Meta-analytic modelling of the incidence–yield and incidence–sclerotial production relationships in soybean white mould epidemics

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White mould (\textit{Sclerotinia sclerotiorum}) is a destructive disease of soybean worldwide. However, little is known of its impact on soybean production in Brazil. A meta-analytic approach was used to assess the relationship between disease incidence and soybean yield (35 trials) and between incidence and sclerotia production (29 trials) in experiments conducted in 14 locations across four seasons. Region, site elevation and season included as moderators in random-effects and random-coefficients models did not significantly explain the variability in the slopes of the incidence–yield relationship. The Pearson’s $r$, obtained from back-transforming the Fisher’s $Z$ estimated by an overall random-effects model, showed that incidence of white mould was moderately and negatively correlated with yield ($r = -0.76$, $P < 0.0001$). A random-coefficients model estimated a slope of $-17.2$ kg ha$^{-1}$%$^{-1}$, for a mean attainable yield of 3455 kg ha$^{-1}$, indicating that a 10% increase in white mould incidence would result in a mean yield reduction of 172 kg ha$^{-1}$. White mould incidence and production of sclerotia were strongly and positively correlated ($r = 0.85$, $P < 0.0001$). For every 10% increase in white mould incidence, 1 kg ha$^{-1}$ of sclerotia was produced. The relationship between disease incidence and production of sclerotia was stronger in southern regions and at higher elevation. In the absence of management, economic losses associated with white mould epidemics, assuming 43% incidence in 22% of the soybean area, were estimated at approximately US $1.47 billion annually within Brazil.

Keywords: \textit{Glycine max}, meta-analysis, \textit{Sclerotinia sclerotiorum}, sclerotinia stem rot

Introduction

White mould is one of the most damaging diseases of soybean worldwide (Grau & Hartman, 1999). In Brazil, the disease causes significant crop losses, with highly prevalent and severe epidemics in production regions at high ($>600$ m a.s.l.) elevations (Meyer \textit{et al.}, 2014). In the USA, annual economic loss estimates due to white mould increased from $10$ million to $560$ million between 1996 and 2009, and was considered the second most important disease of soybean (Peltier \textit{et al.}, 2012).

White mould is caused by \textit{Sclerotinia sclerotiorum}, an ascomycete fungus with a broad host range including crops such as beans, canola, sunflower, tomato, potato and cotton (Boland & Hall, 1994). The fungus is able to survive as sclerotia, which are long-term survival structures with an outer black rind containing melanin (Bolton \textit{et al.}, 2006). Sclerotia are abundantly produced on diseased soybean tissue and can remain viable for up to 8 years in soil (Adams & Ayers, 1979). Sclerotia can germinate myceliogenically and grow as vegetative hyphae and directly infect some plants, such as in stem rot of canola. Alternatively, sclerotia can germinate carpogenically, resulting in the production of apothecia and liberation of ascospores. These spores can be dispersed via air currents resulting in infection of blossom tissues, which are used as a nutrient source to enable growth and infection of other green tissues (Abawi & Grogan, 1979). Infected petals deposited on leaves serve as inoculum sources for mycelia that can directly infect healthy tissues. However, the disease typically has a monocyclic progression because of the rare or minimal contribution of secondary inoculum (mycelia) to plant-to-plant spread (Grau & Hartman, 1999).

The intensity of white mould is usually quantified as incidence, or the proportion of plants with symptoms. White mould intensity can also be quantified as severity, or the proportion of tissue area affected by the disease (Madden \textit{et al.}, 2007). The latter is commonly estimated using an ordinal rating scale (Hall & Phillips, 1996), or a severity index calculated based on the frequency of the ordinal scores (Kolkman & Kelly, 2002). A standard area diagram has also been developed to aid visual assessment of white mould severity in soybean (Garcia & Juliatti, 2012). Other disease-related variables such as the number of sclerotia per seed weight and the number of apothecia per m$^2$ on the soil surface have also been
used as an indirect measure of white mould intensity (Huang et al., 2000; Zeng et al., 2012). Nevertheless, incidence remains a less subjective and rapid method of describing white mould intensity for field assessments, with utility for comparing management tactics, such as fungicide efficacy or evaluating resistance (Hoffman et al., 1998; Yang et al., 1999; del Río et al., 2007).

The empirical relationship between white mould incidence and yield has been described for common bean (del Río et al., 2004), soybean (Chun et al., 1987; Hoffman et al., 1998; Yang et al., 1999; Danielson et al., 2004) and canola (del Río et al., 2007). These studies were conducted in the mid-western USA with markedly different microclimates, cropping systems and soybean genotypes compared to Brazil. The analysis of the coefficients of the linear regression models fitted to soybean field data shows relative reductions ranging from 4.6% to 8.4% in attainable yield for each 10% increase in white mould incidence across nine independent experiments (Chun et al., 1987; Hoffman et al., 1998; Yang et al., 1999). It is not known whether these models have predictive value for soybean production in Brazil, where soybean is grown in subtropical and tropical climates and white mould is intensively managed with fungicides. Soybean production in tropical regions is increasing annually, accelerating the need to model crop loss relationships in these environments to fully understand the impact of white mould on actual yield.

In Brazil, trials have been conducted since 2008 using a standardized research protocol to evaluate fungicide efficacy against white mould across several years and regions. Results of these studies have been summarized for each trial and published as summaries of fungicide efficacy for white mould control in soybean (Meyer et al., 2014). Available summary statistics at the trial level can be selected using defined criteria, to extract and combine data to address specific questions using meta-analysis. The latter is a quantitative method of combining and summarizing results of individual studies using a statistically robust framework (Madden & Paul, 2011; Scherm et al., 2014). There are multiple examples across a range of pathosystems focusing on quantitative summaries of treatment effects on disease reduction and increases in yield (Ojiambo et al., 2010; Ngugi et al., 2011; Paul et al., 2011) and on relationships between disease and agronomic-related variables such as yield, disease severity and mycotoxin concentration in other pathosystems (Paul et al., 2005, 2006; Shah & Dillard, 2006; Madden & Paul, 2009; Dalla Lana et al., 2015). Traditionally, meta-analysis is usually preceded by a systematic review of multiple literature sources (Shah & Dillard, 2006; Scherm et al., 2014). However, results may also be obtained directly from researchers or extracted from summaries in non-peer reviewed literature and analysed using data at the individual level (plots) or aggregated (treatment means) (Paul et al., 2006; Dalla Lana et al., 2015).

This study synthesized aggregated data (means across replicated plots) from multiple field trials designed to evaluate fungicide efficacy for white mould control over 4 years in Brazil (Meyer et al., 2014). The primary objective of the study was to summarize and quantify the heterogeneity of the relationships between mean estimates of white mould incidence and soybean yield or production of sclerotia. Quantitative knowledge of the disease–yield relationship and contribution of inoculum to future growing seasons can assist in crop loss assessment studies and in practical disease management decisions for white mould (del Río et al., 2004).

**Materials and methods**

**White mould incidence, soybean yield and sclerotia production**

Data on the estimated mean white mould incidence, soybean yield and weight of sclerotia were obtained from the tables of published reports of 36 trials conducted in Brazil during four growing seasons (2008/9 to 2011/12; Meyer et al., 2014). The trials were conducted in 14 municipalities located in regions of six states where white mould was previously recorded in years prior to the experiments (Fig. 1). Together, these states comprise approximately 70% of the soybean production in Brazil (CONAB, 2016). Field-specific information for the trials selected for this study is described in Table S1.

![Figure 1](image.png)

**Figure 1** Location and names of the municipalities where 35 uniform fungicide trials were conducted and white mould incidence was recorded and related to sclerotia weight and soybean yield. The size of the circle is proportional to the mean incidence in the untreated control plot across trials and years. Refer to Table S1 for other field-specific information.
Trials were conducted following a standard protocol as described by Meyer et al. (2014). Briefly, fungicides were applied two to four times during the season using a backpack sprayer pressurized by CO₂. The spray volume ranged from 200 to 300 L ha⁻¹. The first fungicide application occurred between the R1 and R2 growth stages and subsequent applications were made at approximately 10-day intervals. In some trials, a mixture of azoxystrobin and cyproconazole (60 + 24 g a.i. ha⁻¹) was used to control soybean rust across the entire trial area. In all trials, the experimental design was a randomized complete block with four replications, including a nontreated control. Each plot was 6 m long × 4 rows wide (12 m²).

White mould incidence was quantified between the R5 and R6 growth stage as the percentage of diseased plants within the two central rows of each plot. Sclerotia produced during the white mould epidemics were collected and weighed after threshing. Sclerotia incidence and sclerotia – incidence relationships, respectively.

Disease and yield variables

Three dependent variables were obtained from each study: white mould incidence (%), crop yield (kg ha⁻¹) and weight of sclerotia (g ha⁻¹), all available as the mean across four replicated plots for each trial (Meyer et al., 2014). Two relationships, the yield-incidence and sclerotia – incidence relationships, respectively.

Establishment of exclusion criteria for studies

Data from each of the 36 studies constituted an independent study in the meta-analysis. The data were explored and one study was excluded because of a too narrow range (5%) between the minimum and maximum incidence, which was considered insufficient to reliably quantify the relationship. Six studies did not provide information on sclerotial weight. Hence, data from 35 and 29 studies were analysed for the study of the yield–incidence and sclerotia–incidence relationships, respectively.

Disease and yield variables

Three independent variables were obtained from each study: white mould incidence (%), crop yield (kg ha⁻¹) and weight of sclerotia (g ha⁻¹), all available as the mean across four replicated plots for each trial (Meyer et al., 2014). Two relationships, the yield-incidence and sclerotia – incidence, were explored and the estimated coefficients and statistics constituted the effect size within each study. Three effect sizes per study were summarized for each relationship, including the Fisher’s Z (Z) transformation of the Pearson correlation coefficient (r) that summarizes the strength of the relationship (Paul et al., 2005; Dalla Lana et al., 2015). Pearson’s r was calculated for each study using the cor.test function within R. The Fisher’s Z, was calculated from r and number of pairs of the relationship (Paul et al., 2005) using the escalc function of the metafor package of R. The two other effect sizes tested were the intercept and slopes of the relationship estimated by two different approaches as described below.

Meta-analytic models

Correlation coefficients

For the Fisher’s Z, and respective sampling variances calculated (Paul et al., 2005; Dalla Lana et al., 2015), a standard univariate random-effect meta-analysis was performed using the rma function of the metafor package of R with parameters estimated via maximum likelihood. Overall means and 95% confidence interval (95% CI) and prediction intervals (95% PI) were calculated (Dalla Lana et al., 2015). Heterogeneity among the true effect sizes was evaluated based on significance of the Cochran Q test and the I² index that measures the extent of heterogeneity of the true effect sizes (Higgins & Thompson, 2002).

Relative yield loss estimation

The scale of the estimated slopes was kg ha⁻¹ per unitary increase in white mould incidence. Because damage functions are commonly reported in relative terms (% increase in yield loss or % yield reduction), and also for the purpose of comparison with other studies, the percentage yield loss was calculated by dividing the estimated slope (kg ha⁻¹%⁻¹) with the estimated intercept (kg ha⁻¹), both derived from the fit of random-effects model, and multiplying by 100 (Madden & Paul, 2009; Dalla Lana et al., 2015).
Results

Study-level variables and relationships

There was substantial variation in white mould incidence (15.4–90.3%), maximum soybean yield (1451–4056 kg ha$^{-1}$) and sclerotal weights (461–9480 g ha$^{-1}$) in the non-treated control treatments among the studies (Fig. 2; Table S1). The fit of the linear regression at the study level demonstrated that, in general, yield decreased as white mould incidence increased (Figs 3c & S1). The gradients in white mould incidence were due to variability in the efficacy of the fungicide treatments in reducing disease intensity. In some cases (e.g. trials 5 and 32; Fig. S1), the most effective treatment (lower incidence) did not result in the highest yield within the study. In other cases, fungicides with a similar level of disease control led to variable yield (e.g. trials 13 and 27; Fig. S1).

The amount of sclerotia produced generally increased with the increase in white mould incidence across all trials, but the slopes of these relationships within individual trials were highly variable (15.3–406.9 k ha$^{-1}$%–1) (Fig. 4c; Table S3). There were always some sclerotia present within the plots even in the treatments with lowest incidence, because the fungicides were not 100% effective. The maximum amount of sclerotia produced varied greatly across the trials. In some cases (e.g. trials 5 and 32; Fig. S2), maximum sclerotal production did not occur in the treatments with highest incidence within the study. In general there was a gradient in sclerotial production with a few exceptions (e.g. trials 21 and 23; Fig. S2). In these cases, the fungicides reduced the sclerotal production similarly and at lower levels than in the non-treated control treatment (Fig. S2).

Yield–incidence relationship

Correlation coefficients

Pearson’s correlation coefficients ($r$) varied from –0.96 to 0.11 (Fig. 3a). However, the null hypothesis of homogeneity in the estimated mean Fisher’s $Z$ ($Z_r$) among studies was rejected ($P = 0.0029$). The estimated $Z_r$ by the random-effects model varied from –1.90 to 0.11 (Fig. 3b) and the mean was $–1.02$ (95% CI = $–0.85$ to $–1.16$), corresponding to a mean back-transformed correlation coefficient across all studies of $–0.76$ (95% CI = $–0.36$ to $–0.92$). The between-study variability ($\tau^2$) estimated using maximum likelihood was high ($\tau^2 = 0.095$, SE = 0.0531) and confirmed by the significance of the $Q$ test ($Q = 61.17$, d.f. = 34, $P = 0.002$), and high values of the $I^2$ (42.97%) and $H^2$ (1.75) statistics. In the mixed-model, none of the categorical or continuous (year) moderator variables tested individually as fixed effects significantly affected $Z_r$ ($P > 0.3$).

Intercept and slopes

The random-coefficients model successfully fitted the data across the 35 studies. The estimated mean for the slope and intercept was highly similar between the two modelling approaches (data not shown). Estimates of the population-average intercept and slope were $\beta_0 = 3455.7$ kg ha$^{-1}$ (SE = 132.93) and $\beta_1 = 17.24$ kg ha$^{-1}$%–1 (SE = 1.45), respectively. Moreover, both estimated parameters differed significantly from zero ($P < 0.001$). The estimates of the random effects variances of the intercept and slope were $\sigma_{\beta_0}^2 = 602$ 192.89 and $\sigma_{\beta_1}^2 = 37.60$, respectively. The estimated covariance was $\sigma_{\beta_0,\beta_1} = –1477.49$. The among-study correlation of the effects of study on the intercept and slope was $–0.31$, estimated based on the between-study variance components. The estimated best linear unbiased predictions (EBLUPs) for the intercepts ($\hat{b}_0$) ranged from 1821 to 4888.9 kg ha$^{-1}$ (Table S2), and the interdecile (ID) range was 1900 kg ha$^{-1}$. The ID was similar between estimates by the simple linear model and random-coefficients model, depicted by similar density curves (Fig. S3). The EBLUPs for the slopes ($\hat{b}_1$) ranged from 7.57 to 28.02 kg ha$^{-1}$%–1 (Table S2), and the ID was 10.41. This range was smaller than for the slopes from fitting the simple linear model to the data, for which the distribution was wider than the

![Figure 2](https://example.com/fi.png) Histograms for the observations of white mould and soybean variables in 35 independent uniform trials (study) conducted in Brazil from 2008/9 to 2011/12. White mould mean incidence in the control treatment of the study (a); maximum mean soybean yield in one fungicide treatment of the study (b); maximum mean sclerota weight in one treatment of the study (c).
EBLUPs (Fig. S2b). The observations and the fit of the linear model for 35 individual regressions are shown in Figure 3c (individual plots in Fig. S1). The study-specific prediction lines and population-average predictions of yield and respective 95% CI are shown in Figure 3d. The conditional (pseudo-) \( R^2 \) of the entire model and the Akaike information criterion (AIC) was 0.94 and 5319.29, respectively. Similar to Fisher’s \( Z \), none of the moderator variables significantly affected the population-average predictions of yield. Based on the overall mean (95% CI) of the intercept and slope estimated by the random-coefficients model, the overall relative damage coefficient was estimated to be 0.49%\(^{-1}\) (0.45–0.54).

Sclerotia–white mould incidence relationship

Correlation coefficients

The Pearson’s correlation coefficients varied from 0.37 to 0.99 (Fig. 4a). The null hypothesis of homogeneity in the estimated mean Fisher’s \( Z \) (\( Z_r \)) among studies was rejected. The estimated \( Z_r \) by the random-effects model varied from 0.39 to 2.94 (Fig. 4b) and the mean was 1.25 (95% CI = 0.085–14.75). This value corresponded to a mean back-transformed correlation coefficient across all studies of 0.85 (95% CI = 0.79–0.89, 95% PI = 0.58–0.95). The between-study variability (\( \tau^2 \)) estimated using maximum likelihood was high (\( \tau^2 = 0.0819, \) SE = 0.0549) and confirmed by the significance of the \( Q \) test (\( Q = 48.68, \) d.f. = 28, \( P = 0.009 \)), and high values of the \( I^2 \) (39.24%) and \( H^2 \) (1.65) statistics. In the mixed-model, the inclusion of region significantly affected \( Z_r \) (\( P < 0.003 \)) and accounted for 62% of the heterogeneity. Stronger associations were estimated in trials within the southern region (\( r_Z = 0.93, \) 95% CI = 0.87–0.96, 95% PI = 0.83–0.97) compared to the trials in the northern region (\( r_Z = 0.81, \) 95% CI = 0.75–0.86, 95% PI = 0.64–0.90). Elevation was marginally significant (\( P = 0.06 \)) and accounted for 26.98% of the heterogeneity of \( Z_r \). Stronger associations were found in fields at higher elevation (\( r_Z = 0.88, \) 95% CI = 0.82–0.92, 95% PI = 0.70–0.95) than the alternative (\( r_Z = 0.80, \) 95% CI = 0.70–0.86, 95% PI = 0.51–0.92). Year, incidence and yield classifications did not significantly affect \( Z_r \) (\( P > 0.5 \)).

Intercept and slopes

Estimates of the population-average intercept and slope were \( \beta_0 = 280.5 \) g ha\(^{-1} \) (SE = 163) and \( \beta_1 = 98.59 \) g ha\(^{-1}\)%\(^{-1} \) (SE = 13.3), respectively, and both estimated parameters differed significantly from zero (\( P < 0.001 \)). The estimates of the random effects variances of the intercept and slope were \( \sigma_{\beta_0}^2 = 431 \) 433 and \( \sigma_{\beta_1}^2 = 4201 \), respectively. The estimated covariance was \( \sigma_{\beta_0,\beta_1} = 40 \) 115. The among-study correlation of the effects of study on the intercept and slope was 0.94, estimated based on the between-study variance components. The EBLUPs for the intercepts ranged from –498.7 to 2312.8 g ha\(^{-1} \) (Table S3), and the ID was 1420.5. The ID of the predictions for the intercepts by random-
coefficients model was lower than the estimates at the study level with the simple linear model (ID = 1793.5),
but the density curves were similar (Fig. S4a). The EBL-UPs for the slopes ranged from 18.4 to 307.8 g ha\(^{-1}\) %\(^{-1}\) (Table S3), and the ID was 145.87, which was slightly smaller from fitting the simple linear model to the data (ID = 163.71; Fig. S4b). Observations and the fit of linear model for the 29 individual linear regressions and the study-specific prediction lines are show in Figure 4c (individual plots in Fig. S2). The population-average predictions of yield and respective 95% CI are presented in Figure 4d. The conditional (pseudo-) R\(^2\) of the entire model and the AIC was 0.90 and 5379, respectively. None of the moderator variables significantly affected the population-average intercept and slope.

Discussion

The strength and heterogeneity of relationships between white mould incidence and soybean yield or sclerotial production was summarized using multilevel and random-effects meta-analytic models, following approaches previously used to address related questions in plant pathology (Madden & Paul, 2009; Dalla Lana et al., 2015). To the best of the authors’ knowledge, this is the first description of these relationships for soybean grown in Brazil, and provides a useful approach to explore these relationships in other subtropical and tropical regions of soybean production. The wide range of microclimates where the trials were conducted, spanning four seasons and several growing regions, may be one factor contributing to the wide variation in mean white mould incidence, production of sclerotia and soybean yield in non-treated control plots across the locations.

As expected, while soybean yield tended to decrease, sclerotial production tended to be positively associated with white mould incidence. Similar negative relationships between yield and white mould incidence have been reported for soybean (Chun et al., 1987; Hoffman et al., 1998; Yang et al., 1999; Danielson et al., 2004), dry bean (del Río et al., 2004) and canola (del Río et al., 2007), using correlation and regression analyses. In soybean, Pearson’s correlation coefficients reported for nine trials conducted in the USA varied from 0.67 to –0.94, with a mean r of –0.84 across three studies (Chun et al., 1987; Hoffman et al., 1998; Yang et al., 1999). This is higher than the mean estimated correlation coefficient obtained in the present study (r = –0.76). Differences among studies are expected and may be due to field-specific conditions, but also the approach used to generate variation in disease incidence gradients. In this study, disease gradients were generated using fungicides with various efficacy levels for white mould control. In the previous studies, disease gradients were obtained by three different approaches: (i) selecting sub-areas within a larger plot representing variable white mould incidence (Hoffman et al., 1998), (ii) using 16 or 20 cultivars with variable white mould susceptibility, and artificial
inoculation of fields (Chun et al., 1987), (iii) using 63–64 cultivars in three small-plot, replicated trials relying upon natural soilborne inoculum (Yang et al., 1999). Hence, in addition to the differential effect of fungicides it is likely that the different cultivars used in the Brazilian trials may have contributed to the variability associated with the incidence–yield relationships.

Results reported here corroborate previous reports claiming white mould as one of the most damaging diseases of soybean (Peltier et al., 2012). The mean estimate of potential maximum relative crop loss of 50% is only slightly lower than similar estimations made for soybean rust (60% crop loss; Dalla Lana et al., 2015). For a mean attainable yield of 3455 kg ha\(^{-1}\) (estimated intercept in this study), an average of 172.4 kg ha\(^{-1}\) would be lost for every 10% increase in white mould incidence. In relative terms, this translates into a decrease of 4.9% in attainable yield for each 10% unitary increase in white mould incidence. This relative loss estimate is lower than findings reported in studies conducted in the mid-western USA, where the same relative loss (10% incidence) across nine trials varied from 4.5% to 8.4% and the mean estimated yield reduction was of 6.3% (Chun et al., 1987; Hoffman et al., 1998; Yang et al., 1999). Although a range of edaphic factors adds stochasticity to direct comparisons, in general, white mould epidemics appear less damaging to soybean in Brazil compared to the mid-western production region of the USA.

None of the moderator variables tested (region, elevation, year, incidence and yield) significantly affected the strength and magnitude of the relationship between white mould incidence and yield. Other epidemiological factors that were unable to be qualified in this study that may be potentially affecting these relationships include primary inoculum densities, environmental conditions, ascosporic infection timing and disease severity (Grau & Hartman, 1999). For the latter, the strength of the disease intensity–yield loss relationships may be increased because severity encapsulates the cumulative effect of epidemic progression in infected plants (Savary et al., 2006), such as the within-plant spread of mycelia in white mould. Analogous observations have been reported for similar diseases such as fusarium head blight in wheat (Spolti et al., 2015), where secondary inoculum has a limited contribution to disease increase.

Studies on the relationships between white mould incidence and sclerotial production under field conditions are scarce. Studies conducted in vitro for evaluating isolate variability in sclerotial production (Li et al., 2008; Lehner et al., 2014) may be of limited value to extrapolate to field conditions. Hoffman et al. (1998) estimated the number of sclerotia per 300 g of seed across five soybean cultivars, but the disease incidence–sclerotial production relationship was not investigated. In the present study, sclerotial production was found to be strongly associated with white mould incidence and there was high heterogeneity in the estimated slopes. Some potential contributors to the high variability in these relationships may be the variation across geographical locations and microclimatic conditions (19 locations across six states), and genetic differences of the S. sclerotiorum populations at these locations.

The mean estimated slope allowed for the determination that approximately 1 kg ha\(^{-1}\) of sclerotia were produced for every 10% increase in white mould incidence. The estimated weight of an individual sclerotium produced under field conditions is unknown. However, studies conducted in vitro have reported average weights of a sclerotium ranging between 0.006 g (Irani et al., 2011) and 0.01 g (Kuang et al., 2011). Based on the mean estimated value in these studies (0.008 g each sclerotium), 10% incidence of white mould may result in 125 000 sclerotia ha\(^{-1}\) or 12.5 sclerotia m\(^{-2}\). One sclerotium may produce several apothecia (Bolton et al., 2006) and each apothecia may release up to 7.6 \(\times\) 10\(^3\) ascospores over 20 days (Clarkson et al., 2003). This data highlights the potential for substantial increases in inoculum densities following the introduction of white mould primary inoculum into a field.

Two moderator variables, region and elevation, significantly explained the heterogeneity of the correlation coefficients for relationships between white mould incidence and sclerotial production. Stronger associations were found in trials conducted in the southern regions and at high elevation areas. It is difficult to explain this result, but it might be related to climatic conditions. The trials classified as southern were those conducted in the Paraná State, Brazil. In this region, temperatures are typically milder than those in the northern region. It is likely that more favourable and uniform weather conditions favouring the disease might have occurred across the trials conducted in the southern region and higher elevations. For the latter, the longer dew period is another factor that would favour disease development.

The results of the present study may be useful for additional studies on risk assessments and economic analyses as a foundation for strategic and tactical disease management decisions. For example, considering a hypothetical scenario of 43% white mould incidence (mean incidence in the non-treated plots across the trials), 651 kg ha\(^{-1}\) (or US $217.00, assuming US $20.00/60 kg) would be lost at harvest in the absence of fungicides, with an expected production of 3000 kg ha\(^{-1}\) (in the absence of white mould). In Brazil, white mould is estimated to occur on approximately 22% of the total soybean area, or 6.8 million hectares (Meyer et al., 2015). In the above scenario, white mould could potentially result in an economic loss of US $1.47 billion in soybean production in Brazil. To manage the disease, two applications of the fluazinam fungicide can be effective, leading to 73% reduction of white mould incidence (Meyer et al., 2014). Therefore, a reduction from 43% to 11.6% in white mould incidence from fungicide application would still result in a reduction of 170.4 kg ha\(^{-1}\), or US $56.8 ha\(^{-1}\). Thus, even when using the most effective fungicides currently available, US $386.24 million would still be lost due to white mould. When this is added to the total fungicide cost of US $408 million (US
$30.00 per application), a total economic loss of US $794.24 would be expected, which is approximately 50% of the total economic loss without fungicides. These results highlight the potential impact of white mould in Brazilian agriculture if not well managed and the need to combine other strategies such as host resistance, biological control and cultural practices that help avoid or reduce the damage caused by the disease.

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Impact of white mould in Brazilian agriculture if not well managed and the need to combine other strategies such as host resistance, biological control and cultural practices that help avoid or reduce the damage caused by the disease.


Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher’s web-site. The raw data and R codes for conducting the analyses are freely available for download at https://github.com/emdelponte/paper-white-mold-meta-analysis.

**Figure S1.** Observations and fit of a simple linear model for the relationship between soybean yield and white mould incidence across treatments (fungicide and one non-treated control) in 35 (coded as 1–35) independent uniform trials conducted in Brazil from 2009/2010 to 2011/2012 (data source: Meyer et al., 2014).

**Figure S2.** Observations and fit of a simple linear model for the relationship between sclerotial weight and white mould incidence across treatments (fungicide and one non-treated control) in 29 (coded as 1–33) independent uniform trials conducted in Brazil from 2008/9 to 2011/12 (data source: Meyer et al., 2014).

**Figure S3.** Density curves for the frequency counts of 35 intercept (a) and slope (b) estimated from fitting a simple linear model (dark grey dots) or predicted (best linear unbiased predictions, BLUPs) from fitting a random effects coefficients model (light grey dots), respectively, for the relationship between soybean yield and white mould incidence. In (b), note the narrower range of the predicted (BLUPs) estimates of individual linear regression models.

**Figure S4.** Density curves for the frequency counts of 29 intercept (a) and slope (b) estimated from fitting a simple linear model (dark grey dots) or predicted (BLUPs) from fitting a random effects coefficients model (light grey dots), respectively, for the relationship between sclerotial weight and white mould incidence. In (b), note the narrower range of the predicted (BLUPs) estimates of individual linear regression models.

**Table S1.** Field-specific information for 35 uniform fungicide trials conducted in Brazil during four seasons (2009–2012 harvest years) and summaries of the mean white mould incidence (INCchk) in non-treated control of the trial, maximum mean soybean yield (YLDmax), and maximum mean sclerotial production at harvest (SCLmax) in the trial (Meyer et al., 2014).

**Table S2.** Pearson’s correlation coefficients (r) and respective P-value; coefficients estimated by a simple linear regression model fitted to data at the study level; and estimated coefficients (best linear unbiased prediction, EBLUP) by a random-coefficients model fitted to data on the relationship between soybean yield (kg ha⁻¹) and white mould incidence (%) in 35 field trials conducted in Brazil with variable number of observations (N), or the mean of a specific fungicide treatment across four replicated plots in a randomized complete block design (Meyer et al., 2014).

**Table S3.** Pearson’s correlation coefficients (r) and their respective P-value; coefficients estimated by a simple linear regression model fitted to data at the study level; and estimated coefficients (best linear unbiased prediction, EBLUP) by a random-coefficients model fitted to data on the relationship between soybean yield (kg ha⁻¹) and sclerotial production (g ha⁻¹) in 29 field trials conducted in Brazil with variable number of observations (N), or the mean of a specific fungicide treatment across four replicated plots in a randomized complete block design (Meyer et al., 2014).