Diseases Caused by Fungi and Fungus-Like Organisms

First Report of Crown Rot of Banana Caused by *Fusarium proliferatum* in Georgia, U.S.A.

S. Waliullah,¹ E. G. Fonsah,² J. Brock,¹ Y. Li,³ and M. E. Ali^{1,†}

¹ Department of Plant Pathology, University of Georgia, Tifton, GA 31793

² Department of Agricultural & Applied Economics, University of Georgia, Tifton, GA 31793

³ Agricultural College, Northeast Agricultural University, Harbin 150030, P.R. China

Plant Dis. 106:1526, 2022; published online as <https://doi.org/10.1094/PDIS-08-21-1809-PDN>. Accepted for publication 22 October 2021.

In November 2020, mature banana fruits (cv. 'Pisang Awak') with disease symptoms were received from the UGA Banana Research Plot, Tifton, GA (31°28'48.0"N 83°31'23.2"W). Based on initial observation, approximately 30% of the fruits (3 out of 11 crowns with 25% disease severity) were infected showing typical postharvest crown rot symptoms. Two weeks later during the postharvest stage, 100% of the fruits (all 11 crowns with 90% disease severity) were decayed. At the early stage of field infection, the symptoms appeared in the crown of green banana fruits. As the infection progressed, the crown tissues became blackened and softened, followed by an internal development of infection affecting the peduncle and the fruit, which triggered early ripening of bananas. Subsequently, necrosis of the pedicels and fruits appeared and caused the fingers to fall off. To identify the pathogen, tissue pieces (~0.25 cm²) from the infected crown and pedicels were surface sterilized in a 10% bleach solution (0.75% NaOCl) for 1 min, followed by 30 s in 70% EtOH. The disinfected tissues were rinsed in sterilized water three times and cultured on potato dextrose agar (PDA) amended with 50 µg/ml of streptomycin at 25°C in the dark for 5 to 10 days. Four isolates were purified using a single-spore isolation method (Leslie and Summerell 2006). On PDA, the isolates produced abundant, fluffy, white aerial mycelium and developed violet-to-dark purple pigment when viewed from the underside. Colonies produced abundant, single-celled, ovoid, hyaline, or

elliptical microconidia and curved septate macroconidia. These morphological features were consistent with the description of *Fusarium proliferatum* (Leslie and Summerell 2006). To further identify the isolates, genomic DNA was extracted from two isolates. The internal transcribed spacer (ITS) region, partial elongation factor (*TEF1-α*) gene, and β-tubulin gene (*TUB2*) were amplified and sequenced using the primers ITS1/ITS4 (Yin et al. 2012), EF-1/EF-2 (O'Donnell et al. 1998), and T1/T22 (O'Donnell and Cigelnik 1997), respectively. Sequences were deposited in NCBI (accession nos. MZ292989 and MZ293071 for ITS; MZ346602 and MZ346603 for *TEF1-α*; and MZ346600 and MZ346601 for *B-tub*). The ITS, *TEF1-α*, and *B-tub* sequences of the isolates showed 100% sequence identity to *F. proliferatum* isolates using BLASTn in GenBank (accession nos. MT560212, LS42312, and LT575130, respectively). For pathogenicity testing, three whole bunched bananas sterilized with 10% bleach solution (0.75% NaOCl) and washed with sterilized water were cut into three bananas per bunch. The cut surface of the banana crown was inoculated with a conidial suspension (1.0 × 10⁷ CFU/ml) of isolate 1. Equal number of bananas were treated with the same amount of sterilized water as a control. All bananas were sealed in a plastic bag and incubated at 25°C in the same incubator. Seven days postinoculation, all inoculated bananas showed initial crown rot symptoms while no symptoms were observed on the control bananas. The reisolated fungus from the symptomatic tissues was confirmed to be genetically identical to *F. proliferatum* by sequencing of amplified ITS, *TEF1-α*, and *B-tub* genes, fulfilling Koch's postulates. No pathogen was isolated from control fruit tissues on PDA media. To the best of our knowledge, this is the first report of *F. proliferatum* causing crown rot on bananas in Georgia, U.S.A.

References:

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The author(s) declare no conflict of interest.

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Keywords: banana, crown rot, fruit rot, *Fusarium proliferatum*

[†]Indicates the corresponding author.
M. E. Ali; emran.ali@uga.edu