RIO CUARTO, ARGENTINA
Universidad Nacional de Río Cuarto
CASTELAR, ARGENTINA
Instituto Nacional de Tecnología Agropecuaria

Path coefficient analysis of "Mal de Río Cuarto" disease components.

--Dallo, MD; Ibañez, MA; Borghi, ML; Girardi, VN; Bonamico, NC; Salerno, JC; Díaz, DG; Di Renzo, MA.

INTRODUCTION

The "Mal de Río Cuarto" (MRC) is considered the most important viral disease of corn in Argentina, causing increase damage and high yield losses to susceptible cultivars (Lenardón, SL et al., Plant Dis 82:448, 1998; Di Renzo, MA, et al., J Agr Sci 139:47-53, 2002). The disease is caused by MRC virus which belongs to the genus *Fijivirus*, within the family *Reoviridae* and is transmitted by the planthopper *Delphacodes kuscheli* Fennah (Hemiptera: Delphacidae) (Nome, SF et al., Phytopathol Z 101:7-15, 1981; Ornaghi, JA et al., J Gen Breed 47:227-282, 1993). Its symptomatology depends of the plant phenological state when it is infected, the genotype and the environmental conditions where the culture grown. This includes stunting, short internodes, galls or enations on the abaxial side of the leaves, size reduction and malformation of spikes, ears and leaves (Lenardón, SL et al, SL Lenardón Ed., IFFIVE, INTA, JICA, 1999; Di Renzo, MA et al., J Agric Sci 142:289-295, 2004). Since both economic and environmental reasons prevent a raise in agrochemicals usage, future cereal improvement will rely on germplasm that optimize present genetic tolerance to plant pathogens (Abeledo, LG et al., Euphytica 130:325-334, 2003). This is an effective way of both increasing and stabilizing production in affected areas.

Selection for one trait usually affects several traits. The correlated response to selection is a change in one or more traits due to selection of another as a result of genetic relations between them. Genetic correlations are useful to decide on selection strategies since they express the relative importance of pleiotropy and linkage between loci (Kang, MS et al., Crop Sci 23:643-647, 1983; Kang, MS, Applied quantitative genetics. MS Kang Publisher, Baton Rouge, LA, 1994). The genetic correlations can be estimated from phenotypic values, which are influenced by type of gene action, environmental effects and genotype x environment interactions (Falconer and Mackay, Introduction to quantitative genetics. Longman Technical, Essex, UK, 1996).

Nevertheless, while correlation coefficient only measures the association magnitude between variables, the path coefficient analysis (Wright, S, J Agric Res 20:557-587, 1921; Wright, S, Ann Math Stat 5:161-215, 1934) allows dissecting the correlation between them into effects, direct and indirect. This method has been commonly used in crop breeding studies to establish the relationships between grain yield and its contributing components (Mohammadi, SA et al., Crop Sci 43:1690-1697, 2003). In addition, there are many references of its application in plant pathology studies (Van Bruggen and Arneson, Phytopathology 76:874-878, 1986; Nayak, P et al., J Phytopathol 119:312-318, 1987; Neher, DA et al., Plant Dis 77:1106-1111, 1993; Birhman and Singh, Ann Appl Biol 127:353-362, 1995; Desprez-Loustau and Wagner, Eur J Plant Pathol 103:653-665, 1997; Garcia, D et al., Eur J Forest Pathol 29:323-338, 1999). Therefore, it would be useful to get information regarding direct and indirect relationships among MRC disease tolerance and different related traits. The objectives of this work were: i) to determine phenotypic and genetic correlation coefficients among MRC disease tolerance and different related traits and ii) to present a path coefficient analysis to show how these traits affect MRC disease tolerance.

MATERIALS AND METHODS

Plant material

One hundred and forty four $F_{2:6}$ recombinant inbred lines (RIL) were developed from a cross between Mo17, a susceptible dent line, and BLS14, a partially resistance red flint line. The RILs were assessed to MRC disease and parental genotypes were used as controls in each plot.

Field trials

The RILs were tested during the 2004 summer cycle in field experiments at two locations belonging to the endemic area: Río Cuarto (R4) and Sampacho (S4). The field trials were conducted in a randomized complete block design with two replicates of single-row plots. Each plot consisted of 3.0 m rows with 0.7 m spacing. Plants were thinned to a distance of 0.15 m in the row. The sowing date determination was made through insect vector monitoring during spring, in order to *Delphacodes kuscheli* population reach the peak during early stages of maize development.

Symptoms observed and scored

Disease symptoms were scored 60-70 days after male flowering. Individual plants from each plot were phenotypically screened for traits related to MRC disease: plant height (PH), internodes (IN), enations (EN), flag leaf edge (LE), width (LW) and length (LL),

panicle (PA) and ear (EA). A disease severity grade was estimated for each plant according to a 0 - 3 scale proposed by Ornaghi *et al.* (Maydica 44:219-223, 1999). The response variable is the disease severity index (DSI) based on the disease severity grades of individual plants. DSI was calculated for each plot and used to rate RIL for their reaction to MRC disease according to Grau *et al.* (Plant Dis 66:506–508, 1982). The details of the rating for MRC severity were described in Di Renzo *et al.* (J Agr Sci 139:47-53, 2002).

Statistical analysis

Data from each trial location were subjected to variance and covariance analyses using the PROC GLM procedure of SAS (SAS Institute ver 9.1.3). Genetic and phenotypic correlation coefficients among DSI and traits related to MRC disease were determined from variance and covariance components. The path coefficient analysis was performed to calculate direct and indirect effects among the response variable, DSI, and MRC related predictor variables. Direct and indirect path coefficients were calculated as firstly proposed Wright (J Agric Res 20:557-587, 1921; Ann Math Stat 5:161-215, 1934), and then Dewey and Lu (Agron J 51:515-518, 1959) and Li (Path analysis: a primer. Boxwood Press, Pacific Grove, CA, 1975). For each trait, direct path coefficient was solved by means of PROC IML (SAS Institute ver 9.1.3). The product of appropriate correlation coefficient (*r*) and path coefficient values provides the indirect path coefficient. Correlations and path coefficient analyses based on genetic values define more precisely what factors affect DSI genetically. Previous to analysis, the original data was logarithmically transformed to satisfy the assumption of additivity among the components. The presence of multicollinearity among variables was measured using the variance inflation factor (VIF) and the condition number (CN). Residual effects and determination coefficients were estimated according to Kang (Applied quantitative genetics. MS Kang Publisher, Baton Rouge, LA, 1994). The criterion followed to evaluate the extent of effects of MRC related traits on DSI magnitude was according to Board *et al.* (Crop Sci 37:879-884, 1997).

RESULTS AND DISCUSSIONS

Phenotypic and genetic correlations coefficients among all pairs of traits are shown in Table 1. In general, phenotypic and genetic correlation coefficients agreed in sign and the magnitude of the phenotypic correlation coefficient was practically the same that the genetic correlation coefficient indicating that the influence of environment on these relationships was little or negligible. However, in most cases, the genetic correlation estimates between DSI and MRC disease related traits were slightly high showing that they are genetically associated or that they are physiologically related (Sidwell, RJ et al.,

Crop Sci 16:650-654, 1976). At R4 location, EN had the highest positive phenotypic correlation on DSI (0.94) followed by IN (0.91) and EA (0.64) but, IN had the highest positive genetic correlation on DSI (0.96) followed by EN (0.95) and EA (0.69). However at S4 environment, both phenotypic and genetic correlation coefficient of EN on DSI showed the highest positive values (0.96 and 0.98, respectively) followed by IN (0.90 and 0.94, respectively) and PA (0.69 and 0.77, respectively).

Table 1. Phenotypic and genetic correlations among all pairs of traits at R4 and S4 environments.

	PA	LL	LW	LE	EN	IN	EA	DSI
PA		0.490 **0.535**	0.408 **0.399**	0.237 **0.225**	0.576 ++ **0.596**	0.552 **0.581**	0.255 **0.298**	0.591 ++ **0.611**
LL	0.577 **0.703**		0.411 **0.412**	0.175 **0.128**	0.353 **0.358**	0.309 ++ **0.363**	0.019 **0.002**	0.352 ++ **0.375**
LW	0.444 **0.320**	0.362 **0.340**		0.119 **0.042**	0.155 **0.092**	0.148 **0.137**	-0.044 **-0.143 ++**	0.189 **0.166**
LE	0.324 **0.350**	0.480 ++ **0.530**	0.128 **-0.038**		0.534 **0.558**	0.518 ++ **0.582**	0.596 ++ **0.679**	0.541 **0.556**
EN	0.666 **0.762**	0.622 ++ **0.747**	0.226 **0.243**	0.566 **0.714**		0.867 ++ **0.913 ++**	0.600 ++ **0.648**	0.937 ++ **0.955**
IN	0.616 ++ **0.697**	0.530 **0.646**	0.212 **0.221**	0.483 **0.603**	0.849 ++ **0.891 ++**		0.559 ++ **0.651**	0.914 ++ **0.961**
EA	0.427 **0.469**	0.233 **0.272**	0.049 **-0.016**	0.282 ++ **0.331**	0.528 **0.600**	0.486 **0.570**		0.642 ++ **0.691**
DSI	0.685 ++ **0.774**	0.597 ++ **0.707**	0.232 **0.240**	0.532 **0.660**	0.961 ++ **0.979**	0.898 ++ **0.941**	0.549 ++ **0.628**	

† R4 = Río Cuarto 2004; S4 = Sampacho. DSI = disease severity index; PA = panicle; LL = leaf lenght; LW = leaf width; LE = leaf edge; EN = e-nations; IN = internodes; EA = ear.
++, + Significant at the 0.01 and 0.05 levels, respectively, by bootstrap method.
§ Values above/below the diagonal: correlations among all pairs of traits at R4 and S4 environments, respectively. Genetic values are shown in bold.

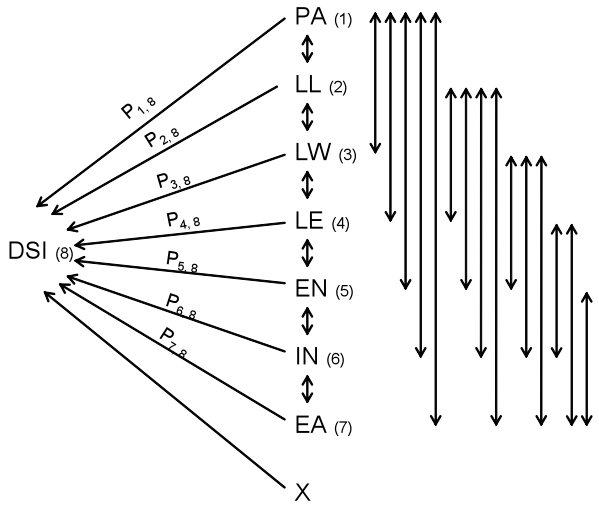

Fig. 1. Path diagram showing causal relationships between disease severity index (DSI) and Mal de Río Cuarto (MRC) disease related traits. One-headed arrow represents direct paths and double-headed arrows represent correlations (r).

Because of the similarity between phenotypic and genetic correlations, the phenotypic and genetic path coefficients were also quite similar. The path diagram based both phenotypic and genetic correlation coefficients were carried out according to shown in Fig. 1. The almost equal magnitude of EN and IN direct effects on DSI at both phenotypic and genetic levels indicated that these effects were under genetic control with unimportant environmental effects. However, at R4 and S4 environments, the genetic direct effects of EN and IN increased hardly over their respective phenotypic direct effects which suggest a negative relationship between DSI and environmental effects. In general, EN and IN were the primary and secondary direct determinants of DSI over both environments at the phenotypic and genetic levels (Table 2). Nevertheless, at R4 location, IN showed the highest positive genetic direct effect on DSI (0.490) followed by EN (0.445). Similarly, the large positive phenotypic and genetic direct effects of EN and IN on DSI was counterbalanced by a small phenotypic and genetic indirect effects via the remaining traits included in the analysis. In most cases, the remaining traits had negligible phenotypic and genetic direct effects but the phenotypic and genetic indirect effects were intermediate via EN and IN on both environments (Table 2).

Table 2. Phenotypic and genetic path coefficient analysis of DSI and MRC components at R4 and S4 environments.

Pathway	R4		S4	
DSI vs. PA				
Direct effect	0.032	**-0.010**	0.052	**0.053**
Indirect effect via				
LL	0.012	**0.011**	-0.004	**-0.032**
LW	0.016	**0.032**	-0.001	**0.000**
LE	-0.005	**-0.020**	-0.004	**-0.011**
EN	0.290	**0.266**	0.454	**0.532**
IN	0.213	**0.285**	0.173	**0.219**
EA	0.033	**0.047**	0.016	**0.013**
Correlation	0.591	**0.611**	0.685	**0.774**
DSI vs. LL				
Direct effect	0.025	**0.021**	-0.007	**-0.046**
Indirect effect via				
PA	0.016	**-0.005**	0.030	**0.038**
LW	0.016	**0.033**	-0.001	**0.000**
LE	-0.004	**-0.011**	-0.007	**-0.017**
EN	0.178	**0.159**	0.424	**0.522**
IN	0.119	**0.178**	0.149	**0.203**
EA	0.003	**0.000**	0.008	**0.007**
Correlation	0.352	**0.375**	0.597	**0.707**
DSI vs. LW				
Direct effect	0.038	**0.080**	-0.002	**-0.001**
Indirect effect via				
PA	0.013	**-0.004**	0.023	**0.017**
LL	0.010	**0.009**	-0.002	**-0.016**
LE	-0.003	**-0.004**	-0.002	**0.001**
EN	0.078	**0.041**	0.154	**0.169**
IN	0.057	**0.067**	0.060	**0.070**
EA	-0.006	**-0.023**	0.002	**0.000**
Correlation	0.189	**0.166**	0.232	**0.240**
DSI vs. LE				
Direct effect	-0.022	**-0.089**	-0.014	**-0.031**
Indirect effect via				
PA	0.008	**-0.002**	0.017	**0.019**
LL	0.004	**0.003**	-0.003	**-0.024**
LW	0.005	**0.003**	0.000	**0.000**
EN	0.269	**0.249**	0.386	**0.498**
IN	0.200	**0.285**	0.136	**0.190**
EA	0.077	**0.108**	0.010	**0.009**
Correlation	0.541	**0.556**	0.532	**0.660**
DSI vs. EN				
Direct effect	0.504	**0.445**	0.681	**0.698**
Indirect effect via				
PA	0.019	**-0.006**	0.035	**0.041**
LL	0.009	**0.008**	-0.004	**-0.034**
LW	0.006	**0.007**	-0.001	**0.000**
LE	-0.012	**-0.049**	-0.008	**-0.022**
IN	0.334	**0.447**	0.238	**0.280**
EA	0.078	**0.103**	0.019	**0.016**
Correlation	0.937	**0.955**	0.961	**0.979**
DSI vs. IN				
Direct effect	0.385	**0.490**	0.281	**0.315**
Indirect effect via				
PA	0.018	**-0.006**	0.032	**0.037**
LL	0.008	**0.008**	-0.003	**-0.030**
LW	0.006	**0.011**	0.000	**0.000**
LE	-0.011	**-0.052**	-0.007	**-0.019**
EN	0.436	**0.407**	0.578	**0.622**
EA	0.073	**0.103**	0.018	**0.016**
Correlation	0.914	**0.961**	0.898	**0.941**
DSI vs. EA				
Direct effect	0.130	**0.158**	0.036	**0.027**
Indirect effect via				
PA	0.008	**-0.003**	0.022	**0.025**
LL	0.000	**0.000**	-0.002	**-0.012**
LW	-0.002	**-0.011**	0.000	**0.000**
LE	-0.013	**-0.060**	-0.004	**-0.010**
EN	0.302	**0.288**	0.359	**0.419**
IN	0.215	**0.319**	0.137	**0.179**
Correlation	0.642	**0.691**	0.549	**0.628**
Coefficient of determination	0.931	**0.971**	0.951	**0.985**
Residual effect	0.262	**0.170**	0.222	**0.123**

† R4 = Río Cuarto 2004; S4 = Sampacho. DSI = disease severity index; PA = panicle; LL = leaf lenght; LW = leaf width; LE = leaf edge; EN = enations; IN = internodes; EA = ear.
§ Genetic values are shown in bold.

The phenotypic and genetic path coefficients for DSI accounted for a large proportion of phenotypic and genetic variation on both environments as indicated by a large coefficient of determination and by the corresponding small residual effect. In the phenotypic and genetic path analysis, the residual effect represents the failure of the estimated genetic correlations among the variables to account for the total genetic variation in a trait (Sidwell, RJ et al., Crop Sci 16:650-654, 1976).

The most important components of DSI, if we just consider phenotypic and genetic correlation coefficients, were EN, IN, EA, PA and LE. Nevertheless, phenotypic and genetic path coefficient analysis showed that among these traits only EN and IN were the most important to increase DSI. The direct effects of EA, PA and LE were little or negligible. The results of this study suggest that EN and IN are the best traits in determining DSI on both environments and may be useful as an indirect selection criteria in breeding and selection programs related to MRC disease.

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