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Gray Mold of Castor: A Review

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1. Introduction

Castor plant (*Ricinus communis* L.) is a non-edible oilseed crop with unique oil features for the chemistry industry. The crop was very important in the mid and late nineteenth century and also during WWI. After that the crop lost its importance in developed countries (Godfrey, 1923), but in India and Brazil it has remained as the most important non-edible oilseed crop of the arid and semi-arid regions (Dange et al., 2005; Santos et al., 2007). Nowadays, due the constant pressure for renewable fuels, castor has been investigated as a potential source of biofuel, mainly in Brazil due to governmental stimulus, and this has raised the crop importance once again. Regardless of the lack of a well established crop system, castor hosts several pests and diseases which cause heavy losses in the crop yield. One of the most destructive diseases of castor is gray mold, caused by the fungus *Botryotinia ricini* (Godfrey) Whetzel. Actually, it is the anamorphic phase of *B. ricini*, known as *Amphobotrys ricini* (N.F. Buchw.) Hennebert, that is responsible for disease epidemics and heavy yield losses frequently observed in castor crops. The first epidemic outbreak caused by this fungus was reported by H.E. Stevens of the Florida Experiment Station, Gainesville, Florida (Godfrey, 1919, 1923). At that time, a meticulous study was conducted and much of our knowledge regarding the disease and its causal agent was published in the classic work of Godfrey (1923). Subsequently, only sporadic works were conducted by other scientists around the world, consequently few advances have been made on management of gray mold. Breeding programs have failed in developing varieties with satisfactory resistance levels (Kolte, 1995), and chemical control is still ineffective and economically prohibitive, mainly due to the lack of basal information about the causal organism and its biology. In this chapter, the major aspects of castor gray mold will be reviewed.

2. Gray mold of castor

2.1 Historic and economic importance

Castor gray mold was first reported in the USA in 1918, following pioneering investigations by H.E. Stevens and F. W. Patterson, who promptly suggested that the causal organism of castor gray mold was an unknown *Botrytis* species (Godfrey, 1919, 1923). This fungus had caused serious losses of castor crop in the summer of 1918 mainly in Florida and others southern States, where it was responsible for losses up to 100% of castor yield (Godfrey,

1923). Later, the disease was reported in almost all countries where castor has been cultivated (Kolte, 1995), having nowadays a worldwide distribution.

The first occurrence of this disease in USA was directly linked to seeds imported from Bombay (now Mumbai), India, even though until that time, such disease had not been described in that country (Godfrey, 1923). In his work, Godfrey (1923) did a detailed account of the destructive potential of the gray mold of castor under favourable condition. By attacking mainly reproductive organs of the castor plant, gray mold disease is implicated in direct losses of yield whatever the level of infection.

In India, today the major castor producer, gray mold is found in few states and is regarded as troublesome only in Andhra Pradesh and Tamil Nadu, in the South, where the weather conditions are more favourable for disease development where in 1987, an epidemic outbreak of gray mold occurred (Dange et al., 2005).

In Brazil, the disease was first reported in the São Paulo state in 1932. However, it was only in 1936 that any attention was given to the disease due to the serious losses which occurred that year (Gonçalves, 1936). Currently, gray mold is present in almost all Brazilian states and its importance has grown at the same time that the crop cultivation has been intensified, mainly in those regions where the weather conditions are favourable for disease development, including the Southern and South-eastern Brazilian states (Araújo et al., 2007; Freire et al., 2007). In the region of the “Brejo Paraibano”, where the recommended sowing period is between mid-April to early-May (Amorim Neto et al., 2000), the flowering period (mid-June to early-August) usually coincides with highly favourable conditions for disease development (Moraes et al., 2009). Yield losses of up to 100% are quite frequent when highly susceptible cultivars are planted. Conversely, in Bahia the major castor producer in Brazil gray mold is not a problem because the weather conditions are usually not favourable for disease development.

2.2 Etiology, taxonomy and population structure

The causal agent of gray mold of castor was originally described by Godfrey (1919) as *Sclerotinia ricini* Godfrey, based on the holomorph. Later, Whetzel (1945) transferred the species *S. ricini* to the genus *Botryotinia*, which since then has been known as *Botryotinia ricini* (Godfrey) Whetzel. Subsequently, the anamorphic state of *Botryotinia ricini* was named as *Botrytis ricini* N.F. Buchw. (Buchwald, 1949). This led to general confusion between the non-mycologist communities, which adopted the name *Botrytis ricini* N.F. Buchw., instead of *Botryotinia ricini* (Godfrey) Whetzel. In 1973, Hennebert erected the genus *Amphobotrys* to accommodate the anamorphic state of *B. ricini*, based mainly on the distinctive pattern of conidiophore ramification, and since then the anamorphic state became known as *Amphobotrys ricini* (N.F. Buchw.) Hennebert (Hennebert, 1973). Even so, several authors used, and still use, the erroneous name “*Botrytis ricini*” attributing its authority to Godfrey (Barreto & Evans, 1998; Batista et al., 1998; Dange et al., 2005; Lima & Soares, 1990).

Although the correct name to be applied to the causal agent of gray mold of castor is *Botryotinia ricini*, only the anamorphic state is observed in the field by most authors, and thus the name of the anamorph, at the expense of the name of the holomorph, is preferred (Holcomb et al., 1989; Lima et al., 2008).

Botryotinia ricini belongs to Sclerotiniaceae (Helotiales, Ascomycota) and is characterized by its dark, plane-convex, elongated sclerotia (Fig.1), which give rise to cinnamon brown to chestnut brown, long stipitate apothecia, with cylindrical to cylindro-clavate asci, apex slightly thickened, 8-spored; ascospores ellipsoidal, often sub-fusoid, one-celled, bi-guttulate and hyaline; paraphyses hyaline, filiform, septate (Godfrey, 1919). Its anamorphic phase is characterized by cylindrical, straight, dichotomously branched, pale brown conidiophores, with conidiogenous cell not inflated, thin-walled; conidia globose, maturing synchronically, on short denticles, smooth, one-celled, sub-hyaline to pale brown (Fig.1) (Godfrey, 1919; Hennebert, 1973; Lima et al., 2008). A synanamorph (*Myrioconium* sp. – spermatial state) may sometimes be present on culture media (Godfrey, 1923; Hennebert, 1973; Seifert et al., 2011). According to Kirk et al. (2008), the genus *Amphobotrys* remains monotypic.

Botryotinia ricini is regarded as a homothallic species (Beveer & Weeds, 2007), so sexual reproduction will readily take place. If sexual reproduction had taken place, a high degree of diversity would be expected within the *B. ricini* population; however, Bezerra (2007) shows evidence that, in the state of Paraíba (Northeast Brazil) populations of *B. ricini* are clonal, which means that sexual reproduction had not taken place within those populations. Nonetheless, this conclusion must be viewed with caution because the population sampled was relatively small. Unfortunately, there is no other work on this subject and, therefore, the population structure of *B. ricini* remains unknown.

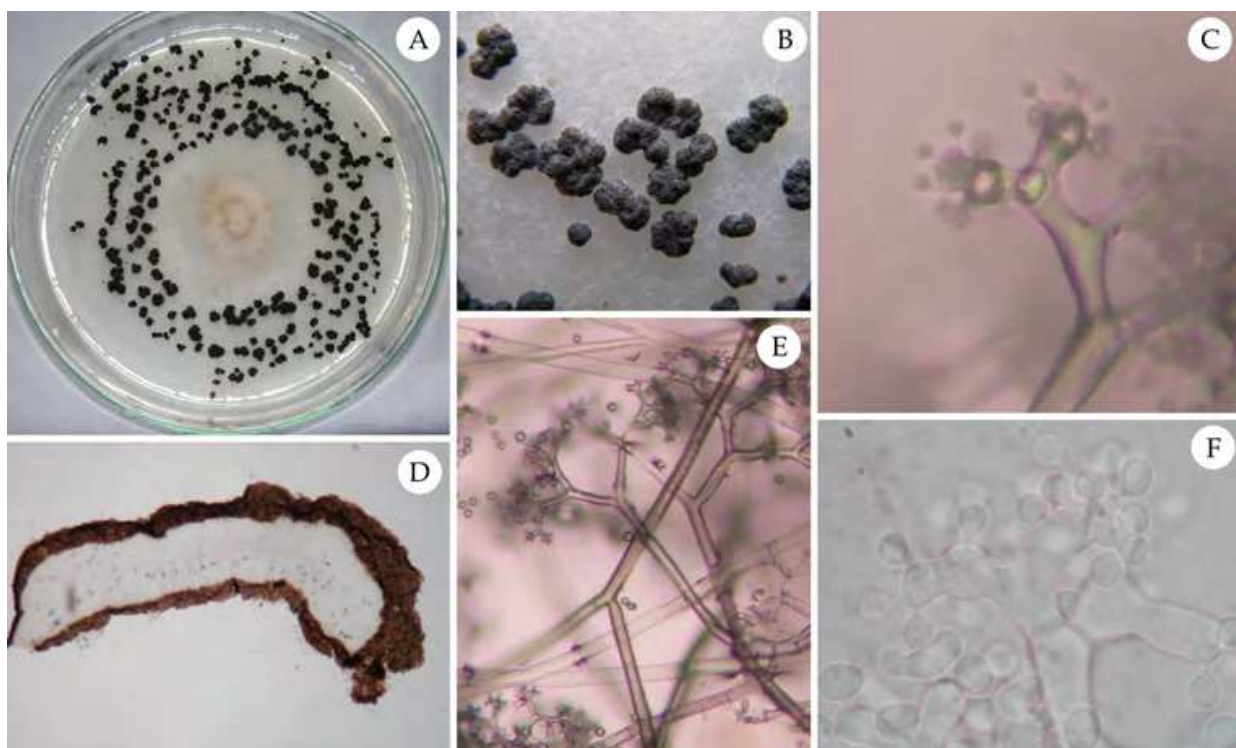


Fig. 1. Dark sclerotia on culture medium (A); close-up view of the sclerotia (B); transversal section through a sclerotium to show its plane-convex form (D); dichotomous branch of the conidiophores (E); conidiogenous cells showing the synchronic conidiogenesis (C); and close-up the conidiogenous cell to show the denticles and globose conidia (F). Photos: D.J. Soares.

2.3 Host penetration and colonization

In his classical work, G.H. Godfrey also investigated the infection process of *B. ricini* on leaves of the castor plant and concluded that penetration occurs directly through the host cuticle, in a process similar to *Botrytis cinerea* (Godfrey, 1923). After penetrating the cuticle, the fungus quickly spreads over the host tissues leading to a complete disorganization and breakdown. Although Godfrey had made mention as to the possible role of an enzymatic action in the penetration process, his conclusion pointed out to a mechanical penetration of the germ-tube, without tissues dissolving, prior to infection. On the other hand, Thomas & Orellana (1963b) found that it was not possible to verify the direct germ-tube penetration of *B. ricini*, through the cuticle or stomata, on castor capsules, before tissue maceration by pectic enzymes action, suggesting that the fungus first degraded the cuticle and later penetrated the host tissues (Orellana & Thomas, 1962). Probably *B. ricini* uses both mechanical and chemical processes to penetrate undamaged host tissue, however, no further studies have been done to clarify these questions.

Although the infection process of *B. ricini* needs to be better understood, it is likely that enzymes, such as lipases and cutinases, play an important role in the infection process similar to several other *Botrytis*-host interactions (Kars & van Kan, 2007). Additionally, *Botrytis* species can also affect the 'redox' process in the host plants, during the colonization of host tissues, through the production of enzymes like superoxide dismutase (Lyon et al., 2007). Due the great biological similarity of *Botrytis* spp. and *B. ricini*, probably, such enzymes also have an important role in the infection process of the causal agent of castor gray mold, as already evidenced by Orellana & Thomas (1962). Hoffmann et al. (2004) had, for example, extracted alpha and beta esterase and superoxide dismutase from *B. ricini*, however they did not perform a study to determine the role of such enzymes in the infection process of this fungus.

It is important to highlight the great distinction between the penetration process of a fungus under controlled and highly favourable condition in contrast with the natural process in the field. In the latter case, all aerial parts of the host are potential targets for deposition and penetration of *B. ricini*, because not only the conidia, regarded as the major propagative unit, usually responsible for the epidemic outbreak, but also the ascospores, sclerotia and mycelia fragments can give rise to infection, as observed in *Botrytis* spp. (Jarvis, 1978). So, besides direct penetration, probably natural openings and wounds also serve as a point of entrance for the fungus. Growth of the fungus on the host surface and consequently its penetration in the host tissues will depend on factors such as inoculum type, free water and nutrient availability, cuticle features, presence of exudates on floral organs and other glands, besides the abundance of natural openings and the size and age of wounds, as pointed out by Holz et al., (2007) for *Botrytis* species.

2.4 Symptoms

The primary targets of the fungus are the inflorescence and the capsules, in any development stage (Fig.2) (Araújo et al., 2007; Dange et al., 2005; Gonçalves, 1936; Lima et al., 2001). Some authors (Drumond & Coelho, 1981; Batista et al., 1996) claim that the male flowers are the first to be infected, but it is not always the case because any part of the inflorescence can be infected, the female flowers being the preferential target. That claim

came from the fact that the male flowers are the first to be exposed, at the earlier stage of inflorescence formation; consequently such flowers are exposed longer to the infection units of the fungus. However, as soon as the male flowers suffer anthesis they are no longer a target and are hardly infected, contradicting the statement of Drumond & Coelho (1981) that “the fungus attacks first the male flowers because the anthers, being soaked with the rain water or dew, easily retain the fungus spores carried by the wind”.



Fig. 2. Symptoms of gray mold attack on castor inflorescence and raceme. A-B. Symptoms on young inflorescences, before fertilization of female flowers. C-H. Symptoms on capsules at distinct development stages. Photos: D.J. Soares.

The first symptoms are visible as bluish spots on the inflorescences, on both female and male (before anthesis) flowers, and on developing fruits. On fruits, the symptoms can evolve to circular or elliptic, sunken, dark coloured spots that can result in rupture of the capsule (Fig.3 A-B) (Araújo et al., 2007). These symptoms are usually more frequent when a period of low relative humidity unfavourable to fungal sporulation occurs soon after the fungus penetrates the host tissues.

Depending on weather conditions (e.g. long periods with high relative humidity soon after the fungus penetrates the host), the occurrence of yellow ooze at the point of infection is frequent (Fig. 3 C-D) (Batista et al., 1996; Dange et al., 2005) as a result of the rapid enzymatic tissue degradation. The symptoms on the male flowers, before anthesis, are small, pale brown, necrotic spots, which can evolve to larger brown spots with a darker edge (Fig.3 E). The infected flowers and young capsules became softened due the fungal colonization and mycelial growth is, at first, pale gray and later dark olivaceous. A profuse sporulation is usually observed in such stage (Fig.3 F). When the infection starts on immature capsules, they become rotten; if the infection starts later, with fully developed capsules, the seeds usually became hollow, with coat discoloration and weight loss (Dange et al., 2005). On the

inflorescence, the male flowers can be infected first, but the fungus has a clear preference for the female flowers (Fig.3 G). Infection can lead to complete destruction of the raceme (Fig.3 H), particularly if it reaches the main stem and the weather conditions are favourable for the disease. Several other plant parts, e.g. leaves, petioles and stem can also be infected, mainly due to the deposition or fall of infected material from the inflorescence or racemes. On leaves, the lesions are usually irregular, but can assume an elliptic or circular pattern, the size is very variable, sometimes coalescing and resulting in a foliar blight (Fig.3 I-J). On petioles and stems, necrotic, sunken lesions usually are formed which can cause the strangulation and consequently death of the parts above the infection point (Fig. K-N) (Batista et al., 1996; Dange et al., 2005).



Fig. 3. Symptoms of gray mold on castor plants. Dark-bluish spot (A) and capsule rupture (B) under unfavourable conditions; Gray-bluish spot with yellowish-brown ooze (C-D) under favourable conditions; Dark brown spot on male flowers before anthesis (E); Profuse sporulation on infected capsule (F); Infected inflorescence showing the fungus preference for female flowers (G); Completely destroyed inflorescence (H); Leaf spot (I) and blight (J); Apical infection (K); Petiole infection (L); Stem (M) and rachis infection (N). Photos: D.J. Soares.

2.5 Epidemiology

There are few studies of the epidemiological aspects of gray mold on castor. Godfrey (1923) mentioned that temperatures around 25°C and high relative humidity are highly favourable to disease development. Such statements have been exhaustively repeated in almost all publications about the subject over the last decades (Araújo et al., 2007; Batista et al., 1998; Gonçalves, 1936; Kimati, 1980; Lima & Soares, 1990; Massola JR & Bedendo, 1997; Melhorança & Staut, 2005;). The minimum and maximum temperature for mycelial growth was established by Godfrey as 12 and 35°C, respectively (Godfrey, 1923).

Some complementary studies have confirmed that temperatures around 25°C are favourable to fungal growth and disease development (Araújo et al., 2003; Suassuna et al., 2003; Sussel, 2008). At temperatures below 20°C, the disease is little expressed and highly dependent on long periods of high relative humidity (Sussel, 2008). According to Sussel et al. (2011), there is a high correlation between the temperature and duration of leaf wetness with the disease incidence and severity. The same authors also concluded that the disease was more intense with a temperature of 28°C and 72 hours of leaf wetness, and that for temperatures below 15° the fungus needs more than 6 hours of leaf wetness, otherwise the disease does not occur (Sussel et al., 2011). Even when under optimal temperatures (near 25°C), the fungus appears to be highly dependent on periods of high humidity, since in a study developed by Esuruoso (1966) with 39 castor varieties in two distinct places, Ibadan and Ilora (West Nigeria), that share similar temperatures (24.4 to 27.9 and 24.2 to 27.2°C, respectively), but that rainfall was twofold higher in Ibadan than Ilora, the disease level in Ibadan was 1.5 fold higher than in Ilora. This dependence on high rainfall, and not only on high relative humidity, was also observed in a study conducted in the Paraíba state (Northeast Brazil) where a high correlation between the disease progress and the accumulated rainfall between the seventh and fifth day before the evaluation was observed (D.J. Soares, unpublished data). The correlation, however, was inversely proportional to the rainfall intensity which means that long periods of low-intensity rains are more favourable than short periods of high-intensity rains.

Sussel (2008) concluded that the disease shows a random distribution pattern, matching its airborne nature. However, under high rainfall, the disease assumes an aggregate pattern, typical of those dispersed by water splash. With reference to aerobiology, Sussel (2008) obtained a positive correlation between the average number of conidia collected daily in air and the weather variables: minimum temperature, mean relative humidity, mean precipitation and leaf wetness. There was an increase in the number of conidia collected with the elevation of the minimum temperature from 14.6 to 18.1°C; the same occurred when the mean relative humidity was raised from 42 to 95% and when the daily precipitation increased from 1 to 20 hours (Sussel, 2008, 2009a).

Under controlled conditions, at 25°C and relative humidity near saturation, Soares et al. (2010) had determined that the incubation period of *B. ricini* can vary from 44 to 88 hours (average 72 h) and the latent period from 72 to 144 hours (average 96 h), depending on the genotype.

Although in the recent years, there have been some advances in knowledge about the epidemiology of gray mold of castor, several issues remains unresolved. Further studies are needed to understand the role of each potential dispersal unit; how the fungus survives

between the growth seasons, for example, as sclerotia, on secondary hosts, on spontaneous (volunteer) castor plants. Additionally, it is also crucial to determine whether it is possible to predict disease development based on environmental variables, like precipitation or surface wetness.

2.5.1 Live cycle and host range

The disease starts with spore deposition on the host surface, followed by penetration and colonization of the host tissues. Soon after colonization, the fungus, under favourable conditions, sporulates profusely on the dead tissues, and then the conidia became the main inoculum source for new infection sites. Although most authors recognized the major role of conidia after the disease had been established in the field, there is much speculation about the primary inoculum source of *B. ricini*.

Godfrey (1923) claims that the fungus survives on soil or crop residues as sclerotia and, under the right conditions, these can produce sexual structures, which will be responsible for the initial infection. However, there is no report of sexual reproduction under natural conditions, other than the original reports of Godfrey (1919, 1923), so the purported role of ascospores as the initial inoculum source remains unclear, despite the fact that apothecia are easily overlooked in the field.

Under tropical climate, the initial inoculum source are probably the conidia from wild castor plants which grows spontaneously near the crop areas all year (Gonçalves, 1936). Wild castor plants can produce flowers throughout the year and consequently new susceptible tissue will be available for the fungus to self perpetuate in its anamorphic state through the year. By infecting the first inflorescence under favourable conditions, the fungus produces abundant sporulation, thus allowing multiple rounds of re-infection, since this pathogen is easily spread by wind, rain splash and, probably, by insects (Fig. 4 A-C) (Dange et al., 2005).

It was also mentioned by Godfrey (1923), that the fungus is seed-borne, the seeds being regarded as the primary inoculum source (Fig. 4 D-E). However, the role of seeds as a primary inoculum source requires further study. Probably the seeds have no essential role at the beginning of the epidemic because there is usually an interval of almost two months between sowing and flowering, so the inoculum originating from the seeds will not be available to infect the flowers. This means that, although *B. ricini* is a seed-borne fungus, it is probably not seed transmitted, because it could hardly infect the crops which grow from them, as conceived by Maude (1996). Thus, the most important role of the seeds is to carry the pathogen to new areas, rather than to act as a primary inoculum source for epidemic on the crop season.

Initially, it was suspected that *B. ricini* was host-specific and that it has a very narrow host range (Godfrey, 1923). Through artificial inoculation, Godfrey (1923) showed that the fungus is extremely dependent on high humidity and it was not able to cause disease, at same levels observed on castor plant, when inoculated on several other hosts, including members of Euphorbiaceae. In field inspections, the same author did not detect any other plant species showing symptoms of natural infection in the surroundings of castor-growing areas which were severely affected by the disease. Nevertheless, since the 1980s several reports of natural infection of *B. ricini* on members of Euphorbiaceae have been made, including both weeds and ornamentals: such as *Caperonia palustris* (Whitney & Taber, 1986), *Euphorbia supina*

(Holcomb et al., 1989; Russo & Rossman, 1991), *Euphorbia milli* (Sanoamuang, 1990), *Euphorbia pulcherrima* (Holcomb & Brown, 1990), *Euphorbia heterophylla* (Barreto & Evans, 1998), *Euphorbia inarticulata* (Alwadie & Baka, 2003), *Acalypha hispida* and *Jatropha podagrica* (Lima et al., 2008). Besides these reports of natural infection, artificial inoculations tests have shown that this pathogen has a wide host range within the Euphorbiaceae, including species of economic interest, e.g. cassava (*Manihot utilissima*) (Holcomb et al., 1989; Kumar et al., 2007; Lima et al., 2008). The sole report of natural infection by *B. ricini* outside the Euphorbiaceae family was made by Hansen & Bega (1955) on *Caladium bicolor* (Araceae) and, it is quite likely a case of fungus misidentification.

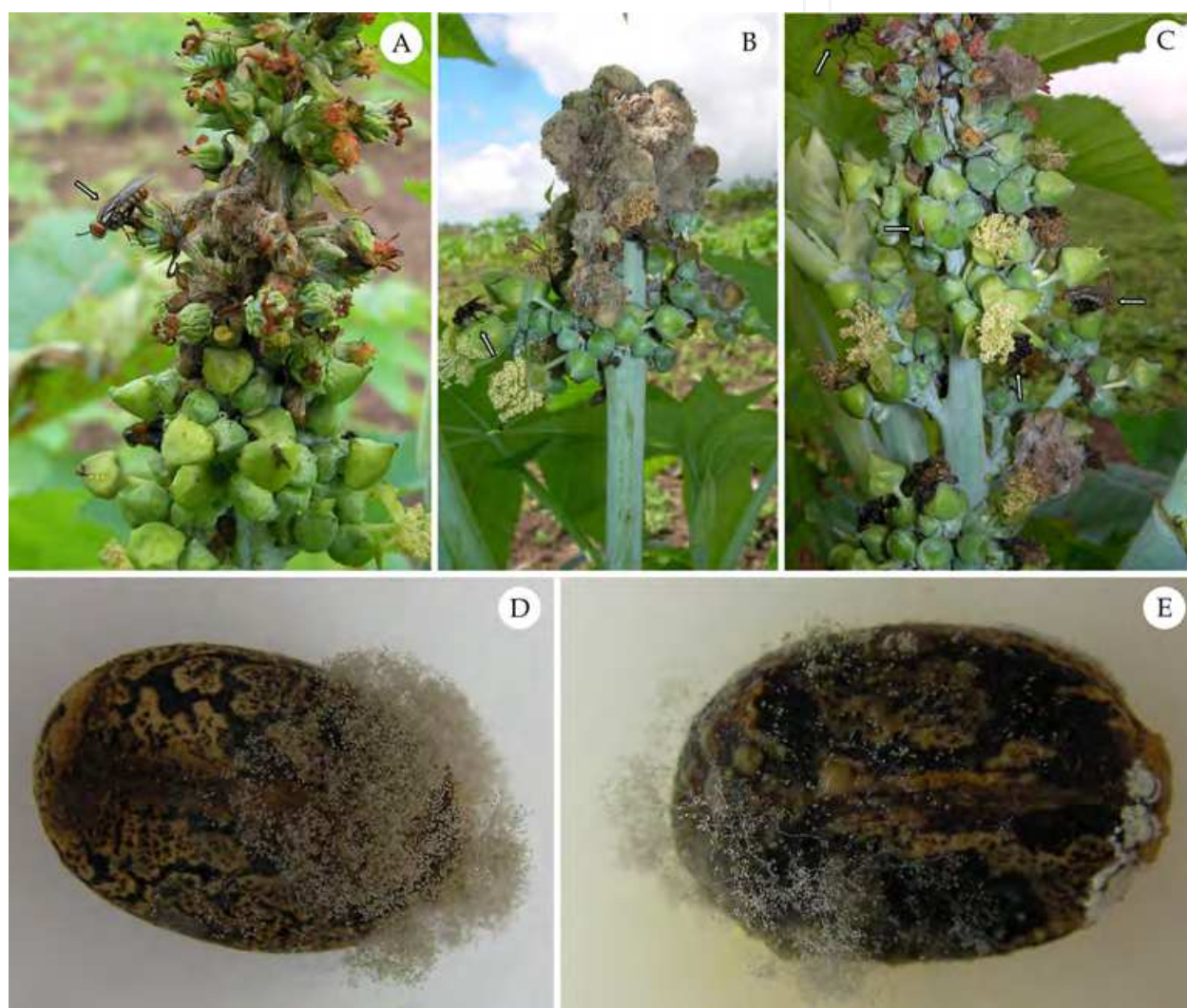


Fig. 4. Visitor insects (flies and stingless bees) on castor inflorescence infected by *B. ricini* (A-C). Seeds with profuse gray mold growth and sporulation (D-E). Photos: D.J. Soares.

Hence, it is possible that the fungus can survive in the field on several other plant species belonging to Euphorbiaceae, and these can be an inoculum reservoir of the fungus in such a way that it would be ready to infect its preferential host, the castor plant, as soon as susceptible tissues become available.

2.6 Host resistance

The search for resistance to gray mold has been investigated ever since the disease was described. In his classic work, Godfrey (1923) pointed out general conclusions that clearly have been overlooked over the decades by plant breeders and plant pathologists alike. Among these, three are worth mentioning: “(1) Plants of more ornamental type, with stalk, foliage, and sometimes pods in different shades of red or reddish green were more resistant; (2) All smaller, many branched plants, which by their yield indicated commercial possibilities, showed high susceptibility to the disease; and (3) Cross pollination in castor-bean fields probably occurs very extensively. It would require years of work to develop pure strains and then to select and breed for desirable qualities combined with resistance before permanent results could be secured” (Godfrey, 1923). Based on this information, it is clear that only minor progress has been achieved toward the development of resistant cultivars during the last century. As a result, breeding programs have failed to develop a resistant cultivar or hybrid until today.

Gonçalves (1936) reported that “spontaneous varieties” are highly resistant to the disease, since the fungus attacks only few capsules, while most of the capsules of the same raceme or from others raceme remain healthy. Although this behaviour is frequently observed in wild types, and even in some commercial cultivars, this statement must be viewed with caution since it has no scientific basis and probably such behaviour is a result of the wide genetic variation within castor plants.

According to several authors, varieties with more compact racemes, shorter internodes, and male flowers distributed all long the inflorescence are considered to be more susceptible to the pathogen (Batista et al., 1998; Costa et al., 2004; Dange et al., 2005; Milani et al., 2005; Thomas & Orellana, 1963b; Ueno et al., 2006; Zarzycka, 1958): a fact already noted by Godfrey (1923), and also by Esuruoso (1966), who concluded that the disease severity was more intense on short-stalked varieties than on those with longer stalks. Another relevant aspect is that, apparently, the presence of spines in the capsule predisposes them to pathogen attack (Alcântara et al., 2008; Cook, 1981; Lima & Soares, 1990).

Plants with capsules containing high soluble sugar concentrations are more susceptible to fungal development than plants with low soluble sugar concentrations (Orellana & Thomas, 1962). According to these authors, capsule resistance is intimately associated with its capacity of inactivation of pectic, cellulolytic and others hydrolytic enzymes through the products of the phenol oxidation (Thomas & Orellana, 1963a), as well as lower content of water-soluble pectin, higher content of calcium and magnesium and lower sodium and potassium contents (Thomas & Orellana, 1964).

Although several studies have been conducted to assess the resistance of castor bean genotypes, none of them deals with the inheritance of resistance to *B. ricini*, nor how it is governed. Probably, the resistance to gray mold is quantitative and possibly governed by several minor genes.

Quantitative resistance, also called horizontal resistance, is, according to Robinson (1976), universal and occurs in all plants against all parasites; it is also permanent and permits cumulative plant breeding. Horizontal resistance can be either passive or active, and

usually is conferred by numerous and complex mechanisms and can include traits such as tolerance and disease-escape which are not strictly resistance mechanisms *per se* (Robinson, 1976). It is also important to highlight the fact that horizontal resistance usually is an efficient way to achieve disease control, and that in the recent years selection for horizontal resistance has become easier with the use of marker-assisted breeding (Keane, 2012).

In Brazil, several studies have been carried out aiming to select for a resistance source to gray mold. However, what has become clear is that whilst there are differences in susceptibility among the assayed genotypes, none has the desired resistance level (Batista et al., 1998; Costa et al., 2004; Lima & Soares, 1990; Milani et al., 2005; Rego Filho et al., 2007). Among the genotypes assessed in different countries, thus far, several distinct levels of susceptibility have been observed, but not immunity (Anjani et al. 2004; Esuruoso, 1996; Zarzycka, 1958). The way that the breeding programs are being conducted, using only information generated by field investigations, and which are usually affected by uncontrollable factors, is much more likely to select for “field resistance” rather than for genetic resistance, i.e., the plant and raceme architecture, together with the weather conditions, play a bigger role in the disease development, by inducing a micro-climate formation, which might be more or less favourable to the pathogen. This means that unless we redirect our thinking to incorporate horizontal resistance, through a careful marker-assisted breeding program, whether it be genetic or morphological, it will probably never be possible to obtain a variety with satisfactory levels of resistance.

Some castor plant varieties have a thick outer wax layer which can act as a constitutive barrier to pathogen infection. However, there is no information about the role of this wax layer in the infection process of *B. ricini* and, although the adhesion of the conidia and subsequently their penetration into the host tissue could be difficult, the wax could also act as an elicitor; being responsible for the initial recognition of the host-pathogen interaction. Thus, the wax layer could actually favour the pathogen rather than inhibit its development. Field observations lead us to hypothesize that the latter, is the most probable scenario in the present pathosystem.

2.6.1 Host resistance assessment

As commented previously, most of the research involving the assessment of genotypes resistance has been conducted under field conditions and without any standard to quantify the disease and, worst still, sometimes using very subjective assessment methods, like that adopted by Lima & Soares (1990). This has been reflected by the fact that it is almost impossible perform a comparison among the different already studies undertaken. However, a diagrammatic scale to assess gray mold severity in castor- was published recently (Fig.5) (Sussel et al., 2009; Sussel, 2009b). This was developed in order to standardize disease assessment in field experiments, and was constructed based on the Weber-Fechner law and divided into 10 levels. Although useful, such a scale faces two crucial factors which might constitute an impediment to its wider adoption: first, there is great variation in raceme architecture among the castor genotypes, and; secondly, it only considers the fully developed raceme. In the first case, as the diagrammatic scale was drawn based on long conical racemes, its use to assess disease severity in genotypes with more or

less globose raceme will be difficult. In the second case, the scale does not take into account disease severity in inflorescences or even in immature racemes, where the disease is usually more severe and more difficult to estimate. Therefore, it is possible that the scale will underestimate disease severity. Nonetheless, the simple fact that such a diagrammatic scale is now available is a huge advance towards more reliable disease assessment and the possibility of comparing assays conducted by different researchers in different localities. Chagas et al. (2010) also developed a diagrammatic scale, with six levels, to assess the disease severity of gray mold on castor, but similar to the scale developed by Sussel et al. (2009), this scale was also developed based on long conical, full developed, racemes, and thus, will face the same problem mentioned above.

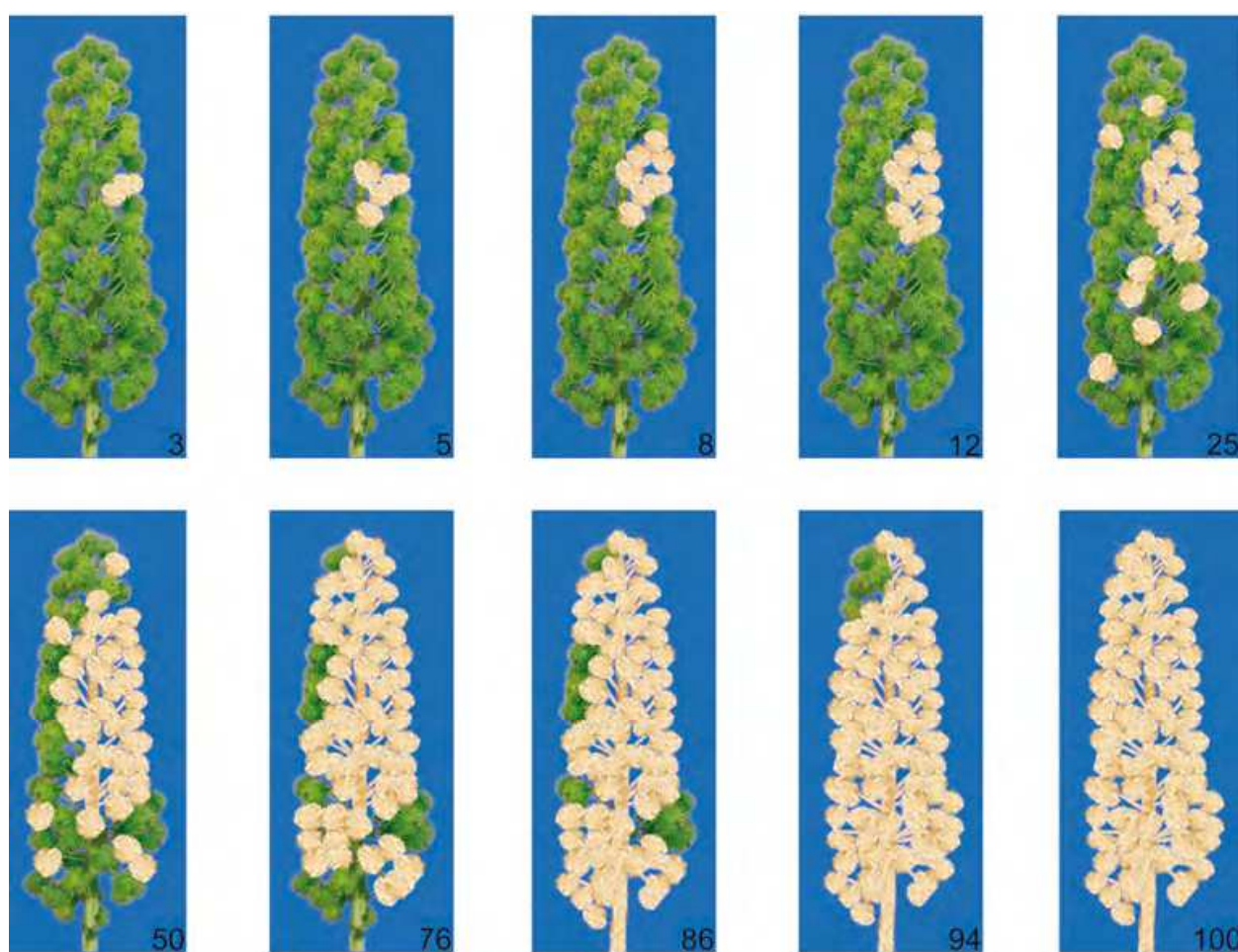


Fig. 5. Diagrammatic scale to assess the gray mold severity on castor (Reprinted from: Sussel et al. *Tropical Plant Pathology*, Vol. 34, No.3, pp.186-191, 2009, by permission). Numbers represents the percent area affected by the disease.

The first attempt to evaluate disease resistance under controlled conditions was made by C.A. Thomas & R.G. Orellana in 1963 using a biochemical test (Thomas & Orellana, 1963b). This methodology was reproduced by O.F. Esuruoso in Nigeria, a few years later (Esuruoso, 1969). This last author concluded that the laboratory results were similar to previous observations from field tests, and none of the tested varieties were resistant to

the disease (Esuruoso 1969). At the Embrapa Algodão Station - a branch of the Brazilian Agricultural Research Agency, responsible for developing research on castor - we have developed a controlled method to assess castor resistance to gray mold. The principal advantage of this method is to eliminate the unpredictable changes usually present under field evaluation, and then to select the castor plants based solely on their genetic background rather than select a phenotype. Based on such studies, it has been possible to obtain a clear difference among the so-called susceptible and resistant genotypes, which had been previously screened under field conditions. It has also been possible to stratify the genotypes within at least three ranks: highly susceptible, moderately susceptible and less susceptible (Silva et al., 2008; Soares et al., 2010), although none of the screened genotypes were regarded as resistant.

2.7 Disease management

Without doubt, protection of the inflorescences and immature capsules is crucial to avoid heavy yield losses when castor is cultivated under favourable disease conditions. Fully developed capsules are less susceptible to pathogen attack and the severity levels are usually lower when compared with infection in young capsules or on the inflorescence. However, it is important to note that under highly favourable conditions with high inoculum pressure, losses of 100% are relatively frequent (Anjani et al., 2004). There is no single measure to keep the disease under acceptable levels; as well as there is no knowledge about any acceptable disease level. As the pathogen has a very short incubation period, and is easily wind-dispersed, its destructive potential is very high and usually the growers do not want to take their chances and wait passively, they usually prefer to act before or at the first signs of the disease.

2.7.1 Cultural

Cultural practices are usually applied at aiming to prevent the introduction of inoculum into the field, reducing its survival, spread or build-up, or rendering the host less prone to disease attack (Palti & Rotem, 1983; Termorshuizen, 2001). The use of varietal resistance is regarded as the better method for disease management. However, as highlighted previously, there are no varieties with satisfactory resistance levels to gray mold (Anjani et al., 2003; Araújo et al., 2007; Cook, 1981; Dange et al., 2005; Kolte, 1995; Milani et al., 2005).

Several authors have recommended the use of healthy seeds, removal of plant debris, adequate choice of planting area and growing season, and use of less susceptible cultivars (Galli et al., 1968; Massola Jr. & Bedendo, 2005; Sussel, 2009a). It is also recommended to use plant spacing adjusted for maximum aeration (Kolte, 1995; Lima et al., 2005). The use of healthy seeds, including seed treatment with fungicides, is always a desirable practice, however, its practical benefits for the management gray mold are questionable, because as mentioned previously, it is unlikely that such seeds will serve as an inoculum source for that crop season, so this practice has much more value in promoting vigorous plant growth and avoiding the introduction of the pathogen into new areas. Elimination of alternate and reservoir hosts (euphorbiaceous hosts), as well as removal and destruction of inoculum persisting in plant residues, are welcome practices for management of gray mold, and

usually result in lower disease levels. Nevertheless, this practice must be followed by rigorous field inspection since the fungus is easily wind-dispersed, and once established in an area, such practice will become unfeasible. Perhaps, among the recognized cultural practices, the choice of growing season, or sowing time in such a way that spike development and maturity occur during the dry season, consequently avoiding the long, wet periods favourable to disease development, should be the most efficient one (Dange et al., 2005; Kolte, 1995); as evidenced by the fact that in locations where dry weather prevails, the disease does not occur (Godfrey, 1923; Kolte, 1995). This situation is typically observed in the Bahia state of Brazil, as mentioned previously.

2.7.2 Chemical

Seed treatment has been the management strategy most frequently recommended (Araújo et al., 2007; Batista et al., 1996; Godfrey, 1923; Gonçalves, 1936; Massola Jr. & Bedendo, 2005; Milani et al., 2005; Sussel, 2009), mainly to avoid the introduction of the pathogen into new areas. However, as the pathogen is air-borne and since it is already reported in almost all countries where castor is cultivated, the efficacy of such measures needs to be corroborated, because doubts about the role of the seeds in this pathosystem, as a primary inoculum source, still need to be clarified.

After disease establishment, fungicide spraying is usually the only way to stop or reduce the disease progress. However, there are few studies on chemical control of gray mold, and, of the fungicides tested, none is registered for use on castor crops in Brazil. According to Araújo et al. (2007), spraying of systemic fungicides soon after the appearance of the first symptoms delayed the epidemic and reduced disease progress.

In India Dange et al. (2005) recommended two prophylactic sprays with carbendazim (0.05%): the first at 50% of flowering; and the second, when the first disease symptoms appear. Although, Anjani et al. (2004) considered that non-genetic management measures have failed to control gray mold.

In the last decades, significant progress has been achieved regarding the use of fungicides to control plant diseases. Several new active fungicides with distinct modes of action, and usually with high specificity, have provided satisfactory levels of control of many plant diseases. However, for gray mold of castor, the main issue is not the ineffectiveness of fungicidal products, but the lack of research on the most appropriate timing of fungicide application and optimum dose, including also cost-benefit analyses.

Preliminary studies under controlled conditions have shown that carbendazim and azoxystrobin are effective against the gray mold pathogen (Bezerra, 2007). According to Chagas (2009), however, azoxystrobin was ineffective against *B. ricini*, while carbendazim and several others fungicides, including tebuconazole, iprodione and procymidone, were highly effective.

A field study, conducted under highly favourable conditions for disease development and using susceptible varieties, has confirmed that procymidone and iprodione are effective in disease control, but only if applied at the beginning of the epidemic and at weekly intervals. Where the timing of the first spraying was lost, and the application intervals were longer (15

days), the use of the same fungicides could not stop the disease and the losses reached 100% (D.J. Soares unpublished data).

Despite the limited studies dealing with chemical control of gray mold of castor, there are several fungicides known as “botryticides” which are effective in protecting crops against *Botrytis* spp. (Leroux, 2007), and probably we can assume that these fungicides also are effective against *B. ricini*. In several *Botrytis* pathosystems, there is a high concern about the use of fungicides just before, or even after the harvest because of the toxicological risks of their residues (Leroux, 2007). In contrast, in castor crops, such a restriction is not a concern and fungicides can be applied just before harvest. However, if used indiscriminately, there must be concern about resistance phenomena associated with several major botryticide families, including benzimidazoles, phenylcarbamates and dicarboximides (Leroux, 2007). Hence, control of gray mold of castor must consider several, rather than just one, management practices, as is currently recommended for the management of other *Botrytis* diseases (Leroux, 2007).

2.7.3 Biological

There are several studies dealing with the use of biological control agents, mainly *Trichoderma* spp. and *Clonostachys rosea* to control diseases caused by *Botrytis* spp. (Elad & Stewart, 2007). If we consider the fact that the genera *Botrytis* and *Amphobotrys* are biologically similar and, probably, phylogenetically related, we could expect that the use of such biological control agents could be applied as an effective strategy in the pathosystem *B. ricini* × *R. communis*. Actually, there are some studies conducted with *Trichoderma* and *Clonostachys rosea* for the control of gray mold of castor and promising results have been obtained (Bhattiprolu & Bhattiprolu, 2006; Chagas, 2009; Demant et al., 2006; Raoof et al., 2003; Tirupathi et al., 2006). However, it is clear that, although promising, these results are still experimental and much work needs to be done before permanent recommendations regarding the use of biological control agents can be secured for gray mold management.

3. Future challenges

The major problem about castor gray mold remains the lack of basic knowledge about its causal agent, how the disease develops, which factors are conducive to epidemics, and how we can manage it. It is necessary to elucidate, for example, what is the role of the climatic variables over the monocyclic components of the disease and how they affect the development of epidemics in the field. A better understanding of such relationships will determine which areas are suitable to grow castor. It is also imperative to know whether sexual reproduction is occurring within *B. ricini* populations in order to prevent fungicide resistance developing, perhaps by recommending permutations of fungicides with distinct active molecules and to determine the role of ascospores during the beginning of epidemics. However, we must first determine which fungicides are effective against gray mold, as well as their timing and frequency of application. We must also determine how resistance to the disease is inherited and if there are any phenotypic or genetic markers associated with it, so that breeders can more effectively generate resistant varieties using marker-assisted programs. Additionally, economic and cost-benefit analyses should be conducted to

determine which practices for disease management are worthwhile recommending. In others words, there is still much work to be done before we can better define the best strategies to avoid economic losses due to gray mold in castor crops.

4. Conclusion

Despite being one of the most important diseases of castor worldwide, causing severe losses on castor yield for almost a century, since its first report, gray mold is still poorly studied. The recent concern about renewable energy sources has proportioned a unique opportunity to draw attention back to this pathosystem. So, researchers involved with castor cultivation must now take this opportunity to try to elucidate the many still to be answered questions about this disease in order to mitigate the constant menace of gray mold to castor crops.

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6. References

- Alcântara, M.J.; Nassur, R.C.M.R.; Sá, G.A.; Fraga, A.C. & Neto, P.C. (2008). Avaliação de materiais de mamoneira visando obtenção de resistência ao mofo cinzento, *Proceedings of 5th Congresso Brasileiro de Plantas Oleaginosas, Óleos, Gorduras e Biodiesel*. 04.06.2010. Available from http://oleo.ufla.br/anais_05/artigos/a5_464.pdf
- Alwadie, H.M. & Baka, Z.A.M. (2003). New records of fungal pathogens of *Euphorbia inarticulate* from Aseer region. *Archives of Phytopathology and Plant Protection*, Vol.36, No.3/4, pp.195-209, ISSN 0323-5408
- Amorim Neto, M. S.; Araújo, A.E.; Beltrão, N. E.M.; Silva, L.C. & Gomes, D.C. (2000) Zoneamento e época de plantio para a mamoneira – estado da Paraíba. Embrapa Algodão, [Comunicado Técnico 108], ISSN 0102-0099, Campina Grande, Brazil
- Anjani, K.; Raoof, M.A.; Ashoka Vardhana Reddy, V. & Hanumanata Rao, C. (2004). Sources of resistance to major castor (*Ricinus communis* L.) diseases. *Plant Genetic Resources Newsletter*. Vol.137, (March 2004) pp.46-48, ISSN 0048-4334
- Araújo, A.E.; Suassuna, N.D.; Bandeira, C.M. & Agra, K.N. (2003). Efeito da temperatura na germinação de esporos de *Amphobotrys ricini* (= *Botrytis ricini*). *Fitopatologia Brasileira*, Vol.28, pp.S200 [Abstract], ISSN 0100-4158
- Araújo, A.E.; Suassuna, N.D. & Coutinho, W.M. (2007). *Doenças e seu Manejo*. In: *O Agronegócio da Mamona no Brasil*, D.M.P. Azevedo & N.E. de M. Beltrão, (Eds.), 283-303, Embrapa Informação Tecnológica, ISBN 978-85-7383-381-2, Brasília, Brazil

- Barreto, R.W. & Evans H.C. (1998). Fungal pathogens of *Euphorbia heterophylla* and *E. hirta* in Brazil and their potential as weed biocontrol agents. *Mycopathologia*, Vol.141, pp.21-36, ISSN 0301-486X
- Batista, F.A.S.; Lima, E.F.; Moreira, J.A.N.; Azevedo, D.M.P.; Pires, V.A.; Vieira, R.M. & Santos, J.W. (1998). Avaliação da resistência de genótipos de mamoneira, *Ricinus communis* L., ao mofo cinzento causado por *Botrytis ricini* Godfrey. Embrapa Algodão. [Comunicado Técnico 73], ISSN 0102-0099, Campina Grande, Brazil
- Batista, F.A.S.; Lima, E.F.; Soares, J.J. & Azevedo, D.M.P. (1996). Doenças e pragas da mamoneira (*Ricinus communis* L.) e seu controle. Embrapa Algodão, [Circular Técnica 21], ISSN 0100-6460, Campina Grande, Brazil
- Beever, R.E. & Weeds, P.L. (2007) *Taxonomy and Genetic Variation of Botrytis and Botryotinia*. In: *Botrytis: Biology, Pathology and Control*, Y. Elad; B. Williamson; P. Tudzynski & N. Delen (Eds), 29-52, Springer, ISBN 978-1-4020-6586-6, Dordrecht, The Netherlands
- Bezerra, C.S. (2007). Estrutura genética e sensibilidade a fungicidas de *Amphobotrys ricini* agente causal do mofo cinzento da mamoneira. MSc Dissertation (Genetic and Molecular Biology), Universidade Federal do Rio Grande do Norte, Natal, Brazil
- Bhattiprolu, S.L. & Bhattiprolu, G.R. (2006). Management of castor grey rot disease using botanical and biological agents. *Indian Journal of Plant Protection*, Vol.34, No.1, pp.101-104, ISSN 0253-4355
- Buchwald, N. F. (1949). Studies in the Sclerotiniaceae: I. Taxonomy of the Sclerotiniaceae. *Kongelige Veterinær- og Landbohøjskole, Aarsskrift*, Vol.32, pp.1-116. ISSN 03687171
- Chagas, H.A. (2009). Controle de mofo-cinzento (*Amphobotrys ricini*) da mamoneira (*Ricinus communis* L.) por métodos químico, biológico e com óleos essenciais. MSc Dissertation, (Agronomy), Universidade Estadual Paulista "Júlio de Mesquita Filho", Botucatu, Brazil
- Chagas, H.A.; Basseto, M.A.; Rosa, D.D.; Zanutto, M.D.; & Furtado E.L. (2010). Escala diagramática para avaliação de mofo cinzento (*Amphobotrys ricini*) da mamoneira (*Ricinus communis* L.). *Summa Phytopathologica*, Vol.36, No.2, pp.164-167, ISSN 0100-5405
- Cook, A.A. (1981). *Disease of Tropical and Subtropical Field, Fiber and Oil Plants*, Macmillan, ISBN 0-02-949300-5, New York, United States
- Costa, R. S.; Suassuna, T. M .F.; Milani, M.; Costa, M. N. & Suassuna, N. D. (2004). Avaliação de resistência de genótipos de mamoneira ao mofo cinzento (*Amphobotrys ricini*), *Proccedings of 1st Congresso Brasileiro de Mamona*. 04.06.2010. Available from: http://www.cnpa.embrapa.br/produtos/mamona/publicacoes/trabalhos_cbm2/049.pdf
- Dange, S.R.S; Desal, A.G. & Patel, S.I. (2005). *Diseases of castor*. In: G.S. Saharan; N. Mehta & M.S. Sangwan (Eds), 211-234, *Diseases of Oilseed Crops*, Indus Publishing Co, ISBN 81-7387-176-0, New Delhi, India
- Demant, C.A.R.; Furtado, E.L.; Zanutto, M. & Chagas, A.A. Controle do mofo cinzento com o uso de *Trichoderma*, *Proccedings of 2nd Congresso Brasileiro de Mamona*. 04.06.2010. Available from: http://www.cnpa.embrapa.br/produtos/mamona/publicacoes/trabalhos_cbm2/043.pdf

- Drumond, O. A. & Coelho S. J. (1981). Doenças da mamoneira. *Informe Agropecuário* Vol.7, No.82, pp.38-42, ISSN 0100-3364
- Elad, Y & Stewart, A. (2007). *Microbial Control of Botrytis spp.* In: *Botrytis: Biology, Pathology and Control*, Y. Elad; B. Williamson; P. Tudzynski & N. Delen (Eds), 223-236, Springer, ISBN 978-1-4020-6586-6, Dordrecht, The Netherlands
- Esuruoso, O.F. (1966). A preliminary study on the susceptibility of certain varieties of castor (*Ricinus communis* L.) to inflorescence blight disease caused by *Sclerotinia* (*Botrytis*) *ricini* (Godfrey) Whet. *The Nigerian Agricultural Journal*, Vol.3, No.1, pp.15-17, ISSN 0300-368X
- Esuruoso, O.F. (1969). The reaction of the capsules of certain varieties of castor to a biochemical test for susceptibility to the inflorescence blight diseases. *The Nigerian Agricultural Journal*, Vol.6, No.1, pp.15-17, ISSN 0300-368X
- Freire, E.C.; Lima, E.F.; Andrade, F.P.; Milani, M. & Nóbrega, M.B.M. (2007). *Melhoramento Genético*. In: *O Agronegócio da Mamona no Brasil*, D.M.P. Azevedo & N.E. de M. Beltrão, (Eds.), 171-194, Embrapa Informação Tecnológica, ISBN 978-85-7383-381-2, Brasília, Brazil
- Galli, F.; Tokeshi, H.; Carvalho, P.C.T.; Balmer, E.; Cardoso, C.O.N. & Salgado, C.L. (1968). *Doenças da Mamoneira*. In: *Manual de Fitopatologia: Doenças das Plantas e seu Controle*, Galli, F.; Tokeshi, H.; Carvalho, P.C.T.; Balmer, E.; Cardoso, C.O.N. & Salgado, C.L., 292-197, Editora Agronomica Ceres, São Paulo, Brazil
- Godfrey, G.H. (1919). *Sclerotinia ricini* n. sp. on the castor bean (*Ricinus communis*). *Phytopathology*, Vol.9, pp.565-567, ISSN 0031-949X
- Godfrey, G. H. (1923). Gray mold of castor bean. *Journal of Agricultural Research*, Vol.23, No. 9, pp.679-715 + 13 plates, ISSN 0095-9758
- Gonçalves, R.D. (1936). Mofo cinzento da mamoneira. *O Biológico*, Vol.2, No.7, pp.232-235, ISSN 0366-0567
- Hansen H.N. & Bega, R.V. (1955). *Botrytis* rot of *Caladium* tubers. *Plant Disease Reporter*, Vol.39, No.3, pp.283, ISSN 0032-0811
- Hennebert, G.L. (1973). *Botrytis* and *Botrytis*-like genera. *Persoonia*, Vol.7, No.2, pp.183-204, ISSN 0031-5850
- Hoffmann, L.V.; Coutinho, T.C.; Duarte, E.A.A.; Bandeira, C.M. & Suassuna, N. D. (2004) Cultivo de *Amphobotrys ricini* e detecção das enzimas málica, superóxido dismutase e esterase, *Proceedings of 1st Congresso Brasileiro de Mamona*, 14.06.2010. Available from: http://www.cnpa.embrapa.br/produtos/mamona/publicacoes/trabalhos_cbm1/126.PDF
- Holcomb, G.E. & Brown, W.L. (1990). Basal stem rot of cultivated *Poinsettia* caused by *Amphobotrys ricini*. *Plant Disease*, Vol.74, pp.828, ISSN 0191-2917
- Holcomb, G.E.; Jones, J.P. & Wells, D.W. (1989). Blight of prostate spurge and cultivated *Poinsettia* caused by *Amphobotrys ricini*. *Plant Disease*, Vol.73, pp.74-75, ISSN 0191-2917
- Holz, G.; Coertze, S. & Williamson, B. (2007). *The Ecology of Botrytis on Plant Surfaces*. In: *Botrytis: Biology, Pathology and Control*, Y. Elad; B. Williamson; P. Tudzynski & N. Delen (Eds), 9-28, Springer, ISBN 978-1-4020-6586-6, Dordrecht, The Netherlands
- Jarvis, W.R. (1978). Epidemiology. In: *The Biology of Botrytis*, J.R. Coley-Smith, K. Verhoeff & W.R. Jarvis (Eds), 219-250, Academic Press, ISBN 0-12-179850-X, London, UK

- Kars, I. & van Kan, J.A.L. (2007) *Extracellular Enzymes and Metabolites Involved in Pathogenesis of Botrytis*. In: *Botrytis: Biology, Pathology and Control*, Y. Elad; B. Williamson; P. Tudzynski & N. Delen (Eds), 99-118, Springer, ISBN 978-1-4020-6586-6, Dordrecht, The Netherlands
- Keane, P.J. (2012) *Horizontal or Generalized Resistance to Pathogens in Plants*. In: *Plant Pathology*, C.J.R. Cumagun (Ed), 317-352, InTech, ISBN 978-953-307-933-2, Viena, Austria
- Kimati, H. (1980). *Doenças da Mamoneira*. In: *Manual de Fitopatologia: Doenças das Plantas Cultivadas*, F. Galli, (Ed), 347-351, Editora Agronomica Ceres, São Paulo, Brazil
- Kirk, P.M.; Cannon, P.F.; Minter, D.W. & Stalpers, J.A. (2008). *Dictionary of the Fungi*, CABI Publishing, ISBN 978-0-85199-826-8, Wallingford, UK
- Kolte, J.S. (1995). *Castor: Diseases and Crop Improvement*. Shipra Publications, ISBN 81-85402-54-X, Delhi, India
- Kumar, A.; Reddy, P.N. & Rao, T.G.N. (2007). Host range studies of *Botrytis ricini*, the causal agent of castor grey mold. *Indian Journal of Plant Protection*, Vol.35, No.1, pp.140-141, ISSN 0253-4355
- Leroux, P. (2007) *Chemical Control of Botrytis and its Resistance to Chemical Fungicides*. In: *Botrytis: Biology, Pathology and Control*, Y. Elad; B. Williamson; P. Tudzynski & N. Delen (Eds), 195-222, Springer, ISBN 978-1-4020-6586-6, Dordrecht, The Netherlands
- Lima, B.V.; Soares, D.J.; Perreira, O.L. & Barreto, R.W. (2008). Natural infection of *Acalypha hispida* and *Jatropha podagrica* inflorescences by *Amphobotrys ricini* in brazil. *Australasian Plant Disease Notes* Vol.3, pp.5-7, ISSN 1833-928X
- Lima, E.F.; Araújo, A.E. & Batista, F.A.S. (2001) *Doenças e seu Controle*. In: *O Agronegócio da Mamona no Brasil*, D.M.P. Azevedo & E.F.Lima (Eds), 192-212, Embrapa Informação Tecnológica, ISBN85-7383-116-2, Brasília, Brazil
- Lima, E.F. & Soares, J.J. (1990). Resistência de cultivares de mamoneira ao mofo cinzento, causado por *Botrytis ricini*. *Fitopatologia Brasileira*, Vol.15, No.1, pp.96-98, ISSN 0100-4158
- Lima, V.P.T.; Graça Leite, E.A.; Botrel, E.P.; Fraga, A.C. & Castro Neto, P. (2005). Avaliação de ataque de mofo cinzento da mamoneira, variedade Al Guarany 2002, em diferentes espaçamentos. *Proceedings of 2nd Congresso Brasileiro de Plantas Oleaginosas, Óleos, Gorduras e Biodiesel*. 12.06.2010. Available from: http://oleo.ufla.br/anais_02/artigos/t183.pdf
- Lyon, G.D.; Goodman, B.A. & Williamson, B. (2007). *Botrytis cinerea Perturbs Redox Processes as an Attack Strategy in Plants*. In: *Botrytis: Biology, Pathology and Control*, Y. Elad; B. Williamson; P. Tudzynski & N. Delen (Eds), 119-142, Springer, ISBN 978-1-4020-6586-6, Dordrecht, The Netherlands
- Massola JR, N. S. & Bedendo, I. P. (2005). *Doenças da Mamoneira (Ricinus communis L.)*. In: *Manual de Fitopatologia: Doenças das Plantas Cultivadas*, H. Kimati; L. Amorim; A. Bergamin Filho; L.E.A. Camargo & J.A.M. Rezende (Eds), 497-500,. Agronomica Ceres, ISBN 85-318-0008-0, São Paulo, Brazil
- Maude, R.B. (1996). *Seedborne Diseases and their Control: Principles and Practice*. 978-0851989228, CABI Publishing, Kew, UK
- Melhorança, A. L. & Staut, L.A. (2005). Informações técnicas para a cultura da mamona em Mato Grosso do Sul. Embrapa Agropecuária Oeste [Sistemas de Produção 8], ISSN 1679-1320, Dourados, Brazil

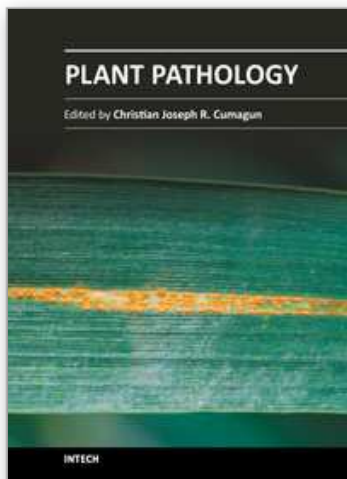
- Milani M.; Nóbrega, M.B.M.; Suassuna, N.D. & Coutinho, W.M. (2005). Resistência da mamoneira (*Ricinus communis* L.) ao mofo cinzento causado por *Amphobotrys ricini*. Embrapa Algodão [Documentos 137], ISSN 0103-0205, Campina Grande, Brazil.
- Moraes, W.B.; Souza, A.F.; Tomas, M.A.; Cecilio, R.A. & Jesus Junior, W.C. (2009). Zoneamento das áreas de risco da ocorrência de mofo cinzento da mamona no Brasil. *Proceedings of 13th Encontro Latino Americano de Iniciação Científica and 9th Encontro Latino Americano de Pós-Graduação*, 01.06.2010. Available from: http://www.inicepg.univap.br/cd/inic_2009/anais/arquivos/0821_1434_04.pdf
- Orellana, R.G. & Thomas, C.A. (1962). Nature of predisposition of castor beans to *Botrytis*. I. Relation of leachable sugar and certain other biochemical constituents of the capsule to varietal susceptibility. *Phytopathology*, Vol.52, No.6, pp.533-538, ISSN 0031-949X
- Palti, J. & Rotem, J. (1983). *Cultural Practices for the Control of Crop Diseases*. In: *Plant Pathologist's Pocketbook*, A. Johnston & C. Booth (Eds), 186-195, CMI, ISBN 0-85-198-460-6, Kew, England
- Raouf, M.A.; Yasmeen, M. & Kausar, R. (2003). Potential of biocontrol agents for the management of castor grey mold, *Botrytis ricini* godfrey. *Indian Journal of Plant Protection*, Vol.31, No.2, pp.124-126, ISSN 0253-4355
- Rego Filho, L.M.; Bezerra Neto, F.V. & Santos, Z.M. (2007). Avaliação da incidência de mofo cinzento em genótipos de mamoneira no período de outono-inverno em campos dos Goytacazes-RJ. *Proceedings of the 2nd Congresso da Rede Brasileira de Tecnologia de Biodiesel 2006*. 27.07.2011. Available from: <http://www.biodiesel.gov.br/docs/congresso2007/agricultura/3.pdf>
- Robinson, R.A. (1976). *Horizontal Pathosystem Analysis*. In: *Plant Pathosystem (Advanced Series in Agricultural Sciences 3)*, R.A. Robinson, 74-89, Springer-Verlag, ISBN 3-540-07712-X, Berlin, Germany
- Russo, V.M. & Rossman, A.Y. (1991). Occurrence of *Amphobotrys ricini* on prostate spurge in Oklahoma. *Plant Disease*, Vol. 75, pp. 750, ISSN 0191-2917
- Santos. R.F.; Kouri, J.; Barros, M.A.L.; Firmino, P.T. & Requião. L.E.G. (2007). *Aspectos Econômicos do Agronegócio da Mamoneira*. In: *O Agronegócio da Mamona no Brasil*, D.M.P. Azevedo & N.E. de M. Beltrão, (Eds.), 22-41, Embrapa Informação Tecnológica, ISBN 978-85-7383-381-2, Brasília, Brazil
- Sanoamuang, N. (1996). First report of gray mold blight caused by *Amphobotrys ricini* on crown of thorns in Thailand. *Plant Disease*, Vol.80, pp.223, ISSN 0191-2917
- Seifert, K.; Morgan-Jones, G.; Gams, W. & Kendrick, B. (2011). *The Genera of Hyphomycetes*, CBS-KNAW Fungal Biodiversity Centre, ISBN 978-90-70351-85-4, Utrecht, The Netherlands
- Silva, J.A.; Suassuna, N.D.; Coutinho, W.M. & Milani, M. (2008). Esporulação de *Amphobotrys ricini* em frutos de mamoneira como componente de resistencia ao mofo cinzento, *Proceedings of the 3th Congresso Brasileiro de Mamona*. 04.06.2010. Available from: <http://www.cnpa.embrapa.br/produtos/mamona/publicacoes/cbm3/trabalhos/FITOSSANIDADE/F%2012.pdf>

- Soares, D.J.; Fernandes J.N. & Araújo, A.E. (2010). Componentes monociclicos do mofo cinzento (*Amphobotrys ricini*) em diferentes genótipos de mamoneira, *Proceedings of 4th Congresso Brasileiro de Mamona*, 12.07.2011. Available from <http://www.cbmamona.com.br/pdfs/FIT-01.pdf>
- Suassuna, N.D.; Araújo, A.E.; Bandeira, C.M.; Agra, K.N. (2003). Efeito de temperatura no crescimento e esporulação de *Amphobotrys ricini* (= *Botrytis ricini*). *Fitopatologia Brasileira* Vol.28, pp.S232 [Abstract], ISSN0100-4158
- Sussel, A.A.B. (2008). Epidemiologia do mofo-cinzento (*Amphobotrys ricini* Buchw.) da mamoneira. PhD Thesis (Phytopathology), (June 2008) Universidade Federal de Lavras, Lavras, Brazil
- Sussel, A.A.B. (2009a) Epidemiologia e Manejo do Mofo-cinzento-da-mamoneira. Embrapa Cerrados, [Documentos 241], ISSN 1517-5111, Brasília, Brazil
- Sussel, A.A.B. (2009b) Escala Diagramática para Avaliação do Mofo-cinzento-da-mamoneira. Embrapa Cerrados, [Documentos 247], ISSN1517-5111, Brasília, Brazil
- Sussel, A.A.B.; Pozza, E.A. & Castro, H.A. (2009). Elaboração e validação de escala diagramática para avaliação da severidade do mofo cinzento da mamoneira. *Tropical Plant Pathology*, Vol.34, No.3, pp.186-191, ISSN 1982-5676
- Sussel, A.A.B.; Pozza, E.A. & Castro, H.A. & Lasmar, E.B.C. (2011). Incidência e severidade do mofo-cinzento-da-mamoneira sob diferentes temperaturas, períodos de molhamento e concentração de conídios. *Summa Phytopatologica* Vol.37, No.1, pp.30-34, ISSN0100-5405
- Termorshuizen, A.J. (2001). *Cutural Control*. In: *Plant Pathologist's Pocketbook*, J.M. Waller; J.M. Lenné & S.J. Waller (Eds), 318-327, CABI Publishing, ISBN 0851994598, Wallingford, UK
- Thomas, C.A. & Orellana, R.G. (1963a) Nature of predisposition of castor beans to *Botrytis*. II. Raceme compactness, internode lengths, position of staminate flowers and bloom in relation to capsule susceptibility. *Phytopathology*, Vol.53, No.2, pp.249-251, ISSN 0031-949X
- Thomas, C.A. & Orellana, R.G. (1963b) Biochemical tests indicative of reaction of castor bean to *Botrytis*. *Science*, Vol.139, pp.334-335, ISSN 0036-8075
- Thomas, C.A. & Orellana, R.G. (1964) Phenols and pectins in relation to browning and maceration of castorbean capsules by *Botrytis*. *Phytopathologische Zeitschrift*, Vol.50, No.4, pp.359-366, ISSN 0931-1785
- Tirupathi, J.; Kumar, C.P.C. & Reddy, D.R.R. (2006). *Trichoderma* as potential biocontrol agents for the management of grey mold of castor. *Journal of Research Angraui*, Vol.34, No.2, pp.31-36
- Ueno, B.; Hellwig, T.C.; Nickel, G.; Silva, S.D.A. (2006). Resistência ao mofo cinzento em 15 genótipos de mamoneira cultivadas na região de Pelotas, RS, na safra 2004/2005, *Proceedings of 2nd Congresso Brasileiro de Mamona*. 12.06.2010. Available from: http://www.cnpa.embrapa.br/produtos/mamona/publicacoes/trabalhos_cbm2/049.pdf
- Whetzel, H.H. (1945). A synopsis of the genera and species of the Sclerotiniaceae, a family of stromatic inoperculate discomycetes. *Mycologia*, Vol.37, pp.648-714, ISSN 0027-5514

- Whitney, N.G. & Taber, R.A. (1986). First report of *Amphobotrys ricini* infecting *Caperonia palustris* in the United States. *Plant Disease*, Vol.70, pp.892, ISSN 0191-2917
- Zarzycka, H. (1958). Resistance of some varieties of castor bean (*Ricinus communis*) to the fungus *Botrytis cinerea* Pers. *Acta Agrobotanica*, Vol.7, pp.117-124, ISSN 0065-0951

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