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Genetic and environmental factors influencing bacterial stalk rot in corn: a comprehensive study of resistance, epidemiology, and weather interactions

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Background: *Dickeya zea*, the cause of bacterial stalk rot (BSR) is one of the important diseases after downy mildew which is often found infecting corn in Indonesia and has the potential to reduce corn yields. The use of resistant corn cultivars is believed to be an effective measure in suppressing BSR disease progression. This study aims to evaluate the resistance level of hybrid corn genotypes and determine the role of phytopathogen-corn genotype-environment interplay in BSR disease progression.

Methods: Resistance evaluation of hybrid corn genotypes was conducted under field conditions using artificial inoculation. Inoculation was performed by injecting a *D. zea* suspension (10^8 – 10^{10} cfu/mL) into the second internode from the base of the corn stalk. Observed variables included disease incidence and severity, disease progression models, infection rate, area under the disease progress curve (AUDPC), protection index, Pearson's correlation, and path analysis.

Results: Three of the nineteen hybrid corn genotypes consistently reacted as resistant or moderately resistant to BSR disease, namely A.26 × 14.12.1, A.26 × Mal.03, and B × Sy-01, with disease incidence and severity at 70%. The disease progression models in A.26 × 14.12.1; A.26 × Mal.03; and B × Sy-01 followed the Gompertz, Monomolecular, and Logistic models, respectively. Pearson correlation and path analysis revealed an interaction between weather factors in influencing the BSR disease progression, both positively and negatively. Based on the two analyses, it was seen that weather factors play a major role in various pathogen growth and progression processes, especially in the generative phase. In-depth and systematic validation of hybrid corn genotypes in suppressing BSR disease through epidemiological, metabolomic, and metagenomic approaches was needed to comprehensively confirm its ability.

KEYWORDS

corn, disease epidemic, GxE, hybrid corn genotype, resistant cultivar

1 Introduction

Corn (*Zea mays* L.) is a vital cereal crop that is used extensively for human and animal use as food, feed, and to produce bioethanol. Growing industrialisation and population growth will directly affect the rising demand for maize (Abduh et al., 2021; Sah et al., 2020; Badr et al., 2020). The primary strategy for lowering imports in Indonesia is to boost maize output. Nevertheless, there are a number of constraints to raising production. One of them is biotic stress, especially caused by phytopathogens that cause major diseases in corn plants. Corn is reported to be susceptible to about 112 diseases globally, caused by fungi, bacteria, viruses, and nematodes, which cause significant yield losses (Jha and Prajapati, 2024; Alvarez-Quinto et al., 2025). In India, approximately 60 diseases that can infect corn have been documented (Hooda et al., 2018).

In Indonesia, there are six major diseases recognized as major threats to corn production and can significantly reduce yields, even causing crop failure (Muis et al., 2019; Mirsam et al., 2025; Mirsam et al., 2022; Suriani et al., 2023a). The six major diseases are downy mildew caused by *Peronosclerospora* spp. (Muis et al., 2019), leaf blight caused by *Bipolaris maydis* (Mirsam et al., 2025), leaf rust caused by *Puccinia* sp. (Mirsam et al., 2021), banded leaf and sheath blight caused by *Rhizoctonia solani* (Mirsam et al., 2023), *Fusarium* Stem Rot caused by *Fusarium verticillioides* (Mirsam et al., 2022), and bacterial stalk rot caused by *Dickeya zae* (Suriani et al., 2023a). Among various leaf diseases, turcicum leaf blight, also known as Northern corn leaf blight, caused by *Exserohilum turcicum* (Pass.) Leonard and Suggs. (syn. *Helminthosporium turcicum* Pass.), plays a globally significant role in reducing corn yields (Aghav et al., 2023; Shinde et al., 2024). In the era of extreme climate change, bacterial stalk rot caused by *Erwinia chrysanthemi* pv. *zae*, currently called *Dickeya zae*, emerged as the most destructive corn disease in Indonesia after downy mildew (Suriani et al., 2023b).

Bacterial stalk rot (BSR) disease has become one of the most important diseases after downy mildew that attacks Indonesian corn crops (Kumar et al., 2017; Muis et al., 2022). BSR is an economically important disease that has the potential to reduce crop yields by 21 to 98.8% (Kumar et al., 2017). In regions with tropical and subtropical maize crops, this disease is a serious problem (Zhu et al., 2021). Several studies have also reported the widespread distribution of BSR disease in various countries, such as Turkey, Korea, Mexico, and Indonesia (Caplik et al., 2022; Martinez-Cisneros et al., 2014; Myung et al., 2010; Suriani et al., 2023a). In Indonesia, *D. zae* was first reported to infect pineapple as its host plant in early 2020 (Aeny et al., 2020), meanwhile *D. zae* was first reported to infect corn in 2022 (Suriani et al., 2023a). The presence of *D. zae* in Indonesia is closely related to its plant host and weather conditions that can support the disease progression caused by this bacteria. High temperature (25–35 °C) and humidity (90%), followed by high rainfall, is a favorable environment for *D. zae* (Kumar et al., 2016; Meena et al., 2023). In addition, corn grown in flood-prone areas has a higher risk of infection than in well-drained soils (Freije and Wise, 2016). These environment conditions can support the physiological and metabolic activities of *D. zae*, providing a good environment for bacteria to grow (Kumar et al., 2017).

D. zae has a wide habitat due to its high adaptability to various ecosystems, both in various types of soil, host and non-host plants, surface water or irrigation, etc. (Charkowski, 2018). This bacterium can initiate infection from a low inoculum potential, a rapid rate of spread

through the plant's vascular tissue, high aggressiveness, and requires an optimal temperature to cause a disease epidemic. In recent years in Indonesia, corn has been reported to be infected by *D. zae* and is one of the susceptible hosts (Suriani et al., 2023b), in addition to 6 families of dicotyledonous plants in 11 orders and 10 families of monocotyledons in 5 orders (Samson et al., 2005). There are many possibilities for *D. zae* to spread from one plant species to another because this bacterium has a wide host range so the risk of spread can increase if supported by favorable weather conditions. Therefore, careful consideration needs to be given to potential infection pathways, as well as the adaptability of this bacterium to plant hosts, the environment, and other weather conditions.

BSR disease management includes agronomic management strategies such as the use of synthetic bactericides, optimal fertilization to support plant growth and health, and crop rotation with non-host crops to reduce bacterial inoculum in the soil (Osdaghi, 2022; Singh et al., 2020). Controlling BSR is very challenging. Currently, no commercially accessible control agents have been created to successfully manage BSR in significant agricultural crops. In addition, no host plants that are completely resistant to BSR have been developed. Although the use of BSR-resistant cultivars assembled through breeding is promising, it has not yet been widely accepted. It is believed that the use of resistant hybrid corn genotypes can manage the BSR disease progression. Resistant hybrid corn genotypes can be obtained through breeding lines that are resistant to BSR. Therefore, evaluation of hybrid corn genotypes for resistance to BSR is necessary to develop lines and hybrids resistant to *D. zae* infection.

Cognition of BSR disease epidemiology is essential for BSR disease management and plant health. Climate change is expected to affect the speed and pattern of distribution, severity, and spread of BSR in certain areas due to changes in weather variables. Effective disease management strategies are needed to control this disease epidemic, such as developing resistant corn cultivars, improving cultivation practices, and using biological control agents. This study is relatively comprehensive in identifying and predicting the disease progression over time so that it can be used as a basis for determining strategies to control the disease. However, there are challenges in implementing this strategy, especially in developing countries that have limited resources. Continued monitoring, assessment, and adaptation of management practices will be critical to reducing the impact of BSR disease on corn and other crops. There is currently little research in Indonesia that evaluates genetic resistance in conjunction with environmental variables and disease epidemiology. Previous studies on BSR in Indonesia have primarily focused on disease occurrence, pathogen identification, and general control strategies. Comprehensive research linking the resistance of hybrid maize genotypes to disease development influenced by environmental conditions is still lacking. The purpose of this study is to assess the resistance level of hybrid maize genotypes to BSR and analyse the effects of the interaction among the phytopathogen, maize genotype, and environmental conditions on BSR disease progression.

2 Materials and methods

2.1 Sampling and isolation of *Dickeya* sp. from corn with BSR symptoms

Sampling and isolation of *Dickeya* sp. were carried out by referring to the modified method of Suriani et al. (2023a), where the sterilized

sample was macerated in a 10% glycerol solution, then the bacterial suspension formed was stored in an effendorf tube and wrapped to be taken to the laboratory. The bacterial suspension was cultured on Nutrient Agar (NA) medium using the Streak Plate Method. *D. zeae* culture propagation was carried out using sterile nutrient broth (NB) medium and incubated at room temperature on a rotary shaker at 170 rpm for 48 h (108–1010 cfu/mL). The pure culture of *D. zeae* was stored in the refrigerator and given the label code BSR_SH100 for further testing.

2.2 Morphological identification of *Dickeya* sp.

Morphological identification was carried out based on macroscopic and microscopic characteristics. The macroscopic characters observed were the shape, edges, elevation, and surface of the colony. Meanwhile, the microscopic characters observed were the shape, arrangement, and size of cells (Cabeen and Jacobs-Wagner, 2005).

2.3 Molecular identification of *Dickeya* sp. based on the housekeeping gene *dnaX*

Molecular identification was conducted using the housekeeping gene *dnaX*, which provides higher phylogenetic resolution than 16S rRNA for soft-rot bacteria. Previous research (Sławiak et al., 2009; Aeny et al., 2020) has effectively employed the *dnaX* gene for phylogenetic analysis and differentiation of *Dickeya* spp. Special-level assignment was based on DNAX sequence analysis and was interpreted with caution. Multilocus sequence analysis was not performed in this study.

2.3.1 Preparation of *Dickeya* sp. pellets

One loop of bacterial isolate from the stock was inserted into an Erlenmeyer flask containing 100 mL of NB medium, and then incubated at room temperature on a rotary shaker at 180 rpm for 48 h. A glass collecting bottle was filled with the bacterial suspension that had been incubated. The incubated bacterial suspension was placed in a 1.5 mL microtube and then centrifuged at 10,000 × g for 5 min. Centrifugation's supernatant was thrown away, but the pellet served as a template for further DNA extraction.

2.3.2 Extraction of *Dickeya* sp. DNA

Dickeya sp. DNA extraction was performed using The Presto™ Mini gDNA Bacteria Kit (Geneaid). The extraction method followed The Presto™ Mini gDNA Bacteria Kit protocol. This kit's working principle was a combination of buffer and lysozyme to efficiently lyse bacterial cell walls containing a peptidoglycan layer. Then, Proteinase K and chaotropic salt continued the lysis process and degraded the protein so that DNA could readily bind to the spin column's glass fibre matrix.

2.3.3 Amplification of *Dickeya* sp. DNA

Dickeya sp. DNA was amplified using a PCR machine (Axygen MaxyGene™ II Thermal Cycler) with the amplification target, namely the housekeeping gene *dnaX*. Amplification using a universal primer

pair, namely *dnaxF* (5'-TATCAGGTYCTTGCCCGTAAAGTGG-3') and *dnaxR* (5'-TCGACATCCARCGCYTTGAGATG-3'). The components of the 25 µL PCR reaction were 1 µL *dnaxF* primer, 1 µL *dnaxR* primer, 8.5 µL nano pure water, 12.5 µL KAPA Taq ReadyMix PCR, and 2 µL DNA template. The PCR program was based on the Aeny et al. (2020) method using annealing at 57 °C for 1 min. The PCR amplicon was subjected to gel agarose 1% electrophoresis at 110 V for 50 min, and ultraviolet (UV) transilluminator was utilised to visualise the results.

2.3.4 Bioinformatics analysis

Bioinformatics analysis of the housekeeping gene *dnaX* includes nucleotide base sequencing, similarity analysis, genetic distance, and phylogenetic reconstruction. DNA amplicons were sent to FirstBase for nucleotide base sequencing. The nucleotide base sequences were then used to perform sequence similarity analysis with databases available through the Basic Local Alignment Search Tool for nucleotide (BLASTn) using the online server of the National Center for Biotechnology Information (NCBI) website¹. Similarity analysis was performed by comparing the nucleotide base sequences of sample isolates with reference sequences contained in GenBank data through the NCBI BLASTn feature, meanwhile the genetic distance matrix was analyzed using the p-distance approach using Bioedit Sequence Alignment Editor version 7.2. (Bioedit 7.2.). The accuracy of the similarity analysis results was based on the low E-value score and the high query cover value. In addition, the phylogenetic tree was reconstructed using Molecular Evolutionary Genetic Analysis Software version 11 (MEGA11) with the Kimura 2-parameter method with Maximum Composite Likelihood model and bootstrap 1000x.

2.4 Evaluation of hybrid corn against BSR in the field condition

2.4.1 Preparation of tested pathogens

Dickeya sp. isolate was repurified in NA and incubated for 24 h. *Dickeya* sp. propagation was carried out using a sterile NB medium and incubated at room temperature on a rotary shaker at 170 rpm for 48 h (108–1010 cfu/mL). The available *Dickeya* sp. suspension was ready to be used for artificial inoculation on tested corn plants.

2.4.2 Planting tested genotypes

This testing was conducted at the Experimental Field of Center for Standard Testing of Cereals Instrument, Lau District, Maros Regency, South Sulawesi. The experimental plot was designed with a length of 5 m, consisting of 4 rows with a planting distance of 70 × 20 cm, and was repeated three times as a test block. Two seeds were planted per hole and given Carbofuran 3G to control soil insect pests. The 7–10 days after planting (DAP), the plants were thinned by leaving one plant per hole. The initial fertilisation was done at 10 DAP with 150 kg/ha of urea and 400 kg/ha of NPK. The second fertilization was carried out at 30 DAP with only 150 kg/ha of Urea.

¹ www.ncbi.nlm.nih.gov

2.4.3 Artificial inoculation of tested bacterial suspension

A total of 1 mL of 24-h-old *Dickeya* sp. suspension was taken using a sterile syringe with a concentration of 108–1010 cfu/mL. A sterile syringe was used to inject the *Dickeya* sp. suspension into the second segment from the base of the corn stem. The injection was done as close as possible to the base of the stem. Inoculation was carried out at 35 DAP.

2.4.4 Experimental design and data analysis

This study was designed using a randomized block design consisting of 19 tested genotypes (14.12-1 × A.13; 14-4-1 × 14-21-1; 14-4-1 × A.13; 14-4-1 × Mal.03; 6 × B; A.13 × Mal.03; A.22 × 1044-14; A.22 × A.26; A.24 × A.18; A.26 × 14.12.1; A.26 × A.13; A.26 × A.6; A.26 × Clyn 231; A.26 × Mal.03; A.6 × A.26; B × Sy-01; N79 × A.13; Sy-02 × Sy-03; Sy-04 × B; Sy-04 × Sy-02) and 4 comparison genotypes (JH37; NK007; P89; NK7328). This study was repeated three times as a test block. The observation data were then analyzed statistically and if the data showed significant differences, then continued with the Least Significant Difference (LSD) at the 5% level ($\alpha = 0.005$).

2.4.5 Observation variables for BSR disease intensity

Observation of BSR disease incidence (DI) was carried out at 7, 14, 21, 28, and 35 days after inoculation (DAI). The data obtained was cumulative data from each observation, then converted into the percentage of plants infected with *Dickeya* sp. using Equation 1. Meanwhile, observations of BSR disease severity (DS) were carried out at 90 DAP. Observations were made by taking 10 plant samples and scoring based on infection symptoms that appeared in the stem vascular tissue based on the Hooda et al. (2018) disease scale. The disease scales were then converted into a percentage of disease severity according to Equation 2. The disease tests' resistance requirements were derived from the Food Crop Varieties Release Procedure (Mirsam et al., 2021) as follows: very resistant (VR), disease intensity of 0–5%; resistant (R), disease intensity of >5–20%; moderately resistant (MR), disease intensity of >20–40%; susceptible (S), disease intensity of >40–60%; very susceptible (VS), disease intensity of >60%. In addition, the level of disease progression suppression was calculated based on DI reduction (Equation 3) and DS reduction (Equation 4).

2.4.6 Observation variables for BSR disease epidemic

Analysis of the disease progression model, infection rate, protection index, and area under the disease progress curve (AUDPC) were the variables that were observed. The analysis of the BSR disease progression model was carried out based on the transformation results of DI and DS values for each observation period. Based on a model accuracy test, the BSR disease progression model was analysed using the three most used models, i.e., Gompertz, Logistic, and Monomolecular (Xu, 2006). The model was selected by transforming the collected disease severity data (x) with the equation $\ln\{1/(1-x)\}$ for Monomolecular, $\ln\{x/(1-x)\}$ for Logistic, and $\{-\ln(-\ln x)\}$ for Gompertz. The transformed data were then analyzed using linear regression against disease progression time (t). The highest coefficient of determination (R^2) value and the lowest mean square error were chosen in order to perform the model accuracy test (MSE) (Xu, 2006).

Furthermore, the data was utilised to determine the infection rate (r) depending on the results of choosing a disease progression model utilising Equations 5–7. The proportion of BSR disease severity within a certain observation period is used to calculate the AUDPC value, which indicates the degree of disease progression during that time. The AUDPC value was calculated using Equation 8 (Mehmood and Khan, 2016). Meanwhile, the protection index is calculated based on the AUDPC value using Equation 9 (Caulier et al., 2018).

$$DI(\%) = \frac{A}{B} \times 100\% \quad (1)$$

Where, DI, incidence of BSR disease; A, number of plants with BSR disease symptoms; B, number of plants observed in each genotype.

$$DS(\%) = \frac{\sum(n \times v)}{Z \times N} \times 100\% \quad (2)$$

Where DS is disease severity; n is the number of affected plants in each category; v is the scale value of each affected plant; Z is the highest scale value; and N is the number of plants observed in each attack.

$$DI \text{ reduction } (\%) = \frac{DI \text{ in susceptible genotype} - DI \text{ in tested genotype}}{DI \text{ in susceptible genotype}} \times 100\% \quad (3)$$

$$DS \text{ reduction } (\%) = \frac{DS \text{ in susceptible genotype} - DS \text{ in tested genotype}}{DS \text{ in susceptible genotype}} \times 100\% \quad (4)$$

Molecular model:

$$r_m = \frac{1}{t} \left(\ln \frac{1}{1-X_t} - \ln \frac{1}{1-X_0} \right) \text{ per unit of time} \quad (5)$$

Logistic model:

$$r_l = \frac{1}{t} \left(\ln \frac{X_t}{1-X_t} - \ln \frac{X_0}{1-X_0} \right) \text{ per unit of time} \quad (6)$$

Gompertz model:

$$r_g = \frac{1}{t} - \ln \{-\ln(X_t)\} + \ln \{-\ln(X_0)\} \text{ per unit of time} \quad (7)$$

Where x_t , the proportion of disease at time t ; x_0 , the proportion at the beginning of the observation ($t = 0$); t , time; r , rate of disease infection.

$$AUDPC = \sum_{i=1}^{n-1} \left(\frac{X_i - X_{i+1}}{2} \right) (t_{i+1} - t_i) \tag{8}$$

where, *n* is the number of observations; *x*, DM intensity, and (*t_i* + 1 – *t_i*) is the time interval between observations.

$$\text{Protection index (\%)} = \left(1 + \frac{\text{AUDPC of tested genotype}}{\text{AUDPC of susceptible genotype}} \right) \times 100\% \tag{9}$$

2.5 Interplay analysis of weather factors on bacterial stalk rot disease based on Pearson’s correlation and path analysis

The influence of weather factors on BSR disease progression was tested using Pearson’s correlation and path analysis. These analyses can describe the direct and indirect influence of weather factors on BSR disease progression and determine which weather factors have the most influence on BSR disease progression. Pearson’s correlation analysis process was performed using The R Project for Statistical Computing software to determine the correlation coefficient value. The conventional approach used to interpret the correlation coefficient values followed Schober et al. (2018), namely 0.00–0.10 = negligible correlation; 0.11–0.39 = weak correlation; 0.40–0.69 = moderately correlation; 0.70–0.89 = strong correlation; 0.90–1.00 = very strong correlation. In path analysis, the severity of BSR disease was the dependent variable (Y), while the weather factor was the independent variable (X). The weather data analyzed includes minimum temperature (Tn), maximum temperature (Tx), average temperature (Tavg), average humidity (RHavg), rain fall (RF), length of sun exposure (LSE), maximum wind velocity (WVx), wind direction at maximum wind velocity (WDx). This data was taken from the website of the Meteorological, Climatological, and Geophysical Agency (BMKG; <https://www.bmkg.go.id/en.html>) (Table 1).

3 Result

3.1 Symptoms and morphological characters of *Dickeya* sp.

The initial symptoms of BSR disease were characterized by brown or gray stem and leaf sheath, the stem base or stem segment near the ground appearing wet, soft, slimy, and smelling rotten, the stem

bending or collapsing easily, and prematurely wilting (Figure 1a). The split stem showed vascular tissue that appeared rotten, wet, and slimy (Figure 1b). While the healthy stem showed clean vascular tissue and no disease symptoms (Figure 1c). Morphological characters of BSR_SH100 isolate grown on NA media and incubated for 24 h have gray-white colonies with a round shape, convex elevation, and flat edges. Observations at 48 to 72 h after incubation showed that the colony shape changed to almost round with irregular wavy edges (Figure 1d). While, the microscopic characters showed rod-shaped bacterial cells with rounded ends, single or paired bacterial cells with a length of 2–3 μm, and did not form spores (Figure 1e).

3.2 Molecular characters of *Dickeya zeae* based on amplification of the housekeeping gene *dnaX*

A deoxyribonucleic Acid (DNA) band of ±535 bp was successfully amplified using the primer pair *dnaxF/dnaxR* based on the polymerase chain reaction (PCR) method (Figure 2). Similarity analysis of nucleotide base sequence showed that BSR_SH100 isolates had very high similarity with *D. zeae* strain KNG Narmada from India, *D. zeae* strain DZ15SB01 from Thailand, *D. zeae* strain DBM_1 from Turkey, and *D. zeae* strain MS32 from Taiwan with homology values of 99.12–99.56% and genetic distance coefficient value of 0.000–0.002. In addition, the BSR_SH100 isolate also had quite high similarity with *D. zeae* strain N_Unila_5 from Lampung (Indonesia), *D. zeae* strain IPO_649 from the Netherlands, and *D. zeae* strain MAFF106502 from Japan with homology values of 98.23, 98.90, and 98.68%, respectively, as well as genetic distance coefficient value of 0.002–0.007. All *D. zeae* strains had very low similarity to *Pectobacterium parmentieri* strain JB106 from the USA as an outgroup strain with a homology value of <80% and distance coefficient value of 0.108–0.125 (Table 2; Figure 3).

3.3 Resistance level of hybrid corn genotypes to bacterial stalk rot disease

BSR disease symptoms were found in all genotypes at 7 days after planting (DAI), except for A.26 × Clyn 231, A.26 × Mal.03, A.6 × A.26, and NK007 genotypes. The BSR disease incidence in all entries at 7 HSI was still relatively low with an attack intensity of 0.42–3.42%. A significant increase in BSR disease incidence occurred at 28 DAI, where P89 genotype showed a fairly high disease incidence of 30.69%. The BSR disease incidence in the last

TABLE 1 Weather conditions during the study period, September–December 2024 (MCGA; <https://www.bmkg.go.id/en.html>).

Month	T (°C)			RH (%)	RF (mm)	LSE (jam)	WV (m/s)		WD (°)
	<i>n</i>	<i>x</i>	avg				<i>x</i>	avg	
September	23.62	34.86	28.52	65.77	7593.17	7.20	4.23	244.84	1.94
October	23.94	34.38	28.21	71.97	3433.38	7.18	3.94	262.58	1.74
November	24.46	32.49	27.46	81.73	1370.33	6.10	3.27	229.67	1.23
December	24.33	29.67	26.31	86.81	638.50	3.76	3.23	220.97	1.39

T, temperature; *n*, minimum; *x*, maximum; avg, average; RH, humidity; RF, rainfall; LSE, length of sun exposure; WV, wind velocity; WD, wind direction.

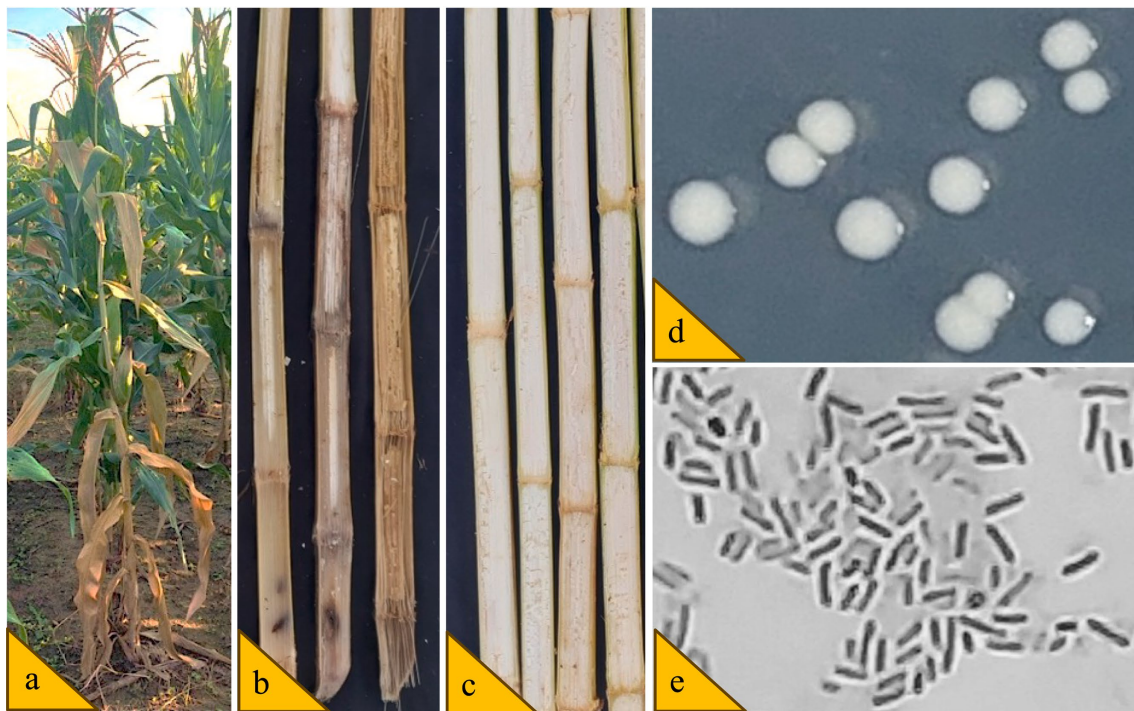


FIGURE 1 BSR disease symptoms and morphological characteristics of *Dickeya* sp. (a) BSR symptoms; (b) infected vascular tissue; (c) healthy vascular tissue; (d) *Dickeya* sp. colony appearance on PDA medium with a microscope magnification of 40x; (e) cell of *Dickeya* sp.

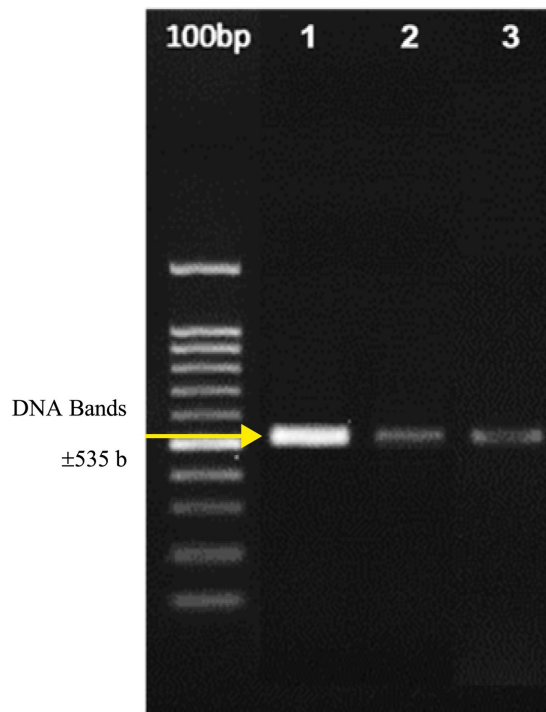


FIGURE 2 Visualization of *D. zeae* DNA bands based on housekeeping gene *dnaX* amplification. 100 bp, Marker DNA; 1, 2, 3, replication.

observation (35 DAI) showed that 18 of 23 hybrid corn genotypes reacted moderately resistant, resistant, and very resistant to BSR disease, namely 14.12-1 × A.13; 14-4-1 × 14-21-1; 14-4-1 × A.13;

14-4-1 × Mal.03; 6 × B; A.13 × Mal.03; A.24 × A.18; A.26 × 14.12.1; A.26 × A.13; A.26 × A.6; A.26 × Clyn 231; A.26 × Mal.03; A.6 × A.26; B × Sy-01; N79 × A.13; Sy-02 × Sy-03; Sy-04 × B; and Sy-04 × Sy-02 with a disease incidence of 4.63–22.49%. The BSR disease incidence in the tested genotype was significantly lower than P89 and NK7328 in the 5% LSD test. The JH37, NK007, and P89 genotypes reacted resistant and moderately resistant with disease incidence of 10.45, 22.56, and 32.34%, respectively. Meanwhile, the NK7328 genotype reacted susceptible with a disease incidence of 40.99% (Table 3).

The disease severity was higher than the disease incidence based on observations of *D. zeae* infection in stem vascular tissue. There were four out of twenty three tested genotypes that reacted moderately resistant to BSR disease, namely A.22 × 1044-14; A.26 × 14.12.1; A.26 × Mal.03; and B × Sy-01 genotypes with disease severity of 39.26, 30.00, 32.96, and 36.30%, respectively. Meanwhile, 11 other test genotypes reacted susceptible and very susceptible with disease severity of 40.00–75.93%. The four comparison genotypes showed high BSR disease severity with an attack intensity of 44.07–61.48% (Table 3).

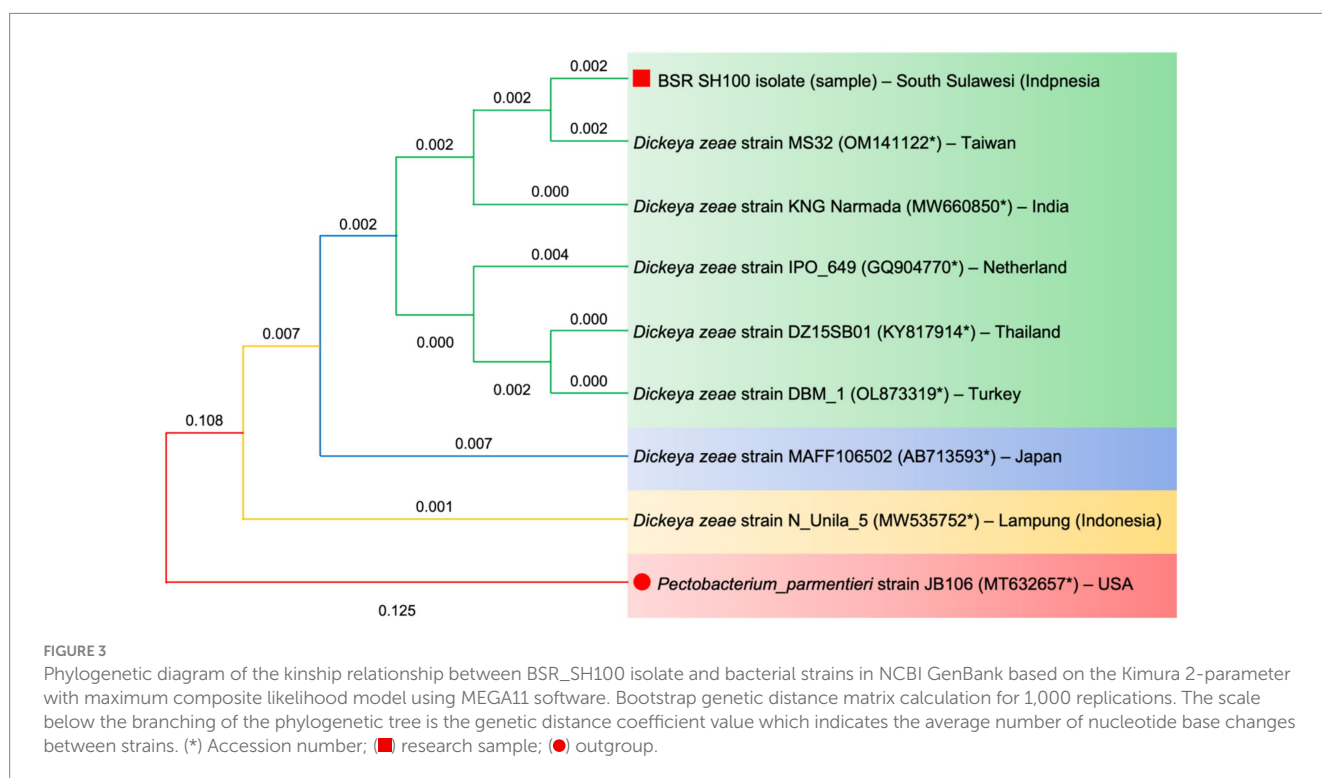
3.4 Bacterial stalk rot disease progression on hybrid corn genotypes

Disease intensity reduction analysis showed that each hybrid corn genotype had a different ability to reduce disease intensity. There were eleven of the nineteen genotypes, namely 14-4-1 × 14-21-1; 6 × B; A.26 × 14.12.1; A.26 × A.13; A.26 × A.6; A.26 × Clyn 231; A.26 × Mal.03; A.6 × A.26; N79 × A.13; Sy-02 × Sy-03; dan Sy-04 × Sy-02,

TABLE 2 The similarity level of the nucleotide sequence of the BSR_SH100 isolates with the strains in national center for biotechnology information (NCBI) genbank.

Isolate/ strain	BSR_SH100*	DZ1	DZ2	DZ3	DZ4	DZ5	DZ6	DZ7	PP**
BSR_SH100*	ID								
DZ1	99.56	ID							
DZ2	99.12	99.56	ID						
DZ3	99.12	99.56	100.00	ID					
DZ4	99.56	99.56	99.12	99.12	ID				
DZ5	98.23	98.67	98.67	98.67	98.23	ID			
DZ6	98.90	99.34	99.34	99.34	98.90	98.45	ID		
DZ7	98.68	98.90	98.90	98.90	98.68	98.45	98.68	ID	
PP**	75.82	76.43	76.43	76.43	75.82	76.76	76.43	75.82	ID

DZ1, *Dickeya zea* strain KNG Narmada from India; DZ2, *Dickeya zea* strain DZ15SB01 from Thailand; DZ3, *Dickeya zea* strain DBM_1 from Turkey; DZ4, *Dickeya zea* strain MS32 from Taiwan; DZ5, *Dickeya zea* strain N_Unila_5 from Lampung (Indonesia); DZ6, *Dickeya zea* strain IPO_649 from Netherland; DZ 7, *Dickeya zea* strain MAFF106502 from Japan; PP, *Pectobacterium parmentieri* strain JB106 from USA; (*) Research sample; (**) outgroup strain.



that had significantly higher DI reduction abilities than the comparison genotypes NK007, P89, and NK7328 at the 5% BNT test. Meanwhile, five genotypes, namely 14.12-1 × A.13; 14-4-1 × 14-21-1; A.26 × 14.12.1; A.26 × Mal.03; and B × Sy-01, in addition to having high DI reduction capabilities, also consistently had higher DS reduction capabilities compared to the comparison genotypes P89 and NK7328 in the 5% BNT test (Figure 5).

AUDPC analysis showed that five of the nineteen genotypes, namely A.26 × 14.12.1; A.26 × A.6; A.26 × Clyn 231; A.6 × A.26; and Sy-02 × Sy-03 had significantly lower AUDPC values than the comparison genotypes JH37, NK007, P89, and NK7328. The AUDPC values of A.26 × 14.12.1; A.26 × A.6; A.26 × Clyn 231; A.6 × A.26; and Sy-02 × Sy-03 genotypes were 81.69, 69.57, 53.34, 77.86 and 53.95,

respectively. Meanwhile, the AUDPC values for JH37, NK007, P89, and NK7328 were 85.17, 134.67, 490.21, and 427.42, respectively. In addition, the five genotypes also had higher protection index values than the comparison genotypes, namely >80% (Table 4).

Analysis of the BSR disease progression model based on the highest coefficient of determination value shows that the disease progression model in various corn genotypes. The BSR disease progression model in A.26 × Clyn 231; A.26 × Mal.03; and A.6 × A.26 was the same as the BSR disease progression model in NK007, P89, and NK7328, namely following the monomolecular model. Genotypes 14.12-1 × A.13; 14-4-1 × 14-21-1; 14-4-1 × A.13; 14-4-1 × Mal.03; 6 × B; A.13 × Mal.03; A.22 × 1044-14; A.24 × A.18; A.26 × A.13; A.26 × A.6; B × Sy-01; N79 × A.13; Sy-02 × Sy-03; Sy-04 × B; dan Sy-04 ×

TABLE 3 Incidence and severity of bacterial stalk rot (BSR) disease in hybrid corn genotypes disease incidence (%) at (DAP).

Genotype	Disease incidence (%)					Resistance criteria
	7 DAI	14 DAI	21 DAI	28 DAI	35 DAI	
14.12-1 × A.13	0.61 ^c	1.53 ^{cd}	1.94 ^{cd}	3.89 ^{cd}	18.66 ^{cd}	Resistant
14-4-1 × 14-21-1	3.41	3.41	3.84 ^{cd}	5.14 ^{cd}	15.77 ^{bcd}	Resistant
14-4-1 × A.13	4.12	4.12	4.1 1 ^{cd}	9.45 ^{cd}	22.49 ^{cd}	Moderately resistant
14-4-1 × Mal.03	1.72	2.58 ^{cd}	6.01 ^{cd}	7.32 ^{cd}	18.90 ^{cd}	Resistant
6 × B	0.84 ^c	1.67 ^{cd}	2.49 ^{cd}	4.57 ^{cd}	11.21 ^{bcd}	Resistant
A.13 × Mal.03	0.42 ^c	1.27 ^{cd}	3.37 ^{cd}	7.16 ^{cd}	18.51 ^{cd}	Resistant
A.22 × 1044-14	1.25 ^c	2.11 ^{cd}	3.81 ^{cd}	27.56	43.95	Susceptible
A.24 × A.18	3.31	3.31	3.31 ^{cd}	4.56 ^{cd}	17.73 ^{cd}	Resistant
A.26 × 14.12.1	0.83 ^c	1.65 ^{cd}	2.50 ^{cd}	3.33 ^{cd}	6.23 ^{bcd}	Resistant
A.26 × A.13	0.83 ^c	1.27 ^{cd}	2.97 ^{cd}	10.97 ^{cd}	12.64 ^{bcd}	Resistant
A.26 × A.6	0.83 ^c	1.23 ^{cd}	1.63 ^{cd}	3.67 ^{cd}	4.92 ^{bcd}	Very resistant
A.26 × Clyn 231	0.00 ^{cd}	0.84 ^{cd}	1.69 ^{cd}	2.53 ^{cd}	4.63 ^{bcd}	Very resistant
A.26 × Mal.03	0.00 ^{cd}	0.42 ^{cd}	2.48 ^{cd}	6.22 ^{cd}	9.56 ^{bcd}	Resistant
A.6 × A.26	0.00 ^{cd}	0.40 ^{cd}	1.66 ^{cd}	3.30 ^{cd}	11.04 ^{bcd}	Resistant
B × Sy-01	1.64 ^c	1.64 ^{cd}	1.64 ^{cd}	3.71 ^{cd}	19.24 ^{cd}	Resistant
N79 × A.13	2.09	2.09 ^{cd}	2.51 ^{cd}	5.03 ^{cd}	10.81 ^{bcd}	Resistant
Sy-02 × Sy-03	0.43 ^c	0.43 ^{cd}	0.43 ^{cd}	2.58 ^{cd}	7.66 ^{bcd}	Resistant
Sy-04 × B	0.82 ^c	1.25 ^{cd}	6.23 ^{cd}	17.01 ^{cd}	32.36 ^d	Moderately resistant
Sy-04 × Sy-02	2.06	2.06	2.06	3.30	9.89	Resistant
JH37 (a)	0.42	1.26	2.09	2.93	10.45	Resistant
NK007 (b)	0.00	1.28	2.94	3.32	22.56	Moderately resistant
P89 (c)	3.42	4.67	13.62	30.69	32.34	Moderately resistant
NK7328 (d)	2.10	4.61	10.48	22.17	40.99	Susceptible
LSD 5%	1.74	1.71	2.78	3.62	6.14	
SE	0.86	0.85	1.38	1.8	3.05	
CV	75.35	48.22	40.62	23.74	19.66	

^aDisease incidence rate was significantly lower than the comparison JH37 at the 5% LSD test level. ^bDisease incidence was significantly lower than the comparison NK007 cultivar at the 5% LSD test level. ^cDisease incidence was significantly lower than the comparison P89 cultivar at the 5% LSD test level. ^dDisease incidence was significantly lower than the comparison NK7328 cultivar at the 5% LSD test level. LSD, least significant differences. SE, standard error. CV, coefficient of variation. DAP, day after planting. MR: Moderately resistant; S: susceptible; VS: very susceptible.

Sy-02 had the same BSR disease progression model as genotype JH37, namely following the Logistic model. Meanwhile, the BSR disease progression model in A.26 × 14.12.1 follows the Gompertz model (Table 4).

The BSR disease progression in each hybrid corn genotype was also confirmed through low infection rate values. There were three of the nineteen hybrid corn genotypes, namely A.26 × Clyn 231, A.26 × Mal.03, and A.6 × A.26 which had relatively low infection rate values compared to the comparison genotypes of 0.0016, 0.0037, and 0.0038, respectively. The infection rate value in NK007 was quite low and equivalent to 26 × Clyn 231, A.26 × Mal.03, and A.6 × A.26, which was 0.0076. Meanwhile, the infection rate values in JH37, P89, and NK7328 were quite high, which were 0.1081, 0.0148, and 0.0174, respectively (Table 4). Nonlinear progression models, namely monomolecular, logistic, and Gompertz, were commonly used to describe plant disease progression dynamics. These models assume that the underlying carrying capacity was constant. These models assumed that the underlying carrying capacity was constant so the

development of more realistic modeling in this study followed the same upper asymptote general nonlinear progression model.

The dendrogram analysis revealed the clustering of maize genotypes based on their resistance levels to BSR disease, as measured by three key variables: Disease severity reduction, disease incidence reduction, and protection index. In general, the genotypes were grouped into three main clusters. The first cluster, consisting of Sy-04 × B, P89 (c), A.22 × 1044-14, and NK7328 (d), appeared on separate branches, indicating a low level of disease reduction and, therefore, representing the most susceptible genotypes. The second cluster included A.26 × 14.12.1, A.26 × Mal.03, 14-4-1 × 14-21-1, 14-4-1 × Mal.03, A.13 × Mal.03, NK007 (b), 14.12-1 × A.13, B × Sy-01, 14-4-1 × A.13, A.24 × A.18, and A.26 × A.13, which exhibited moderate ability in reducing both disease incidence and severity, as well as providing intermediate protection. Meanwhile, the third cluster, comprising A.6 × A.26, Sy-02 × Sy-03, A.26 × A.6, A.26 × Clyn 231, Sy-04 × Sy-02, JH 37 (a), 6 × B, and N79 × A.13, demonstrated the

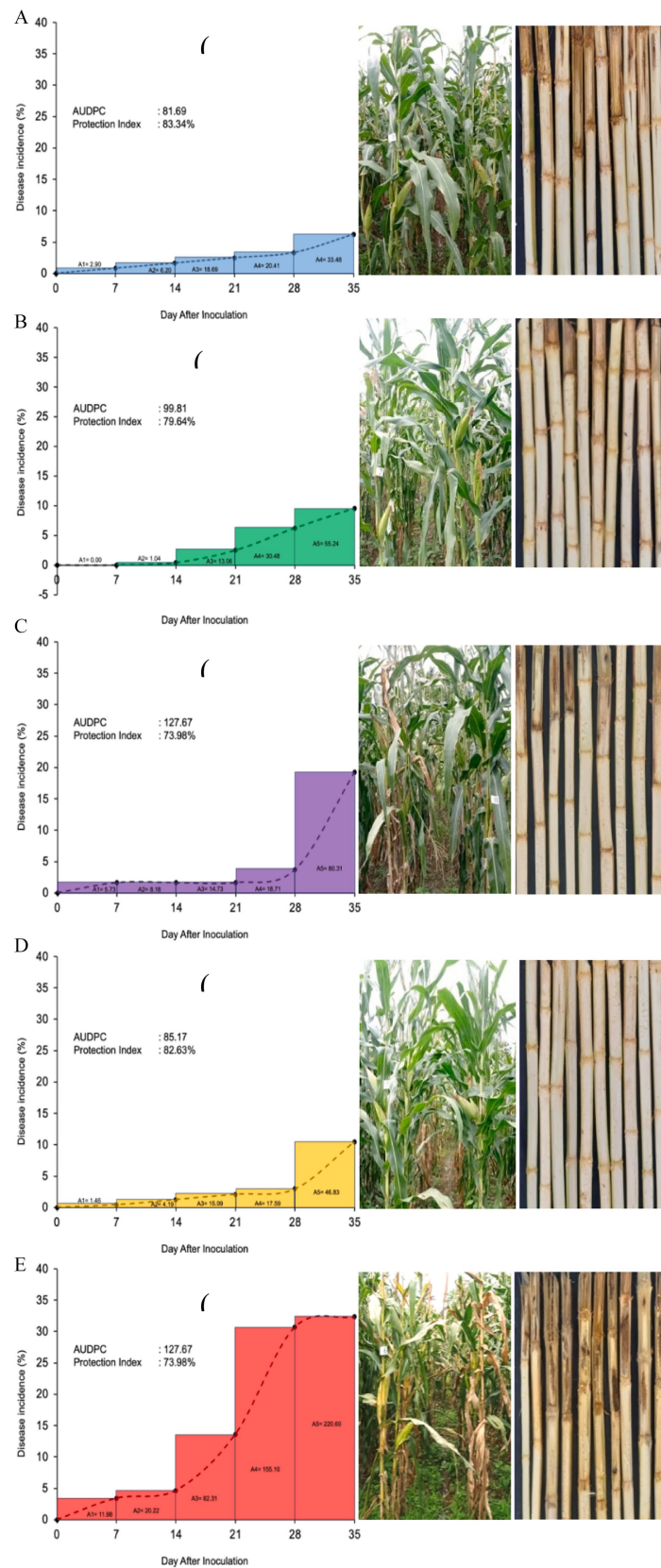


FIGURE 4 Effect of hybrid corn genotype on the area under the disease progression curve (AUDPC) and protection index: **(A)**, A.26 × 14.12.1; **(B)**, A.26 × Mal.03; **(C)**, B × Sy-01; **(D)**, JH37; **(E)**, P89. A₁: AUDPC of the 1st time interval; A₂: AUDPC of the 2nd time interval; A₃: AUDPC of the 3rd time interval; A₄: AUDPC of the 4th time interval; A₅: AUDPC of the 5th time interval.

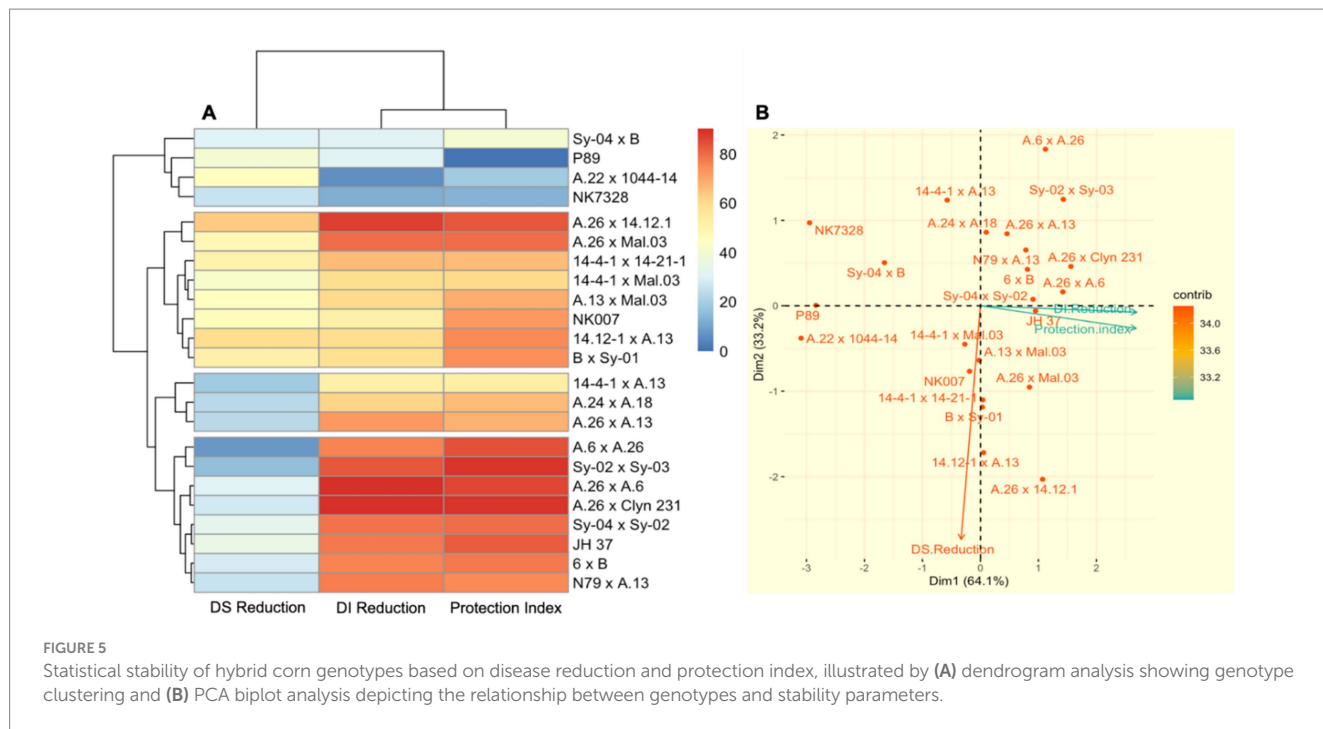


TABLE 4 Disease progression model of bacterial stalk rot (BSR) in hybrid corn genotypes.

Genotype	Disease progression model	AUDPC	Infection rate	Regression equation	R Square
14.12-1 × A.13	Logistic	122.42 ± 29.75	0.1180	y = 0.11801x-6.06081	0.9180
14-4-1 × 14-21-1	Logistic	166.20 ± 25.44	0.0539	y = 0.05386x-4.03555	0.7056
14-4-1 × A.13	Logistic	231.34 ± 39.58	0.0676	y = 0.06756x-4.00837	0.7732
14-4-1 × Mal.03	Logistic	193.86 ± 34.59	0.0898	y = 0.08977x-4.77112	0.9640
6 × B	Logistic	107.84 ± 20.59	0.0932	y = 0.09321x-5.48624	0.9812
A.13 × Mal.03	Logistic	153.23 ± 34.57	0.1406	y = 0.14061x-6.39941	0.9959
A.22 × 1044-14	Logistic	399.44 ± 98.88	0.1579	y = 0.15792x-5.83816	0.9299
A.24 × A.18	Logistic	163.48 ± 27.16	0.0575	y = 0.05753x-4.14861	0.6356
A.26 × 14.12.1	Gompertz	81.69 ± 12.82	0.0183	y = 0.01831x-1.69126	0.9753
A.26 × A.13	Logistic	158.59 ± 32.90	0.1144	y = 0.01144x-5.73251	0.9540
A.26 × A.6	Logistic	69.57 ± 11.35	0.0694	y = 0.06944x-5.37323	0.9690
A.26 × Clyn 231	Monomolecular	53.34 ± 10.07	0.0016	y = 0.00159x-0.01392	0.9467
A.26 × Mal.03	Monomolecular	99.81 ± 22.32	0.0037	y = 0.00374x-0.03970	0.9275
A.6 × A.26	Monomolecular	77.86 ± 19.49	0.0038	y = 0.00375x-0.04454	0.7536
B × Sy-01	Logistic	127.67 ± 29.67	0.0889	y = 0.08891x-5.27782	0.7142
N79 × A.13	Logistic	120.31 ± 19.75	0.0623	y = 0.06227x-4.58835	0.8418
Sy-02 × Sy-03	Logistic	53.95 ± 13.66	0.1137	y = 0.11373x-6.92032	0.7989
Sy-04 × B	Logistic	295.84 ± 67.91	0.1559	y = 0.15591x-6.11167	0.9796
Sy-04 × Sy-02	Logistic	100.94 ± 16.04	0.0533	y = 0.05331x-4.54189	0.6948
JH 37	Logistic	85.17 ± 17.56	0.1081	y = 0.10811x-6.13898	0.9485
NK007	Monomolecular	134.67 ± 34.73	0.0076	y = 0.00761x-0.09340	0.6211
P89	Monomolecular	490.21 ± 89.44	0.0148	y = 0.01475x-0.11232	0.9052
NK7328	Monomolecular	427.42 ± 85.37	0.0174	y = 0.01738x-0.17345	0.8564

highest capacity to suppress disease development and offered maximum protection. The dendrogram analysis confirmed that there were significant differences among genotypes in terms of disease resistance. Genotypes belonging to the third cluster could be considered promising candidates for breeding programs aimed at developing disease-resistant maize varieties, while those in the first cluster represented susceptible genotypes that are less recommended for cultivation in disease-prone environments. Therefore, this clustering provides a scientific basis for genotype selection strategies in the development of maize varieties with enhanced disease resistance (Figure 5A).

Based on the results of the PCA biplot analysis, the resistance of maize genotypes to the disease observed through the parameters of disease incidence reduction, disease severity reduction, and protection index showed a distinct clustering pattern. The vectors of disease reduction and protection index appeared parallel and oriented toward the lower right side of the biplot, indicating a strong positive correlation between these two parameters. Genotypes associated with both parameters, such as A.26 × Clyn 231, A.26 × A.6, A.26 × A.13, and Sy-02 × Sy-03, tended to exhibit high resistance, as they were able to suppress disease incidence while simultaneously enhancing the protection index. Meanwhile, the DS reduction vector pointed toward the lower left side of the biplot, separated from the other two parameters. This suggests that genotypes associated with this vector are more prominent in reducing disease severity, although not necessarily in parallel with reductions in incidence or increases in protection index. Several genotypes positioned near this vector, such as A.26 × 14.12.1, 14.12.1 × A.13, B × Sy-01, and 14-4-1 × 14-21-1, can be categorized as having resistance mechanisms that primarily focus on mitigating symptom severity. Conversely, genotypes such as P89, NK7328, and A.22 × 1044-14, which were positioned far from all resistance-related vectors, tended to exhibit low levels of resistance. This indicates that these genotypes were less effective in reducing both disease incidence and severity, as well as having a low protection index (Figure 5B).

3.5 Interplay of weather factors on bacterial stalk rot disease

Pearson's correlation analysis of the climate factors' influence on the incidence and severity of corn BRS disease showed that at 28 DAI, rainfall was significantly positively correlated with the level of disease incidence. This suggests that higher rainfall may lead to more BSR disease incidences. Other climate factors such as average humidity, length of sun exposure, maximum wind velocity, and wind direction at maximum velocity showed no significant correlation with the incidence and severity of BSR disease. The minimum temperature factors had a weak positive correlation with the incidence of BSR disease at 7–35 DAI, with a correlation coefficient value of 0.11–0.20. At the beginning of the observation, the average humidity was weakly negatively correlated with the disease incidence rate at 7 and 14 DAI (Figure 6). This indicated that environmental humidity levels had a very weak correlation with the penetration and initial spread of *D. zeae*. In this study, *D. zeae* infection achieved by artificial. Unlike naturally occurring pathogen infections, which require environmental conditions that allow penetration into plant tissue, such as

temperatures of approximately 25–35 °C and high relative humidity (around 90%).

Path analysis based on the correlation between weather factors with the incidence and severity of BSR disease showed partial variations in positive and negative path coefficient values. Minimum and maximum temperatures, as well as rainfall, had a positive partial effect on BSR disease incidence with path coefficient values of 0.203, 0.366, and 0.262, respectively. Meanwhile, the average temperature, average humidity, length of sun exposure, maximum wind velocity, wind direction, and average wind velocity had a partial negative effect on BSR disease incidence with path coefficient values of (−0.160), (−0.009), (−0.112), (−0.273), (−0.246), and (−0.212), respectively. The partial effect was slightly different on BSR disease severity, where minimum and maximum temperatures, length of sun exposure, maximum wind velocity, and wind direction had a positive partial effect on BSR disease severity with path coefficient values of 0.051, 0.211, 0.036, 0.183, and 0.014, respectively. Meanwhile, average temperature, average humidity, rainfall, and average wind velocity had a partial negative effect on BSR disease severity with path coefficient values of (−0.254), (−0.033), (−0.101), and (−0.055), respectively. The results of the analysis showed that partial weather factors significantly affect BSR disease progression, both directly and indirectly (Figure 7).

The correlation between weather factors and BSR disease progression was very clearly illustrated. This was because plants that grow and develop in an area require optimal weather conditions and are closely related to the presence of phytopathogens. Therefore, phytopathogens require environmental elements in the form of spatial components, physical environment, physical-chemical environment, weather conditions, and periods in causing disease epidemics. The causal correlation between weather factors and BSR disease progression was known through correlation analysis as well as the magnitude of its direct and indirect influence on the disease progression based on path analysis. Diagram of path analysis on Figure 7 showed that the response of the dependent variable (incidence and severity of BSR disease) was a direct result of the independent variable or an indirect result of other variables from weather factors and genotype, either negatively or positively. The path analysis also showed that there was a negative or positive correlation between weather factors influencing BSR disease progression. The synergistic interaction between weather factors, phytopathogens, and plants increased the disease progression, whereas in absence of synergy, the disease progression would be slow.

4 Discussion

The bacterial isolate BSR_SH100 was identified as *D. zeae* both by colony morphology, microscopically, and nucleotide base sequencing. The colony characters of the BSR_SH100 isolate were the same as the morphological characteristics of *D. zeae* reported by Zhang et al. (2020), viz., circular and convex colonies, white to cream, and colonies like fried eggs on PDA or NA medium enriched with 2% glucose. Meanwhile, the microscopic characteristics were also similar to the report of Liu et al. (2016) which states that *D. zeae* had rod-shaped cells measuring 0.8–3.2 × 0.5–0.8 μm (average 1.8 × 0.6 μm). Nucleotide base sequence analysis of the housekeeping gene dnaX confirmed that the BSR_SH100 isolate had a very close kinship relationship with *D. zeae* with a very small genetic distance coefficient,

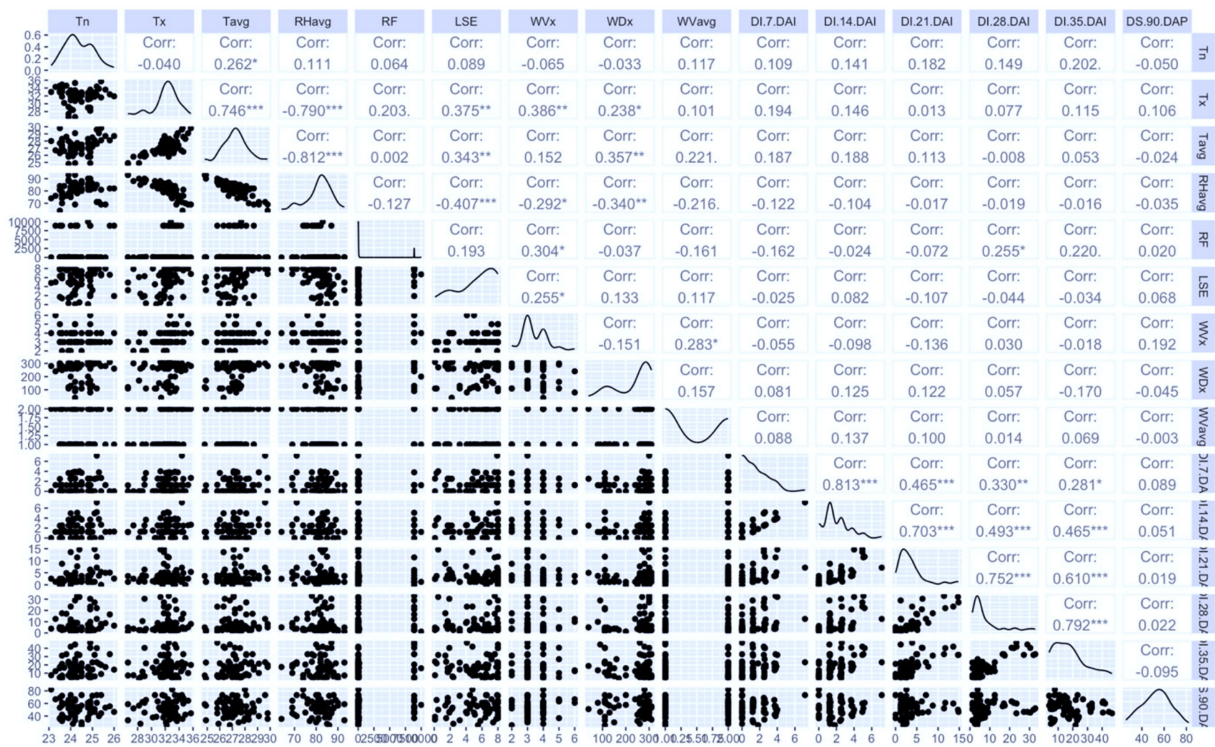


FIGURE 6 Correlation of weather factors with bacterial stalk rot disease. Tn, Temperature minimum; Tx, temperature maximum; Tavg, temperature rata-rata; RHavg, kelembaban rata-rata; LSE, lama penyinaran; WVMax, kecepatan angin maksimum; WDMax, arah angin pada saat kecepatan maksimum; DR, disease reduction; DS, disease severity; DAP, day after planting.

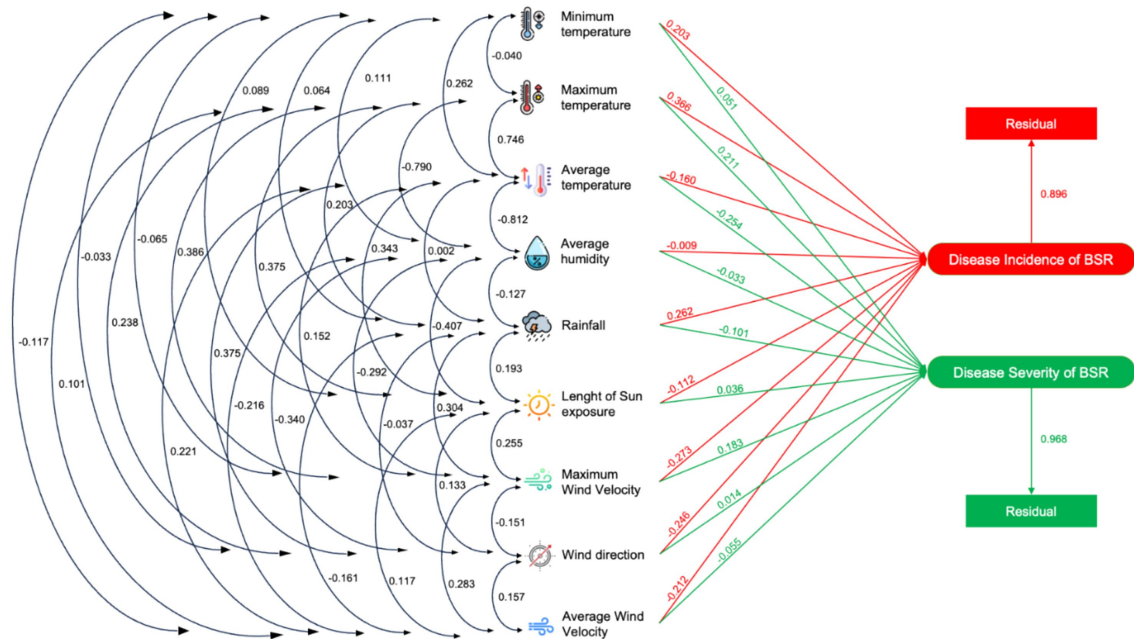


FIGURE 7 Path diagram of the influence of weather factors on bacterial stalk rot disease. The value on the path connecting weather factors and *Maydis* leaf blight disease (black) is the path coefficient. The value on the path connecting between weather factors (red and green) is the correlation coefficient.

ranging from 0.000–0.002. Several studies have reported that the *dnaX* gene encoding the DNA polymerase II subunit is used as a strong genetic marker to distinguish species of *Dickeya* and *Pectobacterium* because it has sequences with conserved and informative variable regions (Zeigler, 2003; Sławiak et al., 2009; Suharjo et al., 2014; Zhang et al., 2014; Potrykus et al., 2016; Aeny et al., 2020). Therefore, morphological characters combined with molecular analysis data have become a trend in identifying *Dickeya* species.

The observations result of BSR disease intensity showed that A.26 × 14.12.1, A.26 × Mal.03, and B × Sy-01 consistently had relatively low disease intensity in both incidence and severity and reacted resistant to moderately resistant to BSR disease. The first phenotypic observation of BSR disease was carried out at 7 DAI with further observations at 14, 21, 28, and 35 DAI. Some of the early symptoms of BSR disease observed in this study were premature wilting of leaves followed by changes in leaf sheath color. According to Subedi et al. (2016), corn yields are more significantly impacted by stalk rot that develops after flowering than by rot that develops during the vegetative period. The leaves of infected maize will begin to yellow at the top and eventually become yellow as a whole (Ahamad et al., 2015). External signs and symptoms on the stem include colour changes in the diseased tissue and maceration of the stem and basal segments, which causes soft rot (Kumar et al., 2017).

Differences in the BSR disease progression in each hybrid corn genotype were also reflected in the AUDPC values and protection index. The A.26 × 14.12.1; A.26 × A.6; A.26 × Clyn 231; A.6 × A.26; and Sy-02 × Sy-03 genotypes had AUDPC values significantly lower than the comparison genotypes, which were 81.69, 69.57, 53.34, 77.86 and 53.95, respectively, and had the highest protection index values of 83.34, 85.81, 89.12, 84.12 and 89.00%, respectively. The AUDPC curve of the BSR disease showed the dynamics of the epidemic over time (Figure 4). The frequency and quantity of inoculum, variations in host sensitivity over the growth season, meteorological phenomena, and the efficacy of cultural and control methods can all be determined using this mathematical tool. A number of variables, including plant age, inoculum concentration, host resistance, bacterial strain, and environmental circumstances, affect how a disease develops after inoculation (Agrios, 2005; Adorada et al., 2013). According to Astiko and Sudantha (2023), the narrower the disease progression area, the more resistant the plant. The difference in AUDPC values for each genotype was thought to be caused by differences in the corn resistance level and the influence of weather factor interactions. This study analyzed the correlation between differences in corn genotypes, and weather factors with the BSR disease progression. Kumar et al. (2017) also explained that the BSR disease progression is greatly influenced by the virulence and aggressiveness of bacteria and differences in the resistance of corn cultivars or germplasm lines. According to Kumar et al. (2017), several strategies can be used to suppress the level of *D. zeae* infection, such as lowering inoculum production, infection levels, or pathogen development by choosing a planting season that is not conducive to the pathogen, lowering inoculum from outside sources during epidemics, and assembling resistant cultivars.

The BSR disease progression model shown in each genotype was different and dominated by the logistic model. These disease progression models described the dynamics of the BSR disease epidemic that occurred during the study period and can be used as a predictive model for the BSR disease progression. The monomolecular model shown in genotypes A.26

× Clyn 231; A.26 × Mal.03; A.6 × A.26; NK007, P89, and NK7328 were suitable for modeling epidemics that do not have secondary spread in one growing season, this is based on the explanation of Chauhan and Chandel (2018) that the monomolecular model describes plant diseases as having only one cycle during the growing season. This model is also called the negative exponential model (Campbell and Madden, 1990). An inoculum from diseased plants can infect nearby plants, or appropriate environmental support can result in recurrent infection cycles. These soil-borne diseases typically follow a monomolecular disease development model and are present in various models in the field (Bande et al., 2015). The logistic model shown in genotypes 14.12-1 × A.13; 14-4-1 × 14-21-1; 14-4-1 × A.13; 14-4-1 × Mal.03; 6 × B; A.13 × Mal.03; A.22 × 1044-14; A.24 × A.18; A.26 × A.13; A.26 × A.6; B × Sy-01; N79 × A.13; Sy-02 × Sy-03; Sy-04 × B; Sy-04 × Sy-02; and JH37 illustrated the polycyclic disease spread, meaning that there was a secondary spread in one growing season. This progression model is most widely used to describe plant disease epidemics (Segarra et al., 2001; Jeger, 2004). Meanwhile, the Gompertz model was shown in genotype A.26 × 14.12.1. The Gompertz model is suitable for polycyclic diseases as an alternative to the logistic model. The absolute rate curve of the Gompertz model peaked faster and decreased more slowly than that of the logistic model (Contreras-Medina et al., 2009). Plant disease progression models that incorporate several factors to depict the dynamics of disease across time generally work effectively, but such models are sometimes not suitable for the process of obtaining key characteristics because they often ignore relevant variables that influence the disease epidemic progression (Xu, 2006), such as plant host development, changing environmental circumstances, duration of infectious and latent phases, etc. However, advances in statistical and computing technology have made it possible to combine several types of characteristics to obtain more reliable models. Critical evaluation of the assumptions underlying plant disease epidemic models is essential to minimize systematic errors and ensure accurate interpretation of disease progression (Xu, 2006; Chauhan and Chandel, 2018).

The BSR disease progression was not only influenced by cultivar resistance, but also by weather factors at the experimental location which caused interactions between phytopathogens, plant host, and weather conditions. Pearson's correlation analysis showed that there was an interaction between weather factors in influencing BSR disease progression, both positively and negatively. In addition, path analysis also showed that weather factors play a major role in the BSR disease progression, where minimum and maximum temperatures, as well as rainfall, had a partial positive effect on BSR disease incidence. Meanwhile, the average temperature, average humidity, length of sun exposure, maximum wind velocity, wind direction, and average wind velocity had a partial negative effect on BSR disease incidence. The partial effects were slightly different on BSR disease severity, where minimum and maximum temperatures, length of sun exposure, maximum wind velocity, and wind direction had a positive partial effect on BSR disease severity. Meanwhile, average temperature, average humidity, rainfall, and average wind velocity had a partial negative effect on BSR disease severity. Based on the two analyses, it was shown that the duration of weather factors played a major role in various pathogen growth and development processes, especially at the generative age of plants. According to Bez et al. (2021), the spread of BSR disease requires conditions of decreased O₂ concentration, high temperature, high humidity, and the presence of a water layer on

the surface of plant organs. *D. zea* thrives and produces plant cell wall degrading enzymes (PCWDEs) that cause disease progression in susceptible hosts. Although the development and presence of *D. zea* may depend on many factors, this study only focused on the influence of corn genotypes and weather conditions on disease progression because these factors are biologically and agroclimatically significant in defining disease progression over time. In many previous studies, these factors were selected to assess habitat and its implications for disease management decision making. Disease epidemic variables and weather factors used for disease progression modeling have been widely reported for other diseases in corn (Mirsam et al., 2025). These variables certainly play an important role in predicting BSR disease progression and its control strategies (Shahid et al., 2024).

5 Conclusion

This study suggested that three out of nineteen hybrid maize genotypes—A.26 × 14.12.1, A.26 × Mal.03, and B × Sy-01—consistently exhibited resistant to moderately resistant reactions to BSR, with disease incidence and severity below 40% and protection index values exceeding 70%, indicating their strong potential to suppress BSR development. Pearson correlation and path analysis showed that there was interaction between weather factors in influencing the BSR disease progression, both positively and negatively. Based on the two analyses, it was seen that weather factors play a major role in various pathogen growth and progression processes, especially in the generative phase. These findings imply that the integration of resistant hybrid maize genotypes with an improved understanding of environmental influences can contribute to more effective BSR disease management strategies. However, further in-depth and systematic validation using epidemiological, metabolomic, and metagenomic approaches is required to comprehensively elucidate the mechanisms underlying BSR suppression by hybrid maize genotypes.

Data availability statement

The datasets presented in this study can be found in online repositories. The names of the repository/repository and accession number(s) can be found in the article/supplementary material.

Author contributions

SS: Resources, Formal analysis, Writing – original draft, Methodology, Conceptualization, Investigation. HM: Methodology, Writing – original draft, Formal analysis, Investigation, Data curation, Validation, Conceptualization. MAZ: Methodology, Writing – review & editing, Supervision, Data curation, Validation. MF: Funding acquisition, Resources, Validation, Project administration, Supervision, Conceptualization, Writing – review & editing, Data curation, Methodology. MAN: Supervision, Writing – review & editing, Data curation, Visualization, Methodology. BP: Methodology, Writing – review & editing, Conceptualization. TK: Writing – review & editing, Supervision,

Data curation. SH: Project administration, Data curation, Writing – review & editing, Validation. AN: Writing – review & editing, Validation, Data curation.

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Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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