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# Locus Interactions Underlie Seed Yield In Soybeans Resistant to *Heterodera glycines*

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## Abstract

In soybean (*Glycine max* L. Merr.) combining resistance to cyst nematode (SCN; *Heterodera glycines* L.) with high seed yield remains problematic. Molecular markers linked to quantitative trait loci (QTL) have not provided a solution. Sets of markers describing a collection of favorable alleles (linkats) may assist plant breeders seeking to combine both traits. The objective of this analysis was to identify linkats in genomic regions underlying seed yield and root SCN resistance QTL. Used were groups of cultivars selected from a single recombinant inbred (RIL) population derived from 'Essex' by 'Forrest' (ExF). The yield was measured at four locations. SCN resistance was determined in greenhouse assays. The mean seed yield was used to define 3 groups (each n=30), high, medium and low. SCN resistance formed 2 groups (SCN resistant (n=21) and SCN susceptible (n=69)). Microsatellite markers (213) alleles were compared with seed yield and root SCN (*Heterodera glycines*) resistance using mean analysis. The number, size and position of

**Abbreviations:** ExF 'Essex' by 'Forrest'. Linkats, a collection of favorable alleles linked in attraction (linkats). RIL, recombinant inbred population. QTL, quantitative trait loci. SCN, soybean cyst nematode.

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potential linkats were determined. Loci, genomic regions and linkats associated with seed yield were identified on linkage group (LG) K and with root resistance to SCN e on LG E, G, and D1b+W. A method to identify co-localized genomic regions is presented.

## Introduction

Soybean [*Glycine max* (L.) Merr.] is one of the world's major crops (Singh and Hymowitz, 1999) grown commercially for its oil (16%) and protein content (42%). Seed yield has doubled over the last 30 years largely by selection of superior adapted cultivars (Specht *et al.*, 2001). However, in the same period soybean has proven susceptible to many diseases that reduce yields. Breeders have struggled to combine high seed yield with resistance to soybean cyst nematode (SCN; caused by *Heterodera glycines* L.) due to linkage drag and other unknown factors (Mudge *et al.*, 2001; Yuan *et al.*, 2002; Concibido *et al.*, 2004; Kopisch-Obuch *et al.*, 2005). In contrast high seed yield has been readily combined with resistance to diseases like soybean sudden death syndrome (SDS; Hnetkovsky *et al.*, 1996; Meksem *et al.*, 1999; Prabhu *et al.*, 1999; Iqbal *et al.*, 2001).

In soybean and other plant species, the majority of economically important agronomic traits like seed yield are controlled in a quantitative fashion (Mansur *et al.*, 1993, 1996). Molecular markers have been used extensively to construct genetic and linkage maps (Song *et al.*, 2004; Zhang *et al.*, 2004; Kassem *et al.*, 2006) and to identify quantitative trait loci (QTL) underlying valuable traits (Yuan *et al.*, 2002; Kopisch-Obuch *et al.*, 2005; Kassem *et al.*, 2006, 2007a,b). However, simple selection of loci for SCN resistance combined with high seed yield has not eliminated the negative relationship between the traits when cyst pressure is low.

Possible explanations for the negative relationship between resistance to SCN and seed yield are sought. One possibility is that introgression of the genes for resistance to SCN has disrupted linkats, a collection of favorable alleles that are linked (Demarly, 1979). In SCN susceptible soybean cultivars large regions where recombination rarely occurs have been detected (Lorenzen *et al.*, 1996; Stefaniak *et al.*, 2005). However, the cultivars with the highest seed yield tend to have more than the mean number of recombination events on several linkage groups (C2, L and M). A second possibility is that the genes for resistance to SCN are themselves unfavorable to seed yield so that selection of new linkats at other genomic locations are needed to ameliorate those negative effects (Lark *et al.*, 1995; Yuan *et al.*, 2002). Indeed there is strong evidence that the resistance allele of major gene for resistance to SCN (*rhg1*) is rigidly co-inherited with an allele on linkage group M, because embryo or pollen in-viability results from breaking this association (Webb *et al.*, 1996). Even

when those alleles are co-inherited negative effects on of *rhg1* on plant growth and development can be observed (Yuan *et al.*, 2002; Kopisch-Obuch *et al.*, 2005; Ruben *et al.*, 2006). Both the *rhg1* and *Rhg4* loci contain several candidate genes, but both include receptor-like kinases that appear likely to play a role in root development (Afzal and Lightfoot, 2007).

Recent studies on analysis of soybean genomes (Obermayer and Greilhuber, 1999; Brown-Guedira *et al.*, 2000; Grant *et al.*, 2000; Shoemaker *et al.*, 1996, 2002; Alkharouf and Mathews, 2004; Shultz *et al.*, 2006) showed that the genome duplicated twice and regions of conserved homeology remain (Shoemaker *et al.*, 1996; Grant *et al.*, 2000; Blanc and Wolfe, 2004; Choi *et al.*, 2004; Shultz *et al.*, 2007; Schlueter *et al.*, 2007). The major genes for resistance to SCN on linkage groups A2 (*Rhg4*) and G (*rhg1*) both have 2 or more homeologous regions where conserved synteny is observed (Campbell *et al.*, 2007; Afzal and Lightfoot, 2007). Therefore, it is likely that regions separate from the loci underlying seed yield and resistance to SCN can be affecting those trait but would escape detection by normal method of QTL map development and locus based detection of epistasis.

The objective of this study was to identify interactions between linkats and loci underlying seed yield and SCN resistance. The analysis identified significant patterns of association between alleles at 213 microsatellite DNA markers, soybean genome structures and mean yield. Prediction was made of the genome structure capable of combining yield with disease resistance.

## Materials and methods

### Genetic material

The genotypes used in this study were the ExF96 population described by Lightfoot *et al.*, 2006. The marker data used was the 213 microsatellite markers from Kassem *et al.*, (2006). Markers were arranged in map order. Trait data were as described in Yuan *et al.*, 2002 for seed yield and in Kassem *et al.*, 2006 for resistance to SCN Hg Type 0.

### Data classification

The groups were selected by ranking the genotypes for yield and disease resistance analysis. For seed yield two equal groups of 30 genotypes with lowest yield and highest yield were used. For disease resistance two groups were defined female index reported for five plants challenged with 2000 eggs of SCN Hg type 0 (race3; Yuan *et al.*, 2002). The two groups are resistant ( $FI < 10$ ) and susceptible ( $FI > 10$ ) with 21 and 69 genotypes respectively.

### Statistical analysis

The ranked genotypes were used to count the number of alleles corresponding to each marker in map order. Allelic chains (multiple As or Bs) and recombination events (A juxtaposed to B) were counted. The means for each genotype corresponding to A, AA, AAA, AAAAA, AAAAA, B, BB, BBB, BBBB, BBBB, BA, AB, values were calculated. The 12 numbers obtained were then subjected to mean

analysis to determine whether phenotype groups differed significantly. The number of allelic chains on each of the chromosome of the genomic structure were calculated and compared.

The mean was the sum of all the scores divided by the number of scores. The formula in summation notation is  $\mu = \Sigma X / N$ , where  $\mu$  is the population mean and N is the number of scores. Since the scores were from a sample, then the symbol M refers to the mean and N refers to the sample size. The formula for M is the same as the formula for  $\mu$ . The mean is a good measure of central tendency for roughly symmetric distributions but can be misleading in skewed distributions since it can be greatly influenced by extreme scores. Therefore, distributions were tested for normality.

## Results and discussion

### Marker allele segregation

The data set used was identical to that reported in Kassem *et al.*, (2006). There were 12, 958 Forrest alleles and 12, 823 Essex alleles at all markers in the data set. That difference was not significant. Further Chi square showed no significant deviations for the expected 1:1 segregation ratio at any of the 240 markers used (heterogeneous scores were excluded). Therefore, segregation was not biased at any locus or across the whole data set. Recombination events showed evidence for non random distribution across linkage groups whether measure directly (not shown) or as linkats of Essex alleles (Fig. 1) or Forrest alleles (Fig. 2). Linkat and recombination frequencies were significantly different in some genotype groups and some linkage groups (see below).

### Correlation among traits

SCN resistance and seed yield in non-SCN infested locations were not negatively correlated ( $r = 0.12$ ,  $p = 0.1$ ; Supplemental Fig. 1). The negative correlation may have been lost due to a low but significant yield loss to SCN in environments considered non-infested. Cysts were present but at less than 10 cyst per 100 cm<sup>3</sup> of rooting soil.

### Disease resistance

Linkats were found on linkage groups (LG) A2, E, D1b and G that were significantly different between the SCN resistant and susceptible phenotype groups ( $n = 21$  and  $n = 69$ ) compared to the overall population distribution. There was a significant increase in the number and length of chains of Forrest alleles associated with the SCN susceptible group at LG E ( $p = 0.0001$ ) and D1b ( $p = 0.01$ ; Supplemental Table 1). Conversely, there was a significant increase in the number and length of chains of Essex alleles associated with the SCN susceptible group at LG A2 ( $p = 0.045$ ) and G ( $p = 0.009$ ) and a significant decrease on LG E ( $p = 0.0005$ ) and D1b ( $p = 0.05$ ; Supplemental Table 2). Equally recombination events were depressed among the SCN resistant lines at these LGs. Compared to the population as a whole there was a significant increase in the number of recombination event at LG G and concomitant decrease in the numbers and lengths of chains of Forrest alleles associated with the

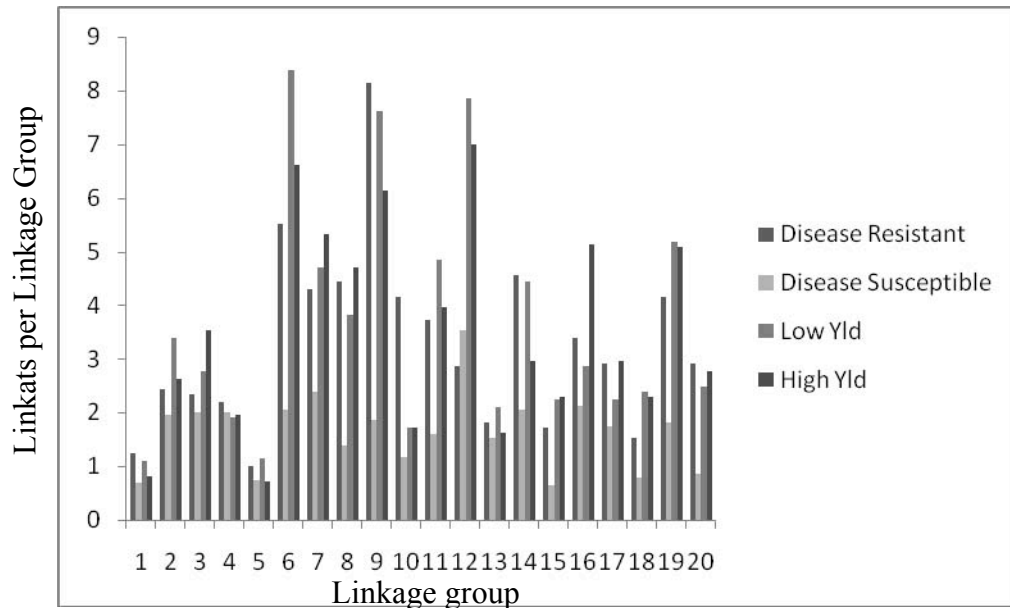


Fig. 1. Linkat distribution of Essex alleles across the 20 linkage groups of soybean within the genotype groups for high yield, low yield, resistance to SCN and susceptibility to SCN. Chromosomes are numbered 1 to 20.

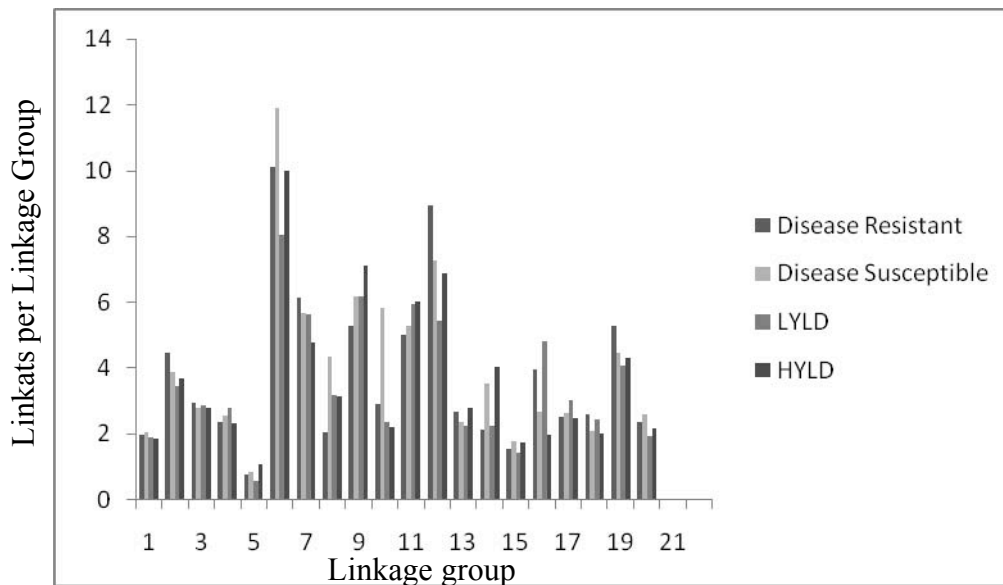


Fig. 2. Linkat distribution of Forrest alleles across the 20 linkage groups of soybean within the genotype groups for high yield, low yield, resistance to SCN and susceptibility to SCN. Chromosomes are numbered 1 to 20.

SCN resistant set. Considering only long linkats, those of more than 3 alleles, there was a significant decrease in the number and length of chains of Essex allele's as among the SCN susceptible lines on LGs A2 ( $p=0.015$ ), G ( $p=0.0067$ ), and M ( $p=0.029$ ). Long linkats of Forrest alleles were found in the disease susceptible group on LG D1b ( $p=0.012$ ) and LG E ( $p=0.0002$ ). Overall linkats and recombination events frequencies appeared to be similar in frequency across all LG of the two phenotypic sets but not at the particular linkage groups associated with *Rhg* genes (A2, G and M) present in Forrest and two linkage groups not known to have effects on resistance to SCN HG Type 0 (E and D1b).

#### Seed yield

Linkats were found on LG I and K that were significantly different between the high yield and low yield phenotypic groups ( $n=30$ ). There was a significant increase ( $p=0.0098$ ) in the number and length of chains of Essex alleles associated with the high yield group at LG K but a decrease on LG I ( $p=0.03$ ; Supplemental Table 3). Conversely, LG I showed a significant increase ( $p=0.002$ ) in the number and length of linkats of Forrest alleles and a significant decrease on LG K ( $p=0.00003$ ; Supplemental Table 4). Longer linkats of 3 or more alleles showed a similar pattern with a significant increase ( $p=0.0026$ ) in the number and length of chains of Essex

alleles associated with the high yield group at LG K but a decrease on LG I ( $p=0.018$ ). Also in the high yield group long linkats significantly increased on LG C1 ( $p=0.026$ ) and significantly decreased LG L ( $p=0.03$ ). Conversely, LG I showed a significant increase ( $p=0.002$ ) in the number and length of linkats of Forrest alleles and a significant decrease on LG K ( $p=0.0003$ ) for both mean and long linkats. Equally recombination events were depressed among both the high and low resistant lines at these LGs. Overall linkats and recombination events frequencies appeared similar in frequency across all LG of the two phenotypic sets except particular LG I and K previously associated with seed yield.

### Discussion

Genome architecture associated with yield was detected in the ExF population. Most impressively the markers revealed a very strong linkat to QTL association between yield on genetic linkage group K and I. Both LGs were shown to contain QTL for seed yield (Yuan *et al.*, 2002; Kassem *et al.*, 2007a). In Williams by Essex there were more recombination events and smaller linkats on LG K among high yielding cultivars compared to the population as a whole (Stephaniak *et al.*, 2005). Linkage group I did not differ in linkat size or recombination events in that study. The beneficial allele for the LG K QTL was from Essex (Yuan *et al.*, 2002; Kassem *et al.*, 2006; 2007a) and so the association with long linkats of Essex alleles may indicate a locus with several underlying genes distributed over a large region of the genome. Equally the beneficial allele for the LG I QTL was from Forrest (Yuan *et al.*, 2002; Kassem *et al.*, 2007a) and so the association with long linkats of Forrest alleles may indicate a locus with several underlying genes distributed over a large region of the genome. Several other yield QTL were detected on LG A2, C1, C2, J, and N but they were not associated with linkats or recombination frequency variation (Lark *et al.*, 1995; Kassem *et al.*, 2006; Guzman *et al.*, 2007). Such loci might be underlain by single genes, small gene clusters or sets of epistatic loci.

Genome architecture was associated with SCN resistance but not only at loci known to encompass QTL (LG A2, G and M; Meksem *et al.*, 2001; Webb *et al.*, 1995). Linkats of Forrest alleles detected on linkage groups E and D1b in the SCN susceptible group and their absence from the SCN resistant group might be related to selection for loci that ameliorate the effect of the resistance loci, like that on LG M (Webb *et al.*, 1995). Equally the decrease in the number and length of chains of Essex alleles associated with the SCN resistant group around the QTL bearing regions of LG A2 and G might indicate the loci are multigenic. However, over the entire genome linkats of Forrest alleles were not more abundant than expected.

The low incidence of polymorphism between Essex and Forrest resulted in a paucity of markers in some regions (C1, J). Gaps between markers in individual maps undoubtedly caused important linkats to be missed. However, marker density was quite high on most of the LG reported to contain linkats here (A2, C2, G, I, K). With continued expansion of soybean yield QTL studies; additional markers need to be tested in the gaps (Shultz

*et al.*, 2007). Further studies can be made by testing the same QTL in a different population and re-examining yield QTL identified without examining the effect of linkats (Orf *et al.*, 1999a; 1999b; Yuan *et al.*, 2002; Kabelka *et al.*, 2004; Smalley *et al.*, 2004; Guzman *et al.*, 2007).

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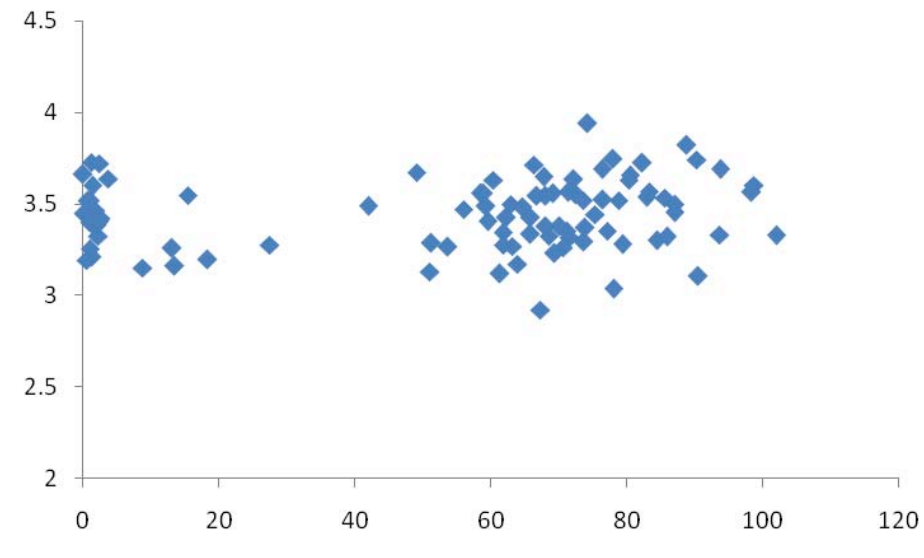
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### Supplementary Material



Supplemental Fig. 1. Correlation between mean seed yield in non-infested locations and resistance to SCN Hg Type 0.

Locus Interactions Underlie Seed Yield In Soybeans Resistant to *Heterodera glycines* i79

Supplemental Table 1. Linkats of Forrest alleles in SCN resistant and susceptible groups.

	Disease Resistant	Disease Susceptible	Ratio	Probability	Linkage Groups
B	2.9048	3	0.96825	0.422409	A1
	6.4286	5.6087	1.1462	0.18244	A2
	5.2857	5.0435	1.048	0.34566	B1
	4.1429	4.3913	0.94342	0.318569	B2
	1.381	1.6957	0.81441	0.151028	<b>C1</b>
	12.81	13.957	0.91782	0.256559	<b>C2</b>
	8.2857	8.0435	1.0301	0.843459	<b>D1a</b>
	3.9524	5.7391	0.68867	0.03494	<b>D1b</b>
	7.5238	8.0435	0.93539	0.363385	D2
	4.3333	7.0435	0.61523	0.000105	<b>E</b>
	6.619	6.913	0.95747	0.382829	F
	13.19	10.696	1.2333	0.029528	<b>G</b>
	3.9524	4.087	0.96707	0.39344	H
	4.1905	5.5217	0.75891	0.044905	I
	2.5238	2.4348	1.0366	0.434052	J
	5.6667	4.5217	1.2532	0.110741	K
	4.381	4.6522	0.9417	0.325899	L
	3.4762	2.913	1.1933	0.157233	M
	7.0952	6.3913	1.1101	0.258952	N
	3.3333	3.8261	0.87121	0.278682	O
BB	2	2.0435	0.97872	0.469067	A1
	4.4762	3.8696	1.1568	0.207282	A2
	2.9524	2.7826	1.061	0.399137	B1
	2.381	2.5652	0.92817	0.384325	B2
	0.7619	0.86957	0.87619	0.373186	<b>C1</b>
	10.095	11.913	0.84741	0.1666	<b>C2</b>
	6.1429	5.6957	1.0785	0.361802	<b>D1a</b>
	2.0476	4.3478	0.47095	0.01442	<b>D1b</b>
	5.2857	6.1739	0.85614	0.286717	D2
	2.9048	5.8261	0.49858	9.52E-05	<b>E</b>
	5	5.3043	0.94262	0.379111	F
	8.9524	7.2609	1.233	0.123539	<b>G</b>
	2.6667	2.3913	1.1152	0.324406	H
	2.1429	3.5217	0.60847	0.028879	I
	1.5714	1.7826	0.88153	0.341171	J
	3.9524	2.6957	1.4662	0.093804	K
	2.5238	2.6522	0.9516	0.415432	L
	2.619	2.087	1.255	0.18487	M
	5.2857	4.4783	1.1803	0.23432	N
	2.381	2.6087	0.9127	0.39237	<b>O</b>



Supplemental Table 1. Continued

	Disease Resistant	Disease Susceptible	Ratio	Probability	Linkage Groups
BBB	1.4286	1.4783	0.96639	0.461257	A1
	3.1905	2.7391	1.1648	0.265637	A2
	1.4286	1.6087	0.88803	0.372965	B1
	1.5714	1.6087	0.97683	0.473989	B2
	0.57143	0.65217	0.87619	0.390453	<b>C1</b>
	8.2857	10.609	0.78103	0.103437	<b>C2</b>
	4.7143	4.2174	1.1178	0.332715	<b>D1a</b>
	1.4762	3.6087	0.40906	0.012039	<b>D1b</b>
	4.0952	5.2609	0.77843	0.227161	D2
	2.2857	5	0.45714	0.000236	<b>E</b>
	4	4.1304	0.96842	0.443994	F
	6.8095	5.5217	1.2332	0.175974	<b>G</b>
	1.9524	1.7826	1.0952	0.388278	H
	1.381	2.2174	0.62278	0.083346	I
	1	1.3478	0.74194	0.06936	J
	2.9524	1.6522	1.787	0.498311	K
	1.4762	1.4783	0.9986	0.223272	L
	2.0476	1.6087	1.2728	0.363838	M
	3.7619	3.3913	1.1093	0.390396	N
	1.8095	2	0.90476		O

Supplemental Table 2. Linkats of Essex alleles in SCN resistant and susceptible groups.

	Disease Resistant	Disease Susceptible	Ratio	Probability	Linkage Groups
A	2.0952	2	1.0476	0.422409	A1
	4.5714	5.3913	0.84793	0.09122	A2
	4.7143	4.9565	0.95113	0.34566	B1
	3.8571	3.6087	1.0688	0.318569	B2
	1.619	1.3043	1.2413	0.151028	<b>C1</b>
	8.1905	7.0435	1.1628	0.256559	<b>C2</b>
	6.7143	6.9565	0.96518	0.42173	<b>D1a</b>
	6.0476	4.2609	1.4193	0.03494	<b>D1b</b>
	10.476	9.9565	1.0522	0.363385	D2
	5.6667	2.9565	1.9167	0.000105	<b>E</b>
	5.381	5.087	1.0578	0.382829	F
	6.8095	9.3043	0.73186	0.029528	<b>G</b>
	3.0476	2.913	1.0462	0.39344	H
	6.8095	5.4783	1.243	0.044905	I
	2.4762	2.5652	0.96529	0.434052	J
	5.3333	6.4783	0.82327	0.110741	K
	4.619	4.3478	1.0624	0.325899	L
	2.5238	3.087	0.81757	0.157233	M
	5.9048	6.6087	0.89348	0.258952	N
	3.6667	4.1739	0.87847	0.27294	<b>O</b>

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Supplemental Table 2. Continued

	Disease Resistant	Disease Susceptible	Ratio	Probability	Linkage Groups
AA	1.2381	0.69565	1.7798	0.439645	A1
	2.4286	1.9565	1.2413	0.045902	A2
	2.3333	2	1.1667	0.180328	B1
	2.1905	2	1.0952	0.122986	B2
	1	0.73913	1.3529	0.077726	<b>C1</b>
	5.5238	2.0435	2.7031	0.371286	<b>C2</b>
	4.2857	2.3913	1.7922	0.408344	<b>D1a</b>
	4.4286	1.3913	3.183	0.050755	<b>D1b</b>
	8.1429	1.8696	4.3555	0.484954	D2
	4.1429	1.1739	3.5291	0.000569	<b>E</b>
	3.7143	1.6087	2.3089	0.400358	F
	2.8571	3.5217	0.81129	0.009315	<b>G</b>
	1.8095	1.5217	1.1891	0.190319	H
	4.5714	2.0435	2.2371	0.084048	I
	1.7143	0.65217	2.6286	0.361069	J
	3.381	2.1304	1.587	0.165115	K
	2.9048	1.7391	1.6702	0.334952	L
	1.5238	0.78261	1.9471	0.06368	M
	4.1429	1.8261	2.2687	0.279609	N
	2.9048	0.86957	3.3405	0.427126	<b>O</b>
AAA	0.71429	0.73913	0.96639	0.472415	A1
	1.1905	2.2174	0.53688	0.015323	A2
	1.3333	1.7826	0.74797	0.247604	B1
	1.2381	0.69565	1.7798	0.064673	B2
	0.52381	0.34783	1.506	0.214849	<b>C1</b>
	4.0476	3.7826	1.0701	0.428494	<b>C2</b>
	3.2381	3.3043	0.97995	0.475663	<b>D1a</b>
	3.2857	2.1304	1.5423	0.07594	<b>D1b</b>
	6.5714	6.7391	0.97512	0.452025	D2
	3.381	1.2174	2.7772	0.000449	<b>E</b>
	2.619	2.4783	1.0568	0.433379	F
	1.5238	4.0435	0.37686	0.006658	<b>G</b>
	1.3333	0.82609	1.614	0.120642	H
	3.0952	2.3478	1.3183	0.16673	I
	1.1429	1.5652	0.73016	0.219074	J
	2.2381	3.1304	0.71495	0.175415	K
	1.9524	1.6957	1.1514	0.34399	L
	0.85714	1.7826	0.48084	0.029304	M
	2.9524	3.3043	0.89348	0.364264	N
	2.381	2.1304	1.1176	0.368596	<b>O</b>

Supplemental Table 3. Linkats of Essex Alleles in high and low yield groups.

	Low Yield	High Yield	Ratio	Probability	Linkage Group
A	2.1667	2	1.0834	0.315483	A1
	5.4667	5.4	1.0124	0.067664	A2
	5.1333	5.0667	1.0131	0.37792	B1
	3.5333	4	0.88333	0.138334	B2
	1.6	1.2333	1.2973	0.058823	C1
	10.5	8.6	1.2209	0.299318	C2
	6.9333	7.9333	0.87395	0.102217	D1a
	5.1333	5.4333	0.94478	0.293946	D1b
	10.033	8.8333	1.1358	0.382622	D2
	2.6	2.9667	0.87639	0.448725	E
	6.2667	6.1333	1.0218	0.349282	F
	11.1	9.7667	1.1365	0.376234	G
	3.3333	3	1.1111	0.141052	H
	<b>6.4667</b>	<b>4.8667</b>	<b>1.3288</b>	<b>0.054894</b>	<b>I</b>
	3.0667	2.5667	1.1948	0.140526	J
	<b>4.5</b>	<b>7.2333</b>	<b>0.62212</b>	<b>1.16E-05</b>	<b>K</b>
	3.9333	4.6333	0.84892	0.065702	L
	2.7667	3.1667	0.87369	0.166634	M
	6.7667	6.9333	0.97597	0.218238	N
	4.9	4.4	1.1136	0.315148	O
5.31	5.2083	1.02843		Mean	
AA	1.0952	0.80952	1.3529	0.221378	A1
	3.381	2.619	1.291	0.10773	A2
	2.7619	3.5238	0.78378	0.1583	B1
	1.9048	1.9524	0.97562	0.468098	B2
	1.1429	0.71429	1.6001	0.108101	C1
	8.381	6.619	1.2662	0.172517	C2
	4.7143	5.3333	0.88394	0.313992	D1a
	3.8095	4.7143	0.80807	0.199123	D1b
	7.619	6.1429	1.2403	0.16879	D2
	1.7143	1.7143	1	0.5	E
	4.8571	3.9524	1.2289	0.215976	F
	7.8571	7	1.1224	0.305542	G
	2.0952	1.619	1.2941	0.221589	H
	<b>4.4286</b>	<b>2.9524</b>	<b>1.5</b>	<b>0.032979</b>	<b>I</b>
	2.2381	2.2857	0.97917	0.468454	J
	<b>2.8571</b>	<b>5.1429</b>	<b>0.55554</b>	<b>0.009804</b>	<b>K</b>
	2.2381	2.9524	0.75806	0.178496	L
	2.381	2.2857	1.0417	0.436078	M
	5.1905	5.0952	1.0187	0.469325	N
	2.4762	2.7619	0.89656	0.317056	O
3.6571	3.50952	1.07985		Mean	

Supplemental Table 3. Continued

	Low Yield	High Yield	Ratio	Probability	Linkage Group
AAA	0.83333	0.6	1.3889	0.227018	A1
	2.1667	2.3	0.94204	0.395092	A2
	1.9333	2	0.96665	0.460807	B1
	1.1	1.0667	1.0312	0.465053	B2
	<b>0.73333</b>	<b>0.3</b>	<b>2.4444</b>	<b>0.026908</b>	C1
	5.7667	5.0667	1.1382	0.304805	C2
	3.5	4.2333	0.82678	0.211233	D1a
	2.5	3.1333	0.79788	0.209991	D1b
	6.6333	5.4	1.2284	0.14822	D2
	1.3	1.6	0.8125	0.253408	E
	<b>3.4333</b>	<b>3.4333</b>	<b>1</b>	<b>0.5</b>	F
	5.4667	4.8333	1.131	0.309594	G
	1.5667	1.2	1.3056	0.207928	H
	<b>3.1</b>	<b>1.8333</b>	<b>1.6909</b>	<b>0.018296</b>	<b>I</b>
	1.7333	1.5667	1.1063	0.359631	J
	<b>1.6667</b>	<b>3.9333</b>	<b>0.42374</b>	<b>0.002573</b>	<b>K</b>
	<b>1.0333</b>	<b>2.0667</b>	<b>0.49998</b>	<b>0.028137</b>	L
	1.4333	1.5333	0.93478	0.407889	M
	3.5667	4.4	0.81061	0.186029	N
	2.6333	2.4667	1.0675	0.396604	O
	2.68999	2.6483			Mean

Supplemental Table 4. Linkats of Forrest alleles in high and low yield groups.

	Low Yield	High Yield	Ratio	Probability	Linkage Group
B	2.8333	3	0.94443	0.339754	A1
	5.5333	5.6	0.98809	0.447336	A2
	4.8667	4.9333	0.9865	0.455683	B1
	4.4667	4	1.1167	0.178333	B2
	1.4	1.7667	0.79244	0.084969	C1
	10.5	12.4	0.84677	0.100339	C2
	8.0667	7.0667	1.1415	0.148827	D1a
	4.8667	4.5667	1.0657	0.357885	D1b
	7.9667	9.1667	0.86909	0.163227	D2
	3.4	3.0333	1.1209	0.232472	E
	7.7333	7.8667	0.98304	0.450061	F
	8.9	10.233	0.86974	0.165103	G
	3.6667	4	0.91668	0.238494	H
	4.5333	6.1333	0.73913	0.006745	<b>I</b>
	1.9333	2.4333	0.79452	0.146559	J
	6.5	3.7667	1.7256	9.48E-05	<b>K</b>
	5.0667	4.3667	1.1603	0.090635	L
	3.2333	2.8333	1.1412	0.19872	M
	6.2333	5.9667	1.0447	0.393425	N
	3.1	3.4333	0.90292	0.328974	O
SEM	2.435599	2.796534	0.212329		
SUM	104.8	106.5664	1.007498	MEAN	

Supplemental Table 4. Continued

	Low Yield	High Yield	Ratio	Probability	Linkage Group
BB	1.9	1.8667	1.0178	0.471703	
	3.4667	3.7	0.93695	0.340583	
	2.8667	2.8	1.0238	0.45589	
	2.8	2.3333	1.2	0.218565	
	0.6	1.1	0.54545	0.035078	
	8.0333	10	0.80333	0.095882	
	5.6333	4.7667	1.1818	0.188766	
	3.2	3.1333	1.0213	0.468363	
	6.2	7.1	0.87324	0.238981	
	2.3667	2.2333	1.0597	0.411216	
	5.9667	6.0333	0.98896	0.473981	
	5.4333	6.9	0.78743	0.133041	
	2.2667	2.8	0.80954	0.146998	
	2.2667	4.0333	0.562	0.00263	
	1.4333	1.7333	0.82692	0.244333	
	4.8333	1.9667	2.4576	2.98E-05	
	3.0333	2.5	1.2133	0.154261	
	2.4333	2.0333	1.1967	0.206271	
	4.1	4.3	0.95349	0.418899	
	1.9333	2.1667	0.89228	0.370876	
SEM	1.901279	2.271715	0.388184		
SUM	70.7666	73.4999	1.01758	MEAN	
BBB	1.2667	1.2	1.0556	0.430955	
	2.4667	2.5667	0.96104	0.426163	
	1.8333	1.5	1.2222	0.257691	
	2	1.6	1.25	0.224322	
	0.3	0.76667	0.3913	0.018609	
	6.5	8.3333	0.78	0.105269	
	4.1667	3.4667	1.2019	0.211313	
	2.3333	2.3667	0.98589	0.482938	
	5	5.9333	0.8427	0.22218	
	1.9667	2	0.98335	0.478212	
	4.5667	4.4	1.0379	0.42911	
	3.7	5.5333	0.66868	0.060731	
	1.5667	1.9667	0.79661	0.213927	
	1.1667	2.7667	0.42169	0.002201	
	1.0333	1.1667	0.88566	0.367227	
	3.5333	1.2	2.9444	0.000314	
	1.7333	1.3667	1.2682	0.202926	
	1.9667	1.4667	1.3409	0.146063	
	2.8	3.1333	0.89363	0.350178	
	1.5333	1.6333	0.93877	0.5	
SEM	1.956247	0.51542	0.168429		
MEAN			1.043521		

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