Meta-Analysis of the Relationship Between Crop Yield and Soybean Rust Severity

Felipe Dalla Lana, Patricia K. Ziegelmann, Aline de H. N. Maia, Cláudia V. Godoy, and Emerson M. Del Ponte

First and fifth authors: Departamento de Fitossanidade, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, 91540-000, Brazil; second author: Departamento de Estatística, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, 91509-900, Brazil; third author: Embrapa Meio Ambiente, Jaguariúna, SP, 13820-000, Brazil; and fourth author: Embrapa Soja, Londrina, PR, 86001-970, Brazil. Current address of E. M. Del Ponte: Departamento de Fitopatologia, Universidade Federal de Viçosa, Viçosa, MG, 36570-900, Brazil. Accepted for publication 5 September 2014.

ABSTRACT

Dalla Lana, F., Ziegelmann, P. K., Maia, A. H. N., Godoy, C. V., and Del Ponte, E. M. 2015. Meta-analysis of the relationship between crop yield and soybean rust severity. *Phytopathology* 105:307-315.

Meta-analytic models were used to summarize and assess the heterogeneity in the relationship between soybean yield (Y , kg/ha) and rust severity (S , %) data from uniform fungicide trials (study, k) conducted over nine growing seasons in Brazil. For each selected study, correlation ($k = 231$) and regression ($k = 210$) analysis for the Y - S relationship were conducted and three effect-sizes were obtained from these analysis: Fisher's transformation of the Pearson's correlation coefficient (Z_r) and the intercept (β_0) and slope (β_1) coefficients. These effect-sizes were summarized through random-effect and mixed-effect models, with the latter incorporating study-specific categorical moderators such as disease onset time (DOT) ($<R1$ or $\geq R1$ reproductive crop stage), disease pressure (DP) (high = $>70\%$, moderate = >40 and $\leq 70\%$, and low = $\leq 40\%$ S the check treatment), and growing season. The overall mean for $\bar{r}$ (back-transformed $\bar{Z}_r$) was -0.61 , based on the random-effects model. DOT and DP explained 14 and 25%, respectively, of the variability in $\bar{Z}_r$. Stronger

associations ($\bar{r} = -0.87$ and -0.90) were estimated by mixed-effects models for the Z_r data from studies with highest DP (DP $> 70\%$) and earliest rust onset (DOT $< R1$), respectively. Overall means (based on a random-effect model) for the regression coefficients $\bar{\beta}_0$ and $\bar{\beta}_1$ were 2,977 and 18 kg/ha/% $^{-1}$, respectively. In other words, S as low as 3% would reduce 60 kg/ha for an expected Y of 3,000 kg/ha. In relative terms, each unitary percent increase in S would lead to a 0.6 percentage point (pp) reduction in Y . The three categorical moderator variables explained some (5 to 10%) of the heterogeneity in $\bar{\beta}_1$ but not in $\bar{\beta}_0$. The estimated relative reduction in Y was 0.41 to 0.79 pp/% $^{-1}$ across seasons. Highest relative yield reductions (>0.73 pp/% $^{-1}$) were estimated for studies with DOT $< R1$ and DP $> 70\%$; the latter possibly due to high fungicide efficacy when DP is low, thus leading to higher yield differences between fungicide-protected and nontreated plots. The critical-point meta-analytic models can provide general estimates of yield loss based on a composite measure of disease severity. They can also be useful for crop loss assessments and economic analysis under scenarios of varying DOT and weather favorableness for epidemic development.

Soybean rust (SBR) is among the most economically important diseases of soybean (12,23). The disease is caused by *Phakopsora pachyrhizi* Syd. & P. Syd., a basidiomycete fungus first reported in Asia (3). Since its discovery in South America in 2001 (50), SBR has affected the soybean crop to a larger extent in Brazil than in any other country (23). The most effective control of this disease in Brazil has been achieved through the use of integrated management practices that include a soybean-free period of 60 to 90 days, sowing of early-maturing varieties, and sequential applications of fungicides that have been shown to effectively suppress the disease and reduce yield losses (22,27,28,40,50).

In botanical epidemiology, yield losses have been studied through the understanding of the relationships between disease intensity and yield (or yield loss) from field experiments, where disease levels are manipulated and assessed and the corresponding yield measured (39). Previous research on yield loss due to SBR have examined the effects of the disease on yield components, in most cases through the assessment of the empirical relationship between SBR and yield (13,46-48). Alternatively, process-based models that integrate a SBR simulation model into

a crop (soybean) model to predict yield have been developed (21,36,48).

Studies on the rust-yield empirical relationship identified disease- or crop-based variables that best explained yield or yield loss variation, such as leaf severity, lesion density, area under the disease progress curve, healthy leaf area duration, and percent defoliation (13,21,46-48). Furthermore, soybean yield was assessed in experiments where the disease levels were manipulated using artificial inoculation, irrigation, or artificial defoliation (13,21,43,46-48). In Brazil, uniform fungicide trials (UFTs) for evaluation of product efficacy have been conducted every year since the 2003-04 growing season. The availability of such data provides an opportunity to explore and analyze large amounts of data on the relationship between crop yield and SBR epidemics that have been manipulated through the use of fungicides.

In this study, these data were combined and analyzed under a meta-analytic framework, generally following the approach taken by Paul et al. (31,32). Meta-analysis is a statistical technique that has gained considerable attention by plant pathologists (26) and has been widely used in the social and health sciences and accepted as a valid and powerful research methodology to quantitatively integrate the results of a collection of primary studies in a given topic (2). In plant pathology, meta-analysis is an emergent technique that has been used mainly to summarize efficacy of management practices such as fungicide control and relationships between variables such as disease and yield and disease and mycotoxins (26,29).

Corresponding author: E. M. Del Ponte; E-mail address: delponte@ufv.br

*The e-Xtra logo stands for "electronic extra" and indicates that the online version contains supplemental material.

In the present study, we used original data (provided by researchers) of the UFTs conducted in Brazil, most of which have been published as yearly reports of fungicide efficacy (Supplemental material). The main objective of this study was to summarize and assess the heterogeneity of the relationship between crop yield and SBR using meta-analysis, which had three of its main common goals: (i) to test whether the relationship described by different effect sizes were homogeneous; (ii) to obtain a global index about the effect magnitude of the studied relation, respective confidence interval (CI), and its statistical significance; and (iii) to identify study-specific variables moderating the effects if significant heterogeneity among studies was present.

MATERIALS AND METHODS

Disease and yield data from UFTs. The UFT datasets consisted of data from 231 trials conducted during nine growing-seasons (2004–05 to 2012–13) across 57 locations from southern to northern Brazil (Table 1). Those trials were conducted under a wide range of climatic conditions, comprising major soybean growing areas in Brazil. Trials were conducted using standardized protocols that consisted primarily of two fungicide applications made at label-recommended rates during reproductive growth stages, usually beginning flowering (R1) and beginning seed (R5) (7). Soybean cultivars differed among trials but all were susceptible to SBR. In all cases, the crop was managed and weed and insect controlled following locally adapted commercial production practices. However, most trials were sown later in the growing season as a strategy for increasing the likelihood of an epidemic developing from inoculum that had built up in the earlier plantings. In all trials, the experimental design was a randomized complete block with four replications. Each replicate plot was at least six rows wide and 6 m long. The middle four rows were treated with a set of foliar fungicides evaluated in the UFTs following the methodology described elsewhere (40). In our extended dataset, most of fungicides evaluated included commercial mixtures of triazole and strobilurin (demethylation

TABLE 1. Attributes of 231 uniform fungicide trials for managing soybean rust conducted in Brazil during nine consecutive seasons

Season, state	Number of			DS ^b
	Studies	Treatments ^a	Sites	
Season				
2004–05	19	15	18	45.2
2005–06	19	8	16	58.5
2006–07	39	18	18	59.3
2007–08	11	10	10	47.4
2008–09	53	16	22	55.8
2009–10	29	13	23	60.2
2010–11	21	18	18	67.8
2011–12	17	16	16	63.3
2012–13	23	18	21	52.9
Total	231	132	55 ^c	57.3
State				
Bahia	7	...	...	40.1
Distrito Federal	9	...	...	56.6
Goiás	41	...	...	57.5
Maranhão	3	...	...	49.8
Minas Gerais	8	...	...	61.0
Mato Grosso	34	...	...	55.6
Mato Grosso do Sul	34	...	...	62.6
Paraná	45	...	...	57.0
Rio Grande do Sul	20	...	...	50.8
Santa Catarina	4	...	...	52.7
São Paulo	24	...	...	64.7
Tocantins	2	...	...	34.4
Total, mean	231	...	...	57.3

^a Numbers of treatments of annual standard protocol.
^b Average disease severity (DS) on untreated check.
^c Total number of unique locations.

inhibitor [DMI] + quinone outside inhibitor [QoI]) or triazole alone (DMI).

SBR developed naturally and epidemic levels were variable across the regions and years (Table 1). Disease and yield data were obtained from the center two rows of each plot. The presence or absence of symptoms was assessed just prior to fungicide application at R1 stage. Plot-level disease severity on leaves was estimated at full-seed stage (R6). Five plants were arbitrarily selected per plot and severity was assessed on three to four leaflets of each of the three canopy heights of each plant with the aid of a standard area diagram (10). In the UFT protocol, estimates of plot severity, conducted in a similar manner across in all trials, took defoliation into account. For instance, if a certain canopy layer was completely defoliated (no leaflet to sample, assuming that early defoliation was caused by the disease), disease severity was recorded as 100%. Therefore, mean plot severity was estimated as the average severity across canopy heights and plants. All plots were hand harvested and mechanically threshed and seed weight and moisture were obtained. Yields were calculated as kilograms per hectare at 13% moisture.

Criteria for exclusion of studies. Each trial was considered an independent, primary study in the meta-analysis. A critical step in meta-analysis, after the systematic review of primary studies addressing the question of interest, is the close inspection of the data and exclusion of studies according to defined criteria (2). First, studies lacking yield (Y) data were excluded ($n = 6$ studies). Second, for the summary of the linear regression coefficients, studies in which disease severity (S) was lower than 10% in the untreated check ($n = 15$) were discarded because one needs an adequate range of S to quantify the yield Y:S relationship (34).

Effect-sizes. The term effect-size in meta-analysis is used to denote the outcome measure. In our study, we used three outcomes to summarize the relationship between disease severity measured at the R6 growth stage and soybean yield. The first effect-size considered was the transformation of the Pearson correlation coefficient (r) (Fisher's Z) that summarized the strength of the relationship (31). For this analysis, the sampling variances were estimated for each study using data available at the plot level (usually four plots of the randomized block design) and not at the aggregate (treatment means) study level, because they were available (35).

The effect-size was calculated for 225 studies, after applying the first criteria of exclusion, using the PROC CORR procedure of SAS (38). For statistical reasons (26), the Fisher's Z, a transformation of r , was the estimated effect size used in the meta-analysis. Z is given by

$$Z_k = \frac{1}{2} \log \left(\frac{1 + r_k}{1 - r_k} \right) \tag{1}$$

where Z_k is the Fisher's transformation of the k th study ($k = 1, 2, \dots, n$) and r_k is the Pearson correlation coefficient of the k study. The sampling variance for each k study is given by

$$v_z = [1/(n_k - 3)] \tag{2}$$

where n_k is the number of pairs of S–Y observations in each k study. For example, a study with 15 treatments had 60 pairs (15 treatments $\times$ 4 blocks) of observations.

The other two effect-sizes considered were the intercept and the slope coefficients of the linear regression model estimated for each of 210 trials, after applying the second criteria of exclusion, using the PROC MIXED procedure in SAS. In the regression model for each study, block was treated as random effect and severity as a fixed effect:

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + \epsilon_{ij} \tag{3}$$

where y_{ij} and x_{ij} are yield (YLD) (kg/ha) and severity (SEV) (%), respectively, in the j block ($j = 1, 2, 3$, or 4) of the i treatment ($i = 1, 2, \dots, nj$); β_0 and β_1 are the intercept and the slope, respectively; and ε_{ij} is the random error associated with each y_{ij} observation. The residuals were assumed to be normally distributed.

The intercept (β_0) represents the estimated attainable yield, which corresponds to yield at zero SEV. The slope (β_1) represents the magnitude of increase or decrease in YLD (kg/ha) for each percentage point (pp) increase in SEV. Estimates of the variance (squared standard error) of the slope and intercept from the linear models were used as the sampling variance to estimate weights in the meta-analytic models.

Random-effect meta-analysis. We tested a standard univariate random effect and a bivariate random effect meta-analysis; the latter because two outcomes (slope and intercept) were estimated in the same study and, therefore, independence could not be assumed (44). The univariate meta-analysis was performed using metafor package of R (45) to estimate overall means and respective 95% CI independently for each effect size. We also calculated the 95% prediction intervals (45). The bivariate model was fitted to the intercept and slope data as described (32) but using the metafor package of R (45).

Preliminary results from these two approaches showed similar values for the mean estimates and their CIs (E. M. Del Ponte, *unpublished data*); therefore, only the results of the univariate meta-analysis are presented here. This may have been due to the low among-study correlation between the intercept and slope ($r = 0.23$) as determined by the bivariate model. In the random-effects model, averages of the effect sizes of all studies were estimated, assuming that the true effect could vary from study to study (14) and then providing an unconditional inference about a larger set of studies (studies that have been conducted, that could have been conducted, or that may be conducted in the future) from which the n studies included in the analysis are assumed to be a random sample, thus addressing the question of how large the true effect is in a larger population (45).

Heterogeneity analysis. We assessed whether there was heterogeneity among the true effect-sizes using the Cochran Q test, which was computed by summing the squared deviations of the effect estimate for each study from the overall effect estimate and weighting of each study by its corresponding inverse of variance (4). We also calculated the I^2 index that measures the extent of heterogeneity of the true effect-sizes (15). This is given by the difference between the result of the Q test and its degrees of freedom ($k - 1$) divided by the Q value itself, and multiplied by 100. Hence, the I^2 index gives the percentage of the total variability in a set of effect sizes due to heterogeneity of the true effect-sizes. Percentages of approximately 25, 50, and 75% would be indicative of low, medium, and high heterogeneity, respectively (15).

Mixed-effects models. Subgroup meta-analysis and meta-regression are models that incorporate one or more moderators (study-specific categorical or continuous covariates) that could account for at least part of the heterogeneity in the true effects (2). These models can be seen as special cases of general linear mixed-effect models (the studies are considered random factors and the covariates as fixed factors). The goal in using those models was to examine to what extent some moderators could explain the heterogeneity among effect-sizes found with the random effects model. A univariate approach was used in our study; that is, a separate mixed-effect model was fitted for each effect-size (slope and intercept) and included one moderator at a time.

In our analysis, we hypothesized that strength, direction, and magnitude of the relationship between disease severity and soybean yield (given by r and β_1), and the yield when severity was zero (given by β_0) are influenced by two key epidemiology-related variables: the time of disease onset relative to the flowering growth stage and the final level of disease. Because these two

variables are strongly dependent on the prevailing seasonal weather conditions (6), we hypothesized that the relationship was variable across growing seasons. These hypotheses were tested by fitting mixed-effects model (31,32), including one of the following categorical covariates, as follows: (i) disease onset time (DOT), with two levels: prior to flowering (<R1 growth stage) or during and after flowering ($\geq R1$), defined based on presence and absence, respectively, of the disease during the first application of fungicide around flowering for each study; (ii) disease pressure (DP), defined based on SBR severity in the untreated check, with three levels according to a previous meta-analysis of fungicide efficacy (40): high ($>70\%$ severity), moderate (>40 and $\leq 70\%$), and low ($\leq 40\%$); and (iii) growing season (SEAS), with nine levels (season 2004–05 to season 2012–13).

Model fitting. The random- or mixed-effects models were fitted using a two-step approach (33). First, the among-study variance was estimated via maximum likelihood (45). In the second step, the mean effect was estimated using weights based on the among-study variance and within-study variance, the latter being held fixed for each study (2,26,45). As a complement, Wald-type tests and CIs were obtained under the assumption of normality.

Relative yield loss. The random- and mixed-effects models provided mean estimates of β_0 and β_1 , representing the attainable yield and its reduction for a unit increase in pp severity, respectively. Because empirical damage functions are commonly reported in relative terms (percent increase in yield loss or percent yield reduction) (25,37,41), we divided the slope, or the damage coefficient (kg/ha/pp⁻¹), by the estimated intercept (kg/ha), both estimated by the meta-analytic models, and multiplied by 100 to calculate percent yield reduction. These calculations were performed for both the global coefficients estimated via the random-effects models and the coefficients estimated via the mixed-effects models that included a moderator variable.

RESULTS

Correlation and regression analysis in the primary studies.

Soybean yield was associated with rust severity across the studies, and the correlation coefficients varied from one study to another. The correlations were mostly negatives, ranging from -0.95 to 0.23 . For several studies, a strong negative association was found; the association was positive for only three studies (Fig. 1A). The distribution of the correlation coefficients was slightly skewed (Fig. 1A) but the distribution of Fisher's transformation of r was more symmetrical about the mean (Fig. 1B), justifying the use of the transformed values in the meta-analysis.

The significance of the linear regression fitted for the Y - S relationship data in 210 studies was very high ($P < 0.001$) for 159 studies, moderate ($0.001 < P < 0.05$) for 35 studies, and nonsignificant ($P > 0.05$) for 16 studies. The coefficient of determination of the linear regression models showed that at least 50% of the variation in Y was explained by variation in S in 43% of the studies. The linear regression parameter estimates varied from one study to another, ranging from 703.8 to 5,063 kg/ha for the intercepts (β_0) (Fig. 1C), and from 3.04 to -101.66 kg/ha/pp⁻¹ for the slopes (β_1) (Fig. 1D). All but one slope (3.04 kg/ha/pp⁻¹) was negative, with 56.2% being between -10.0 and -25.0 kg/ha/pp⁻¹ (Fig. 2A, gray lines).

Meta-analysis of the correlation and regression coefficients.

For all effect-sizes evaluated (z , β_0 , and β_1), the null hypothesis of homogeneity among studies was rejected. The mean Fisher's Z_r estimated by the random-effects model ($\bar{Z}_r$) was -0.7 (95% CI = -0.75 to -0.65), corresponding to a mean back-transformed correlation coefficient ($\bar{r}$) across all studies of -0.61 (95% CI = -0.6 to -0.57) (Table 2). The estimated mean effect size and the corresponding standard error, 95% CI, and 95% prediction interval for the intercept ($\bar{\beta}_0$) and slope ($\bar{\beta}_1$) are shown in Table 2. The mean yield at zero SBR severity was estimated to be 2,977 kg/ha (95%

CI = 2,862 to 3,093). For every percent increase in severity, there was, on average, an 18 kg/ha⁻¹ (95% CI = 19.4 to 16.6) decrease in yield (Table 2). The variability among the intercepts and slopes from individual studies and the mean estimates with their respective 95% CI and 95% prediction interval from the random-effects model are depicted in Figure 2A. The between-study variability (τ^2) estimated via maximum likelihood was very high for all effect-sizes, which was confirmed by the significance of the Q test and high values of the I^2 statistics, suggesting high level of heterogeneity (Table 2) (16).

Influence of study-specific moderator variables. The moderators DP, DOT, and SEAS, tested individually as categorical moderator variables in mixed-effect models, showed significant results on the Fisher's Z_r transformation of the correlation coefficient and on the slope ($P < 0.001$) but not on the intercept ($P > 0.05$) (Table 3). The percentage of the heterogeneity in the random-effects model that was accounted for in the mixed-effects model varied among models and effect-sizes (Table 3). For example, for the estimated mean transformed correlation coefficient ($\bar{Z}_r$), DP accounted for the highest heterogeneity (26%), followed by DOT (15%) and SEAS (12%). For the mean estimate of the slope, DOT and SEAS were the moderators that resulted in the greatest reduction in the magnitude of the between-study variance (9.1 and 10.2%, respectively). Therefore, considerable between-study variability still remained after the fit of the mixed models, especially for the mean estimate of the slope.

Considering DP as a moderator, the higher correlation between SBR and yield was found for trials conducted under the highest DP (−0.90; 95% CI = −0.97; −0.83); that is, when severity in the untreated plots was >70% (Table 4). When disease onset was included as moderator, higher correlation was found when rust onset was prior to flowering (−0.87 95% CI = −0.94; −0.79)

(Table 4). The magnitude of the correlation varied significantly among the seasons, with mean Fisher's $\bar{Z}_r$ and its back-transformation ($\bar{r}$) ranging from −0.54 (−0.49) to −0.95 (−0.73) for the 2004–05 and 2009–10 seasons, respectively (Table 4; Fig. 3). The low precision of the 2007–08 estimated $\bar{Z}_r$, as indicated by the widest 95% CI, was likely due to the small sample size (Fig. 3).

The magnitude of the effect of seasons on the overall mean slope was moderate and likely due to the most negative slope estimated for the 2009–10 season (Table 3; Fig. 2B). The mixed-effects model estimated mean slope values for the intermediate and high DP class similar to the overall mean estimated by the random-effects model (Tables 2 and 4). Although the categorical moderators did not affect overall mean intercepts, overall mean slopes were affected by DOT and DP (Tables 3 and 4; Figs. 2C and 3D).

Relative yield loss. Based on the overall mean intercept and slope, estimated by the random-effects model, the global relative damage coefficient was estimated to be 0.6 pp/%⁻¹. In other words, a reduction of 60 pp in the attainable yield would be expected, on average, for the maximum rust severity (100%). Considering the significant influence of disease onset on the relative damage coefficient, a reduction of 0.74 pp/%⁻¹ in yield was estimated when disease onset was prior to flowering and 0.51 pp/%⁻¹ when it occurred later in the season. For DP, the damage coefficients were 0.78 pp/%⁻¹, 0.59 pp/%⁻¹, and 0.54 pp/%⁻¹ for the low, medium, and high category of DP, respectively. In a hypothetical scenario, a fixed 20-pp reduction in yield would occur at a mean plot severity level of 27% for epidemics initiating early in the season and at a severity level of 39% for epidemics initiating later in the season (Fig. 4A). Moreover, such reduction would occur at a range between 26 and 37% severity, depending on the category of DP (Fig. 4B).

Fig. 1. Frequency distribution of **A**, Pearson correlation coefficients (r) ($k = 225$); **B**, Fisher transformation of r ($k = 225$); and the linear regression coefficients **C**, intercept ($k = 210$) and **D**, slope ($k = 210$) for the relationship between severity of soybean rust in R6 stage and crop yield in individual studies.

DISCUSSION

SBR is well known as a disease of major damage to crop yield, leading to losses as high as 40 to 80% in the field when environmental conditions are favorable for epidemics and no management action is taken (3,12,13,18,42,49,50). In this study, we corroborated these findings by showing that soybean yield was significantly correlated with SBR severity, where the highest estimated mean correlation coefficient was found for studies conducted under high DP. Moreover, we further quantified the strength of the relationship between yield and SBR based on both the correlation (Pearson's product moment) coefficient and the slope the linear regression line. These are two valid and complementary approaches that have been used in similar meta-analysis of the relationship between *Fusarium* head blight and

deoxynivalenol content in wheat (31,32). The linear regression analysis was particularly useful to determine the functional relationship between these two variables based on the slope and intercept of a straight-line equation fitted to the data. In fact, we found that the linear model provided a good fit to data from most trials, especially for those in which pairs of yield-severity were more evenly distributed across a wide severity range due to high variability in fungicide efficacy. In a few trials, the fungicides were similarly efficacious; thus, the severity values of treated plots were close together but very different (much lower) from the severity value of nontreated plots. Linear relationships between measures of disease intensity and yield have been previously reported for SBR (17,47,48) and also for other rusts such as common rust of sweet corn (30), wheat leaf rust (19), and white oat leaf rust (34).

Fig. 2. A, Parameters of the linear regression of the relationship between soybean yield and rust severity data for each of 210 individual trials prior to meta analysis (gray lines) and the estimated mean (black solid thicker line) and respective 95% confidence interval (black solid thin line) and 95% prediction interval (black dashed line) by a meta-analytic random-effects model. Mean parameters were also estimated by mixed-effects models for B, each growing season (where each line represents mean estimated parameter for the season); C, three disease pressure levels (high = >70%, moderate = >40 and ≤70%, and low = ≤40% severity in the check treatment); and D, two disease onset times (prior to or during and after flowering).

Through a random-effects meta-analytic model fitted separately for the correlation and regression coefficients from over 200 field trials, we found that there was a significant, negative straight-line relationship between plot yield at harvest and SBR severity quantified at approximately growth stage R6. Based on the mean intercept and slopes, for an expected yield of 3,000 kg/ha, a severity level as low as 3.3% would lead to a decrease of 60 kg/ha in the

yield. Indirect evidence that relatively low rust levels might reduce yield was found in fungicide evaluation studies in which preventative applications usually led to higher yield compared with curative applications (9,22,27,28,40).

Previous research on the empirical SBR–yield relationship focused extensively on the understanding of which and how components of yields were affected by the disease or which disease

TABLE 2. Estimates, related statistics and heterogeneity measures of the transformed and back-transformed Pearson correlation coefficients (CC) and linear regression slope and intercept for the relationship between soybean rust severity and crop yield based on separate random-effects meta-analytical models

Effect-size	k^a	Statistics ^b						Heterogeneity ^c		
		Estimate	SE	CI_L	CI_U	PI_L	PI_U	$\bar{\tau}^2$	P value	I^2 (%)
Pearson's CC ^d										
$\bar{Z}_r$	225	-0.70	0.026	-0.75	-0.65	-1.40	-0.002	0.127	<0.001	87.2
$\bar{r}$	225	-0.61	...	-0.64	-0.57	-0.88	-0.002	...	...	...
Linear regression ^e										
$\bar{\beta}_0$	210	2,977	58.9	2,862	3,093	1,316	4,638	714,839	<0.001	99.4
$\bar{\beta}_1$	210	-18.0	0.72	-19.4	-16.6	-37.5	1.14	94.9	<0.001	95.4

^a Total number of studies used in each analysis.

^b Mean estimate, standard error (SE), lower (CI_L) and upper (CI_U) limits of the 95% confidence interval, and lower (PI_L) upper (PI_U) limits of the 95% prediction interval.

^c Between-studies variance estimates ($\bar{\tau}^2$), P value of Cochran Q test, and I^2 statistic.

^d Meta-analysis of Fisher's $\bar{Z}_r$ obtained from meta-analysis of transformed Pearson's correlation of each trial and coefficient. $\bar{r}$ is the back-transformed of Fisher's $\bar{Z}_r$.

^e Intercept ($\bar{\beta}_0$) and slope ($\bar{\beta}_1$) of meta-analysis of functional relationship between soybean rust severity in R6 stage and crop yield.

TABLE 3. Significance, heterogeneity, and between-study variance reduction by the inclusion of moderators variables based on mixed-effects meta-analytical models for the relationship between soybean yield and soybean rust severity^a

Moderator ^b	Correlation			Linear regression					
	Transformed correlation (Fisher's $\bar{Z}_r$)			Estimated intercept ($\bar{\beta}_0$)			Estimated slope ($\bar{\beta}_1$)		
	P value	$\bar{\tau}^2$	VAF (%)	P value	$\bar{\tau}^2$	VAF (%)	P value	$\bar{\tau}^2$	VAF (%)
DP	<0.001	0.094	25.75	0.920	714,241.6	0.08	0.001	90.2	5.00
DOT	<0.001	0.110	14.88	0.163	725,542.5	1.04	<0.001	88.4	9.11
SEAS	0.001	0.111	12.36	0.070	668,066.5	6.54	0.017	85.2	10.20

^a P value of omnibus test (Wald type test); $\bar{\tau}^2$ = the between-study variance estimated by maximum likelihood; and variance accounted for (VAF) (following Vietchbauer [45]), is the percentage of the heterogeneity in the reduced, random-effects model (without-moderator) that is accounted for in the full, mixed-effect, model (with the specific moderator).

^b Categorical moderators: disease pressure (DP) = low, medium, and high; disease onset timing (DOT) = prior to R1 and after R1; and nine categories of season (SEAS) tested in mixed-effects models.

TABLE 4. Estimates and related statistics of the transformed Pearson correlation coefficients and linear regression slope and intercept for the relationship between soybean yield and soybean rust severity for each level of moderator variables included in separate mixed-effects meta-analytical models^a

Moderator ^b	Correlation				Linear regression							
	Transformed correlation (Fisher's $\bar{Z}_r$)				Estimated intercept ($\bar{\beta}_0$)				Estimated slope ($\bar{\beta}_1$)			
	k	ES	CI_L	CI_U	k	ES	CI_L	CI_U	k	ES	CI_L	CI_U
DP												
High	82	-0.90	-0.97	-0.83	82	2,979	2,794	3,164	82	-16.02	-18.14	-13.91
Med	77	-0.71	-0.78	-0.63	77	2,951	2,760	3,141	77	-17.48	-19.71	-15.24
Low	66	-0.45	-0.53	-0.36	51	3,014	2,780	3,248	51	-23.45	-26.65	-20.25
DOT												
>R1	120	-0.58	-0.65	-0.52	107	3,065	2,902	3,228	107	-15.69	-17.61	-13.76
<R1	93	-0.87	-0.94	-0.79	92	2,895	2,719	3,071	92	-21.41	-23.49	-19.34
SEAS												
2004–05	19	-0.54	-0.70	-0.38	16	2,669	2,267	3,072	16	-16.67	-21.59	-11.75
2005–06	19	-0.84	-1.01	-0.67	18	2,780	2,397	3,162	18	-17.92	-22.61	-13.23
2006–07	39	-0.72	-0.83	-0.60	38	3,042	2,778	3,306	38	-20.53	-23.72	-17.33
2007–08	10	-0.55	-0.78	-0.32	9	2,710	2,169	3,250	9	-20.17	-27.07	-13.28
2008–09	53	-0.59	-0.69	-0.50	49	2,828	2,597	3,058	49	-15.48	-18.30	-12.66
2009–10	28	-0.95	-1.08	-0.81	26	2,981	2,663	3,299	26	-23.69	-27.53	-19.84
2010–11	21	-0.78	-0.94	-0.63	21	3,429	3,076	3,783	21	-16.78	-20.97	-12.58
2011–12	15	-0.67	-0.85	-0.49	15	3,272	2,854	3,689	15	-13.48	-18.35	-8.62
2012–13	21	-0.67	-0.82	-0.52	18	3,073	2,694	3,452	18	-16.44	-20.89	-11.99

^a Number of trials (k) for each level in each moderator, effect size (ES) estimates for each level of each moderator, and their respective 95% confidence intervals (CI_L and CI_U).

^b Moderator variables using in mixed-model meta-analysis: disease pressure (DP) = disease in untreated treatment at the R6 growth stage (High = severity >70%, Med = severity between >40% and ≤70%, and Low = severity ≤40%), disease onset timing (DOT) = prior to R1 growth stage (>R1) and after R1 (<R1), and SEAS = soybean growing season.

Fig. 3. Back-transformed Pearson correlation coefficients (from Fisher's Z) for the relationship between severity of soybean rust in R6 and grain yield estimated by random-effects (vertical line) and mixed-effects meta-analytic model including one of the three moderator variables: disease pressure (DP): high (>70% severity), moderate (>40 and ≤70%), and low (≤40%); disease onset time (DOT): prior to flowering (<R1 growth stage) or during and after flowering (≥R1), defined based on presence and absence, respectively, of the disease during the first application of fungicide around flowering for each study; and growing season (SEAS): nine consecutive growing seasons (2004–05 to 2012–13).

or crop-related variables would best explain yield losses (13,20, 21,47,48). For example, the analysis of data from a series of field trials conducted in Taiwan showed that relative yield loss was best explained ($R^2 = 0.58$ to 0.46) by the relative area under the severity progress curve and area under the defoliation progress curve, among other variables (47). In another study, the area under the green leaf area (not the diseased area) progress curve, which considered both the foliar severity and defoliation, explained from 52 to 85% of the variation in yield loss (13). A 2-year study conducted in Brazil showed that two variables, area under severity progress curve and a normalized difference vegetation index, were highly correlated with yield, with mean correlation coefficients of 0.83 and 0.75, respectively (17).

In general, our results corroborate these findings, although a direct comparison between the measures of association and damage coefficients from our study and those reported in the literature are not straightforward. First, we analyzed a large dataset of SBR epidemics that developed naturally across a range of production situations in which disease gradients were generated through the use of fungicides with varying efficacy. Moreover, the majority of trials received a maximum of only two applications; therefore, plots were not protected as needed or with a high number of sprays that are known to provide better control and protect yield (40). Second, rust severity estimation took into account both severity for leaves remaining on the soybean plant and early defoliation due to the rust. Finally, severity was estimated at one time in the course of epidemics, just prior to maturity (around R6 stage); thus, data were not available to calculate integrative variables such area under curve, which has been found to be a better predictor of yield in a previous yield loss study on SBR (13,48) as well as in many other pathosystems (24). Leaf area duration has also been found to better explain variation in soybean yield than other measures (20,21).

We showed that the degree of the correlation and the functional relationship, summarized by the regression coefficients, were significantly affected by epidemiology-related moderator variables, while slightly affected or unaffected by season. The significant effect of growing season as a moderator variable in the mixed-effects model relates to varying environmental conditions across years, which affects both the disease and the crop, thus likely

Fig. 4. Relative yield loss by **A**, three categories of disease pressure and **B**, two disease onset times based on estimated parameters from meta-analysis of the relationship between soybean rust severity at the R6 growth stage and yield. Horizontal gray line represents 20% yield loss, whereas the vertical gray lines show the level of severity at which 20% yield loss occurred for each level of each variable.

affecting the relationship. For example, a severe drought occurred in southern Brazil during the 2004–05 season, which affected soybean yield and SBR epidemics (5). Hence, the association between the disease and yield in the trials of that season was probably weak and the large CI for the mean correlation coefficient was due to large variation across locations. Other abiotic and biotic factors may have also affected yield. Conversely, higher associations found for the 2009–10 season were likely related to the above-normal seasonal rain (data not shown), which is beneficial to both crop growth and disease development (6), thus leading to a high difference in the yield response between protected and unprotected plots.

We found that DOT and DP significantly explained the variability among correlation coefficients and slopes while not affecting intercepts. The disease–yield association was stronger in trials conducted under high DP and with early onset of the epidemics and much weaker for trials conducted under low DP and late disease onset. The higher correlation and maximum yield loss potential of around 75% for trials conducted under such conditions are similar to those reported in the literature (21,27,28). Higher associations between SBR and yield in severe epidemic situations have been documented. Correlation coefficients ranging from 0.79 to 0.9 were found in 1986, a severe epidemic year, and from 0.01 to 0.49 in 1987, a year with light epidemics in Taiwan (48).

We found that, for trials conducted under lower DP, the relative mean rate of yield reduction was higher for unitary increase of percent severity compared with trials conducted under more conducive environments for epidemics. This can be due to the fact that fungicides are more efficacious under low DP (38), thus leading to higher response in yield and explaining steeper slopes. For trials grouped by the time of disease onset, the overall mean rate of yield reduction per percent increase in severity was greater when disease onset was prior to flowering. Studies have shown that, when SBR becomes established during vegetative or early reproductive stages, several yield components may be negatively affected, causing reduced number of pods per plant, increased pod abortion, reduced seed growth rate, and reduction of the number of days between R4 and R7 (46,48). Analyzing data from natural epidemics of SBR, Kumudini et al. (21) observed a 68% reduction in yield when infection initiated at the R2 growth stage (full flowering), while a 39% reduction in yield was observed when infections occurred at the R5 growth state (beginning seed). The impact of early SBR onset on yield was further confirmed in a study on the effect of manually induced defoliation from the lower to the upper leaves, mimicking SBR, during the seed filling period. Total defoliation during early seed filling caused the greatest yield loss (78%), compared with defoliation at late seed filling (21). Our results add to those observations in that slopes for the yield loss–SBR relationship were affected by onset time.

Through meta-analysis, we were able to estimate the overall mean damage caused by SBR on soybean yield and also identify epidemiological variables that explained a portion of the variability in the disease–yield relationship. However, a great portion of the variability remained unexplained and might be related to unknown factors or factors inherent in the study design. We examined the disease–yield relationship in trials that used several commercial fungicides with different effects on the disease and yield. The effects of QoI fungicides on yield are known (1,11) and most studies in the UFTs included treatments that consisted of commercial mixtures of DMI and QoI. In this study, we did not categorize the trials based on fungicide active ingredient (as moderator variable) because a trial that tested only DMI, QoI, or the mixture was not available. In most cases, there was a mixture of these fungicides in the treatments. If only specific treatments had been selected from a single trial, fewer disease–yield pairs would be left to model the relationship than using the whole set of fungicides evaluated in the primary studies. Other factors related to the host and the pathogen such as differences in cultivar sus-

ceptibility and pathogen populations across regions may have also influenced the relationship and contributed to the variability. In the 9-year dataset used in this study, more than 50 different varieties were used. Although these were all susceptible to SBR, quantitative information pertaining to other traits was not available.

In summary, there was a significant negative linear relationship between soybean yield and SBR severity, and intercept and slopes were highly variable. Epidemiological factors such as the time of disease onset and final disease severity explained a portion of the variability in the rate of yield reduction per unit increase in SBR. In order to accurately assess yield loss, a critical step is to identify an appropriate metric in empirical modeling (8,39). The damage coefficients estimated in our study were based on a large dataset from fungicide trials conducted over 9 years under a range of conditions. As such, these models may provide more general estimations of yield loss based on SBR severity, and the large variability encountered may preclude accurate site-specific prediction of actual yield due to SBR. Yet, results from this study may provide useful information for regional risk assessment of potential yield loss if rust severity is measured on site or estimated using disease models considering different scenarios of DOT and DP (6).

ACKNOWLEDGMENTS

We thank The National Council for Scientific and Technological Development (CNPq) for providing a scholarship to F. Dalla Lana and a research fellowship to E. M. Del Ponte; many researchers (institutions) who have contributed data, including A. D. Roese (Embrapa Agropecuária Oeste), C. A. Forcelini (Universidade de Passo Fundo), C. M. Utiamada and L. N. Sato (Tagro), D. Cassetari Neto (Universidade Federal do Mato Grosso), E. P. Borges (Fundação Chapadão), F. C. Juliatti (Universidade Federal de Uberlândia), F. V. Siqueri (Fundação Mato Grosso), H. R. Feksa (FAPA), J. Nunes, Jr. and C. B. Pimenta (CTPA/Emater), L. H. C. P. da Silva and H. D. Campos (UniRV), L. G. Carneiro (Universidade Federal de Goiás), L. M. Costamilan (Embrapa Trigo), M. D. Dias (Universidade Federal do Tocantins), M. Madalosso (Instituto Phytus), M. C. Martins (Faculdade Arnaldo Horácio Ferreira), M. C. Meyer (Embrapa Soja), M. F. Ito, M. A. Ito, and E. U. Ramos, Jr. (IAC/DDD/APTA), M. G. Canteri (Universidade Estadual de Londrina), R. Borges and J. F. J. Grigolli (Fundação MS), R. S. Balardin (Universidade Federal de Santa Maria), S. A. da Silva (Embrapa Cerrados), S. H. Furlan (Instituto Biológico), T. D. N. Montecelli (Coodetec), V. J. Carlin (Agrodinâmica), W. S. Venâncio (CWR Pesquisa Agrícola Ltda), D. S. Miguel-Wruck (Epamig); and C. D. M. Nunes (Embrapa Clima Temperado); and P. A. Paul (Ohio State University) for critically reviewing the manuscript prior to submission.

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