offer valuable haplotype-based genetic insights for developing rice cultivars with improved GRH resistance.

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APPENDICES

Appendix 1. Primers used in gene cloning and expression analysis of *GRH6*

Primer	Forward	Reverse	PCR product size in Nipponbare (bp)	Purpose
G6-c60k	AGATGGTGTGGCCTTGAGTC	CTTCAGCGTCAAGGACAGGT	893	BAC clone screening
G6-c70k	GCAAATGCAGCCATCTAAGC	CCTCGCCACTCGATGTACTA	922	BAC clone screening
7L16f	AAATGGCTAATGCGGTTCTG	GTTGCAAATGCACGACACAT	922	BAC clone screening
F2-R1	CTGATTGGTTGCTGTTGGTC	ACGGTGTGTTTGAGAGGAAG	1041	Tandem duplication
F1-R2	CTCCATCGGCTTGTTAGTCG	ATGGTGCTGGATCTGTGTTG	183	Tandem duplication
F1-R1	CTCCATCGGCTTGTTAGTCG	ACGGTGTGTTTGAGAGGAAG	1859	Tandem duplication
GRH6oligo2	TTGAGCCTACACACCCTTG	ACCACATAGGCTTGGCAAA	120	Transgene selection
GRH6RT1	GCTGTATCAACTGCTGCTCAC	GCAAACACTCGAAACCACTGC	422	QRT-PCR
ubiquitin	CTGTCAACTGCCGCAAGAAG	GGCGAGTGACGCTCTAGTTC	358	QRT-PCR
35S_F/HPT_R	ACTATCCTTCGCAAGACCCT	GATCAGCAATCGCGCATATG	536	Transgenic plants selection

Appendix 2. *Agrobacterium*-mediated transformation in rice

1. Callus induction

Healthy and uninfected rice seeds were dehusked and sterilized with 70% ethanol and wash 2 to 3 times with sterilized distilled water. The rice seeds were vigorously shaken in a solution containing 50% sodium hypochlorite (NaClO) with 0.1% Tween 20 on a shaker for 15 min. decant the sodium hypochlorite and washed with SDW for three times until bubbles were disappeared. The procedure of rinsing in 50% NaClO without Tween 20 was repeated again, washed with SDW for three times. About 12 to 16 rice seeds were sown on N6D solid medium in a petridish and incubated at 30-33 ºC under dark condition for about 3 to 4 weeks.

2. *Agrobacterium* inoculation

Agrobacterium strains carrying the *Avr*II fragment were cultured in Lysogeny Broth (LB) solid-medium 3 days before infection. A small piece of *Agrobacterium* pellets was suspended and shaken well in 2N6-AS liquid medium. *Agrobacterium* suspension was added to a 50ml tube containing the calli and shaked slowly for exactly 90 sec for inoculation and collected with stainless wire mesh. Infected calli were first placed on sterilized filter papers to remove extra Agrobacterium solution. The calli were incubated on 2N6-AS solid medium at 25 ºC for three days.

3. selection of transformants

Three days after infection, the infected calli were washed with SDW for about 10 times to remove the *Agrobacterium* until the solution became clear. Finally, the calli were sterilized with SDW containing 12.5 mg/L of meropenem. And then placed on sterilized filter papers to remove extra SDW. The calli were cultured on N6D-S medium containing 12.5 mg/L meropenem and 50 mg/L hygromycin in order to select transformed calli, at

28-30 C under light. The calli were transferred to a new N6D-S medium for 2-3times every 10-14 days.

4. Regeneration of transgenic rice plants

The hygromycin resistant calli were transferred onto the Regeneration III medium containing 12.5 mg/L of meropenem and 50 mg/L hygromycin and cultured at 28-30 ºC. the regenerated calli that formed shoots were transferred to hormone-free medium and maintained at 28-30 ºC until sufficient growth of the transgenic rice plants had been obtained. The regenerated plants were transferred into 50 ml plastic tube and grown using Yoshida solution in the transgenic green house.

The components of various medium used for complementation analysis

Medium	Components	Amount
N6D	Sucrose	30g
	Casamino acids	0.3 g
	CHU(N6) Basal Salt Mixture	3.96 g
	Proline	2.88 g
	CHU(N6) Vitamin solution	1ml
	2,4-D	1ml
	Fill up SDW to adjust volume to	1L
	Adjust pH to 5.8	
	Gelrite	4g
	Autoclave and add to petridish	
2N6-AS	Sucrose	30g
	Casamino acids	0.3 g
	CHU(N6) Basal Salt Mixture	3.96 g
	Proline	2.88 g
	CHU(N6) Vitamin solution	1ml
	2,4-D	1ml
	Fill up SDW to adjust volume to	1L
	Adjust pH to 5.8	
	Gelrite	4g
	Autoclave and wait till temperature of solution is below about 50 ºC	
	Acetosyringone	10-20mg/l
	Streptomycin (500mg/l)	1ml
	Hygromycin (50mg/l)	1ml
	Add to petri dish	
N6D-S	Same components with 2N6-AS except Acetosyringone	
RegenerationIII	Sucrose	30g
	Sorbitol	30g
	Casamino acids	2g
	Murashige and Skoog (MS) Basal medium	4.4g
	NAA	40ul
	Kinetin	20 ml
	Fill up SDW to adjust volume to	1L
	Adjust pH to 5.8	
	Gelrite	4g
	Autoclave and wait till temperature of solution is below about 50 ºC	
	Streptomycin (500mg/l)	1ml
	Hygromycin (50mg/l)	1ml
	Add to petridish	
Hormone free	Sucrose	30g

Murashige and Skoog (MS) Basal medium	4.4g
Fill up SDW to adjust volume to	1L
Adjust pH to 5.8	
Gelrite	4g
Autoclave and wait till temperature of solution is below about 50 ºC	
Hygromycin (50mg/l)	1ml
Add to 50ml plastic tube	

Appendix 3. F_2 populations developed based on haplotype of donor parent

22GRHMYGF$_2$	Origin		Resistance Donor parent (G no.)	Resistance Donor parent (CC no.)	NM[a] of donor	Haplotype of resistance donor		
	Female	Male				*GRH6*	*GRH1*	MTA11
214	G263	T65	G263	CC310	100.0	HapGrh6A	HapGRH1B	HapMTA11B
3	G124	G143	G124	CC143	100.0	unknown	HapGRH1A	HapMTA11B
108	G120	IR24	G120	CC12	100.0	unknown	HapGRH1A	HapMTA11B
109	G124	IR24	G124	CC143	100.0	unknown	HapGRH1A	HapMTA11B
211	G231	T65	G231	CC361	100.0	unknown	HapGRH1A	HapMTA11B
325	KD18	G231	G231	CC361	100.0	unknown	HapGRH1A	HapMTA11B
2	G83	G92	G92	CC98	100.0	HapGrh6E	HapGRH1A	HapMTA11B
107	G92	IR24	G92	CC98	100.0	HapGrh6E	HapGRH1A	HapMTA11B
113	G165	IR24	G165	CC427	100.0	unknown	Unknown	HapMTA11B
114	G169	IR24	G169	CC461	100.0	HapGrh6G	HapGRH1A	HapMTA11B
115	G194	IR24	G194	CC477	100.0	unknown	HapGRH1A	HapMTA11B
117	G245	IR24	G245	CC517	100.0	HapGrh6F	HapGRH1A	HapMTA11B
118	G266	IR24	G266	CC368	100.0	unknown	HapGRH1A	HapMTA11B
202	G92	T65	G92	CC98	100.0	HapGrh6E	HapGRH1A	HapMTA11B
206	G162	T65	G162	CC417	100.0	HapGrh6F	HapGRH1A	HapMTA11B
207	G169	T65	G169	CC461	100.0	HapGrh6G	HapGRH1A	HapMTA11B
209	G203	T65	G203	CC197	100.0	unknown	HapGRH1A	HapMTA11B
218	T65	G123	G123	CC106	97.8	HapGrh6F	HapGRH1A	HapMTA11B
220	T65	G145	G145	CC240	100.0	HapGrh6G	HapGRH1A	HapMTA11B
223	T65	G165	G165	CC427	100.0	unknown	Unknown	HapMTA11B
303	G123	KD18	G123	CC106	97.8	HapGrh6F	HapGRH1A	HapMTA11B
308	G245	KD18	G245	CC517	100.0	HapGrh6F	HapGRH1A	HapMTA11B
311	G138	KD18	G138	CC184	100.0	HapGrh6F	HapGRH1A	HapMTA11B
313	G266	KD18	G266	CC368	100.0	unknown	HapGRH1A	HapMTA11B
327	KD18	G203	G203	CC197	100.0	unknown	HapGRH1A	HapMTA11B
101	G22	IR24	G22	CC379	97.5	HapGrh6E	HapGRH1B	HapMTA11A
105	G85	IR24	G85	CC68	98.0	HapGrh6D	HapGRH1B	HapMTA11A
106	G91	IR24	G91	CC96	97.8	HapGrh6F	HapGRH1B	HapMTA11A
203	G98	T65	G98	CC125	92.8	HapGrh6D	HapGRH1B	HapMTA11A
310	G5	KD18	G5	CC26	96.0	HapGrh6E	HapGRH1B	HapMTA11A
316	G64-2	KD18	G64	CC371	82.3	HapGrh6B	HapGRH1B	HapMTA11A
318	KD18	G22	G22	CC379	97.5	HapGrh6E	HapGRH1B	HapMTA11A
320	KD18	G103	G103	CC142	93.1	unknown	HapGRH1B	HapMTA11A
322	KD18	G136	G136	CC153	100.0	HapGrh6B	HapGRH1B	HapMTA11A
110	G134	IR24	G134	CC36	97.8	unknown	HapGRH1B	HapMTA11A

Appendix 3. Continued

22GRH MYGF$_2$	Origin		Resistance Donor parent (G no.)	Resistance Donor parent (CC no.)	NM[a] of donor	Haplotype of resistance donor		
	Female	Male				*GRH6*	*GRH1*	MTA11
205	G134	T65	G134	CC36	97.8	unknown	HapGRH1B	HapMTA11A
103	G76	IR24	G76	CC15	97.8	HapGrh6A	HapGRH1B	HapMTA11B
116	G216	IR24	G216	CC270	100.0	HapGrh6A	HapGRH1B	HapMTA11B
208	G200	T65	G200	CC13	100.0	HapGrh6A	HapGRH1B	HapMTA11B
212	G247	T65	G247	CC528	100.0	HapGrh6A	HapGRH1B	HapMTA11B
314	G192	KD18	G192	CC444	100.0	HapGrh6A	HapGRH1B	HapMTA11B
224	T65	G228	G228	CC331	100.0	HapGrh6A	HapGRH1A	HapMTA11B
324	KD18	G225	G228	CC331	100.0	HapGrh6A	HapGRH1A	HapMTA11B
4	G143	G148	G148	CC257	100.0	HapGrh6F	HapGRH1A	HapMTA11B
111	G148	IR24	G148	CC257	100.0	HapGrh6F	HapGRH1A	HapMTA11B
221	T65	G148	G148	CC257	100.0	HapGrh6F	HapGRH1A	HapMTA11B
312	G148	KD18	G148	CC257	100.0	HapGrh6F	HapGRH1A	HapMTA11B
326	KD18	G148	G148	CC257	100.0	HapGrh6F	HapGRH1A	HapMTA11B
1	G78	G83	G78	CC34	97.5	HapGrh6G	HapGRH1A	HapMTA11A
104	G78	IR24	G78	CC34	97.5	HapGrh6G	HapGRH1A	HapMTA11A
201	G78	T65	G78	CC34	97.5	HapGrh6G	HapGRH1A	HapMTA11A
5	G143	G228	G228	CC331	97.8	HapGrh6A	HapGRH1A	HapMTA11B
210	G220	T65	G220	CC288	100.0	HapGrh6A	HapGRH1A	HapMTA11B
102	G28	IR24	G28	CC426	87.1	HapGrh6E	HapGRH1B	HapMTA11B
213	G262	T65	G262	CC291	73.9	HapGrh6D	HapGRH1B	HapMTA11B
217	T65	G113	G113	CC242	95.0	unknown	HapGRH1B	HapMTA11B
219	T65	G127	G127	CC259	80.1	unknown	HapGRH1B	HapMTA11B
301	G142	KD18	G142	CC217	100.0	unknown	HapGRH1B	HapMTA11B
309	G2	KD18	G2	CC10	100.0	HapGrh6E	HapGRH1B	HapMTA11B
317	G199	KD18	G199	CC529	89.3	HapGrh6E	HapGRH1B	HapMTA11B
319	KD18	G28	G28	CC426	87.1	HapGrh6E	HapGRH1B	HapMTA11B
321	KD18	G127	G127	CC259	80.1	unknown	HapGRH1B	HapMTA11B
112	G156	IR24	G156	CC342	96.0	unknown	HapGRH1B	HapMTA11B
222	T65	G156	G156	CC342	96.0	unknown	HapGRH1B	HapMTA11B
323	KD18	G156	G156	CC342	96.0	unknown	HapGRH1B	HapMTA11B
119	IR24	G90	G90	CC94	85.2	unknown	HapGRH1B	HapMTA11B
215	T65	G90	G90	CC94	85.2	unknown	HapGRH1B	HapMTA11B

[a]NM represents nymph mortality (%) of GRH at evalulated at 7DAI

Appendix 4. InDel markers used in identification of QTLs conferring resistance in GRH in F_2 populations derived from Myanmar landraces

Marker name	Chr.	Position	Forward primer	Reverse primer	Product size (bp)	Purpose
Grh6Id1	4	2479682	gcaaacgatgagagaagcatg	GGAGTGAGAATGGACTGAACC		To validate MTA4
Grh6Id2	4	4440537	TGGGTTGAAGGGATTATGTTGC	ACAGTACTTGCTAGCTTGAGGT		To validate MTA4
Grh6Id4	4	4521451	CACCCTCTCTGCCTCTGTG	ACACCTCAACACCATCTCCT		To validate MTA4
MTA4Id1	4	5107426	CATGGTTCTCTGAAGTGGCA	AGCCGACGAAACAAGCAAAT		To validate MTA4
Grh6Id6	4	6567563	CTTTCTCTCTCTGTCGGCGA	ACTTCAATCCAAAGCGACCG		To validate MTA4
Grh6Id7	4	2472073	CCGACGTGACATTCCAACG	GTTAGGGGAAAATGCGAGGG		To validate MTA4
Grh6Id9	4	9939257	GACGTCCATAGAGTCGGCAA	GGATGATGAGGTCCGCGAG		To validate MTA4
Grh1Id2	5	8057416	tctgtaccgtagtgtgtagca	ctttccccatcctccccaag		To validate MTA5
Grh1Id4	5	8877648	CGGAAGCCACAAAATAGAGAAAC	acatcgaggattatatagccgga		To validate MTA5
MTA11Id1	11	27177381	CTTCATGTTTCTCGCGGTGA	TGGCACTGAAGGAAGAGATGA		To validate MTA11
MTA11Id3	11	23413916	AACCTAGTAAGCCGTGGTCC	GAGGTCGGATCCCCTTTTCA		To validate MTA11
MTA11Id5	11	21276902	GATGTTTCAAGCTGGCACCA	CTGGAGCTGAGCGGTAACT		To validate MTA11
MTA11Id6	11	24087485	GTACCGGTGATCTCAGGGAC	GTTGTTCTGGCTGTGGGTG		To validate MTA11
MTA11Id8	11	26265353	CCTTCTGCTGTTCCTTGTTGT	GCATGGAAGCAGAAGAGGAT		To validate MTA11
MTA11Id9	11	28967652	GCACTGCATCATGGATCTCC	TGGGTTATTCTCTGTCCAGTGT		To validate MTA11

Appendix 5. Genotypes of F_2 individuals derived from the cross between IR24 (resistance) and CC96 at RM1233 and RM18180

Sr. no.	F_2 plant no.	NM % at 3DAI	RM1233 (at MTA11)	RM18180 (at *GRH1*)
1	2	80.0	H	A
2	11	85.7	A	A
3	16	12.5	B	A
4	25	25.0	H	A
5	36	0.0	B	A
6	39	0.0	H	A
7	44	14.3	H	A
8	47	57.1	H	A
9	48	66.7	H	A
10	66	37.5	A	A
11	67	50.0	H	A
12	69	28.6	H	A
13	70	57.1	H	A
14	71	75.0	H	A
15	78	55.6	H	A
16	79	50.0	H	A
17	81	62.5	H	A
18	85	28.6	H	A
19	86	55.6	H	A
20	89	77.8	H	A
21	91	0.0	H	A
22	94	0.0	B	A
23	97	0.0	B	A
24	100	37.5	A	A
25	108	0.0	B	A
26	110	0.0	H	A
27	118	75.0	H	A
28	120	60.0	H	A
29	129	44.4	A	A
30	134	22.2	H	A
31	141	81.8	H	A
32	149	50.0	A	A
33	150	75.0	H	A
34	152	50.0	A	A
35	154	77.8	H	A
36	156	20.0	H	A
37	157	60.0	A	A
38	161	0.0	B	A
39	167	33.3	H	A
40	168	28.6	B	A
41	176	0.0	B	A
42	178	11.1	H	A
43	181	28.6	H	A
44	186	37.5	H	A

Appendix 5. Continued

Sr. no.	F_2 plant no.	NM % at 3DAI	RM1233 (at MTA11)	RM18180 (at *GRH1*)
45	193	71.4	H	A
46	195	37.5	H	A
47	198	50.0	H	A
48	199	0.0	B	A
49	204	60.0	H	A
50	209	0.0	B	A
51	211	66.7	H	A
52	224	62.5	A	A
53	237	33.3	H	A
54	252	62.5	A	A
55	261	0.0	B	A
56	266	30.0	H	A
57	267	87.5	H	A
58	271	85.7	H	A
59	290	55.6	H	A
60	292	66.7	A	A
61	297	50.0	H	A
62	298	0.0	H	A
63	301	10.0	B	A
64	306	0.0	B	A
65	319	60.0	H	A
66	332	83.3	H	A
67	347	0.0	B	A
68	355	0.0	B	A
69	357	33.3	H	A
70	361	80.0	H	A
71	371	0.0	B	A
72	372	88.9	H	A
73	374	50.0	H	A
74	379	81.8	H	A
75	380	0.0	B	A
76	388	85.7	A	A
77	392	50.0	H	A
78	397	87.5	A	A

A represented CC96 homozygous.
B represented IR24 homozygous.
H represented heterozygous