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Artificial wounds implication for the development of mango (*Mangifera Indica* L. Anacardiaceae) fruit disease caused by *Colletotrichum gloeosporioïdes* (Penz.) Sacc. (Glomerellaceae)

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ABSTRACT

Anthracnose is the most important post-harvest disease of mango caused by *Colletotrichum gloeosporioïdes* in Côte d'Ivoire. This study was conducted to evaluate the pathogenicity of 5 isolates (CA1, CA2, CB2, CB3 and CK2) of *C. gloeosporioïdes*. The isolates were obtained from naturally infected fruits of varieties including Brooks (CB2 and CB3), Kent (CK2) and Amelia (CA1 and CA2). The mycelium plugs of each purified isolate were used for a wound or no wound inoculation of Brooks and Keitt mango fruits. The dates of the first lesions appearance with their sizes were assessed. The inoculation without wounds didn't produce any lesion on both varieties. On the other hand, with the wounded method, all the isolates caused lesions on the varieties studied. The first lesions were induced on the 6th day after inoculation (DAI) on both varieties by isolate CA2. It also produced the largest lesion size on Keitt (3.19 ± 0.39 cm) and Brooks (2.61 ± 0.34 cm). On the opposite, isolate CA1 induced lesions lately with an average at 10.75 and 8.50 DAI, as well as a lower average size of 0.12 ± 0.07 cm and 0.62 ± 0.21 cm, respectively on Brooks and Keitt varieties. Isolates CA2 was the most virulent on the two varieties.

Keywords: Mango, anthracnose, inoculation, pathogenicity, Côte d'Ivoire.

INTRODUCTION

Mango (*Mangifera indica* L.) is a tropical fruit having economical importance in the world market. The world mango production was estimated at over 31 millions tons in 2008 (FAO, 2009). It is cultivated in many tropical and subtropical countries. In Côte d'Ivoire, the annual production is approximately estimated at 100,000 tons. It is the third export fruit after banana and pineapple. Côte d'Ivoire is the first exporting country of mango in Africa, and is the third world provider of mango to European countries after Brasilia (65,000 tons) and Peru (29,000 tons) (Gerbaud, 2007). It exports Kent, Keitt, Zill and Amelia mango varieties. But mango fruit is affected by many diseases including anthracnose caused by *Colletotrichum gloeosporioïdes* (Penz.) Sacc (Glomerellaceae). This disease is considered as

the most devastating and the major constraint in the production and export of mango fruits in all mango production areas (Arauz, 2000; Chrys, 2006). It is the second major problem for mango's production and exportation in Côte d'Ivoire, after fruit flies. Anthracnose deteriorates fruits quality and also causes severe post harvest losses (Arauz, 2000). The fruit can be infected in the field through lenticels or wounds caused during harvest, transportation or storage process. Most green fruits infections remain latent and invisible. The symptoms are most conspicuous and important on ripening fruits (Ploetz, 1999). They are rounded brown to black lesion with an indefinite border on the surface. Lesions of different sizes can coalesce and cover extensive area of the fruit surface. In severe cases, the fungus can invade the pulp and reach the stone (Arauz, 2000; Bally, 2006). These necroses affect the commercial value of the fruits and induce losses of income for the producers and all the actors of mango sector. Some works concerning fruit flies and incidence of the mango mealybug including control strategies against them were done in Côte d'Ivoire (Hala et al., 2004). However very few researches concerning post harvest anthracnose pathogen of mango fruit have been done. To contribute to the knowledge of this pathogen, this study was conducted. It aims at evaluating the pathogenicity of five isolates of *Colletotrichum gloeosporioïdes* (Penz.) Sacc. from mangoes infected fruit.

MATERIALS AND METHODS

Pathogen isolation

The fungal isolates used in this study were taken from mango fruits of Brooks, Kent and Amelia varieties with anthracnose lesions. The anthracnose infected fruits were obtained from a commercial warehouse in Abidjan (South of Côte d'Ivoire). The fruits were transferred to the laboratory where they were washed with soapy water, rinsed three times with tap water and surface was sterilized by spraying with ethanol 70% followed by a washing with sterilized water (Hand et al., 2004). Fragments of decayed fruit were removed with a sterile scalpel from margins of decayed and healthy tissues and placed on PDA (potato dextrose agar) medium amended with chloramphenicol 100 ppm to prevent bacterial development. Plates were incubated at room temperature (28 ± 2 ºC) with a 12 h photoperiod. Petri dishes were observed daily for 8 days, and developing colonies were purified. Purification process has consisted of subculture with mycelium discs of the colonies on new PDA medium to obtain pure colonies. The pathogens obtained were identified by microscopic examination based on identification keys (Barnett and Hunter, 1972) and according to Common Laboratory seed health testing methods for detecting fungi (Mathur and Kongstal, 2003).

Test of pathogenicity

Isolates used in this study were *Colletotrichum gloeosporioïdes* (Penz) Sacc. from Brooks (CB2 and CB3), Amelia (CA1 and CA2) and Kent (CK2). Mango fruits of Brooks and Keitt varieties that had been harvested from a commercial orchard in Korhogo (North of Côte d'Ivoire) without post harvest fungicide treatment were used for the experiment.

They were apparently disease free, uniform in size and mature green (370-450 g). The fruit surface was disinfected for 5 min in 1% sodium hypochlorite solution, rinsed three times with sterile distilled water and then, air dried in the culture room. Each fruit was divided into two regions by equatorial line and inoculated on five points (2 per zone and one on equatorial line) according to modified method (Prusky et al., 2001). The fruits were inoculated using either the wound or non wound method in order to determine the attack mode of each isolate.

- The wound method involved piercing the fruit surface on five points with a sterile 0.66-mm-diameter needle head to a depth of 5 mm according to modified method (Moalemiyan et al., 2007). Fruits were injured in order to simulate the conditions of natural infection due to bite of insects. Inoculations were performed by removing a 5-mm-diameter mycelium plug from the edges of 8 days old colony growing on PDA and placed in each wound of the fruits. The inoculation site was covered for 48 h with a piece of sterile moist cheesecloth (Zainuri et al., 2003; Xiao and Rogers, 2004) to optimize infection conditions. Four

inoculated fruits were put in each cardboard and placed in the laboratory room at 28 ± 2 ºC with 60% of relative humidity. They were five replicates of four fruits inoculated with each isolate of *C. gloeosporioïdes*. For the control, fruits were wounded as describe above and treated with cheesecloth moistened with sterile distilled water. The wounded sites did not receive mycelium plug. The fruits were incubated at the same condition as the assay.

- The unwound method involved placing 5 mm diameter mycelium plug of each isolate on five circles made on five sites on the fruit surface with marker pen. The inoculated sites were covered as describe above. They were also five replicates of four fruits as describe on the first experiment. Fruits used as control treatment were not inoculated with any isolate. Each circle was covered with cheesecloth moistened with sterile water. All the fruits were incubated under the same conditions. After 48 h, moistened cheesecloth was removed; fruits were incubated under the same conditions and were observed daily for two weeks. The experience was carrying out twice.

Data collection

After inoculation, data was collected everyday. Data concerning the day lesion was first (1^{st}) observed and the number of inoculated site that developed lesion.

Incubation period (IP) was calculated using the formula below proposed by Shuman (2001).

IP = day lesion 1^{st} observed – day inoculated

Lesion rate (LR) was also calculated as:

$$LR = \frac{\text{Number of sites that produced lesions}}{\text{Number of total inoculated points}} \times 100$$

Lesion length (along the long axis of the fruit) and width (along the short axis of the fruit) were measured. It was assumed that lesions grew in a circular manner so their diameter (LD) was evaluated as follows:

$$LD\ (mm) = \frac{\text{Lesion length + lesion width}}{2}$$

Statistical analysis

Data collected were analysed using analysis of variance (ANOVA) with statistica version 6.0. Differences between means were tested using Newman Keuls multiple comparison procedure at the 5% level.

RESULTS

Both Brooks and Keitt mango varieties inoculated with the 5 *Colletotrichum gloeosporioïdes* isolates after wounded showed symptoms of anthracnose and lesions. On the opposite no isolate produced symptoms on the fruits inoculated without wound.

Incubation period (IP)

Incubation periods were not different (p= 0.183) among all isolates inoculated to Keitt variety. These periods varied from 6.25 ± 0.25 days after inoculation (DAI) to 8.5 ± 0.87 DAI (Table 1). On Brooks, they were different groups of IP. The shortest IP (6.00 to 7.25 ± 0.25 DAI) was obtained with 4 isolates (CA2, CB2, CB3 and CK2) and the longest (10.75 ± 0.25 DAI) with the isolate CA1 (Table1). The IP among the first group was statistically identical. Moreover the first symptoms were induced at 6.25 ± 0.25 DAI at the latest by the isolate CA2 on the 2 varieties. On the contrary, later isolate CA1 produced symptoms at 10.75 ± 0.25 DAI on Brooks and 8.50 ± 0.87 DAI on Keitt varieties (Table 1). The symptoms were brown, dark spot with more or less circular margins.

Lesion rate (LR)

Lesion rates obtained on wounded fruits varied according to DAI (Fig. 1 and 2) among isolates for each mango variety. So at 8 and 9 DAI, isolate CA1 produced LR statistically different ($P< 0.05$) from that induced by the four other isolates on Brooks variety (Figure 1). Lesion rates induced by these four isolates were statistically identical during all experience time (Figure 1). At day 9^{th}, the lowest lesion rate (5 ± 5%) was notified with isolate CA1. On the opposite, the greatest L R (95 ± 5%) was obtained with isolate CB2. On the Keitt variety, at 8^{th} and 9^{th} DAI, the greatest L R (80 ± 20% and 90 ±10%) was induced by isolate CB3 (Figure 2). On the contrary to the isolate CB3, isolate CA1

produced the smallest L R (15 ± 9.57% and 30 ± 12.91%).

Finally, isolate CA1 which produced global LR (13.75 ± 5.07%) and (1.25 ± 1.25%) respectively on Keitt and Brooks varieties, was less aggressive on the both mango varieties (Table 2). Isolates CK2 and CB2 appeared more aggressive on Brooks than on Keitt, and CB3 was more aggressive on Keitt (Table 2).

Lesion diameter (LD)

All the 5 isolates had produced lesions of different sizes according to DAI on fruit after wounding and inoculated (Figures 3 and 4). On Brooks variety (Figure 3), isolates CA2 and CB3 induced identical lesion diameter at 7, 8, 9 and 10th DAI. At 7th and 8th DAI, isolate CA2 had induced the higher lesion diameters (2.13 ± 0.47 cm and 3.51 ± 0.58 cm). These mean lesion diameters were statistically different from those induced by CA1, CK2 and CB2. At 10th DAI, isolate CA1 provoked the lowest lesion size (0.57 ± 0.28 cm) which was statistically different from those produced by isolate CA2 (3.58 ± 0.56 cm), CB2 (3.55 ± 0.45 cm) CB3 (2.60 ± 1.17 cm) and CK2 (2.59 ± 1.17 cm; Figure 3). With Keitt variety at the 6th DAI the diameter of lesions produced by all isolates were not statistically different ($p>0.05$; Figure 4). In total, isolate CA2 appeared the most virulent on both varieties. It induced the largest lesion diameter (3.19 ± 0.39 cm) on Keitt and 2.61 ± 0.34 cm on Brooks (Table 2). On the opposite, isolate CA1 was less virulent on both mango varieties. Isolates CB3 and CK2 were more active on Keitt than Brook's variety (Table 2).

DISCUSSION

Disease symptoms developed on mango fruits after artificial wounds inoculations with isolates of *Colletotrichum gloeosporioïdes* proved that these isolates were pathogenic. There are causal agents of anthracnose infection on mango fruits. In this study, all the five isolates varied in pathogenicity and virulence in the two mango varieties. Similar results had been found by some authors. Mirko et al. (2007) showed the ability of one *C. gloeosporioïdes* reference isolate, *C. acutatun* and 2 others isolates to induce lesions on inoculated strawberry fruits. They found that the two isolates were the most pathogenic. Isolate CA2 had induced early lesions on both mango varieties, as for isolate CA1, it induced lesion later. This result showed that all the isolates did not have the same pathogenic capacity. Isolate CA2 appeared the most pathogenic. This result is in accordance with those reported by Martinez et al. (2009) on identification of *Colletotrichum* spp from mango fruits and anthracnose disease on

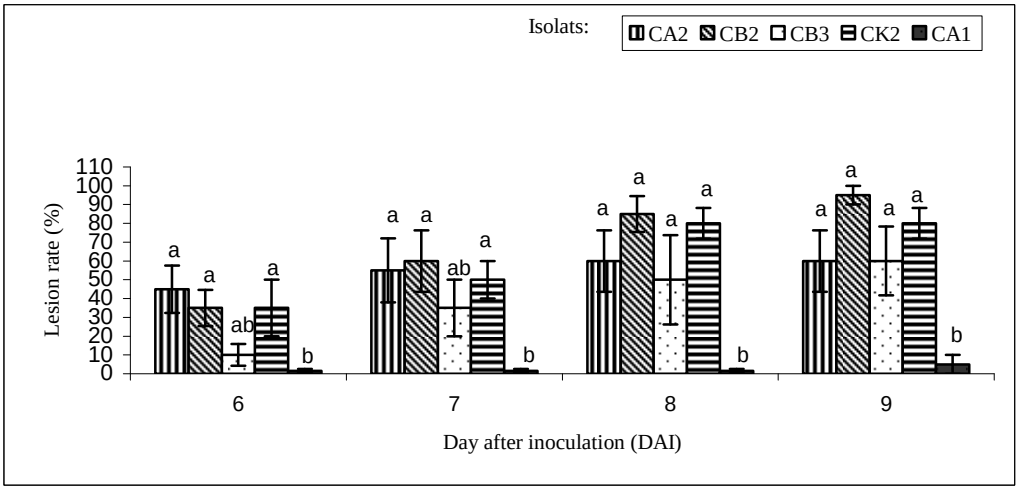

Figure 1: Lesion rate (%) on Brooks mango variety inoculated with 5 isolates of *Colletotrichum gloeosporioïdes* (Penz.) with wound methods.

Significant differences ($p < 0.05$) among averages are indicated by letters above histogram bar. Where the letters are the same, there is no significant difference among different isolate lesion rates.

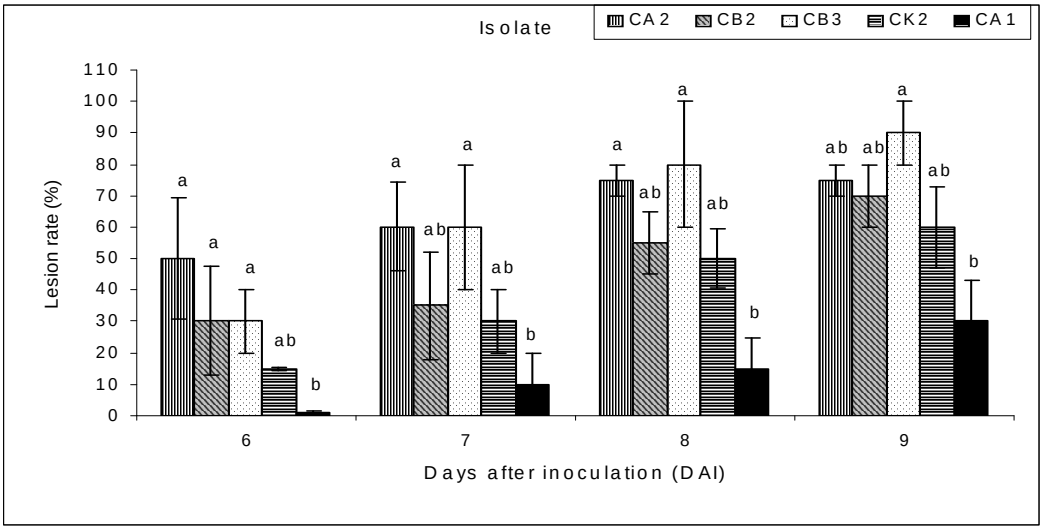

Figure 2: Lesion rate (%) on Keitt mango variety inoculated with 5 isolates of *Colletotrichum gloeosporioïdes* (Penz.) with wound methods.
Significant differences (p < 0.05) among averages are indicated by letters above histogram bar. Where the letters are the same, there is no significant difference among different isolate lesion rates.

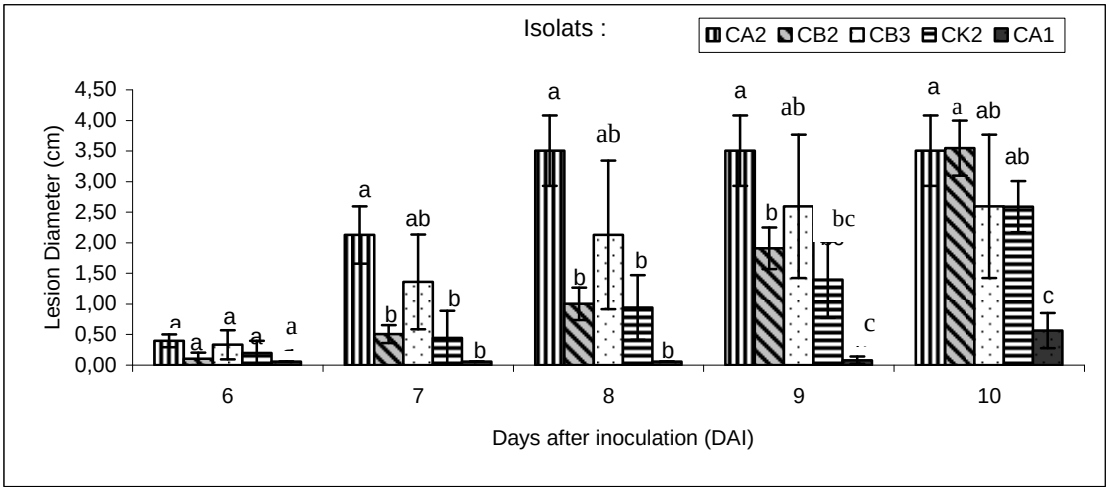

Figure 3: Lesion diameter (cm) on Brooks mango variety inoculated with 5 isolates of *Colletotrichum gloeosporioïdes* (Penz.) with wound methods.
Significant differences (p < 0.05) among averages are indicated by letters above histogram bars. Where the letters are the same, there is no significant difference among different isolate lesion diameters.

tomato. They found that only 19 among 30 isolates inoculated on mango produced the first symptom for 4 days after inoculation. Freeman and Shabi (1996) also proved the pathogenicity of 6 *C. gloeosporioïdes* and 3 *C. acutatun* isolates inoculated on various fruits. They also found lower difference on pathogenicity among isolates from pear. Otherwise, Than et al. (2008) reported that *C. gloeosporioïdes* isolates from mango were not able to produce lesion on pepper fruits 15 days after inoculation either by wound or unwound methods. This difference with our results could be explained by the fact that, the isolates from mango could be susceptible to some compounds in pepper fruits which is not their

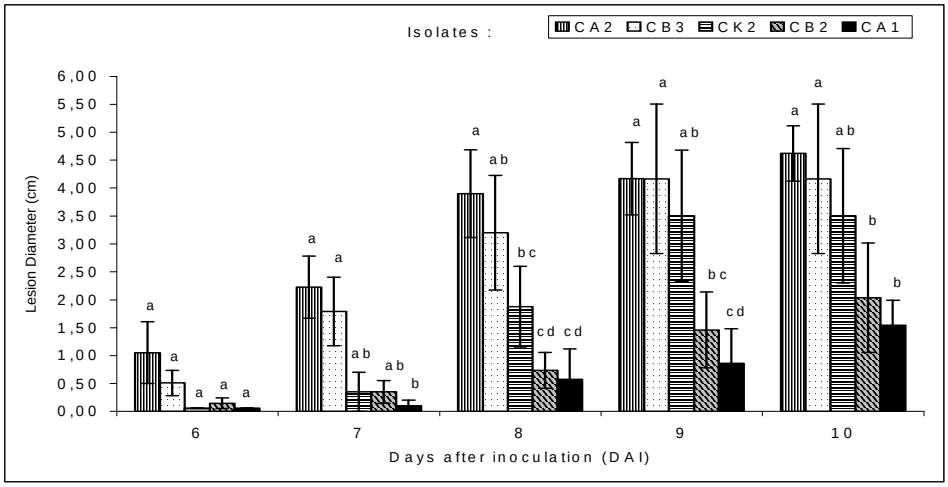

Figure 4: Lesion diameter (cm) on Keitt mango variety inoculated with 5 isolates of *Colletotrichum gloeosporioïdes* (Penz.) with wound methods.

Significant differences ($p < 0.05$) among averages are indicated by letters above histogram bars. Where the letters are the same, there is no significant difference among different isolate lesion diameters.

Table 1: Incubation period (IP) on Brooks and Keitt mango varieties inoculated with 5 isolates of *Colletotrichum gloeosporioïdes (*Penz.) with wound method.

Isolates	**Incubation Period (DAI) on mango fruits**	
	Brooks	**Keitt**
CA2	6.00 ± 00^{a}	6.25 ± 0.25^{a}
CA1	10.75 ± 0.25^{b}	8.5 ± 0.87^{a}
CB3	7.25 ± 0.25^{a}	7.5 ± 0.65^{a}
CB2	6.75 ± 0.25^{a}	6.75 ± 0.25^{a}
CK2	6.75 ± 0.48^{a}	7.5 ± 0.87^{a}

(DAI: Day after Inoculation; Isolates (CB2, CB3), (CK2) and (CA1, CA2) were respectively obtained from naturally infected fruits of Brooks, Kent and Amelia varieties). *Averages followed by the same letter within each column did not differ significantly at $p < 0.05$ according Newman Keuls Test.*

Table 2: Lesion rate (%) and Average lesion diameter (cm) on two mango varieties inoculated with 5 isolates of *Colletotrichum gloeosporioïdes* after wounded.

Isolates	**Lesion rate (%) on the mango fruits**		**Average lesion diameter (cm) on the mango fruits**	
	Brooks	**Keitt**	**Brooks**	**Keitt**
CA2	55.00 ± 19^{ab}	65.00 ± 6.19^{a}	2.61 ± 0.34^{a}	3.19 ± 0.39^{a}
CA1	01.25 ± 1.25^{c}	13.75 ± 5.07^{c}	0.12 ± 0.07^{c}	0.62 ± 0.21^{b}
CB3	38.75 ± 9.03^{b}	65.00 ± 9.22^{a}	1.80 ± 0.44^{b}	2.77 ± 0.52^{a}
CB2	68.00 ± 7.39^{a}	47.50 ± 7.5^{ab}	1.41 ± 0.30^{b}	0.95 ± 0.28^{b}
CK2	61.25 ± 6.94^{a}	38.75 ± 8.65^{b}	1.11 ± 0.27^{b}	1.63 ± 0.46^{b}

Averages followed by the same letter within each column did not differ significantly at $p < 0.05$ according Newman Keuls Test.

natural host. Lesion diameter grew highly on Brooks and Keitt except that induced by CA1. Gina (1999) showed the same result studying *C. gloeosporioïdes* isolates from mango and avocado fruit inoculated with wound method. She found that lesion diameter induced by most isolates on mango fruits varied more than 3 times. Brooks Mango variety was very susceptible to isolate CA2 and CB3. These isolates could secrete abundant quantity of pectate lyase, an enzyme which was able to induce host pectocellulosic wall maceration (Yakoby et al., 2000). This destruction should lead to anthracnose symptom appearance according to some authors (Wattad et al., 1997; Kramer-haimovich et al., 2006). Isolate CA1 was less virulent on both varieties used. It would synthesize less amount of cell wall degrading enzyme. That would make it less virulent. In addition, its action was revealed later when fruits started the process of ripening, which indicated the degradation of some compounds of fruit. Lesion diameters produced by the five isolates increased with wire of time on both mango varieties. This progression could be ascribed to the ripening of the fruits which would also provoke progressive reduction of compounds involved in fruit's defence mechanism. Indeed, the unripe green mature fruits had antifungal compounds level higher than that of ripened fruits (Jinyoung et al., 2002). These compounds could be polyphenols such as tannins (Morrissey and Osbourn, 1999; Macheix et al., 2005). Jinyoung et al. (2002) showed on banana that anthracnose symptom progressed faster on ripe fruit than green fruit. Otherwise, none of the 5 isolates could induce lesion on the fruits of both mangoes variety without wounds. The fruits pericarp would constitute a natural barrier of protection. That justifies the provisions taken by Freeman and Shabi (1996) during their work of inoculation of various fruits with 6 isolates of *C. gloeosporioïdes* and 3 isolates of *C. acutatun* isolates. They made wounds on mango fruits before inoculating them, whereas the other fruits were inoculated without wound. The 5 isolates used would be unable to degrade the pericarp by themselves to reach pulp nutriments source. Then it could be possible that anthracnose symptoms are the results of wounds caused during the harvest and by insects. Thus, wounds promoted and enhanced the pathogens activities on fruits. It is possible to prevent or reduce the severity of the anthracnose on the fruits by avoiding injuring them.

Conclusion

Following up the different types of inoculation, the isolates (CA1, CA2, CB2, CB3 and CK2) of *Colletotrichum gloeosporioïdes* are able to deteriorate the quality of mango fruits after harvest. Isolate CA2 is more virulent than the other isolates. Isolate CA1 is the least virulent. Thus, the 5 isolates do not have identical pathogenicity. Also, the pathological activity of these isolates is favoured by the wounds on the fruits before the infection. No variety was resistant. The results of this study constitute an outline with the characterization of these isolates. Moreover, mango fruits of other varieties should be inoculated for better determination of the isolate specificity. It would be also interesting to study the physiological and molecular aspect of these isolates for a better characterization in order to determine a method of suitable control of *C. gloeosporioïdes.*

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