EVALUATION OF PARTIAL RESISTANCE TO CROWN RUST OF OAT CONFERRED BY MN841801 IN TWO RECOMBINANT INBRED POPULATIONS

M. Acevedo[1]*, E. Jackson[1], J. Chong[2], H.W. Rines[3], Stephen A. Harrison[4], A. Bateman[1], J.M. Bonman[1]

[1] Small Grains and Potato Germplasm Research Unit, USDA-ARS; Aberdeen, ID 83210, USA

[2] Cereal Research Centre, Agric. & Agri-Food Canada, Winnipeg, MB, Canada R3T 2M9

[3] USDA-ARS, Department of Agronomy and Plant Genetics, University of Minnesota; St. Paul, MN 55108, USA

[4] School of Plant, Environmental and Soil Sciences, Louisiana State University; Baton Rouge, LA

*Corresponding author: Maricelis.Acevedo@ars.usda.gov

Introduction

Crown rust, caused by *Puccinia coronata,* is probably the most destructive disease of oat worldwide. Many simply inherited, race- specific resistance genes (major genes) are available and have been used extensively in cultivated oats, but their effectiveness has been short lived. Genetically complex resistance that allows some pathogen reproduction could prove more durable. The oat line MN841801 has been proposed as an "excellent source of…durable adult plant resistance" (Leonard, 2002). The inheritance of resistance in MN841801-1 was previously described via multiple year-location testing as being complex (7 QTL's) (Portyanko, et al., 2005) whereas that of MN841801 was relatively simple (2 genes for adult plant resistance with additive effects) (Chong, 2000). The objectives of this study were: 1) compare the resistance of recombinant inbred (RI) lines from mapping populations generated from MN841801-1 and MN841801 and 2) to map the resistance in MN841801-1 using single-isolate evaluations and three measures of disease: relative fungal DNA (RFDNA) content, diseased leaf area (DLA), and infection type (IT).

Materials and Methods

Oat populations. Partial resistance to crown rust was evaluated in two RI populations, MN841801-1 X Noble-2 and Makuru X MN841801. Two isolates (CR251 and 93MNB236) virulent on all four parents at seedling stage were used in field and greenhouse experiments at Aberdeen, ID. The isolates produced flecks and small pustules on the MN841801 and MN841801-1 on adult plants. Both populations were also evaluated at Baton Rouge, LA in naturally infected field plots.

Fungal DNA (FDNA) quantification. In the Aberdeen experiments RFDNA content was estimated using a multiplex reaction containing two labeled probes, one for the pathogen and one for the host (β-actin probe for oats), and two different primer sets (Fig.1). RFDNA was estimated based on the Cycle Treshold (CT) values for each sample. The RFDNA content for each sample was calculated as $2^{(CTh- CTp)}$ where CTh is the CT value for the host DNA and CTp is the CT value for the pathogen DNA. The mean RFDNA was calculated for each field replicate and genotype.

QTL analysis and mapping. A genetic linkage map was constructed for the MN841801-1 X Noble-2 population using MapManager QTxb20 using previously published genetic data (Portyanko et al., 2005).

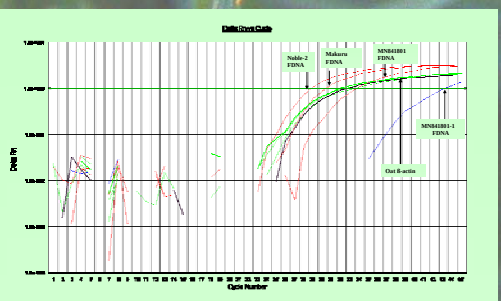

Fig. 1 Amplification of fungal DNA and plant DNA for RFDNA quantification of oat lines used as parents for the RI populations

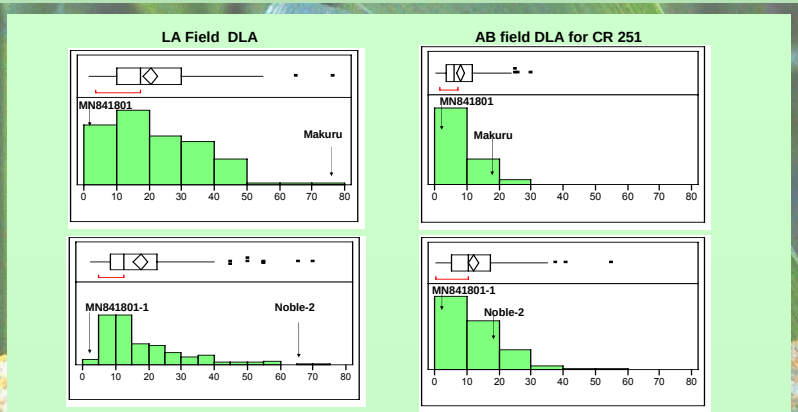

Fig. 2 Frequency distribution of leaf diseased area (%) caused by crown rust for the recombinant inbred lines of the crosses Makuru X Mn841801 and MN841801-1 X Noble-2 under natural disease pressure (LA) and artificially produced single isolate infection (AB).

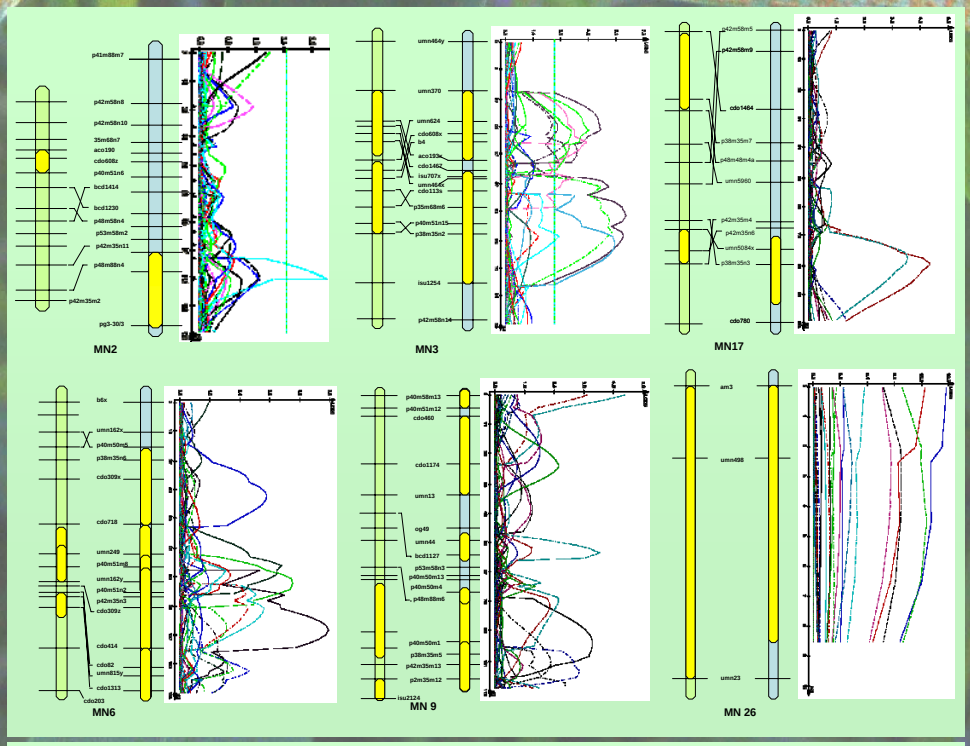

Fig. 3 Linkage groups map of QTLs for partial resistance to crown rust on the MN841801-1 X Noble-2 population (blue). Map based on phenotypic reaction and RFDNA content of 150 F6:8 RILs. Maps in green represent the homologous areas based on previously publish data (Portyanko, 2005). Yellow regions represent support intervals for the QTLs detected.

Table.1 Quantitative trait locus (QTL) analysis summary for crown rust resistance based on mean various assessments of MN841801 X Noble-2 recombinant inbred (RI) lines with different *Puccinia coronata* isolates over greenhouse (GH) and field (F) experiments.

Linkage group	Experiment (Trait/Race/Environment)	QTL marker[a](peak/interval)	LOD[b]	R^2 (%)[c]	Add.[d]
MN2					
	FDNA/CR251/F	p42m35n11(110.9/101.0-112.0)	3.6	10.8	5.70
MN3					
	DLA/CR251/AbF	cdo608x (29.2/27.0-40.0)	3.3	7.1	-0.04
	FDNA/CR251/AbF	cdo1467 (45.1/44.0-46.0)	3.4	7.6	-0.18
	DLA/MN236/AbF	umn37 (29.2/19.0-33.0)	4.8	13.8	-3.00
	FDNA/MN236/AbF	umn37 (33.1/18.0-37.0)	4.1	11.2	-10.80
	DLA/MN236/AbGH	p35m68m6 (64.5/61.0-68.0)	2.7	7.1	-3.10
	DLA-Flag/LSU/LaF	umn37 (33.8/21.0-42.0)	3.0	7.7	-19.40
	DLA-Leaf/LSU/LaF	cdo113x (63.0/48.0-68.0)	5.4	14.1	-33.80
	Composite/LSU/LaF	cdo113x (57.5/51.0-73.0)	6.1	16.3	-0.57
	HD/AbF	p35m68m6 (66.4/61.0-73.0)	5.6	18.8	0.96
MN17					
	DLA/MN236/AbGH	umn508x (82.1/77.5-90.0)	4.4	10.2	-0.02
MN6					
	DLA/MN236/AbGH	p40m51n2 (94.7/82.0-105.0)	3.4	7.8	-3.30
	FDNA/MN236/AbGH	cdo414 (123.4/120.0-127.0)	2.8	6.7	-0.11
	IT/MN236/AbF	umn249 (74.9/68.0-82.0)	4.1	10.9	-0.22
	IT/CR251/AbF	cdo309x (42.8/36.0-53.0)	3.5	9.1	-0.23
	Composite/LSU/LaF	cdo309z (104.7/95.0-113.0)	5.9	23.9	-0.70
	IT/LSU/LaF	p40m51m8 (83.0/76.0-90.0)	4.5	12.0	-0.27
MN9					
	DLA/CR251/AbGH	og49 (59.0/54.0-64.0)	4.3	10.7	1.03
	DLA/MN236/AbGH	p40m58m13 (0.01/0.0-4.0)	3.4	7.8	-3.30
	FDNA/MN236/AbGH	p40m50m1 (104.4/95.0-112.0)	3.4	8.8	-0.12
	DLA/LSU-Flag/LaF	p2m35m12 (109.0/104.0-112.0)	2.9	9.6	-21.3
	IT/LSU-Flag/LaF	cdo460 (27.0/16.5-34.0)	2.6	6.6	-0.19
MN26					
	DLA/CR251/AbF	am3 (2.01/0.0-8.5)	12.8	31.4	-0.07
	FDNA/CR251/AbF	am3 (2.51/0.0-8.5)	9.2	23.1	-0.31
	DLA/CR251/AbGH	am3 (0.01/0.0-8.5)	13.3	29.6	-0.03
	FDNA/CR251/AbGH	am3 (2.01/0.0-8.5)	3.7	9.4	-0.13
	FDNA/MN236/AbGH	am3 (2.01/0.0-8.5)	3.2	8.8	-0.09
	DLA-Leaf/LSU/LaF	am3 (0.01/0.0-8.5)	2.8	6.4	-23.40
	IT/CR251/AbF	am3 (0.01/0.0-8.5)	15.8	33.9	-0.45

[a] Name of the flanking marker to the left of the QTL peak and interval (cM)
[b] QTL were detected using WinQTL Cartographer CIM and were based on a LOD threshold of 2.70 (1,000 permutations and a type I error of 5%).
[c] Percent of the phenotypic variation explained by the QTL
[d] Additive effect

Results and Discussion

The RI line distributions for both populations as measured by DLA were continuous and skewed towards the resistant parent (Fig. 2). Two QTLs, one on linkage group MN3 and one on MN26, were detected in both single isolate trials in Aberdeen and in the field test in LA (Fig. 3). In addition, four other QTLs associated with MN841801-1 alleles on linkage group MN2, 6, 9, and 17 were detected in single-isolate trials. Five of the six QTLs were identified with single-isolate testing as measured by RFDNA and DLA, whereas IT only identified two of the six QTLs. All QTLs detected in this study were comparable to QTLs found in the previous crown rust evaluation of the MN841801-1 X Noble-2 population in Minnesota. Thus, single-isolate experiments can be used to identify QTLs for resistance similar to those obtained by multiple year-location experiments. However, our results indicate major QTLs on two linkage groups (MN3 and MN26) accounting for most of the phenotypic variance in all environments. Our next step will be to test markers associated with the QTLs identified in the MN841801-1 X Noble-2 population for significance on the Makuru X MN841801 population.

Literature Cited

•Chong J. 2000. Acta Phytopathologica et Entom. Hungarica 35 (1-4) 37-40.
•Leonard, K.J. 2002. Plant Dis. 86:593-598.
•Portyanko V.A., G. Chen , H.W. Rines, R.L. Philips, K.J .Leonard, G.E. Ochocki, D.D. Stuthman. 2005. Theor Appl Genet 111:313-324.