

Bayesian inference to study the genetic control of resistance to gray leaf spot in maize

RENZO G. VON PINHO^{1,3}, MÁRCIO BALESTRE¹, ANDRÉ BRITO² IOLANDA V. VON PINHO¹

¹ Agriculture Department, Federal University of Lavras, Lavras, Brazil, ⁽²⁾ Research Department, Dow AgroSciences Ltda, Indianópolis (MG), Brazil,

E-mail: renzo@ufla.br

³CNPq Researcher - Nat. Council of Sci. and Technological Development, Brazilian Ministry of Science and Technology

ABSTRACT

Gray Leaf Spot (GLS) is a major maize disease in Brazil and can significantly affect grain production. Studies on the genetic control of resistance to this disease are scarce and none have been published on the use of Bayesian methods for this purpose. This study, based on Bayesian inference, investigated the nature and magnitude of gene effects related to GLS resistance by the evaluation of contrasting lines and segregating populations. The experiment was arranged in a randomized block design with three replications and the mean values analyzed using Bayesian shrinkage approach. Additive-dominant and epistatic effects and its variances were adjusted in an only over-parametrized model. Bayesian shrinkage analysis one showed an excellent approach to handle with complex models in study of genetic control in GLP. Genetic control of GLS resistance was predominantly additive presented insignificant influence of dominance and epistasis effects.

INTRODUCTION

- Gray leaf spot (*Cercospora zea maydis*), it is currently one of the major foliar maize diseases in Brazil due to its nation-wide distribution and level of damage in susceptible hybrids (Brito et al. 2008). As of 2000, gray leaf spot has reached epidemic proportions in several regions of the country (Julliat et al. 2004).
- In Brazil, there is consensus among maize breeders that a major cause of interruptions in the planting of commercial maize hybrids is the severity of diseases such as gray leaf spot. The emergence of variations in the pathogen population was mainly due to the cultivation of susceptible hybrids and to changes in production systems.
- To study the inheritance of any trait, joint scaling tests have usually been applied, where the main and epistatic effects can be included in the model and tested by chi-square test (Mather and Jinks 1984)
- This methodology has some limitations, mainly when the degrees of freedom are restricted to number of parameters adjusted in the full-model, i.e. epistatic effects. One alternative to get around this limitation is to realize model selection or to adjust complex models where the number of parameters is higher than the number of observations.
- Xu (2003) proposed an approach to handle with complex models in the QTL analysis where the number of parameters is higher than number of observations; The Xu (2003) approach it is a free-model selection methodology and will be applied in this study, since enable us to adjust additive, dominant, epistatic effects and its variances in a single model (Balestre et al 2012).

OBJECTIVE

- The present study was carried out to investigate the nature and magnitude of gene effects related to resistance to GLS based on Bayesian inference by the evaluation of contrasting lines and segregating populations.

MATERIAL AND METHODS

Genotypes

- Four population (F₁, F₂, BC₁₁ and BC₂₁) tracing back to two backgrounds (GNS30 x GNS31 and GNS84 x GNS31) and its parental inbred lines (GNS30, GNS31 and GNS84) were evaluated. The lines GNS30 and GNS31 arose from the same background formed by lines derived from the genotypes Cateto and Caribbean. Both have hard grains, short stature and medium-late cycle. GNS31 is susceptible and GNS30 GLS-resistant. Line GNS84 was obtained from the selfing of varieties derived from genotype Tuxpeno with semident grain, medium sized, early maturity and GLS resistance. The seeds of the parent lines, as well as the F₁ F₂, BC₁₁ and BC₂₁ generations, were obtained in the 2007/2008 growing season by selfing and field crosses.

Disease evaluation

- For these evaluations of disease severity data (grades) were used represented by the percentage of infected leaf area (ILA) on a 1 - 9 rating scale (Von Pinho et al., 2001) as follows: 1 = 0 % ILA and no symptoms, 2 = <1% ILA with a few scattered lesions, 3 = 1% - 20% ILA, 4 = 20% - 40% ILA, 5 = 40% - 50% ILA with lesions on the ear leaf and a few lesions on leaves above the ear, 6 = 50% - 60% ILA with lesions on the leaves above the ear, 7 = 60% - 75% ILA, 8 = 75% - 90% ILA and 9 => 90% ILA with premature plant death prior to physiological maturity (formation of black layer on grain).

Genetic model

$$y|b, a, d, aa, ad, dd, \sigma^2 V \sim N(b + Z_1 a + Z_2 d + Z_3 aa + Z_4 ad + Z_5 dd, \sigma^2 V)$$

Prior distributions

$$p(a) \propto N(0, \sigma_a^2), p(d) \propto N(0, \sigma_d^2), p(aa) \propto N(0, \sigma_{aa}^2), p(ad) \propto N(0, \sigma_{ad}^2), p(dd) \propto N(0, \sigma_{dd}^2), \\ p(\sigma_a^2) \propto 1/\sigma_a^2, p(\sigma_d^2) \propto 1/\sigma_d^2, p(\sigma_{aa}^2) \propto 1/\sigma_{aa}^2, p(\sigma_{ad}^2) \propto 1/\sigma_{ad}^2, p(\sigma_{dd}^2) \propto 1/\sigma_{dd}^2 \text{ and} \\ p(\sigma^2) \propto 1/\sigma^2$$

Posterior distributions

$$b = (1/n) \sum_{i=1}^n V_i^{-1} (y_i - Z_{i1}a - Z_{i2}d - Z_{i3}aa - Z_{i4}ad - Z_{i5}dd)$$

$$(1/n) \sigma^2$$

$$a = (Z_3 V^{-1} Z_1 + \sigma^2 / \sigma_a^2)^{-1} Z_1 V^{-1} (y - b - Z_2 d - Z_3 aa - Z_4 ad - Z_5 dd)$$

$$d = (Z_2 V^{-1} Z_2 + \sigma^2 / \sigma_d^2)^{-1} Z_2 V^{-1} (y - b - Z_1 a - Z_3 aa - Z_4 ad - Z_5 dd)$$

$$(Z_1 V^{-1} Z_1 + \sigma^2 / \sigma_a^2)^{-1} \sigma^2 \text{ and } (Z_2 V^{-1} Z_2 + \sigma^2 / \sigma_d^2)^{-1} \sigma^2$$

$$aa = (Z_3 V^{-1} Z_3 + \sigma^2 / \sigma_{aa}^2)^{-1} Z_3 V^{-1} (y - b - Z_1 a - Z_2 d - Z_4 ad - Z_5 dd)$$

$$(Z_3 V^{-1} Z_3 + \sigma^2 / \sigma_{aa}^2)^{-1} \sigma^2$$

$$\sigma_a^2 = a^2 / \chi_1^2$$

$$ad = (Z_4 V^{-1} Z_4 + \sigma^2 / \sigma_{ad}^2)^{-1} Z_4 V^{-1} (y - b - Z_1 a - Z_2 d - Z_3 aa - Z_5 dd)$$

$$(Z_4 V^{-1} Z_4 + \sigma^2 / \sigma_{ad}^2)^{-1} \sigma^2$$

$$\sigma_d^2 = d^2 / \chi_1^2$$

$$dd = (Z_5 V^{-1} Z_5 + \sigma^2 / \sigma_{dd}^2)^{-1} Z_5 V^{-1} (y - b - Z_1 a - Z_2 d - Z_3 aa - Z_4 ad)$$

$$(Z_5 V^{-1} Z_5 + \sigma^2 / \sigma_{dd}^2)^{-1} \sigma^2$$

$$\sigma_{aa}^2 = aa^2 / \chi_1^2$$

$$\sigma_{ad}^2 = ad^2 / \chi_1^2$$

$$\sigma_{dd}^2 = dd^2 / \chi_1^2$$

$$\sigma^2 = \sum_{i=1}^n (y_i - b - Z_{i1}a - Z_{i2}d - Z_{i3}aa - Z_{i4}ad - Z_{i5}dd)^2 / \chi_n^2$$

- For the analysis a program was developed using the SAS / IML (SAS Institute, 2000) package. Chains of different sizes were used according to the analysis (per cross or combined). The chain sizes as well as the burn-in and jump process were obtained as suggested by Raftery and Lewin (1992). For the stationarity analysis of the chains we used the criterion suggested by Books and Gelman (1998) using the Bayesian Output Analysis package (BOA) available for platform R.

RESULTS



Figure 1- Gray Leaf Spot in maize

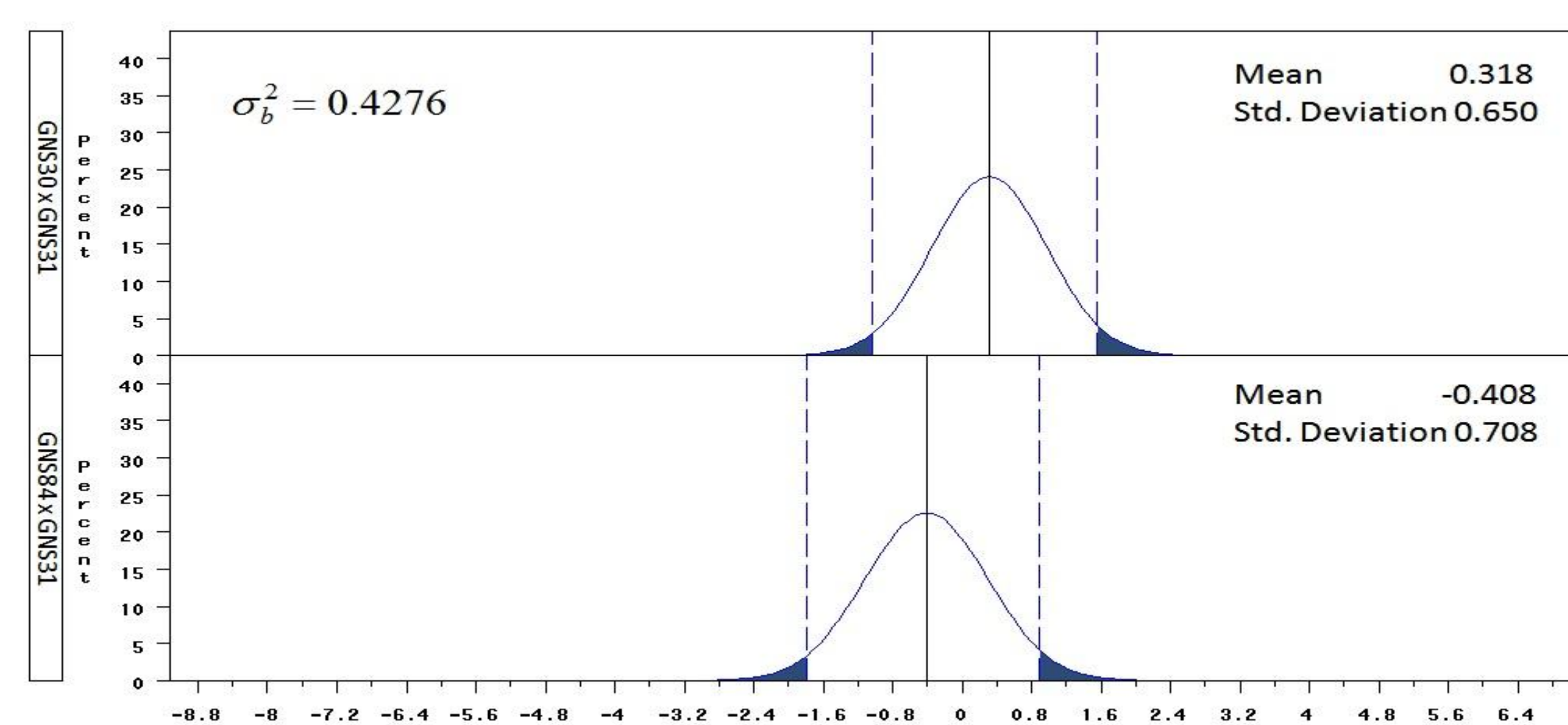


Figure 2- Background effects correspondent to GNS84 x GNS31 and GNS30 x GNS31 crosses and background variance.

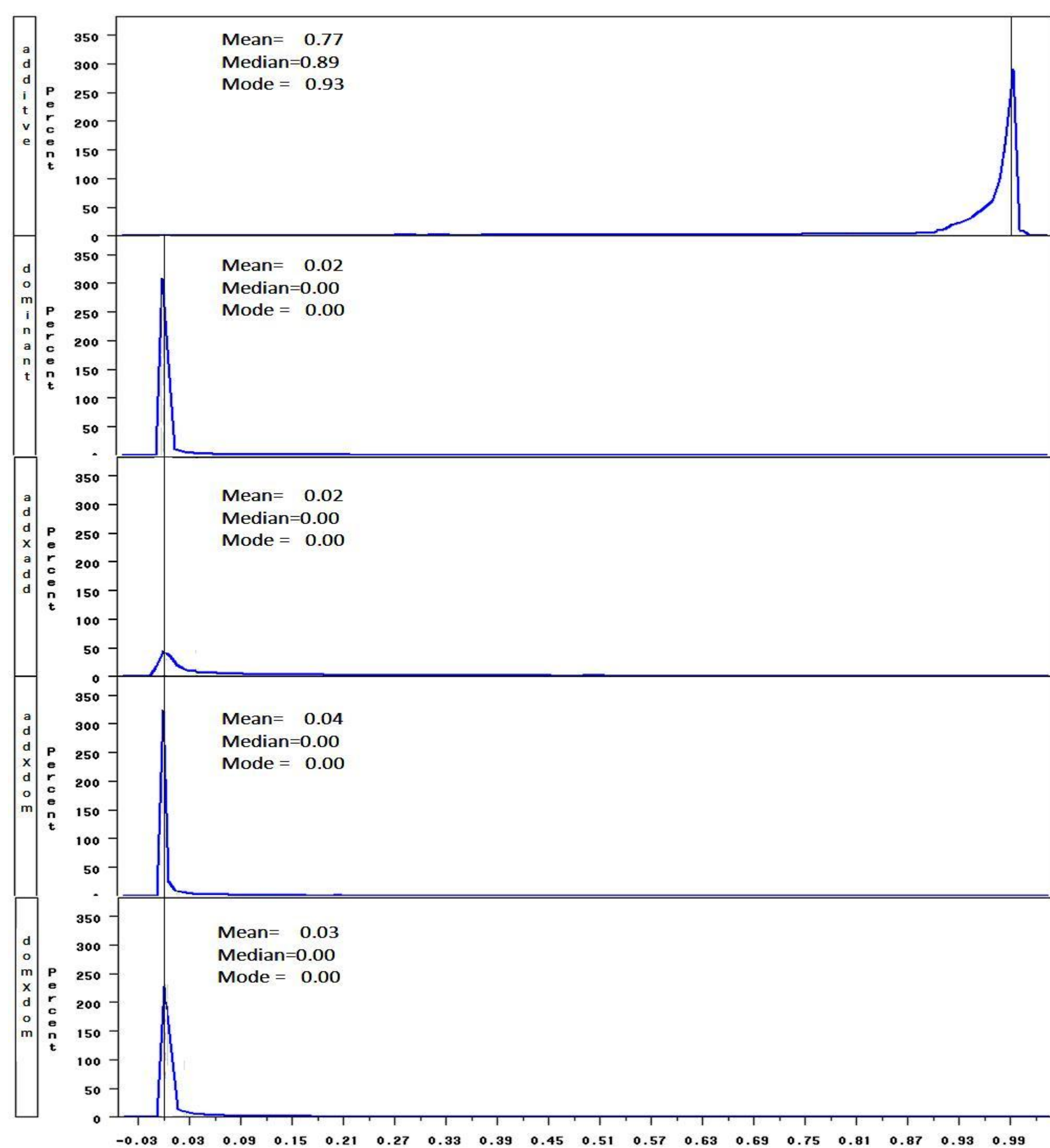


Figure 4 Posterior probability distributions of additive, dominant and epistatic heritability obtained in join analysis

CONCLUSIONS

- ✓ Bayesian shrinkage analysis it is an excellent approach to handle with complex models in study of genetic control in GLS.
- ✓ Genetic control of Cercosporiose resistance was predominantly additive presented insignificant influence of dominance and epistasis effects

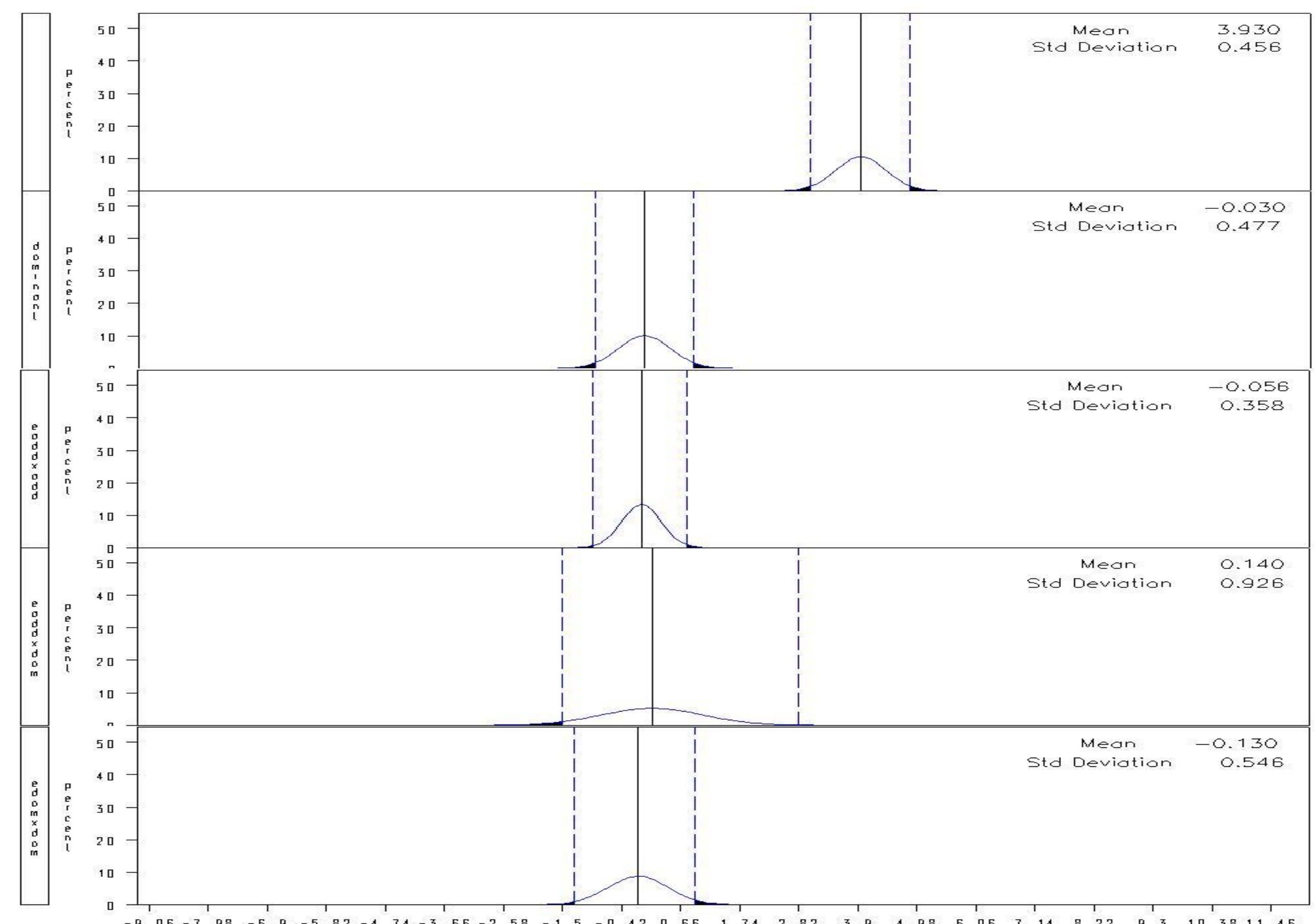


Figure 3 Posterior probability distributions of additive, dominant and epistatic effects and its variances obtained in join analysis.

Table 1 Number of iterations required for convergence..

GNS31 x GNS84		
Grown	Chain	MPSRF
Season 1	330000	1.000
Season 2	400000	1.000
GNS31 x GNS30		
Grown	Chain	MPSRF
Season 1	95000	1.000
Season 2	160000	1.000
Join		
Season1 + Season2	chain	MPSRF
	510000	1.000

MPSRF-Multivariate Potential Scale Reduction Factor

Table 2- Additive, dominant and epistatics effects and its variances obtained under different backgrounds and sowing season.

E effects/parameters	GNS31 x GNS84					
	Mean	Sown 1		Mean	Sown 2	
		0.05	0.95		0.05	0.95
a	3.755	2.293	4.656	2.302	-0.216	5.962
d	-0.365	-2.050	0.104	-0.450	-5.540	2.696
aa	0.083	-0.178	0.909	-0.441	-4.599	1.668
ad	0.242	-0.350	2.242	1.817	-3.364	14.131
dd	-0.430	-1.811	0.078	-0.761	-6.012	1.703
	Mean	Median	Mean	Median	Mean	Median
σ_a^2	4.1×10^2	30.100	49104	7.300		
σ_d^2	3.9×10^2	1.3×10^{-4}	30104	0.150		
σ_{aa}^2	3.4×10^2	6.9×10^{-6}	56103	0.088		
σ_{ad}^2	3.5×10^2	1.2×10^{-5}	62104	0.440		
σ_{dd}^2	1.5×10^2	2.4×10^{-4}	77104	0.180		
σ^2	1.897	0.579	19.458	10.996		
E effects/parameters	GNS31 x GNS30					
	Mean	Sown 1		Mean	Sown 2	
		0.05	0.95		0.05	0.95
a	3.947	3.360	4.608	1.943	-0.021	4.801
d	0.144	-0.208	0.975	0.325	-0.362	2.934
aa	0.257	-0.056	1.280	-0.464	-3.337	0.214
ad	0.926	-0.091	4.781	-0.455	-5.559	1.213
dd	0.046	-0.127	0.236	-0.008	-1.000	1.376
	Mean	Median	Mean	Median	Mean	Median
σ_a^2	5.9×10^2	34.600	4.5×10^4	4.41		
σ_d^2	8.2×10^2	2.1×10^{-5}	1.5×10^3	1.31×10^2		
σ_{aa}^2	1.3×10^2	5.8×10^{-5}	1.2×10^3	3.5×10^2		
σ_{ad}^2	5.6×10^2	0.006	9.1×10^3	3.3×10^2		
σ_{dd}^2	1.1×10^2	2.7×10^{-5}	9.8×10^3	1.1×10^2		
σ^2	0.609	0.230	12.880	6.966		

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