



OVERLAP IN THE GENETIC BASIS OF HOST BASAL AND NONHOST RESISTANCES OF BARLEY TO LEAF RUSTS

Dido, A.A.¹, Yeo, F.K.S.², Marcel, T.C.³, Niks, R.E.²

¹ Oromia Agricultural Research Institute, Adami Tullu Agricultural Research Center, Plant Biotechnology Research Team, P. O. Box 35, Batu (Ziway), Oromia, Ethiopia. E-mail: alloaman2010@gmail.com

² Department of Plant Breeding, Graduate school for Experimental Plant Sciences, Wageningen University, Droevendaalsesteeg 1, 6708 PB Wageningen, The Netherlands

²INRA-AgroParisTech, UMR1290 BIOGER-CPP, Avenue Lucien Brétignières BP01, 78850 Thiverval-Grignon, France



ABSTRACT

Barley host basal resistance (partial resistance) and nonhost basal resistance (nonhost resistance) to leaf rusts are based on prehaustorial mechanism associated with papillae formation. Both are mainly governed by genes with relatively small, quantitative effects, located on quantitative trait loci (QTL). The genes for host basal resistance seem to play similar roles in basal resistance as those governing nonhost basal resistances. We presume that these two resistance types are based on shared principles. Quantitative trait loci-near isogenic lines (QTL-NILs) were developed for basal resistance QTLs of our interest using SusPtrit as recurrent parent. SusPtrit is an experimental line which is exceptionally susceptible to leaf rusts for which normally barley is a nonhost. Three host basal resistance QTL-NILs and a nonhost basal resistance QTL-NIL were used in this study. They were challenged with one homologous and three heterologous leaf rusts. The result showed that, the 3 QTLs for host basal resistance and the QTL for nonhost basal resistance have a significant effect on both homologous and heterologous rusts. This gives an indication that indeed, host and nonhost basal resistance are associated.

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Introduction

Barley host basal resistance (alias partial resistance) and nonhost resistance to leaf rusts are based on a prehaustorial mechanism associated with papilla formation at sites of cell wall penetration attempt. Both types of resistance are typically governed by genes with relatively small, quantitative effects, located on quantitative trait loci (QTL). Jafary *et al.* (2006 and 2008) reported that the QTLs for host basal resistance and nonhost resistance of barley tended to co-locate on a consensus map of barley. Based on the similarity in resistance mechanism and the co-localisation of QTLs for host basal resistance and nonhost resistance, we presume that these two resistance types are based on shared principles.

Five QTL-near isogenic lines (QTL-NILs) were developed by introgressing four host basal resistance QTLs and a nonhost resistance QTL into SusPtrit genetic background. SusPtrit is an experimental line which not only



susceptible to the homologous rust, *P. hordei*, but also exceptionally susceptible to leaf rusts for which normally barley is a nonhost (Figure 1). The QTL-NILs developed allow us to study the possible association between the QTLs for host basal resistance and nonhost resistance.

Crop/Variety	<i>Puccinia hordei</i>	<i>Puccinia triticina</i>
	host	Non-host
Barley-Vada		
Barley-SusPtrit		
Wheat-8860		

Figure 1. Susceptibility of SusPtrit to homologous rust *P. hordei* (barley is a host) and heterologous rust *P. triticina* (barley is a nonhost) at seedling stage (Atienza *et.al*, 2004).

Host basal resistance QTLs and a nonhost resistance QTL confer longer latency period for *P. hordei*, a homologous rust

P. hordei had a longer latency period on QTL-NILs introgressed respectively with, host basal resistance QTLs *Rphq2*, *Rphq3*, *Rphq11*, *Rphq16*, and nonhost resistance QTL, *Rnhq.v* compared to the control SusPtrit. The host basal resistance QTLs prolonged the latency period more than the nonhost resistance QTL (Figure 2).

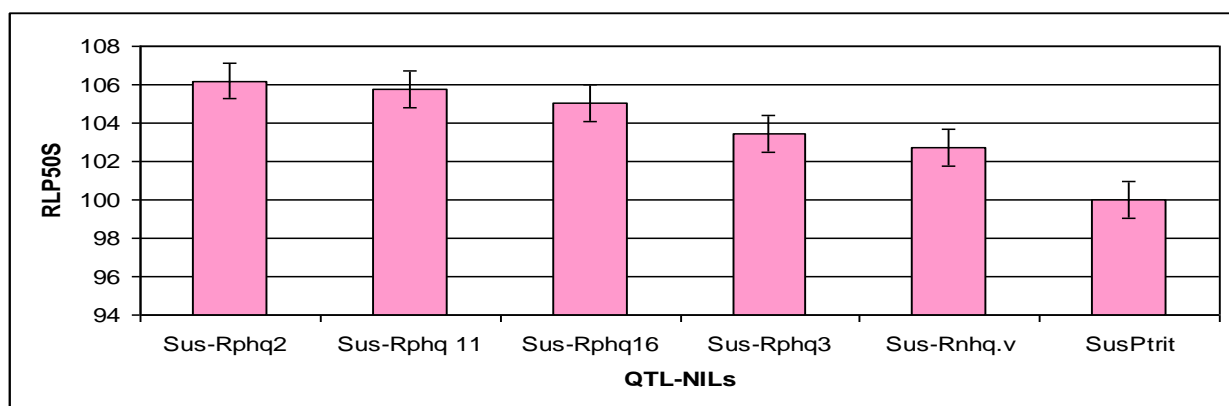


Figure 2. The relative latency period of *P. hordei* (RLP50S) on the QTL-NILs introgressed with host basal resistance QTLs *Rphq2*, *Rphq3*, *Rphq11*, *Rphq16* and nonhost resistance QTL, *Rnhq.v*, respectively.



Host basal resistance QTLs and a nonhost resistance QTL confer higher levels of prehaustorial resistance to heterologous rusts

Both host basal resistance (*Rphq2*, *Rphq3*, *Rphq11*, *Rphq16*) and nonhost resistance (*Rnhq.v*) QTLs significantly reduced the infection frequency of heterologous rusts *P. hordei-murini* (*Phm*), *P. hordei-secalini* (*Phs*) and *P. triticina* (*Pt*) (Figure 3a). Histology demonstrated a significantly higher percentage of early aborted colonies and smaller size of established colonies on all QTL-NILs compared to SusPtrit (Figure 3b and 3c). These results suggest that less colonies are established on QTL-NILs and that the growth of the established colonies is restricted on the QTL-NILs. All the QTLs of both host basal and nonhost resistances had higher effect against *Phs* and *Phm* than against *Pt*.

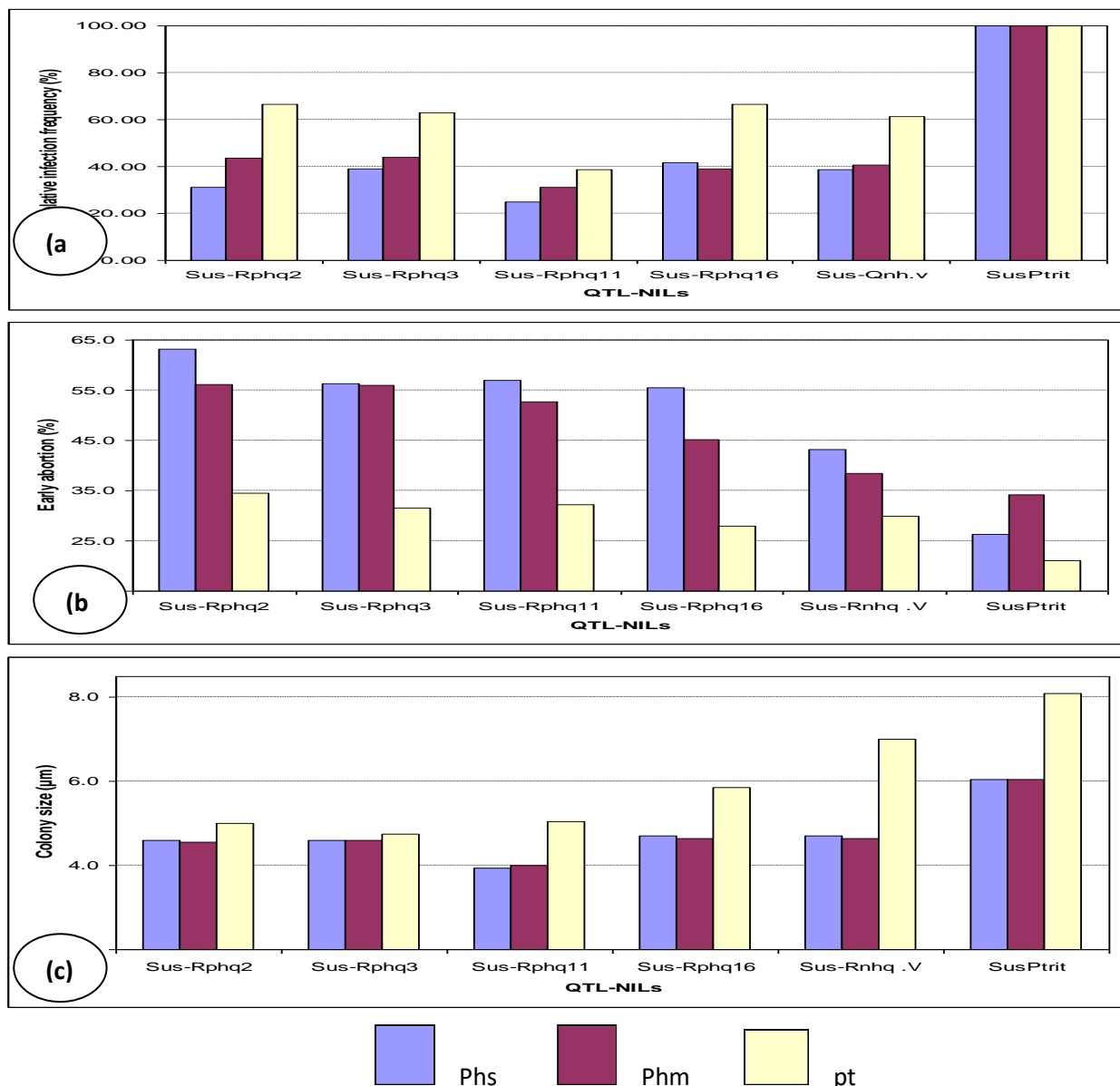


Figure 3. The effect of host basal resistance QTLs *kpnq2*, *kpnq3*, *kpnq11*, *kpnq16* and nonhost resistance QTL, *Rnhq.v*, respectively on heterologous rusts, *P. hordei murini*, *P. hordei secalini* and *P. triticina*. (a) The infection frequency of the heterologous rusts on the QTL-NILs in relative to SusPtrit. (b) The percentage of early aborted



colonies on QTL-NILs and SusPtrit. (c) The colony size of established colonies with sporogenic tissues on QTL-NILs and SusPtrit.

Conclusion

The present results suggests that most barley genes conferring a basal resistance level to the homologous rust *P. hordei* may also confer resistance to heterologous rusts that normally do not infect barley. To our knowledge, it is the first time that such a clear overlap in the genes involved in the natural variation of host basal and nonhost resistances is obtained. Those results will have important implications in the development of strategies to achieve durable resistance against such fungal pathogens.

Laboratory of plant breeding (Group: nonhost and insect resistance)

Most plant species are completely resistant to almost all potential pathogen species. This is due to the narrow specialization of most plant pathogens: by far most pathogens have only a limited host range. Recent advances in molecular biology of plant pathogen interactions suggest that adapted pathogens are able to suppress the basal defence of their host plants, but not in, sometimes related, nonhost plant species. It is not known which plant genes are responsible for allowing a potential pathogen to succeed (or not) in suppression of this basal defence. For plant breeding it is useful to understand the molecular basis of (non) host status of plants to potential pathogens. When understood, it might be possible to mimic nonhost resistance in a host plant species somehow, creating a pseudo-nonhost resistance in a host species. Such a resistance would be complete and hard to overcome by the pathogen. In our group, we have developed three research systems to investigate the inheritance underlying (non)host status to biotrophic specialized pathogens: barley/*Puccinia* rusts and powdery mildews and Lettuce/*Bremia lactucae*

Acknowledgements

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