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Review Article

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Botryotinia ricini (Gray Mold); A Major Disease in Castor Bean (*Ricinus communis* L.) – A Review

Akwasi Yeboah[1], Jiannong Lu[1], Kwadwo Gyapong Agyenim-Boateng[1], Yuzhen Shi[1], Hanna Amoanimaa-Dede[1], Kwame Obeng Dankwa[2] and Xuegui Yin [1,*]

[1]Department of Crop Breeding and Genetics, College of Agricultural Sciences, Guangdong Ocean University, Zhanjiang 524088, China

[2]Council for Scientific and Industrial Research - Crops Research Institute, Kumasi, Ghana

*Corresponding Author E-mail: yinxuegui@126.com

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ABSTRACT

Castor is an economically important oilseed crop with 3-5% increase in demand per annum. The castor oil has over 700 industrial uses, and its oil is sometimes considered as an alternative for biodiesel production in several countries. However, its worldwide demand is hardly met due to hampered production caused by biotic stress. One of the most critical biotic factors affecting castor production is the fungal disease, Botryotinia ricini. The study of the B. ricini disease is very essential as it affects the economic part of plant, the seed, from which castor oil is extracted. Despite the devastating harm caused by B. ricini in castor production, there is limited research and literature. Meanwhile, the disease continues to spread and destroy castor crops. The disease severity is enhanced by an increase in relative humidity, temperature (around 25ºC), and high rainfall. Suggested control of the disease includes timely planting, breaking of infected panicles, wide planting interval, use of resistant varieties, and seed treatment with fungicides. Use of resistant cultivars is most appropriate against the gray mold disease, however, only few cultivars have been identified. Marker-assisted selection, a cost effective and fast method of identifying resistant cultivars and ultimately resistant genes has not been employed to date. This review investigates the current status of the gray mold disease in castor production and also discusses some effective control measures.

Keywords: *Gray mold disease; Castor; Botryotinia ricini; Amphobotrytis; Capsules.*

INTRODUCTION

Castor (*Ricinus communis* L.), is a member of the spurge family (*Euphorbiaceae)*, and a commercially valued nonedible oilseed crop (Singh et al., 2015). The castor plant can be grown as an indeterminate annual or perennial crop based on the soil type and the climatic conditions (Wang et al., 2013, Kallamadi et al., 2018). Currently, castor is widely grown over 30 different countries, with India, China, Brazil, Mozambique, Ethiopia, and Thailand as the most dominant countries contributing to about 90% of the world's castor production (Manjunath & Sannappa, 2014, Lakhani et al., 2015).

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Ethiopia is speculated to be the center of origin owing to the presence of high diversity (Lakhani et al., 2015, Anjani, 2012). Nevertheless, castor is distributed in almost all the tropical and semi-tropical regions of the world (Chaudhari et al., 2019, Kalogiannis et al., 2016). In terms of cultivated area, India accounts for 66.5% (Ramanjaneyulu et al., 2017). India, the leading producer of castor in the world, earns more than Rs. 8,000 crores (equivalent as 115.28 USD) annually by exporting castor oil and its derivatives (Mohsin et al., 2018).

Due to the many uses of castor oil, its worldwide demand rises at an average of 3-5% per annum (Gahukar, 2018). Castor oil, derived from the castor seed, is unique among all other oils because it is the sole source of hydroxy fatty acid; ricinoleic acid (about 85%), conferring a lot of its distinctive industrial properties (Vivodík et al., 2014, Mubofu, 2016). Castor oil is widely used for many industrial products like; ointments, fiber optics, nylon, varnishes, airplane engine lubricants, hydraulic fluids, dyes, detergents, plastics, soaps, greases, paints, synthetic leather, cosmetics and perfumes (Venegas-Calerón et al., 2016, Patel et al., 2016). The leaves of castor are also used in sericulture as feed for eri and muga silkworms (Manjunath, & Sannappa, 2014, Shifa et al., 2016).

Generally, the castor plant is greatly attacked by several pathogens such as fungi, bacteria, virus, mycoplasma, and nematode diseases, yielding to heavy yield losses and poor market value of seeds (Manoharachary, & Kunwar, 2014, Dix, 2012). In India alone, about 80% of yield loss in castor crop is attributed to fungal disease (Prasad, & Bhuvaneswari, 2014). Regardless of the tolerance ability of the castor plant to biotic stresses; gray mold, vascular wilt (bacterial and fusarium wilt) and charcoal rot, are the three major diseases that undermining its productivity (Severino, 2012, Anjani et al., 2004). Among these, gray mold is the most destructive and difficult to deal with. It causes direct damage to castor capsules and inflorescence (Severino, 2012).

Under conditions that enhance disease severity (high rainfall, temperature 25ºC), castor yield loss could be as high as 100% (Anjani, 2018, Lourenço Jr, 2017). The occurrence of castor gray mold disease is polycyclic; thus, infection can reoccur when the pathogen is blown by air to a new site (Prasad, 2016). *Botryotinia ricini* (Godfrey), is the fungal pathogen that causes gray mold in castor bean and its anamorphic phase is *Amphobotrys ricini* (Prasad & Kumaraswamy, 2017). The anamorphic form is known to incite gray mold disease and capable of causing an epidemic (Yamuna et al., 2015). Initial symptoms of the gray mold disease include the appearance of small bluish spots on the capsules and inflorescence followed by a darkening of elliptical sunken spots (Gahukar, 2018, Anjani et al., 2014). A detailed study about gray mold of castor was conducted in the 20th century (Godfrey, 1923). However, not many studies have been done on it in recent times (Soares, & Cumagun, 2012). Hence, minor improvements in its management have been achieved. The gray mold is a major obstacle that hinders castor production. An understanding of the disease cycle, resistant cultivars identified, and control methods employed by scientists will be much beneficial to castor breeders all around the world.

Etiology and taxonomy of the gray mold disease

Godfrey (Godfrey, 1919) pioneered the naming and description of the gray mold disease of castor and, named the causative agent mainly based on the teleomorph (sexual reproductive form) as *Sclerotinia ricini*. However, in 1945 Whetzel transferred the species *S. ricini* to the genus *Botryotinia.* Till now, the fungal pathogen of castor gray mold is called *Botryotinia ricini* (Godfrey) Whetzel, which has been recorded by several authors in their literature (de Oliveira Datovo, & Soares, 2019, Nagaraja & Krishnappa, 2016). In 1949, the anamorph (asexual reproductive form) phase for *Botryotinia ricini* was given the name *Botrytis ricini* (Buchwald, 1949). Later in 1973, Hennebert denoted a new name for the anamorph based on the unique pattern of the conidiophore development and named it *Amphobotrys* (Hennebert, 1973). The anamorphic state for *Botryotinia ricini* has remained *Amphobotrys ricini* to date and has been reported by several researchers (Nascimento, 2014, Chagas, 2014).

Botryotinia ricini of castor belongs to the kingdom-Fungi, division-Ascomycota, class-Leotiomycetes, order-Helotiales, family-Sclerotiniaceae, genus-Botryotinia (Godfrey, 1919). The fungus *Botryotinia ricini* (Godfrey) Whet. is morphologically characterised as grey, conidia dusty, conidiophores erect, seldom curved, septate, 11-23µ thick, wall blackish brown approaching the tip closely, and hyaline with numerous projection at the tip from which conidia are formed (Soares, 2012). *Amphobotrys ricini* is characterized morphologically and molecularly by its erect, cylindrical, light brown hyaline, straight, curved dichotomously, with uninflated and thin-walled conidiogenous cell; conidia globose developing synchronically or sterigmata, on shorter denticle, smooth, unicellular, sub-hyaline to light brown (Yamuna et al., 2015, Soares, 2012, Lima et al., 2008).

History and economic importance of gray mold disease

In 1918, Stevens and Patterson reported the outbreak of gray mold disease from Florida (U.S.A) where castor was cultivated on a large scale in order to meet the demand for castor oil (Godfrey, 1923). This was the first report of gray mold disease and was believed to have been due to castor seeds imported from an Indian town called Bombay, currently known as Mumbai (Soares, 2012). The prevalence of the disease caused massive yield loss to castor farms, especially during the summer.

In 1932, the gray mold disease of castor was mentioned for the first time in the Brazilian state of São Paulo but was overlooked until its epidemic in 1936 (Gonçalves, 1936). Currently, the gray mold epidemic has increased in almost all Brazilian states like Brejo Paraibano with the exception of Bahia state, due to its arid environment (Nascimento, 2014, Soares, 2012).

In 1971, gray mold disease affected over 25% of castor racemes when planted on a large scale in Mississippi state (Kumari, 2011). India, the major producer of castor in the world, is highly burdened with the gray mold disease affecting its production (Dange et al., 2005). In 1987, the epidemic of gray mold disease was reported for the first time in Tamil Nadu and Andhra Pradesh; a major growing state of castor in India (Kallamadi & Sujatha, 2018, Nagaraja et al., 2009). Also, an incidence of the gray mold disease in Telangana states seriously decreased about 3.92 lakh ha castor cultivation from 2000 to 2001 (Prasad, & Kumaraswamy, 2017). In 2013, Campbell reported of the occurrence of gray mold again in Florida, the first known area of castor infestation. This time around, he observed a reduction in the oil production of the affected plants (Campbell, 2013).

The anamorph of *Botryotinia ricini* was found to incite gray mold in various countries where castor are cultivated (Yu et al., 2012). In 2001, the manifestation of the anamorph of *B. ricini* disease was reported on castor leaves for the first time in South Korea (Hong et al., 2001). 25 pathogens known to be associated with the gray mold disease were isolated and evaluated. In the end, only 5 out of the 25 isolated pathogens were identified as *Botrytis cinera* and the remaining 20 as *Amphobotrys ricini* (Hong et al., 2001). Aside castor, *Ricinus communis* (Nascimento, 2014, Moraes, 2011), gray mold caused by its anamorph has been reported in other plants including, *Acalypha herzogiana* (Duarte et al., 2013,Coutinho et al., 2014), *Fragaria ananassa* (Amiri et al., 2016), *Vitis vinifera* (Lorenzini, & Zapparoli, 2016), and in *Solanum lycopersicum* (Zhang, 2013).

Currently, gray mold incidence is reported in other parts of the world where castor is cultivated including Russia, Ukraine, Germany, Thailand, Texas, Jamaica, and Morocco (Patel et al., 2016, Sharma & Rana, 2017, de Oliveira Datovo & Soares, 2018).

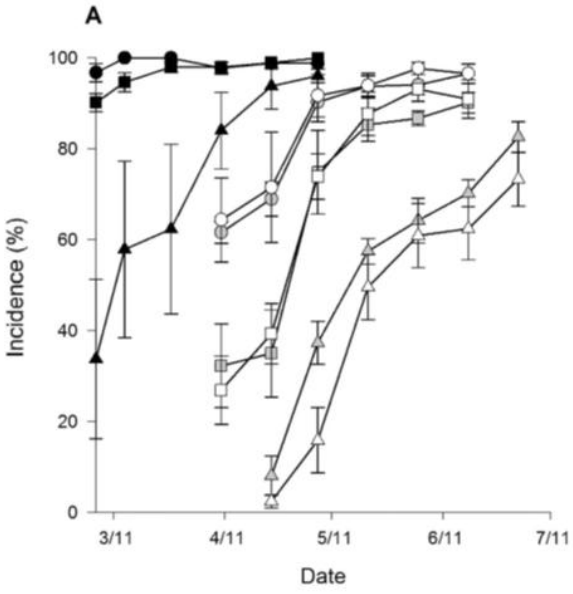
Epidemiology

In any castor production, high relative humidity, high rainfall, and temperature around 25°C are well known to foster the development of the fungus *Botryotinia ricini* (Godfrey) (Lourenço Jr, 2017, Sussel et al., 2011). The maximum and minimum temperature for mycelial growth is 35°C and 12°C, respectively (Yamuna et al., 2015). The disease is partially expressed at temperatures below 20°C, and its occurrence depends on long periods of high relative humidity (Sussel, 2008). This statement confirms preliminary studies conducted by Esuruoso (Esuruoso, 1966), on the susceptibility of some castor cultivars in Nigeria at two different locations, Ibadan and Ilora. Both places experience

relatively same temperature. But, the rainfall in Ibadan was higher than Ilora, which led to higher disease level in Ibadan compared to Ilora (Esuruoso, 1966). The occurrence and degree of disease severity have a corresponding relationship with temperature and time of leaf wetness. At temperature 28°C, a time of 72hours is required before the disease can occur. Also, for a temperature below 15°C, a time of 6 hours of leaf wetness is needed for the disease to occur (Sussel et al., 2011).

Moreover, studies by Sussel (Sussel, 2008) proved that gray mold disease exhibits a randomized distribution pattern, corresponding to its airborne nature. According to the author, castor plants that experienced high rainfall showed an aggregate pattern and correlated positively to the mean number of conidia and the weather parameters such as temperature. The number of conidia increased with an increase in temperature ranging from 14.6°C to 18.1°C. However, there was no difference in the number of conidia with increasing relative humidity. Under relative humidity close to saturation, the incubation and latent period of *B. ricini* vary with time, and this is mainly dependent on the castor genotype (Soares et al., 2012).

Sowing date also affects the occurrence and intensity of gray mold. An experiment conducted in Brazil on the effects sowing dates (January and February) on gray mold intensity in castor bean cultivars IAC 80, IAC 226, IAC 2028, BRS Paraguaçu, and Guarani, revealed gray mold less occurrence and severity in all cultivars sown in February because the flowering stage meets low rainfall compared to those planted in January (Costa et al., 2009). Lourenço et al., (2017) assessed the response of castor cultivars to gray mold incidence at different planting season, a clear conclusion about proper sowing dates were not given (Figure 1 A). However, cultivar IAC Guarani, exhibited the least symptom of gray mold occurrence and severity when planted in December 2010 and October 2011 (Figure 1 B). This result was attributed to the late flowering nature of the cultivar. This cultivar reached the flowering stage around 70 days after emergence and rainfall pattern was infrequent at that time compared to other months. This confirmed the work of other scientists who reported that prolonged rains at the flowering stage contribute immensely to a rise in the gray mold infestation (Dange et al., 2005, Sussel et al., 2011, Costa et al., 2009). However, further studies are required to ascertain if environmental parameters like surface wetness also affect the incidence of gray mold disease.



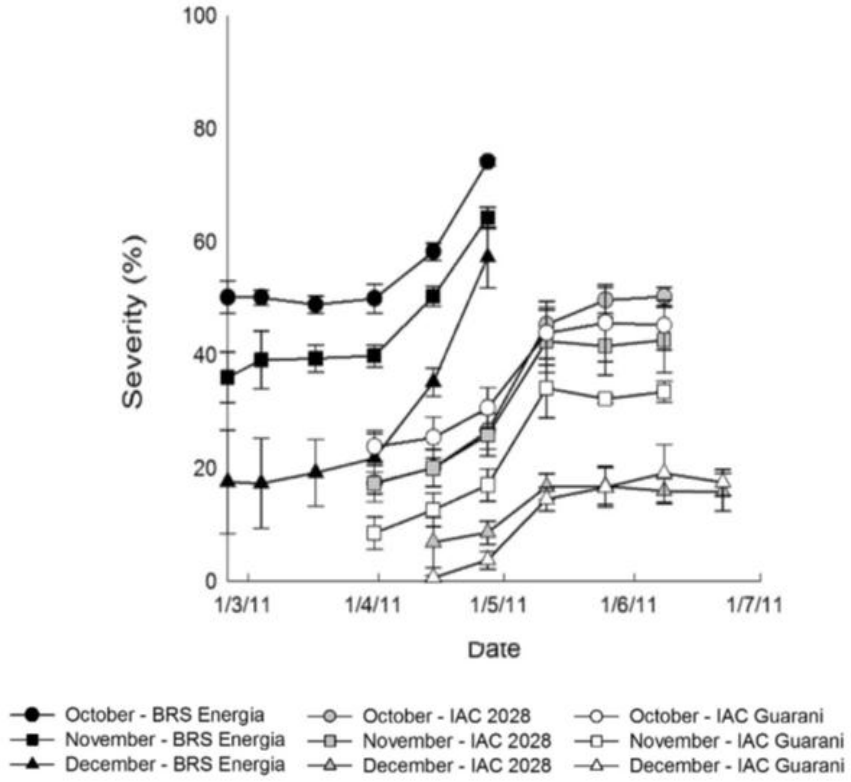


Fig. 1: A and B. Disease incidence and severity curves of castor cultivars sown in different months to examine gray mold occurrence on the 15th day of October, November, and December in the 2010/11 seasons. Each bar represents the standard error of mean (Lourenço Jr, 2017).

Gray mold disease cycle and host range

The pathogen is a seed-borne and survives in soil and crop residues in the form of sclerotia, and produces sexual structures during favorable conditions to initiate disease infection (Dange et al., 2005) Fig 2. *B. ricini* can easily be transmitted from the infected plant to healthy plants through rain splash, insects, and by wind. Under favourable conditions, the spores of the pathogen sporulate profusely on the new host to cause infection. The castor seed is the most important source of *B. ricini* primary inoculum which begins with an infected seeds. Some researchers, however, dispute castor seeds as being the primary sources for infection, and rather acknowledge the conidia as the initial infection agent (Soares, 2012, Gonçalves, 1936, Maude, 1996). Gonçalves (1936), reported that, for humid climates and for wild castor types, the primary inoculum source could be the conidia because the castor plant can grow spontaneously throughout the year. Therefore, it is possible for new susceptible plant types that grow close to infected old plants to enhance the fungus to re-germinate in its anamorphic state for the next crop season. (Costa et al., 2009) also reported thet although *B. ricini* is seed-borne, it is unlikely for infection to begin with the seed. Soares (2012), also argues the castor seeds cannot be the primary source for inoculum due to the long periods (about two months) that exist between the time of planting and the time of flowering, which makes it impossible for the inoculum to originate from the seeds to cause epidemic. Therefore, the author concluded that, the seed has no essential role for the initiation of the disease cycle but only serves as a medium through which the pathogen is disseminated to a new site for infection to occur.

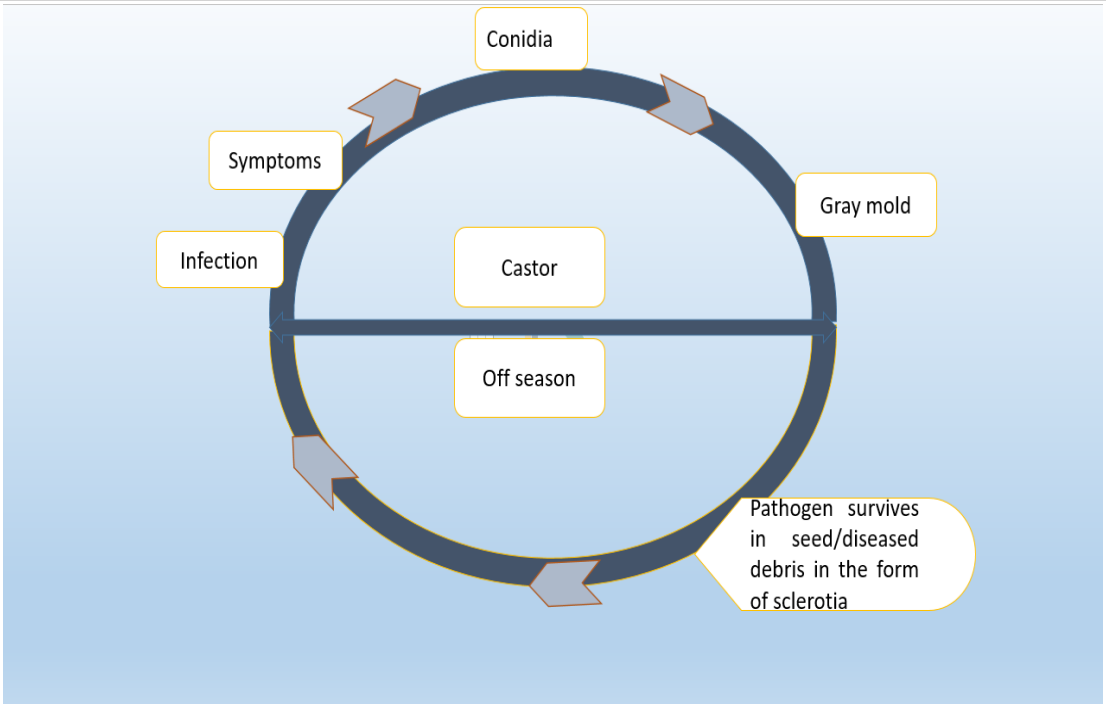


Fig. 2: The infection cycle of gray mold disease The disease cycle begins with infected castor seeds. The pathogen can survive in seed and reproduce to cause infection under right conditions. The off season is the dormant period where no infection occurs. The conidia becomes the secondary source of inoculation for infection to re-occur.

Godfrey (1923), pre-supposed *B. ricini* to be host-specific having a narrow host range. However, employing artificial inoculation method, it was realized that the fungus was unable to cause disease on castor plants so as other members of the *Euphorbiaceae* family, but its occurrence was highly dependent on relative humidity. In a field inspection, no other plant species showed signs of infection even in areas of a high prevalence of *B. ricini*. However, several reports have been made since 1980 regarding *B. ricini* naturally infecting members of the Spurge family such as *Acalypha hispida* and *Jatropha podagrica*, *Euphorbia inarticulata* (Nascimento, 2014, Alwadie & Baka, 2003). Aside from these records of natural infection, artificial inoculation tests have portrayed that host range of *B. ricini* in the Euphorbiaceae family is extensive, affecting economically essential crops like cassava (*Manihot utilissima*) (Lima et al., 2008, Kumar et al., 2007).

Host penetration and colonization

Explicit investigations have not been conducted towards the host penetration and colonization of *Botryotinia ricini* on castor (Soares, & Cumagun, 2012). The mycelium of the fungus first degrades the cuticle and later penetrates the host tissues (Prasad, 2016). The fungus extends very fast immediately after penetrating, causing destruction and breakdown of the tissue. Inoculum type, availability of nutrients and water, cuticle features and exudates on floral organs are some of the factors that enhance the fungal growth and penetration process on the host plant (Holz et al., 2007). Enzymatic action from enzymes such as cutinase and lipase have also been found to partake in the penetration process (Soares & Cumagun, 2012). Some researchers also claim that the fungus penetration takes place directly through cuticles. (Soares, 2012), also suggested that the penetration process of *B. ricini* into the host tissue occurs via both mechanical and chemical means. The fungus infection process should be precisely known as well as a clear distinction of how the penetration process occurs under highly controlled experimental conditions and penetration under natural processes in the field. In some cases, penetration of *B. ricini* fungus on castor plants can take place on all aerial parts such as the stem (Soares, 2012). However, the ascospores and conidia are also infectious, as witnessed in other *Botrytis* spp (Walker, 2016). Natural

opening and wounds on the host surface can also serve as a medium for fungus penetration into the host tissue.

Symptoms of gray mold

Gray mold disease has symptoms that can easily be seen in infected castor fields. In any developmental stage, the inflorescence and capsule are the main targets of the fungus (Costa et al., 2009) Fig 3. The fungus has a preference for female flowers mainly due to their succulence and retention of water droplets for longer periods, which helps in spore germination (Prasad, 2016). Gray mold firstly appears as small bluish spots on developing fruits from which droplets of yellowish fluid percolates (Yamuna, 2015). The symptoms on the fruits can emerge as circular or elliptic, sunken, dark-colored spots that can cause rupture of the capsule and strands of fungal hyphae to emerge out from these spots within a short period (Gahukar, 2018, Anjani et al., 2014). These symptoms are mostly recurrent, especially in low relative humidity after the invasion of the fungus into the host plant. Symptoms on the inflorescence are seen as greyish colored moldy growth which later metamorphoses to olive-gray (Nagaraja & Krishnappa, 2016). The unified racemes alongside the flowers and seeds become infected as the infection spreads upwards, leading to a destruction of other plant parts (Lima et al., 2008).

On the capsules, greyish brown, water-soaked lesions appear, extending from one capsule to the other (Yamuna et al., 2015). Infected immature capsules become rotten whiles the seeds of matured capsules lose weight, become hollow, and the coats turn discoloured (Gahukar, 2018). Fungal colonization of young capsules and both flowers (male and female) become softened, and mycelial growth turns from pale gray to dark olive (Yamuna, 2015). Usually, the symptoms on the leaves are noticeable by small dots where the inoculum accumulated during infection. The fungus then spreads on the leaf generating light brown dead spots followed by gray mold. The lesion is circular to sub-circular and also differs in size of diameter ranging from 5 to 9mm (Nascimento, 2014, Yamuna, 2015). As the lesion assimilate, they expand in the area creating massive necrosis of leaves. Gray mold infection also transpires apically or marginally over the leaf base where dew or rain typically hoard. Under humid conditions, the pale brown lesion is shielded with tender web mycelium. Sometimes, the periodic advancement of the disease is observed by broad blighted areas on the leaves (Sussel et al., 2011). Infection does not easily exist on the stalks and branches because matured tissues are more resistant to infection (Soares, 2012). The disease can occur on delicate peduncles and shoots where water-soaked lesions develop into dull white as the affected tissue breaks off from the stem and spike (Yamuna et al., 2015).

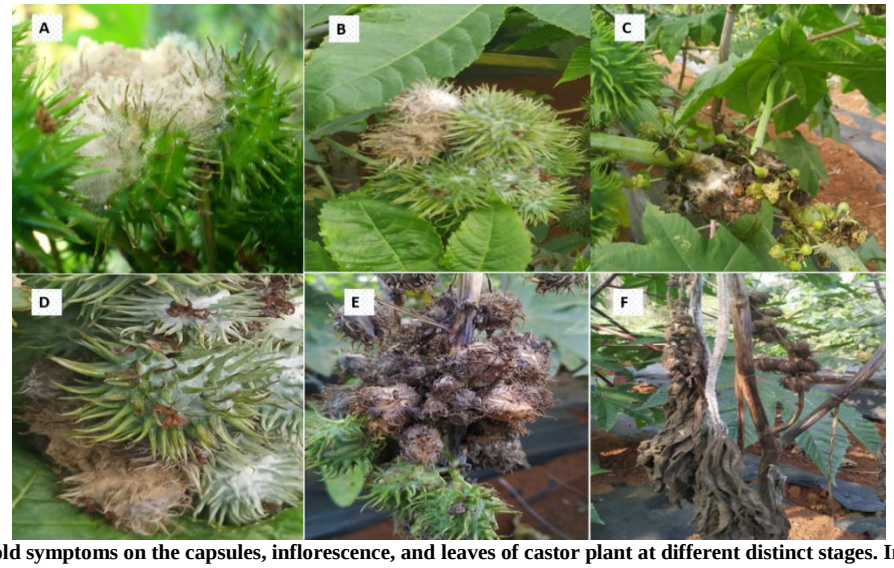


Fig. 3: Gray mold symptoms on the capsules, inflorescence, and leaves of castor plant at different distinct stages. Infected capsules with whitish mycelial growth A-B; Infected inflorescence C; Completely damaged capsules at different stages D-E; Severely affected castor leaves with mold on the petiole F.

Sources of host resistance

In view of the high losses caused by gray mold in castor, there is an urgent need to concentrate on identifying tolerant sources and breed resistant varieties which can withstand high inoculum loads of the pathogen at the seedling, flowering and capsule formation stages (Sudhakar et al., 2010). Though plant breeders and pathologist have tried to develop resistant variety over the past decade, however, only minimal success has been achieved (Ramanjaneyulu et al., 2013). Notwithstanding, some plant breeders attempt to select for resistant genotype only based on field observation, conversely, this method could be misleading since other unperceived environmental factors could be responsible for the observed resistance other than genetic resistance. Godfrey (Godfrey, 1923) suggested that plants with more ornamental type of different shades of reddish-green are more resistant to the fungus. Till date, no research has been carried on to prove it. Generally, the pigmentation of castor plants is caused by the presence of some secondary metabolites like anthocyanin. These metabolites have specific functions, and possibly, the reddish green castor plants possess some disease resistant metabolites giving them the antifungal properties. Gonçalves (Gonçalves, 1936), published that "spontaneous varieties" showed much resistance to the diseases because infection normally affects only small portions of the capsules leaving most of the other capsules healthy. Although this is witnessed in wild castor plant as well as some commercial cultivars. This requires further studies, possibly such happens because of the extensive genetic variation within the members of *Euphorbiaceae*. Wax blooms castor phenotypes have been studied to be much effective against pests like the leafhopper in castor, but not much confirmatory research has been done in the area of gray mold; hence the information is limited (Lopes et al., 2014).

Researchers in Brazil have tried to develop resistant cultivars against gray mold infestation, but only cultivars with partial resistance have been identified (Chagas, 2014, Lopes et al., 2014). Similar results have been observed worldwide with different castor varieties having moderate levels of resistance against gray mold (Prasad & Kumaraswamy, 2017). The castor accession RG2752 has been found to be resistant to *Botrytis ricini* (DOR, 2004). Milani et al., (2005) by screening the castor germplasm under natural infestation identified six (6) resistant genotypes; Baker H 66, Cinnamon Juriti, Cnpa Sm1, Mpai T63/6, Sipeal 28, and 2004 Sipeal. Lima et al., (2008) discovered three (3) resistant cultivars which were Sipeal 04, Sipeal 28 and Canela de Junti by numbering the whole racemes and infected racemes under natural infestation. Anjani and Raoof (2010), conducted a study by screening the castor germplasm to select cultivars which were highly resistant to gray mold disease when cultivated under the epiphytotic conditions of the disease in both glasshouse and field. Some cultivars which were found to be resistant for the first year became susceptible to the disease in the subsequent years. Five major castor accession (RG 2787, RG 2836, RG 2980, RG 3126, RG 3139) showed resistance for three consecutive years. However, after the sixth year of screening, only two cultivars RG 2787 and RG 2836 were identified to be resistant with a percentage of 14% and 8% respectively. Some Brazilian castor genotype (CNPAM-93-168, CNPAM-89-34) have also been identified with moderate resistance under artificial inoculation condition (Nascimento, 2014). Prasad and Kumaraswamy (2017), by screening castor germplasm under artificial epiphytotic conditions in field and glasshouse with fifty accessions for gray mold disease identified only RG-1826, which showed partial resistance to the disease by using detached spike method.

B. ricini is reported to be controlled by a quantitative trait (Keane, 2012). Quantitative trait is controlled by polygene with a few minor genes. An in-depth search into this phenomenon could enhance the development of *B. ricini* resistant genotype with the use of genetic marker-assisted selection. These markers can aid in the identification disease resistant genes/QTLs within the shortest possible time.

Gray mold disease management

Under favorable weather conditions for gray mold infections, it is essential to protect the

young capsules and inflorescence to prevent further infection and huge yield losses. Gray mold fungus is seed-borne and therefore challenging to manage. Castor growers always want to take precautions even before any visible symptoms are seen in their field. To manage the pathogen, pathogen-free seeds should be used and much precaution must be taken to reduce the primary inoculum in the soil. A single method may not efficiently control the castor gray mold. Therefore different management practices need to be integrated judiciously to keep the disease losses below economic threshold. Management practices may include cultural practices that make the environment unfavourable for disease development, chemical control by using effective and active fungicides, and biological control.

Controlling gray mold disease through cultural practices

Cultural practice is one of the practical and widely used methods to control *Botryotinia ricini* disease in castor cultivation. The advantage of this method is to prevent inoculum initiation into the field and to reduce pathogen survival and its spread or making the host less vulnerable to fungal disease. Selection of good seeds, plant debris removal, suitable planting area, and growing season are recommended before planting (Gahukar, 2018). The practice of plant spacing and removal of weeds is also advisable for maximum aeration (Soares, 2012, Soratto et al., 2012). The use of viable seeds is always an appropriate practice because the fungus can hide under the seed coat and serve as a primary source for inoculation during the next planting season (Moraes, 2011). Removal of any alternative host (*Euphorbiaceous* hosts) and damaging of inoculum that persists in crop residues are also appropriate to reduce the disease incidence. All the same, this practice should be backed by regular field inspection because the wind easily carries the seeds, and once the diseases are established in a field, this method becomes unfeasible. During dry seasons, the disease seldom occurs. Therefore, the sowing time and the season should be factored since wet seasons enhances disease development (Prasad, 2016).

Chemical control of gray mold

Over the years, seed treatment has been a common practice adopted against gray mold disease (Moraes, 2011). Spraying with fungicides reduces disease progress. However, the application of fungicides either by spraying or dusting is ineffective once the disease has been well established in the field. Fungicides with high specificity and distinction in their mode of action are effective in controlling many plant diseases (Chagas, 2009). Nevertheless, for *B. ricini*, the problem is not about the ineffectiveness of fungicides but the right timing as well as the optimum dose for application (Bhat & Rajasri, 2015). The use of two prophylactic sprays with carbendazim (0.05% and 0.1%) at the flowering stage and immediately after the appearance of the first symptoms decreases the disease spread (Sudhakar et al., 2010). Preliminary studies have proved that under favorable conditions, both azoxystrobin and carbendazim are active against *B. ricini* pathogen (Sudhakar et al., 2010, Bezerra, 2007). Application of tebuconazole, iprodione, and procymidone are known to control fungal pathogen. Among these, procymidone and iprodione fungicides are highly effective against gray mold, only if they are applied at the start of disease development and on a weekly bases (Soares, 2012). In an experimental study by Bhat and Rajasri (Bhat & Rajasri, 2015) in India, propiconazole at 1 mL/L and Carbendazim at 1 g/L fungicides were applied to assess their effectiveness against gray mold of castor. Spraying was done twice, with the first spraying before disease incidence and the second spraying immediately after 10 days of the first spray. It was observed that out of the two fungicide applications with nine treatments, propiconazole, which was applied at 1 mL/L recorded the lowest incidence (9.6%) of gray mold. Studies have shown that both *Botrytis* spp. and *Botrytis ricini* are phylogenetically and biologically similar as stated earlier (Walker, 2016, Yamuna, 2015). Therefore, fungicides found to be effective against *Botrytis* spp. can also be applied to that of *B. ricini*. An extensive study conducted by (Leroux, 2007), on chemical control of *Botrytis* revealed that fungicides such as "botryoticides" are very efficient in protecting crops from *Botrytis* spp. Several in *vitro*

studies have identified other fungicides such as Fluazinam and thiophanate-methyl as effective against *Botrytis* spp (Leroux, 2007, Shao, 2015). Studies conducted by Oliveira and Soares (de Oliveira Datovo, & Soares, 2018), to assess the sensitivity of *B. ricini* isolates to Fluazinam and thiophanate-methyl showed that both fungicides were extremely intrinsically toxic to *B. ricini;* however, fluazinam was found to inhibit the growth of *B. ricini*.

Knowledge about the genetic basis of fungicide resistance is very crucial to develop strategic methods to overcome the disease. The importance of this knowledge is well documented, mostly for *Botrytis* spp (Walker, 2016). Unfortunately, there is little to no research reports on the genetic basis for *B. ricini* fungicide resistance in castor.

Biological control of gray mold

Biological control may arise either as a direct or indirect relationship among useful organisms and pathogens (Vos et al., 2015). The genus *Trichoderma* consists of several fungal strains with biological control ability (Woo, 2014). Although *Trichoderma* species are soil fungi, their ability to reduce plant disease and to survive under unfavorable weather conditions have been extensively studied (Vos et al., 2015, Alonso-Ramírez, 2014). *Trichoderma* spp. has been used as a bio-control agent against several fungal diseases such as *Pythium, Rhizoctonia*, *Fusarium*, and *Phytophthora* (Lorito et al., 2010, Sharma, 2011). Research on *Clonostachys rosea* have been found to be highly effective in controlling fungal diseases specifically caused by *Botrytis* spp (Chagas, 2009, Elad, & Stewart, 2007, Tirupathi et al., 2006). Bhattiprolu (2008) found that both *T. viride* and *T. harzianum* were capable of inhibiting the mycelia growth of *B. ricini*. Raoof and Yasmeen (2003), experimented on *in vitro* and detached spike method of strains of *Trichoderma* species and *Pseudomonas* species against *B. ricini*. The authors observed that the inhibition growth of *Trichoderma* spp. occurred after 72hours of incubation, and within 96hours, the hyphae of *Trichoderma* were matured. Treatment of gray mold infected castor spikes with *T. viride* (10^6 spores/mL) soon after early symptom reduced the disease development drastically. However, for detached spike method, about 45% of disease decrease was recorded with the use of *T. viride* and *P. fluorescenes*. Bhattiprolu (2008), studied on the effectiveness of *Trichoderma viride* against *B. ricini* as influenced by culture medium pH (4.0 – 6.0), incubation temperature (25°C), fungicides and exposure to mutagenic agents (gamma rays). The isolate was found compatible at 10% leaf extracts of *Azadirachta indica, Curcuma longa, Datura stramonium, Emblica indica, Lantana camera, Memordica dioica,* and *Ricinus communis* and other fungicides such as mancozeb 0.25%, copper oxychloride 0.3%, and thiram 0.3% but not with benomyl 0.1%, carbendazim 0.1%, thiophanate-methyl 0.1%, and 0.2% hexaconazole. In the end, mutant TV4 obtained the highest inhibition of *B. ricini* of about 88% when compared to the wild castor isolate.

Navaneetha et al., (2015) conducted research on the efficacy of *Trichoderma* species against *Botrytis ricini* in a greenhouse. The concentration suspension of liquid formulation at $2x10^7$ conidia/mL sprayed at 2 mL/L water within the period of capsule formation decreased (65%) of the castor diseased spike. Also at the same concentration of liquid formulation but with 1 mL/L spray of *T. harzianum* + *Trichoderma asperelium* gave the same results. The liquid formulation was kept in room temperature for about 540 days but was still stable. Because liquid formulations are easier to apply on the field most castor growers prefer its use.

CONCLUSION

Despite the increasing demand for castor oil worldwide, the attack on the crop by fungal diseases that undermines its productivity has not been curtailed. The main challenge gray mold disease in castor ever since its emergence presents to the scientific community, stems from the difficulty in breeding highly resistant genotype, and lack of understanding of the diseases development cycle. Available literature suggests that the ultimate way to overcome gray mold disease in castor is through the use of resistance varieties coupled with chemical treatment and crop rotation practice. Nevertheless, an idea about how sexual reproduction occurs within *B. ricini* is vital to avoid fungi developing

fungicide resistance. Fungicides used against gray mold disease should be well studied to recommend most effective ones and also suggest the most appropriate time of application for optimum results. Moreover, it's also necessary to know how complete integrated resistance to the diseases could be achieved, and how to use marker-assisted selection to develop effective resistance genotypes. Likewise, it is imperative to do an economic and cost-benefit analysis to know the most appropriate management practice for controlling gray mold disease.

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