Bioagro 34(3): 301-310. 2022 doi: http://www.doi.org/10.51372/bioagro343.9

ADAPTABILITY OF *Rhizoctonia solani* AG-1 IA FOR MANCOZEB SENSITIVITY UNDER TEMPERATURE STRESS

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ABSTRACT

The genetic architecture of quantitative characters in plants can be influenced by stress due to environmental changes, in combination with the decrease in the organism's average performance, resulting in genetic and environmental variances. The main objective of this study was to determine how the high-temperature stress affects the sensitivity of three populations of the soybean foliar blight pathogen *Rhizoctonia solani* AG-1 IA from Mato Grosso, Maranhão, and Tocantins to a broad-spectrum fungicide. The specific objective was to determine the effect of environmental stress on evolvability components (i.e., the selection response measures such as genotypic, environmental, and phenotypic variances) associated with sensitivity to the broad spectrum dithiocarbamate fungicide mancozeb. The fungal isolates from the three pathogen populations were grown under two temperatures (25 °C and 33.5 ºC, optimum and stress, respectively) and three fungicide concentrations (0.0, 0.32, and 0.64 $g \cdot L^{-1}$ of active ingredient). The mycelial growth was measured, and evolvability components, such as the genotypic variance coefficient (I_G), the environmental variance (I_E), and the broad-sense heritability (h^2), were determined. The results showed that high-temperature stress decreased (≈ 0.1 units, in a scale from 0 to 1.0) the genotypic variance and the heritability for mancozeb sensitivity in three populations of the soybean foliar blight pathogen *R. solani* AG-1 IA.
Additional keywords: Evolvability, genetic and environmental variation, heritability, soybean foliar blight, thermal adaptability

RESUMEN

Adaptabilidad de Rhizoctonia solani AG-1 IA a la sensitividad al mancozeb bajo estrés de temperatura

La arquitectura genética de los caracteres cuantitativos en las plantas puede verse influenciada por el estrés debido a cambios ambientales, en combinación con la disminución en el desempeño promedio del organismo, lo que resulta en cambios en las variaciones genéticas y ambientales. El objetivo principal de este estudio fue determinar cómo el estrés por altas temperaturas afecta la sensibilidad de tres poblaciones del tizón foliar de la soya *Rhizoctonia solani* AG-1 IA de Mato Grosso, Maranhão y Tocantins a un fungicida de amplio espectro. El objetivo específico fue determinar el efecto del estrés ambiental sobre los componentes de la capacidad de evolución (es decir, las medidas de respuesta de selección, como las variaciones genotípicas, ambientales y fenotípicas) asociadas con la sensibilidad al fungicida mancozeb, un ditiocarbamato de amplio espectro. Los aislados fúngicos de las tres poblaciones del patógeno se cultivaron bajo dos temperaturas (25 y 33,5 º C, óptima y de estrés, respectivamente) y tres concentraciones de fungicida (0,0, 0,32 y 0,64 $g \cdot L^{-1}$ de ingrediente activo). Se midió el crecimiento del micelio y se determinaron los componentes de la capacidad de evolución como el coeficiente de varianza genotípica (I_G), la varianza ambiental (I_E) y la heredabilidad en sentido amplio (h^2). Se encontró que el estrés por alta temperatura disminuyó la varianza genotípica y la heredabilidad (≈ 0,1 unidades, en una escala de 0 a 1,0) de la sensibilidad a mancozeb en las tres poblaciones del patógeno del tizón foliar de la soya (*R. solani* AG-1 IA).
Palabras clave adicionales: Adaptabilidad térmica, evolución, heredabilidad, tizón foliar de la soya, variación genética y ambiental

INTRODUCTION

Because of climate changes, the temperature increase may facilitate the evolution of quantitative resistance to fungicides in populations of plant pathogenic fungi (Willi et al., 2011). To understand whether plant pathogens can adapt to stress conditions due to temperature increases, it is

Received: December 1, 2021 Accepted: August 2, 2022
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necessary to investigate how the temperature stress affects their evolutionary potential and adaptability (McDonald and Linde, 2002; Zhan and McDonald, 2011; Yang et al., 2016). The ability of organisms to adapt to environmental changes correlates with the level of genetic diversity of their populations (Gunter et al., 2000).

The adaptation of organisms to heat stress may occur due to phenotypic plasticity or genetic differentiation. Phenotypic plasticity is the phenomenon whereby a genotype produces different phenotypes in response to different environmental conditions. In contrast to phenotypic plasticity, genetic differentiation is an intrinsic property of organisms that produces permanent genetic adaptations, allowing them to cope with local temperature fluctuations (Yamori et al., 2010).

The plant pathogenic basidiomycetous fungus *Thanatephorus cucumeris* (sexual phase of *Rhizoctonia solani*) anastomosis group AG-1 IA has worldwide distribution as the rice sheath blight and soybean foliar blight pathogen along with a range of different climate zones. The populations of this pathogen are genetically differentiated along a latitudinal gradient, both north and south of Ecuador (Bernardes et al., 2009).

Populations of the soybean foliar blight pathogen obtained from the most important soybean cropping areas in the central-west and northern Brazil were highly subdivided (Ciampi et al., 2008; Chavarro et al., 2020). Selection and genetic drift may have enhanced genetic differentiation since many populations are geographically isolated (Pauls et al., 2013). Because genetic variation is considered the basis for future adaptation to climate change (Gunter et al., 2000), it is postulated that the soybean foliar blight pathogen *R. solani* AG-1 IA confers the highly diverse genetic nature of the pathogen the potential for thermal adaptation (Bernardes et al., 2009).

Management of soybean foliar diseases has relied mainly upon chemical control with systemic fungicides, but fungicide efficacy has decreased steadily over the past 17 years in Brazil from complete control to around 20 % efficacy only (Godoy et al., 2016; Meyer et al., 2006), probably due to the emergence of resistance to the two major classes of fungicides, i.e., quinone-outside-inhibitors (QoIs) and demethylation-inhibitors (DMIs) (Schmitz et al., 2014). The emergence of fungicide resistance is attributed to the high selection pressure on the soybean foliar disease pathogens populations by the large-scale usage of prophylactic, calendar-based spray timing of high-risk fungicide programs (Godoy et al., 2016).

The current options for chemically managing soybean foliar diseases with some acceptable efficacy have relied upon old broad-spectrum protectant fungicides such as copper and dithiocarbamates (Godoy et al., 2016). The mancozeb-based dithiocarbamate fungicides had multiple sites and a broad action spectrum interfering nonspecifically with the general pathogen enzymatic activity (Lucas et al., 2015). For this reason, it is assumed that fungal growth in the presence of dithiocarbamate is a polygenic base character. A recent study indicated that the populations of *R. solani* AG-1 IA from soybean under high-temperature stress conditions (33.5 °C) increased genetic variance and heritability for sensitivity to copper oxychloride, another broad-spectrum fungicide (Ferro et al., 2019).

The objective of this study was to determine how the stress through temperature increase affects *R. solani* AG-1 IA sensitivity to a broad-spectrum fungicide. Specifically, the objective was to determine the effect of environmental stress due to temperature increase on evolvability components associated with the sensitivity to the dithiocarbamate fungicide mancozeb.

MATERIALS AND METHODS

Rhizoctonia solani AG-1 IA isolates were obtained in 2006 from soybean infected plants sampled from cropping areas in Maranhão, Mato Grosso, and Tocantins States, Brazil (Ciampi et al., 2008; Ferro et al., 2019). For this study, twelve genetically distinct isolates were randomly selected from each of these three populations.

These fungal isolates were genotyped by Ciampi et al., (2008) with ten highly polymorphic microsatellite markers (Zala et al., 2008). Fungal sclerotia from isolates stored on silica gel at -20 °C were transferred to 90 mm Petri dishes containing 15 mL potato-dextrose agar medium, PDA (18.5 g potato dextrose broth, 15 g agar, supplemented with 50 $\mu g \cdot mL^{-1}$ of chloramphenicol and streptomycin). Two series of transfers were

carried out to reactivate the cultures. Mycelial discs of 7 mm in diameter were cut off from colony margins, and each disc was transferred to the center of each experimental Petri dish containing PDA amended with 50 $\mu g \cdot mL^{-1}$ of the antibiotics chloramphenicol and streptomycin. Three doses of mancozeb (Manzate WG, Dupont, containing about 80 % active ingredient) were tested: 0.00, 0.32, and 0.64 $g \cdot L^{-1}$.

The fungi were incubated at 25 °C, considered the optimal growth temperature for *R. solani* AG-1 IA (Costa et al., 2007) and at 33.5 °C, considered stress temperature (Ferro et al., 2019). After 48 h incubation, the mean radial mycelial growth was measured along the line of the longest diameter and its perpendicular line. After ten days of incubation, the colonies were evaluated for the presence and abundance of sclerotia.

The experimental design was a completely randomized design with five replicates, in a 3×2×3 factorial scheme: three pathogen populations (Maranhão, Mato Grosso, and Tocantins populations), two temperatures (25 and 33.5 °C), and three mancozeb concentrations (0.0, 0.32 and 0.64 $g \cdot L^{-1}$). Each population consisted of 12 fungal isolates. The experiment was repeated once. No treatment by experiment interaction was detected, indicating full reproducibility, and allowing for merging the two experiments into one dataset with ten replicates. The mycelial growth data (x) were transformed into a logarithm of x+1.

The effect of strains, environments (temperature and fungicide concentration), and the interaction strains*environments were analyzed independently within each population and jointly. The effects of mancozeb doses and temperature on the isolates' sensitivity to the fungicide were tested to reveal their potential as stressing agents. The isolates were considered as random effects and the temperature and concentration of fungicide (here considered as different environments) as fixed effects.

The analysis of variance (*F* test) was performed using the Proc Mixed statistical procedure implemented in the SAS program 9.1.3 (Cary, NC, USA). The significance of the *F* values for each source of variation was determined using the GLM Procedure for mixed model analysis of variance. Mancozeb sensitivity means within each population and at two different temperatures were compared by the *t*-test.

The environment effect on the components of variance and evolvability for temperature and fungicide sensitivity was analyzed after that. The variance components were determined using the Proc VarComp procedure implemented in the same program. About the effects of the environment on the components of variance, the variance explained by the factor strain within each population was interpreted as genetic variance (V_G), and the experimental error was considered as environmental variance (V_E).

The variances obtained were standardized by the mean square of the mycelial growth in each environment to reflect the measures of evolvability (or response to selection) I_G and I_E (Houle, 1992). Heritability was calculated as the ratio between the genotypic variance (I_G) and phenotypic variance ($I_P = I_G + I_E$) coefficients, both standardized. The 95 % confidence interval for heritability estimates was calculated through a bootstrap analysis based on 1,000 data resampling using a statistical program developed for R.

RESULTS

The non-significant population effect indicated no differences between populations. In contrast, a significant isolate within population effect (F test) indicated differences between *R. solani* AG-1 IA isolates, probably attributed to genetic variation (for mycelial growth) among pathogen genotypes within each population. Mancozeb concentration and temperature also had a significant effect on the mycelial growth rate (Table 1).

The interaction temperature*fungicide within each population was not significant (by *F* test), indicating that the fungicide concentration effect on the fungal isolates mycelial growth was similar at different temperatures. The interactions fungicide*isolates" and temperature*isolates were significant (by *F* test), indicating that, depending on the temperature and fungicide concentration, the isolates varied in mycelial growth (Tables 1 and 2).

Taking only the temperature effect (Tables 2 and 3), the increase from 25 to 33.5 °C caused a significant decrease in the mean mycelial growth of isolates from Maranhão and Tocantins populations. However, there was no significant effect of temperature on the Mato Grosso population.

Table 1. Analysis of variance for populations, isolates within a population, fungicide concentration, and temperature effects, and their interactions on the *in vitro* mycelial growth of *Rhizoctonia solani* AG-1 IA in soybean

Sources of variation	df	MS	*F* value
Population[1]	2	1.73	0.33 ns
Isolates (population)[2]	33	5.26	3.30 ***
Fungicide[3]	2	312.43	525.59 ***
Temperature[4]	1	36.20	34.31 ***
Temperature*fungicide[5]	2	0.14	2.62 ns
Fungicide*isolates (population)[5]	70	0.59	10.89 ***
Temperature*isolates (population)[5]	35	1.06	19.33 ***
Coefficient of variation (%)	17.28		

MS: Mean sum of squares; [1]MS error for population = 5.2580; [2]MS error for isolates(population) = 1.5950; [3]MS error for fungicide = 0.5944; [4]MS error for temperature = 1.0551; [5]MS error for temperature*fungicide, fungicide*isolates (population), and temperature*isolates (within population) = 0.0546. ***: $P \leq 0.001$; ns: non-significant, df: degrees of freedom

Table 2. *F* values within population variance analysis for isolates, fungicide concentration, temperature, and their interactions on the *in vitro* mycelial growth of *Rhizoctonia solani* AG-1 IA in soybean

Sources of variation	df	Population		
		Maranhão	Mato Grosso	Tocantins
Isolate	11	5.01 **	4.42 ***	5.41 ***
Fungicide	2	294.51 ***	151.27 ***	253.36 ***
Temperature	1	8.63 *	2.11 ns	101.52 ***
Temperature*fungicide	2	0.45 ns	0.51 ns	1.09 ns
Fungicide*isolates	22	1.89 ns	6.52 ***	2.86 **
Temperature*isolates	11	6.92 ***	3.36 **	2.90 *
Coefficient of variation (%)		15.97	19.12	15.01

*: $P \leq 0.05$; **: $P \leq 0.01$; ***: $P \leq 0.001$; df: degrees of freedom, ns: non-significant ($P > 0.05$)

Table 3. Effect of temperature and mancozeb concentration on in vitro mycelial growth of *Rhizoctonia solani* AG-1 IA populations from soybean

Population	Mean diameter ($cm \cdot 48h^{-1}$) of colonies in three concentrations of mancozeb					
	0 $g \cdot L^{-1}$		0.32 $g \cdot L^{-1}$		0.64 $g \cdot L^{-1}$	
	25 °C	33.5 °C	25 °C	33.5 °C	25 °C	33.5 °C
Maranhão	9.05 b	7.05 b	1.97 b	1.12 b	2.49 a	1.44 a
Mato Grosso	7.85 b	7.42 a	2.23 b	1.97 a	1.84 b	1.45 a
Tocantins	9.76 a	5.80 c	2.93 a	1.68 a	2.61 a	1.23 a

Means followed by distinct letters in the columns differ from each other by the t-test ($P \leq 0.05$). Data were transformed to the logarithm (x+1)

For the mancozeb effect, the increase in the concentration (from 0.0 to 0.32 or 0.64 $g \cdot L^{-1}$ active ingredient) reduced the mycelial growth rate. There was a significant difference in the adaptability of the pathogen populations, observed by the mean mycelial growth in different stress environments. At 25°C and 0.32 $g \cdot L^{-1}$ mancozeb, the population from Tocantins

showed the highest mycelial growth (Table 3 and Figure 1). At 33.5 °C, the Tocantins and Mato Grosso populations were similarly adapted to the same fungicide concentration. With the increase in fungicide stress (at 25 °C and 0.64 g·L^{-1} mancozeb), there was no difference between the Tocantins and Maranhão populations, both of which had the highest mycelial growth. There was no significant difference among the three populations in the environment of maximum stress (at 33.5 °C and 0.64 g·L^{-1} mancozeb).

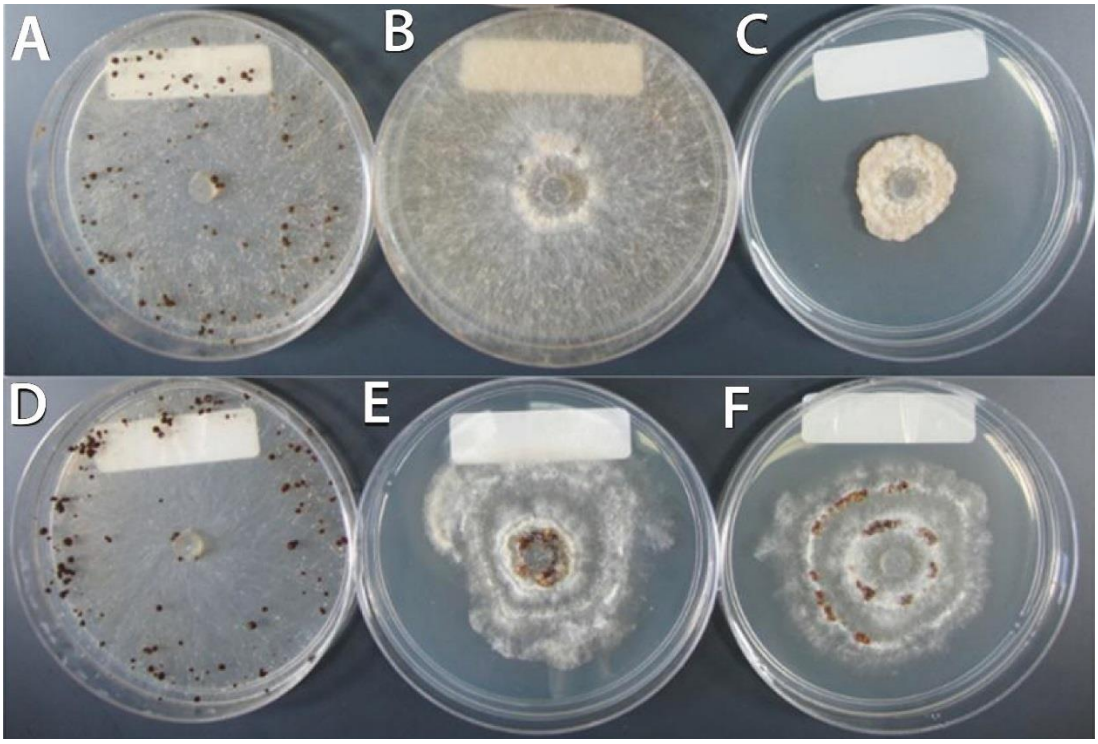

Figure 1. Morphological characteristics and sclerotia production of *Rhizoctonia solani* AG-1 IA colonies under optimum (25.0 °C) and stress temperature (33.5 °C) and three mancozeb concentrations (0.0, 0.32 and 0.64 g·L^{-1}) after ten days of growth. (A) 25°C and 0.0 g·L^{-1} mancozeb; (B) 25 °C and 0.32 g·L^{-1} mancozeb; (C) 25 °C and 0.64 g·L^{-1} mancozeb; (D) 33.5 °C and 0.0 g·L^{-1} mancozeb; (E) 33.5 °C and 0.32 g·L^{-1} mancozeb; (F) 33.5 °C and 0.64 g·L^{-1} mancozeb

The sclerotia production was abundant in the absence of fungicide, while under fungicide stress, it was completely inhibited at both 25 °C and 33.5 °C (Figure 1).

In regard to the effects of the high-temperature stress on the pathogen´s evolvability components, in general, the I_G for fungal mycelial growth was significantly higher than the *Ie*, resulting in the median to high heritability for this trait (minimum h^2 = 0.52 ± 0,06; maximum h^2 = 0.85 ± 0.02) (Figure 2). There were significant differences among the heritability values for mancozeb sensitivity at 25 °C and 33.5°C (five out of six treatments, except for the population from Tocantins growing at 0.64 g·L^{-1}). The high-temperature stress decreased the genotypic variance (I_G), hence the heritability (h^2), for mancozeb sensitivity in all three pathogen populations either at 0.32 or at 0.64 g·L^{1} of mancozeb (Figure 2).

The boxplot distribution of the evolvability components in each treatment (fungicide concentration or temperature) depicts the median line, the lower quantile at 0.25, and the upper quantile at 0.75 of the values. A notch around the median indicates the 95 % confidence interval.

DISCUSSION

Regarding the temperature effect as stress factor, the known optimum and maximum for mycelial growth of *R. solani* AG-1 IA from

soybean are 25 °C and 35 °C, respectively (Costa et al., 2007; Fenille et al., 2002). As there was a restriction in mycelial growth at 33.5 °C for the pathogen populations sampled from Maranhão and Tocantins, this was considered a stress temperature. In contrast, the mean of mycelial growth for the population from Mato Grosso was not affected at 33.5 °C, indicating adaptation to more extreme fluctuations in temperatures (Ferro et al., 2019). This potential for adaptation to higher temperature was reported for populations of *R. solani* AG-1 IA associated with rice and signal grass pastures in Colombia (Ramos et al., 2019).

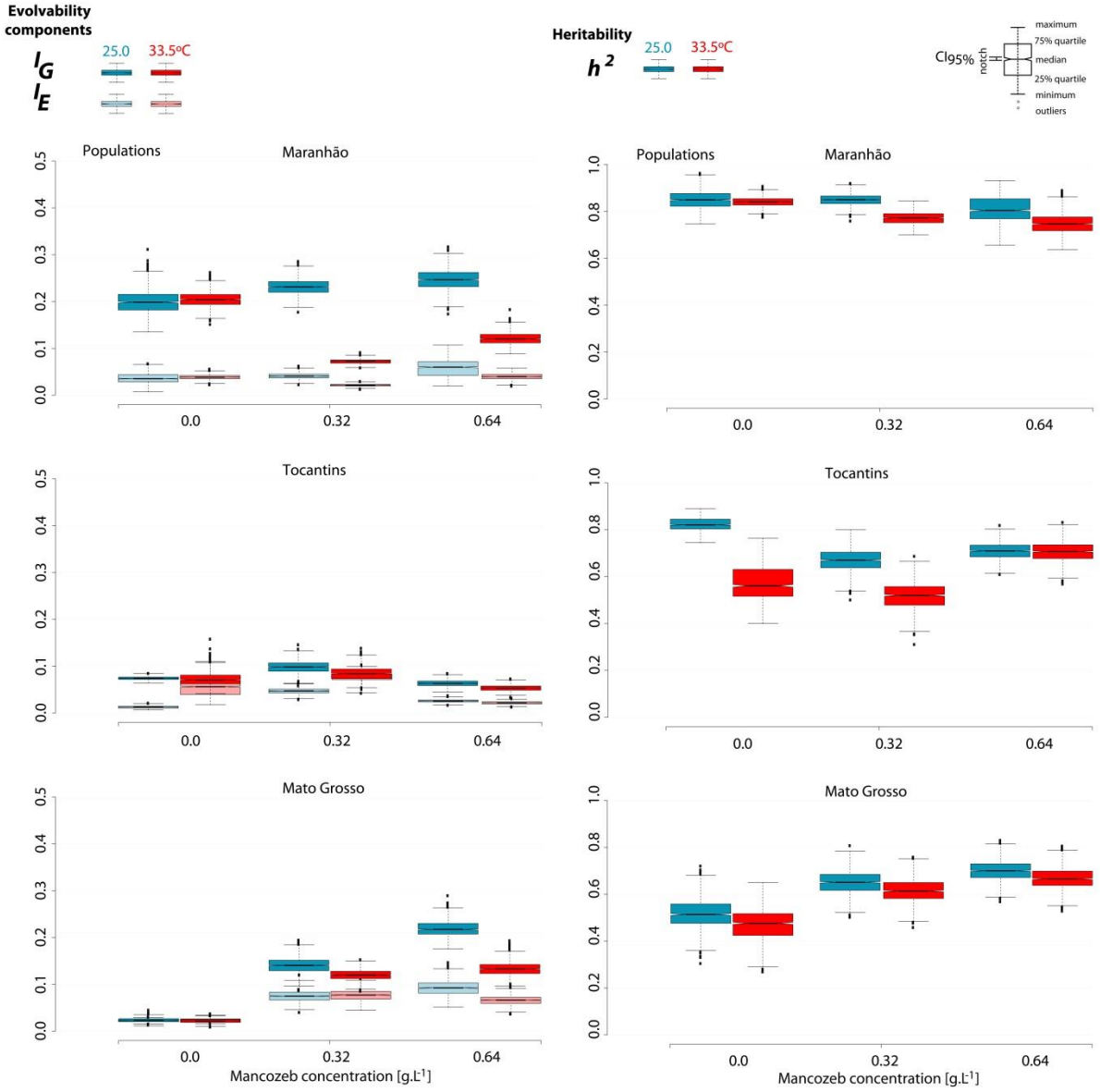

Figure 2. Depiction of the evolvability components genotypic variance (I_G), environmental variance (I_E), and heritability (h^2) for mycelial growth rate as a response to temperature and fungicide stress on three populations of the soybean foliar blight pathogen *Rhizoctonia solani* AG-1 IA sampled from Maranhão (MA), Mato Grosso (MT) and Tocantins (TO) states in Brazil. The dark or light blue boxplots indicate the resulting evolvability component measured from the fungal populations growing at 25 °C, while dark or light red boxplots indicate measures from populations growing at 33.5°C as stress temperature

Considering the fungicide effect as a stress factor, mancozeb reduced the mycelial growth rate of *R. solani* AG-1 IA and inhibited sclerotia production at 25 °C or 35 °C. Similarly, copper oxychloride, another broad-spectrum fungicide, inhibited sclerotia production (Ferro et al., 2019). The inhibition of sclerotia production under fungicide stress occurred due to the resulting lower fungal mycelia growth, as large amounts of mycelium are required for sclerotia production (Youssef et al., 2012).

In regard to the effects of the stress conditions on the evolvability components determined in our study, the relatively high contribution of genotypic variance to the phenotypic variance for mycelial growth at distinct environments resulted in the median to high heritability for this trait ($0.52 \leq h^2 \leq 0{,}85$) (Figure 2). Higher heritability indicates that the phenotypic variation in mycelial growth of the isolates is an excellent predictor of *R. solani* AG-1 IA adaptive potential, as it could indicate a more significant contribution of genetic components to the phenotype (Willi et al., 2011).

In fact, *R. solani* AG-1 IA has been described as a pathogen with high evolutionary potential, derived from high population size, a predominantly mixed reproductive system that provides genetic diversity to populations, besides adaptation to a broad host range. These evolutionary and biological traits could contribute to high adaptability potential to diverse climatic and environmental stresses (Bernardes et al., 2009; González et al., 2010).

In our study, the high-temperature stress significantly decreased genotypic variance, hence on heritability (h^2), for mancozeb sensitivity in all three pathogen populations. These results contrasted with a previous study from Ferro et al. (2019), by which an increase in the I_G for sensitivity to copper oxychloride under temperature stress (33.5 °C) was detected for the same three populations of the pathogen, with a significant increase in the broad sense heritability. On average, at 33.5 °C, there was an increase of 0.37±0.18 units in h^2. Differences in fungitoxicity between mancozeb and copper oxychloride fungicides (and the inherent differences in sensitivity of *R. solani* AG-1 IA to the distinct active ingredients) could explain the discrepancies between the two studies (Kataria and Grover, 1978; Meyer et al., 2006).

The results from Ferro et al. (2019) were in line with the first hypothesis of Hoffmann and Merilä's (1999), concerning the effect of environmental stress on genetic variation and heritability in populations of several organisms. This hypothesis associated the increased heritability under stress conditions to a mechanism they termed "decanalization". Under the decanalization mechanism, some genetic variants are expressed only in stressful environments or by temporally insufficient selection to remove deleterious alleles in new and stressful environments.

On the other hand, our results (Figure 2) fit Hoffmann and Merilä's second hypothesis that predicts a decrease in the population's broad sense of heritability because stress increases environmental variance or because organisms, under conditions of nutrition restriction, for example, do not reach their genetic potential.

Our study was not the only one that did not fit the decanalization mechanism above mentioned. Willi et al. (2011) detected no significant change in heritability for the mycelial growth rate of the potato stem canker pathogen *R. solani* AG-3 PT under temperature and copper-based fungicide stress once genetic variance increases were counteracted by a simultaneous increase in environmental variance.

As a final remark, increasing temperature, such as the effect of climate change, may facilitate the evolution of quantitative fungicide resistance in populations of plant pathogens (Ghini et al., 2008). However, only a few empirical studies are available to support this hypothesis, among which are Willi et al. (2011), Ferro et al. (2019), and our current contribution.

In summary, these few studies available on *Rhizoctonia* pathosystems showed that there were changes in the fungus evolvability components for sensitivity to broad spectrum fungicides under high temperature stress. Impacts were positive, negative, or neutral as there was a decrease, increase, or no effect on the evolvability components, indicating that changes in heritability under stress conditions are challenging to predict because multiple evolvability components can change simultaneously, in addition to the population-dependent effects, as suggested by Hoffmann and Merilä (1999).

CONCLUSION

High-temperature stress decreased the genotypic variance and heritability for mancozeb sensitivity in all three soybean foliar blight pathogen *R. solani* AG-1 IA populations.

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