

## Technical Note

# REACTION OF SWEET CORN GENOTYPES TO BACTERIAL STALK ROT

Isadora Gonçalves da Silva<sup>1</sup>, Renata Castoldi<sup>2</sup>, Nilvanira Donizete Tebaldi<sup>3</sup>, Dahis Ramalho Moura<sup>3</sup>, Gabriel Mascarenhas Maciel<sup>2</sup>, Ana Carolina Pires Jacinto<sup>3</sup> and Patricia Diniz Graciano<sup>3</sup>

### ABSTRACT

Bacterial stem rot, caused by *Dickeya zea*, is among the main bacterial diseases of sweet corn, and finding sources of resistance is important. The objective was to evaluate the reaction of sweet corn genotypes to *Dickeya zea*. The experiment was carried out at the Federal University of Uberlândia, in an 11 x 2 factorial scheme with four replications and in a randomized block design. The treatments consisted of 10 sweet corn genotypes (L2P1, L2P11, L2P33, L2P37, L2P45, L3P27, L4P19, L5P3, L5P18, L5P42), belonging to the Plant Germplasm Bank of UFU, Campus Monte Carmelo, a common commercial hybrid susceptible to *Dickeya zea* (AS1633), and two *D. zea* isolates (UFU J23 and UFU H113). At 45 days after planting, inoculation was performed using the syringe method. Evaluations were performed at 4, 8, 12 and 16 days after inoculation, observing the size of the lesion. In addition, the incubation period, disease severity and area under the disease progress curve were determined. Analysis of variance was performed and means were compared using the Scott-Knott test. The onset of the disease occurred between 96 and 204 hours after inoculation. The L2P33 and L2P37 genotypes showed high values of area under the disease progress curve and disease severity, regardless of the isolate. The genotypes were classified as moderately susceptible, with emphasis on the L2P11 genotype.

**Additional keywords:** *Dickeya zea*, resistance, *Zea mays*

### RESUMEN

#### Reacción de genotipos de maíz dulce a la pudrición bacteriana del tallo

La pudrición bacteriana del tallo, causada por *Dickeya zea*, se encuentra entre las principales enfermedades bacterianas del maíz dulce, y es importante encontrar fuentes de resistencia. El objetivo fue evaluar la reacción de genotipos de maíz dulce a *Dickeya zea*. El experimento fue realizado en la Universidad Federal de Uberlândia, en arreglo factorial 11 x 2 con cuatro repeticiones y en diseño de bloques al azar. Los tratamientos consistieron en 10 genotipos de maíz dulce (L2P1, L2P11, L2P33, L2P37, L2P45, L3P27, L4P19, L5P3, L5P18, L5P42), pertenecientes al Banco de Germoplasma Vegetal de la UFU, Campus Monte Carmelo, un híbrido comercial común susceptible a *Dickeya zea* (AS1633) y dos aislamientos de *D. zea* (UFU J23 y UFU H113). A los 45 días después de la siembra se realizó la inoculación por el método de la jeringa. Se realizaron evaluaciones a los 4, 8, 12 y 16 días después de la inoculación, observando el tamaño de la lesión. Además, se determinaron el período de incubación, la severidad de la enfermedad y el área bajo la curva de progreso de la enfermedad. Se realizó análisis de variancia y comparación de medias mediante la prueba de Scott-Knott. El inicio de la enfermedad ocurrió entre 96 y 204 horas después de la inoculación. Los genotipos L2P33 y L2P37 mostraron altos valores de área bajo la curva de progreso de la enfermedad y severidad de la enfermedad, independientemente del aislado. Los genotipos se clasificaron como moderadamente susceptibles, con énfasis en el genotipo L2P11.

**Palabras clave adicionales:** *Dickeya zea*, resistencia, *Zea mays*

### INTRODUCTION

Breeding programs for sweet corn (*Zea mays* L. subsp. *saccharata*) have many similarities to those for common maize; however, the fact that sweet corn is considered a vegetable and not a grain means its qualitative characteristics are more

relevant (Pereira and Teixeira, 2016).

As such, biotic factors such as pest attack and disease, which affect traits related to appearance, flavor and texture, directly impact the final product (Kumar et al., 2016), reducing the commercial value of the crop or making it unsuitable for commercialization.

Received: December 26, 2022

Accepted: October 23, 2023

<sup>1</sup>Departamento de Agricultura, Universidade Federal de Lavras, Campus Universitário. Lavras, MG, Brazil  
e-mail: isadoragsilva@live.com

<sup>2</sup>Instituto de Ciências Agrárias, Universidade Federal de Uberlândia, Campus Monte Carmelo, Rodovia. Monte Carmelo, MG, Brazil. e-mail: rcastoldi@ufu.br; gabrielmaciel@ufu.br

<sup>3</sup>Instituto de Ciências Agrárias, Universidade Federal de Uberlândia, Campus Glória. Uberlândia, MG, Brazil  
e-mail: nilvanira.tebaldi@ufu.br; dahis\_ramalho@hotmail.com; carol.agro.ufu@gmail.com (corresponding author); patriciagraciano01@hotmail.com

Sweet corn cultivation has gained ground in Brazil because it is an excellent alternative for center-pivot irrigation systems, for its wide diversification of use and due to the growing number of vegetable processing plants, particularly in the states of Goiás, São Paulo, Rio Grande do Sul and Minas Gerais (Pereira and Teixeira, 2016).

However, the expansion of cultivated areas has also exacerbated crop management difficulties, owing to the emergence of several diseases that can significantly compromise grain yield and quality.

Among the diseases that occur in sweet corn, bacterial stalk rot, caused by *Dickeya zeae* (Samson et al., 2005), is considered the most severe (Kumar et al., 2017a). Nevertheless, there are currently no tropical sweet corn hybrids available that are resistant to this pathogen.

The genus *Dickeya* was initially named *Erwinia* by Winslow et al. (1917) and in 1945, Waldee proposed that the pectinolytic species were transferred to the genus *Pectobacterium* (Robbs, 1981). The last alteration, with a change to the new genus, was performed by Samson et al. (2005).

Their rapid dissemination and multiplication, wide range of hosts and highly variable physiological characteristics make bacteria difficult to control (Kumar et al., 2015); and although several chemical compounds are used for this purpose, none have proved efficient (Soleimani et al., 2015).

Given that bacterial stalk rot causes significant losses, is difficult to control and that Brazilian climate conditions favor its development, further research on sweet corn resistance to *Dickeya zeae* is needed. Thus, the aim of this study was to assess the reaction of sweet corn genotypes to *Dickeya zeae*.

## MATERIALS AND METHODS

The experiment was conducted from October 2018 to January 2019, in a greenhouse and the Plant Bacteriology Laboratory (LABAC) at the Institute of Agricultural Sciences of Universidade Federal de Uberlândia – UFU, in Minas Gerais State (MG), Brazil. The temperature in the greenhouse ranged from 18 to 35 °C and the relative humidity ranged from 48 to 88 % during the experiment. A randomized block design was

used, with an 11 x 2 factorial scheme and four repetitions, totaling 88 plot. Each experimental plot consisted of one 5-liter pot containing four plants and filled with substrate composed of sterile soil, sand and vermiculite at a proportion of 3:1:1 (v/v). At the end, 352 plants were evaluated.

The treatments consisted of ten F3 generation sweet corn genotypes (L2P1, L2P11, L2P33, L2P37, L2P45, L3P27, L4P19, L5P3, L5P18, L5P42) from the UFU Vegetable Germplasm (Monte Carmelo campus), one common commercial sweet corn hybrid susceptible to *Dickeya zeae* used as control (AS1633), and two *Dickeya zeae* isolates (UFU J23 and UFU H113) from the working collection of LABAC.

Isolates were taken from the stalks of infected maize plants. Furthermore, pre-tests were carried out to choose the most aggressive and stable isolates under extreme environmental conditions of temperature and relative humidity. To determine whether the soft rot symptoms (steam pith soaked, macerated and greenish) present in the genotypes were in fact caused by bacteria, one plant from each genotype was inoculated with sterile water.

The isolates were cultivated in bacteria screening medium 523 (Kado and Heskett, 1970), consisting of 10 g sucrose, 8 g hydrolyzed casein, 4 g yeast extract, 2 g K<sub>2</sub>HPO<sub>4</sub>, 0,3 g MgSO<sub>4</sub>.7H<sub>2</sub>O, 20 g agar and 1000 mL water, for 48 h at 28 °C. Inoculation was performed 45 days after planting, when plants were at vegetative growth stage V<sub>4</sub> (four fully expanded leaves).

To that end, sterilized filtered water was added to the Petri dish containing the bacterial growth and a suspension concentration was adjusted in a spectrophotometer to 600 nm ( $D_{600} = 1.5$ ). This corresponding to approximately  $9 \times 10^{13}$  UFC mL<sup>-1</sup>. A syringe was used to deposit 0.02 mL of the suspension containing Tween 80 (0.05 %) on the second basal internode (Rangarajan and Chakravarti, 1967). The plants were placed in a humidity chamber 24 h after inoculation. Irrigation was performed frequently in order to keep the soil moist, which was important for disease development.

At 4, 8, 12 and 16 days after inoculation, lesion length was measured on four plant per treatment, cutting the stalks longitudinally and classifying infected plants on a scale of 1 to 5 (Lal, 1981) to determine the following epidemiological

components: A) incubation period of the disease (IP), corresponding to the time between inoculation and symptom emergence in four predefined days. B) Disease severity (SEV), measured 16 days after inoculation on a descriptive scale from 1 to 5 (Lal, 1981), in which 1 = infection is limited to a very small spot in the pith at the site of inoculation; 2 = disease infection spreads on half of the length of inoculated internode in the pith and cortical tissues, rind not infected; 3 = disease infection covers the entire length of the inoculated internode but does not cross nodal plates; the rind is green and symptoms are not visible externally, but plant shows signs of wilting, 4 = nodal plates are crossed and increasing infection also covers adjacent internode of the inoculated plants, the pith and cortical tissue are degenerated, the rind of the inoculated internode is affected and plant wilts; 5 = the disease spreads in three or more internodes; the pith, cortical tissue and vascular bundles rot and disorganized, plants wilt and may topple down. C) Area under the disease progress curve (AUDPC), calculated using the formula:

$$\text{AUDPC} = \sum [((y_1 + y_2) / 2) \times (t_2 - t_1)]$$

where,  $y_1$  and  $y_2$  are two consecutive assessments of the proportion of damaged tissue performed at time  $t_1$  and  $t_2$ , respectively.

The reaction of the ten sweet corn genotypes to *Dickeya zea* was classified into levels of resistance, based on the scoring system proposed by Kumar et al. (2017a), with some adaptations, in

association with Lal's scale (Lal, 1981), where 0 = resistant; 0.1-1 = moderately resistant; 1.1-3 = moderately susceptible; 3.1-5 = susceptible.

The data were submitted to analysis of variance and means compared with the Scott-Knott test at 0.05 probability, using R software. The disease progress curves and bidirected graph were constructed in SigmaPlot software, version 10.

## RESULTS AND DISCUSSION

There was interaction between genotypes and isolates for the variables AUDPC and SEV, according to the F-test at 1% probability. With respect to IP, interaction and isolates did not differ significantly according to the F-test at 5% probability, and as such, genotype (main effect) was presented and discussed separately (Table 1).

Since IP analysis requires destruction of the plant, it was evaluated on the same days as the other characteristics, making it impossible to pinpoint the exact time that symptoms emerged. For example, symptom onset in genotypes L2P1, L2P33, L2P45, L4P19 and L5P18 occurred 0 to 96 h after inoculation and not necessarily at the upper limit of this time period (Table 1). This time band also encompasses the symptom onset period in *Brassica rapa* which is of 15.6 to 23.7 hours (Melo et al., 2017) and 14.6 to 51.4 hours (Silva et al., 2014), respectively, after inoculation with *Pectobacterium carotovorum* subsp. *carotovorum* (Jones) Hauben et al. (1998).

**Table 1.** Mean values for the area under the disease progress curve (AUDPC), disease severity (SEV) and incubation period (IP) of eleven sweet corn genotypes and two *Dickeya zea* isolates. Universidade Federal de Uberlândia, Uberlândia-MG, 2019

Genotype	AUDPC		SEV		IP
	Isolate		UFU J23	UFU H113	
	UFU J23	UFU H113			
L2P1	37.5 Aa	39.5 Ab	3.00 Ab	2.50 Ab	96 b
L2P11	17.5 Ab	16.5 Ac	2.00 Ab	1.50 Ac	192 a
L2P33	43.0 Ba	57.5 Aa	4.25 Aa	4.00 Aa	96 b
L2P37	45.0 Aa	40.0 Ab	4.50 Aa	3.50 Aa	108 b
L2P45	44.0 Aa	22.0 Bc	4.75 Aa	2.75 Bb	96 b
L3P27	18.5 Ab	24.5 Ac	1.75 Ab	1.75 Ac	156 a
L4P19	31.5 Aa	39.0 Ab	4.75 Aa	2.75 Bb	96 b
L5P3	36.5 Aa	40.5 Ab	1.75 Ab	1.75 Ac	132 b
L5P18	38.5 Aa	25.5 Bc	2.00 Ab	2.25 Ac	96 b
L5P42	24.0 Ab	28.0 Ac	1.50 Bb	2.75 Ab	144 a
AS1633	7.0 Ac	17.0 Ac	1.25 Ab	1.75 Ac	204 a

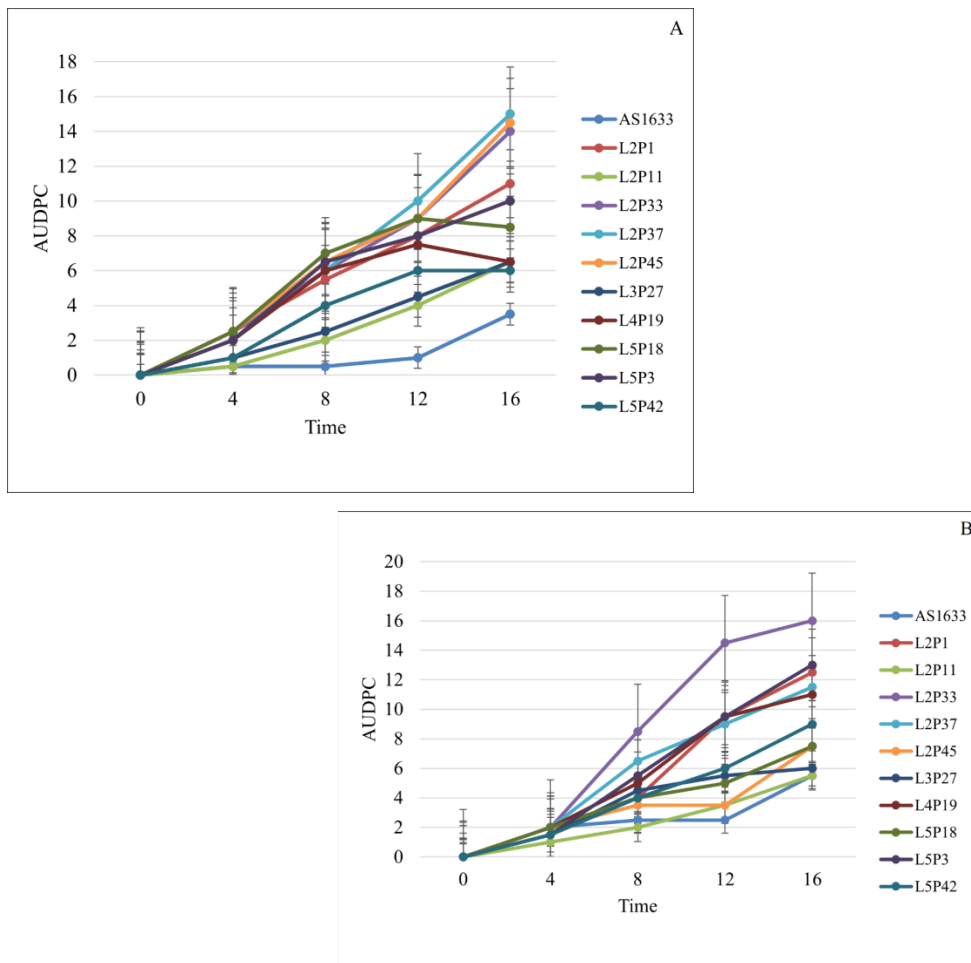
\*Means followed by the same uppercase letter in the rows and lowercase letter in the columns belong to the same group according to the Scott-Knott test at 5 % probability.

The hybrid used as a susceptible control showed symptoms 204 hours after inoculation. Although the symptom onset period was longer than that of the remaining genotypes, Ferreira et al. (1994) inoculated maize plants with *Erwinia chrysanthemi* using the syringe method and observed the first symptoms about 288 hours after inoculation. This fact shows that the incubation period depends on the susceptible genotype. Isolate UFU H113 produced a larger AUDPC in genotype L2P33, but lower values than isolate UFU J23 for genotypes L2P45 and L5P18. This did not occur for disease severity (Table 1), indicating that isolate aggressiveness varies according to the genotype studied, i.e., the ability of isolates to cause different levels of disease depending on the host (Soliman et al., 2018).

The data obtained here corroborate those reported by Kumar et al. (2015), where studies

emphasizing the significant variation in aggressiveness of different *Dickeya zae* isolates in maize. This is because stalk rot caused by different pectolytic bacterial species depends on a number of factors, such as inoculum concentration, environmental temperature, the cultivar used, physiological age of the plant and the virulence of the pathogen (Catara and Bella, 2020).

All the genotypes exhibited disease progress over the 16 days of assessment for isolate UFU J23, except L4P19, L5P18 and L5P42, in which the disease progressed only up to 12 days after inoculation (Figure 1A). This was not observed for isolate UFU H113, since all the genotypes showed an increase in symptoms throughout the assessment period (Figure 1B).



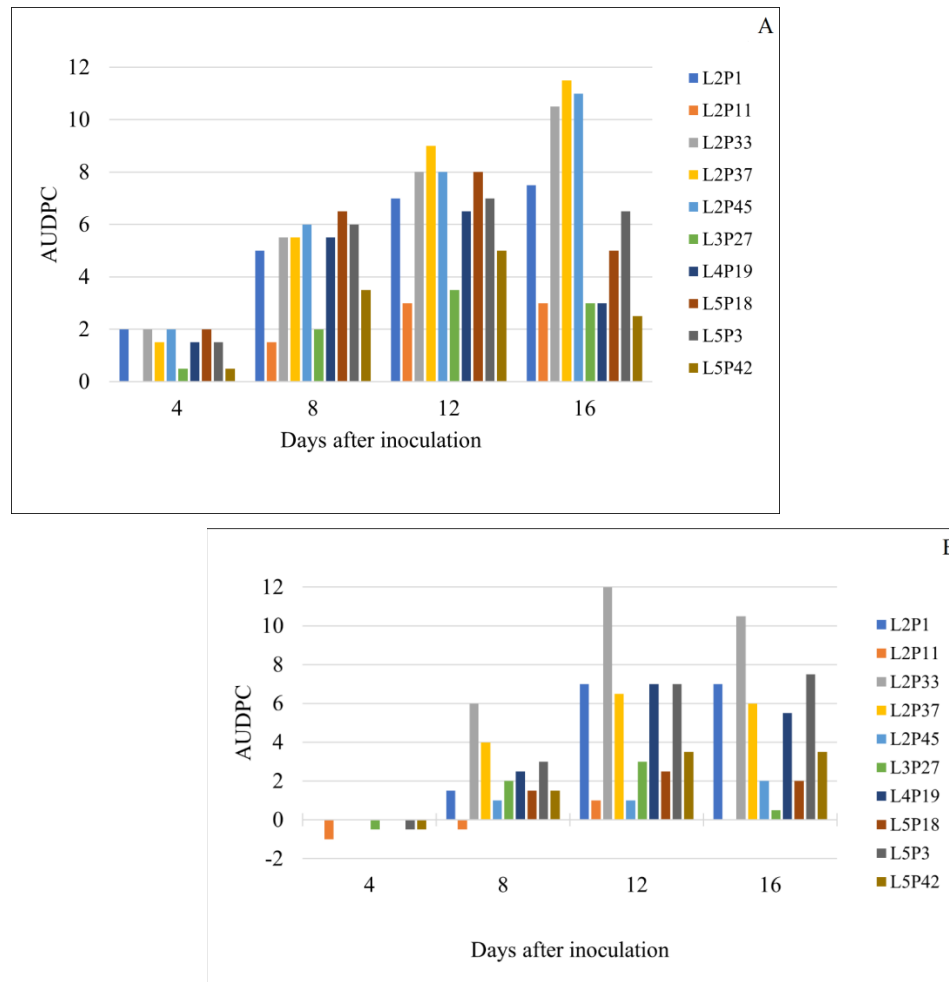
**Figure 1.** Area under the disease progress curve (AUDPC) across the assessment period for eleven sweet corn genotypes and two *Dickeya zae* isolates. A: isolate UFU J23; B: isolate UFU H113

Similarly, Benelli et al. (2004) studied the AUDPC of potato cultivars inoculated with *Pectobacterium chrysanthemi* and found that disease severity was very high under favorable conditions, worsening over time.

Bacterial soft rot is diseases of maize severe under high temperature and humidity conditions. A serious outbreak in Iran led to up to 50 % of plants in the diseased area being severely affected. And a bacterial soft rot outbreak in China caused

by *D. zea* resulted in up to 82 % yield loss in the infected area, with plants dying one week after the appearance of symptoms (Askari et al., 2018; Van Gijsegem et al., 2021).

Figures 2A and 2B present the relative increases in AUDPC for the genotypes studied on each assessment day, based on AUDPC reference values obtained for the susceptible hybrid AS1633.



**Figure 2.** Bidirectional graph comparing all the treatments with the susceptible control (AS1633), based on AUDPC of genotypes minus AUDPC of susceptible control, on the different assessment days.

A: isolate UFU J23; B: isolate UFU H113

In all the treatments, inoculation with UFU J23 resulted in higher AUDPC values than those of the control (Figure 2A), whereas UFU H113 produced equal or lower AUDPC in all the genotypes when compared to the AS1633, four days after

inoculation (Figure 2B). On the remaining assessment days, the AUDPC exceeded that of the hybrid, except genotype L2P11, 8 and 16 days after inoculation.

Concomitantly, genotypes L2P11 and L3P27 and hybrid AS1633 displayed statistically lower AUDPC and SEV values for both isolates (Table 1). However, despite exhibiting less disease severity 16 days after inoculation, neither of these

genotypes were classified as resistant (Table 2). This is in line with Kumar et al. (2017a), who reported that the frequency of genotypes moderately or highly resistant against this pathogen is rare.

**Table 2.** Reaction of ten sweet corn genotypes to the *Dickeya zea* isolates, classified into levels of resistance based on the scoring system proposed by Kumar et al. (2017a) with adaptation

	R	MR	MS	S
Number of genotypes <sup>1</sup>	0	0	6	4
Number of genotypes <sup>2</sup>	0	0	8	2
Disease severity <sup>1</sup>	-	-	2.00*	4.56*
Disease severity <sup>2</sup>	-	-	2.25*	3.75*
Range (disease score)	0	0.1-1	1.1-3	3.1-5

R: Resistant; MR: Moderately resistant; MS: Moderately susceptible; S: Susceptible. 1 Isolate UFU J23; 2 Isolate UFU H113. \* Mean of the SEV values of the genotypes included in the corresponding level of resistance.

Host plant resistance is the most economical and effective strategy to control this disease (Kumar et al., 2017b). Different approaches for characterizing plant responses to Soft Rot Pectobacteriaceae have been used to identify the mechanisms involved in disease development. Such as induction of the jasmonic acid related defense pathway, the strong oxidative burst associated with cell wall protein cross-linking and the necrosis around the maceration zone (Van Gijsegem et al., 2021).

However, complete resistance has yet to be reported, although several authors have attempted to identify qualitative traits that confer multigenic resistance to bacterial stalk rot (Canama and Hautea, 2010).

Ebron et al. (1987) assessed the reaction of 107 maize accessions to bacterial stalk rot and classified only 7% of accessions as resistant. The susceptibility of maize varieties may be the result of changes in stem and leaf protein and total amino acid content (Chagas et al., 2018).

There are also asymptomatic infections induced by *Pectobacterium* and *Dickeya* spp., unrelated to the formation of soft rots and wilts. The wilt is known to be a transitory infection stage, followed by either rot development-susceptibility-related, or by plant recovery-related-resistance or tolerance (Ansermet et al., 2016).

In the framework of latent infections, the bacteria appear to thrive within the host without necessarily requiring the formation of disease

symptoms (Czajkowski et al., 2011). Moderately susceptible genotypes were identified in the present study, including L2P11, which also exhibited low AUDPC and SEV values. Understanding the physiological processes and the genes involved that may determine pathosystem development, may provide the basis for control option for soft rot diseases.

## CONCLUSION

Bacterial stalk rot emerged 96 to 204 hours after inoculation. However, the aggressiveness of the isolates varied according to the studied genotype. Genotype L2P11, classified as moderately susceptible, exhibited the lowest AUDPC and SEV values. It can be used in other researches as a parameter of tolerance to the pathogen. With the study it was possible to standardize the method to characterize the reaction of sweet corn cultivars to *Dickeya zea* under tropical conditions.

## ACKNOWLEDGEMENTS

To the Coordination for the Improvement of Higher Education Personnel (CAPES) for the grant awarded.

## LITERATURE CITED

1. Ansermet, M., S. Schaerer, I. Kellenberger, M. Tallant and B. Dupuis. 2016. Influence of

- seed-borne and soil-carried inocula of *Dickeya* spp. on potato plant transpiration and symptom expression. *European Journal of Plant Pathology* 145: 459-467.
2. Askari, S., R. Rezaei, M. Sadravi and S.M. Taghavi. 2018. Identification of *Dickeya dadantii* Samson, et al 2005 sp. Nov. (*Erwinia chrysanthemi* 3937) as a causal agent of bacterial stalk rot of maize in southwest of Iran. *Tropical Agriculture* 95: 146-153.
  3. Benelli, A.I.H., N.D. Denardin, C.A. Forcelini and V. Duarte. 2004. Reação de cultivares de batata à podridão mole causada por *Pectobacterium carotovorum* subsp. *atrosepticum*, por *P. carotovorum* subsp. *carotovorum* e por *P. chrysanthemi*. *Fitopatologia Brasileira* 29(2): 155-159.
  4. Canama, A.O. and D.M. Hautea. 2010. Molecular mapping of resistance to bacterial stalk rot (*Pectobacterium chrysanthemi* pv. *zeae* Burk., McFad. and Dim.) in tropical white maize (*Zea mays* L.). *Philippine Agricultural Scientist* 93(4): 429-438.
  5. Chagas, J.F.R., G.R. Santos, R.V. Costa, J.F. Alves and I.R. Nascimento. 2018. Adução nitrogenada na severidade de doenças foliares, produtividade e respostas bioquímicas em híbridos de milho. *Revista Brasileira de Milho e Sorgo* 17(1): 1-14.
  6. Catara, V. and P. Bella. 2020. Bacterial Diseases. In: Gullino, M., Albajes, R and Nicot, P. *Integrated Pest and Disease Management in Greenhouse Crops. Plant Pathology in the 21st Century*. Springer, Cham, pp. 33-54.
  7. Czajkowski, R., M.C.M. Pérombelon, J.A. Van Veen and J.M. Van der Wolf. 2011. Control of blackleg and tuber soft rot of potato caused by *Pectobacterium* and *Dickeya* species: a review. *Plant Pathology* 60: 999-1013.
  8. Ebron, L.A., M.S. Tolentino and M.M. Lantim. 1987. Screening for bacterial stalk rot resistance in corn. *Philippine Journal of Crop Science* 12(1): 31-32.
  9. Ferreira-Pinto, M.M., M.L. Cruz and H. Oliveira. 1994. The occurrence in Portugal of the bacterial disease of maize crops caused by *Erwinia chrysanthemi*. *Plant Disease* 43(6): 1050-1054.
  10. Kado, C.I and M.G. Heskett. 1970. Selective media for isolation of *Agrobacterium*, *Corynebacterium*, *Erwinia*, *Pseudomonas* and *Xanthomonas*. *Phytopathology* 60: 969-976.
  11. Kumar, A., M.S. Hunjan, H. Kaur, H.K. Dhillon and P.P. Singh. 2017a. Biochemical responses associated with resistance to bacterial stalk rot caused by *Dickeya zeae* in maize. *Journal of Phytopathology* 165(11-12): 822-832.
  12. Kumar, A., M.S. Hunjan, H. Kaur, R. Rawal, A. Kumar and P.P. Singh. 2017b. A review on bacterial stalk rot disease of maize caused by *Dickeya zeae*. *Journal of Applied and Natural Science* 9(2): 1214-1225.
  13. Kumar, A., M.S. Hunjan, H. Kaur, R. Kaur and P.P. Singh. 2016. Evaluation of management of bacterial stalk rot of maize (*Dickeya zeae*) using some chemicals and bio-agents. *Journal of Applied and Natural Science* 8(3): 1146-1151.
  14. Kumar, A., M.S. Hunjan, H. Kaur and P.P. Singh. 2015. Characterization of *Dickeya zeae* isolates causing stalk rot of maize based on biochemical assays and antibiotic sensitivity. *Indian Phytopathology* 68(4): 375-379.
  15. Lal, S. 1981. Bacterial stalk rot of maize. Techniques of scoring for resistance to important diseases of maize. All India coordinated maize improvement project. IARI, New Dehli.
  16. Melo, MRF., E.B. Souza, K.M.S. Pinto, M.A.S. Gama and R.L.R. Mariano. 2017. Redução da podridão mole em couve-chinesa mediada por indutores de resistência. *Revista CIENTEC* 9(2): 15-24.
  17. Pereira Filho, I. A and F.F. Teixeira. 2016. O cultivo do milho-doce. 1ª edição. Embrapa. Brasília, DF. 298 p. <http://livimagens.sct.embrapa.br/amostras/00084810.pdf>
  18. Rangarajan, M. and B.P. Chakravarti. 1967. A strain of *Pseudomonas lapsa* isolated from corn seed causing stalk rot in India. *Plant Disease Reporter* 51(9): 764-766.

19. Robbs, C.F. 1981. Taxonomia, bio-ecologia e principais representantes do gênero *Erwinia* no Brasil. *Fitopatologia Brasileira* 6: 304-305.
20. Samson, R., J.B. Legendre, R. Christen, M.F. Saux, W. Achouak and L. Gardan. 2005. Transfer of *Pectobacterium chrysanthemi* (Burkholder et al. 1953) Brenner et al. 1973 and *Brenneria paradisiaca* to the genus *Dickeya* gen. nov. as *Dickeya chrysanthemi* comb. nov. and *Dickeya paradisiaca* comb. nov. and delineation of four novel species, *Dickeya dadantii* sp. nov., *Dickeya dianthicola* sp. nov., *Dickeya dieffenbachiae* sp. nov. and *Dickeya zae* sp. nov. *International Journal of Systematic and Evolutionary Microbiology* 55(4): 1415-1427.
21. Silva, M.S., F.C.Q. Carvalho, J.R. Silva, S.R.O. Lins and S.M.A. Oliveira. 2014. Uso de antagonistas e produtos alternativos no manejo pós-colheita de podridão mole em pimentão. *Revista Ciência Agronômica* 45(4): 718-725.
22. Soleimani-Delfan, A., Z. Etemadifar, G. Emtiazi and M. Bouzari. 2015. Isolation of *Dickeya dadantii* strains from potato disease and biocontrol by their bacteriophages. *Brazilian Journal of Microbiology* 46(3): 791-797.
23. Soliman, A., R.H. Gulden, J.T. Tambong, P. Bajracharya, L.R. Adam, R. Xu et al. 2018. Developed and validated inoculation and disease assessment methods for Goss's bacterial wilt and leaf blight disease of corn. *Crop Protection* 112: 159-167.
24. Van Gijsegem, F., N. Hugouvieux Cotte-Pattat, Y. Kraepiel, E. Lojkowska, L.N. Moleleki, V. Gorshkov et al. 2021. Molecular interactions of *Pectobacterium* and *Dickeya* with plants. In: F. Van Gijsegem, F., J.M. Van der Wolf and I.K. Toth. 2021. *Plant Diseases Caused by Dickeya and Pectobacterium Species*. Springer, Berlin. pp. 85-147.
25. Winslow, C.E.A., J. Broadhurst, R.E. Buchanan, C. Krumwiede Junior, L.A. Rogers and G.H. Smith. 1917. The families and genera of the bacteria. Preliminary report of the Committee of the Society of American Bacteriologists on characterization and classification of bacterial types. *Journal of Bacteriology* 2: 505-566.