VARIETAL SCREENING OF BANANA AGAINST ANTHRACNOSE DISEASE

By

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THESIS

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DEDICATED TO

MY BELOVED PARENTS

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DECLARATION

I hereby declare that this thesis entitled "Varietal screening of banana against anthracnose disease" is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title of any other University or Society.

K.L. Srinagesh

Vellanikkara,
16-4-, 1984.

CERTIFICATE

Certified that this thesis entitled "Varietal screening of banana against anthracnose disease" is a record of research work done independently by Sri. K.L. Srinagesh, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

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INTRODUCTION

Banana (*Musa* spp.) is one of the most important fruit crops in India. Not only it is cultivated on a large scale as a field crop, but it is also widely grown in backyards of house holds. This crop is cultivated in India in an area of 1,53,200 ha with an annual production of 1.7 to 2.0 million tonnes (Anonymous, 1962). Among the banana growing states, Kerala ranks first with an area of 49,262 ha with an annual production of 3,27,405 tonnes (Anonymous, 1983).

The average production per hectare in Kerala is very low (6.70 tonnes/ha) when compared to the neighbouring state of Tamil Nadu (17.75 tonnes/ha). Among the reasons attributed to the low yield, the incidence of diseases plays an important role. The most serious and wide spread disease of banana in the world is Panama disease (Vascular wilt) caused by *Fusarium oxysporum* Schlect *var. cubense* (E.F. Smith) Wollen W. However, this disease is not having much importance to Kerala state. The most devastating disease of banana in this state is Bunchy Top. It is a virus disease transmitted by the aphid *Pentalonia nigronervosa* Coq. Other important disease, in Kerala are leaf spot (Sigatoka) caused by *Cercospora musae* Zimm and Cordana leaf spot caused by *Cordana musae* (Zimm) Von Hohn. The anthracnose disease of banana caused by *Colletotrichum gloeosporioides* Penz (Sacc), the imperfect

stage of *Glomerella cingulata* Stonem (Spauld) and Shrenk is considered to be one of the serious post harvest diseases in Kerala which also causes some loss in the field. The actual loss due to this disease in Kerala is not known. But in Tamil Nadu loss due to this disease was estimated to be 10 to 15 per cent (Damodaran and Ramakrishnan, 1964). Though this is an important disease of banana, no study has been undertaken on this disease in Kerala. Therefore, an attempt has been made to study some aspects of this disease.

The detailed symptomatology of the disease, both in field and after harvest was not studied in Kerala. Therefore, in the present investigation, an attempt has been made to study the symptoms of the disease.

On perusal of the literature it is evident that there is some confusion of the nomenclature of the pathogen. Therefore morphological study of the pathogen is undertaken with the modern taxonomical concepts to avoid the confusion regarding the nomenclature.

Kerala state is blessed with a good number of varieties suitable for both culinary and dessert purposes. The relative susceptibility/resistance of these varieties against anthracnose disease was not known. Therefore a study was taken up to screen the varieties *in vitro* against the disease.

The importance of biochemical constituents in the host in imparting resistance or susceptibility to diseases is a well established factor in plant pathology. The banana

fruit is found to vary with regard to chemical constituents at different stages of development. These chemical constituents may be responsible for susceptibility or resistance of banana fruit to infection and development of the disease. The influence of chemical constituents of banana varieties in the incidence of anthracnose disease was stressed by Damodaran and Ramakrishnan, 1964; Thakur, 1969. Keeping this in view, chemical constituents of banana fruit of different varieties at various developmental stages have been worked out to study whether any correlation can be established with the incidence of anthracnose disease.

To achieve the above goal, the present investigation was undertaken with the following objectives.

1. To study the symptomatology of the disease both in the field as well as after harvest.
2. To study in detail, the morphological characters of the fungus with modern taxonomical concepts.
3. To find out the reaction of different banana varieties to infection.
4. To find out the biochemical aspects contributing to susceptibility/resistance in different banana varieties.

Review of Literature

REVIEW OF LITERATURE

The anthracnose of banana is considered, the most important and widely distributed post harvest disease. This disease is also known as Gloeosporium fruit rot, black rot or ripe fruit rot and in Traders' language, it is known as 'finger stalk rot'. This disease has been found wherever bananas are grown. The fruits are liable to infection in the field, in local markets and in large consignments during transport especially in the ripening time (Wardlaw, 1972). However, this pathogen has been reported to attack leaves also by Petch (1917) from Ceylon, Campbell (1925) from Fiji and Wardlaw (1934) from Trinidad.

The incidence and wastage caused by banana anthracnose has been recorded by various workers. Agati (1922) recorded 15.62 per cent loss; Castellani (1956) observed 15 per cent loss and Damodaran and Ramakrishnan (1964) recorded 10 to 15 per cent loss.

Gloeosporium musarum Cooke and Massee was first recorded (Grev., 16, 3, 1873) on the skin of ripe banana fruit from Queensland. Later Von Arx (1957a) redescribed the pathogen as Colletotrichum musae (Berk and Curt) Von Arx. After the report of the perfect stage of the pathogen Glomerella cingulata (Stonem) Spauld and Shrenk, Von Arx (1957b) made a detailed study in then

known species of Colletotrichum and he placed Gloeosporium musarum Cooke and Massee and Colletotrichum musae as synonyms of C. gloeosporioides (Penz) Sacc.

Symptoms of the disease are mainly seen on the fruit. Detailed descriptions of symptoms in the field have been given by Cobb from West Indies (1906); Agati from Phillippines (1922); Park from Ceylon (1930 and 1933); Parham (1935) from Fiji islands.

In India the symptomatology had been described by Dastur (1916), Chona (1933), Roy and Sharma (1952) and Damodaran and Ramakrishnan (1958).

Cobb (1906) observed that in case of Cavendish varieties, when the fruit attain more or less three inches in length, they began to change colour and to shrival. The colour changes passed through greenish yellow, yellow brown or french grey to almost black. The final blackening was accompanied by pink eruptions of Gloeosporium acervuli. Agati (1922) also described the symptomatology of the disease and according to him, the disease was marked on the fruit by small, circular, black specks usually on skin, distal ends of hands and flowers. These spots enlarged and advanced towards the hands, then to petiole and finally to bunch stalk. In the advanced stages of infection, the specks became sunken and

coalesced forming larger spots. In the case of severe infection, the entire fruit becomes dark. In these sunken areas, he observed characteristic red bright moist mass of spores which later dried. The infected fruit were observed to ripen prematurely, finally turning black and rotting. In advanced cases the stalk became stunted and dry and leaves drooped and shrivelled up.

Park (1930) reported that the infection may begin at the flower end which becomes black and the disease spreads further to involve the whole fruit which finally becomes black and shrivelled. Sometimes infection takes place from the stalk, infected finger turning black progressively from the point of infection which was found to be very common. The spore masses of G. musarum can be seen on the small, shrivelled fruit. The spore masses were at first moist and bright pink and on drying become a dull light pink. Park (1933) further confirmed his earlier findings and in addition to that reported that the main axis of the bunch was sometimes infected first and the disease may spread from there to immature fruit through stalk end. The diseased fruit turned black from point of attack and then the whole fruit was involved in a short time. The infected fruit finally shrivelled and dried up but remain attached to the central stalk. Some times the whole bunch may be attacked

but more commonly one or two hands are involved.

Parham (1935) observed that the pathogen C. musarum was commonly present on the persistent bracts of Cavendish variety of banana. He also noted that the disease in association with insect injury caused severe rotting and cracking of immature fruit.

In India, the symptoms of anthracnose disease of banana have been described by different workers. The first report of this disease in India was by Dastur (1916). He observed that the disease first appeared on immature fruit of the plant occurring as a distinct black depressed lesions. Later the lesions coalesced and eventually covered the whole fruit resulting in premature ripening and shrivelling of the fruit. Such fruit was covered with pink spore masses of the fungus. Chona (1933) observed the stalk rot symptoms. The rots gradually spread downwards to the fruit. Roy and Sharma (1952) agreed with the symptoms described by the earlier workers. Damodaran and Ramakrishnan (1958), while working on the disease, fully agreed to the symptoms described by Dastur (1916). In addition to that, they also observed, circular specks on the rachis and splitting of the rachis. They also stated that the symptoms are extremely variable and seem to depend on the varieties. Wardlaw (1930) has given a good account of banana fruit diseases. He compiled the symptomatological studies of earlier workers also.

Few workers have described the post harvest symptom of the anthracnose disease (Wardlaw and McGuire, 1931; Wardlaw, 1935; Damodaran and Ramakrishnan, 1958 and Meredith, 1960).

Wardlaw and McGuire (1931) studied the behaviour of the diseases of banana in storage and transport with special reference to chilling, wherein most consignments, the attack of G. musarum was found to be more frequent than other fungi. When the fruit reached maturity in the ripening room after a long storage, the pathogen infected the fruit at the tip of the finger and the perianth decayed and the disease spread slowly backwards from the finger tip. This type of symptom was noticed both on Cavendish and Gros Michel varieties. Later the affected tips became dark brown in colour with gradual transition through a watery looking greenish margin to the normal healthy skin. By that time the flesh with in was slowly infected by the pathogen and the pulp became moist and disintegrated with a watery margin, wherever deeper invasion had taken place. In advanced stage, the brownish black region became covered with brownish acervuli of the pathogen. Artificial inoculation also showed the same symptoms which was observed in natural infection.

Wardlaw (1935) has described the symptomatology of the disease on green banana before placing in cold storage

and after keeping in cold storage. If the organism is inoculated on green bananas, sunken black diseased areas were developed. The same inoculated fruit when kept in cold storage and when fruit approach final maturity, these spots became more conspicuous and consist of more or less superficial brown spots or patches contrasting with the yellow skin. Under a moist atmosphere, these spots or blemished areas became more conspicuous with orange to salmon pink acervuli or conidial masses. When the infected area was dried up, the acervuli appeared as small coral pustules.

Damodaran and Ramakrishnan (1958) also observed browning and blackening of infected harvested fruits. The expression of symptom varied according to the varieties.

Meredith (1960) reported that the anthracnose infection was very negligible on mature Jamaican varieties, Lacatan and Gros Michel. He observed only few lesions at full maturity stage. These lesions were small, more or less circular, light brown with a back ground of yellow skin. He also noted that the infected, matured fruit when kept above 70°F, the lesions increased in diameter and became black or dark brown in colour. Whenever several lesions were observed on one finger, they coalesced extensively. At this stage, the fungus invaded the pulp to a depth of several mm causing watery rot.

Cooke and Massee (Grev. 16. 3, 1873) while describing the G. musarum on the ripe fruit of banana, gave a conidial measurement of the fungus as 10 - 12 x 4 µ. Later Miss Wakefield has noted that the dimensions ranged from 12 - 20 x 5 µ with a mean of about 15 x 5 µ indicating that the original measurements were incorrect (Wardlaw, 1972). But Laubert (1910) found that the conidia of the pathogen on the banana fruit which were imported to Germany ranged from 9 - 24 x 5 - 7 µ and he proposed the name G. musarum var importatum. Krüger (1913) isolated an ascosporic strain from the ripe banana fruit and found the conidia on the fruit measuring 15 - 17.5 x 4.6 - 6.0 µ but in culture they were mostly measured 14 - 18 x 4.6 - 5.7 µ.

Agati (1922) has given a detailed description of the morphological characters of the fungus. According to him, the mycelium grows abundantly on almost all the media tested. It was cottony white growth in the beginning and mycelium was hyaline and septate. Later, it became dark brown and finally turned grey. Numerous acervuli have been observed in the culture as pink concentric rings and the conidia were seldom attached to conidiophores as they separate before they mature. Conidia were borne singly and apically on long branching conidiophores. The conidiophores arose from abundantly packed mycelia. The spores were hyaline, generally elongated, ellipsoid

and rounded at the ends. They were thin walled and granular within. They varied in size and shape from 11.5 - 13.5 /u x 3.5 - 9.49 /u in dimension. The young ones were more rounded than older ones. The germ tube generally arose from one of the ends of the spore. The spores germinated rapidly in a drop of water.

Toro (1922) indicated that this pathogen exists as eleven morphological strains, with different conidial measurements and the colony characters. He also indicated that, these strain variations will lead to range in the pathogenecity. Ashby (1931) studied the pathogen on two varieties of banana namely, Cavendish and Gros Michel showing different type of symptoms and accordingly he categorised them into three groups based on conidial measurement and cultural characters. According to him the conidial measurements varied from 11 - 16 /u x 5 - 7 /u in different groups. He also compared the G. musarum isolates from Trinidad, Ceylon and India and found that all the isolates were similar in growth, conidial production and measurements on Brown's agar media and Oats meal agar.

Jain (1950) studied the pathogen on host itself and found that the hyphae of pathogen were inter and intracellular. He also observed that at the time of acervuli formation, the acervuli accumulated in the

vicinity of epidermal tissue immediately below the cuticular region. The growth of conidiophores pushed cuticle upwards and the acervuli in young stages were held together by delicate mass of hyphae. This mass of hyphae held the spores together. The size of conidia varied from 7.68 - 21.3 x 2.8 - 5.52/u in dimension. Chakravarty (1957) examined the Gros Michel bananas from Jamaica and Cameroons and Cavendish bananas from Canary islands on arrival in Britain. According to her all the isolates are comparable.

Damodaran and Ramakrishnan (1958) studied the cultural characters of the fungus *in vitro* and observed four types of mycelial growth and acervuli formation. They were submerged mycelium without any black stromata, aerial or submerged mycelium with few black stromata, slightly aerial or white crust like submerged mycelium with profuse production of large black stromata and submerged white mycelium with acervuli in concentric rings.

Krüger (1913) reported that the ascospores of the pathogen varied from 13.5 - 20.5 x 4.6 - 8.5/u. All these types of spores were mainly allantoid but he observed few straight spores also which measured 12.5 - 17 x 5 - 6.3/u.

Ashby (1931) had given a detailed morphological description of *G. cingulata*, the perfect stage of

G. musarum. According to him the perithecia measured 120 to 122 /u. Asci measured 60 - 80 x 11 - 13 /u. Ascospores measured 16.5 x 5 /u with a range of 13.5 - 20.5 x 4 - 6 /u.

Wardlaw (1935) also observed perithecia on the G. musarum infected fruit and according to him perithecia are sub spherical and cross aggregated without stroma measuring 100 - 150 /u with beaks 80 - 90 /u x 600 /u at base and 4 to 5 /u at the apex. Asci are ellipsoid with a pedicellate base measuring 50 - 75 x 10 - 13 /u. Paraphyses are absent. Ascospores are allantoid, the curvature being frequently pronounced measuring 14.8 - 23.8 x 4 - 5 /u with a mean of 19.4 x 4.4 /u. He also observed a few straight spored ascospores either equally or unequally convex laterally with obtuse ends measuring 14.4 x 5.0 /u with a range of 12.5 - 17 x 5 - 6 /u. They are hyaline, continuous and granular with a clear spot at the centre with equally rounded ends. The observation on the size of perithecia noted by Hoette (1935) also confirmed the earlier findings of Wardlaw.

Few workers have made an attempt to find out the relative resistance and susceptibility of different banana varieties. Agati (1922) conducted a survey and found that the sweet varieties of banana were most susceptible to infection by G. musarum, than the culinary varieties. He also reported that sweet varieties

required six to ten days for infection and less sweet varieties which required 15 to 23 days. Similar observation was noted by Toro (1922) who found that the fruit of sweet varieties or Dwarf bananas (M. cavendishii Lamb) were more susceptible than those of plantains (M. paradisiaca L). Rios (1931) supported the earlier findings and stated that M. cavendishii was the most susceptible variety to G. musarum. Wardlaw and McGuire (1931) observed that the finger tip disease caused by Gloeosporium sp. was more common on both Cavendish and Gros Michel varieties.

Park (1933) reported that under field conditions the variety Ash plantain was more susceptible to the anthracnose pathogen. Parham (1938) stated that Blue Java bananas were highly susceptible to the anthracnose disease and even immature bunches were destroyed in the field. Simmonds and Mitchell (1940) also suggested that Cavendish variety was more susceptible to anthracnose than the other varieties tested.

Roy and Sharma (1952) found that all table varieties were susceptible to G. musarum in Punjab and Bihar region of India. They also reported that the varieties Kothia and Muthia were fairly resistant. Damodaran and Ramakrishnan (1964) conducted a survey and according to them, all commercial banana varieties were found to be

susceptible to C. [redacted] in Tamil Nadu. Meredith (1970) reported that most of the 'Cavendish' clones are more susceptible to C. [redacted] infection than the variety Gros Michel. Shilling Ford and Sinclair (1977) screened five banana cultivars against anthracnose disease and they found that Valery was highly susceptible while Lacatan, Robusta and Dwarf Cavendish were susceptible. The tetraploid banana, Coded 65-3405-1 was found to be resistant.

The infection caused by C. gloeosporioides is mainly of two types. They are the infections occurring on immature fruit in the field and the infections occurring after harvest. Generally the young fruit are infected in the field, the infection remaining latent until favourable conditions for its development are brought by full maturity and ripening of the fruit.

Shear and Wood (1913) have given a detailed description of latent infection of anthracnose of banana. According to them when the conidia germinate on the surface of fruit, thick walled bodies, the appressoria are formed and at first germ tube only penetrates a short distance and does apparently a little damage to host cells. Thus the pathogen becomes inactive and remain so until favourable conditions for further development are provided by the maturation of the fruit.

Dastur (1916) also observed the latent infection in the field which remained latent till favourable conditions for their development occurred (Maturity of the fruit). Baker and Wardlaw (1937) also noted the latent infection in the field and according to them, this infection could extend for a long period. This observation was further confirmed by Wardlaw, Baker and Crowdy (1939).

While working on latent infections of tropical fruit in Queensland, Simmonds (1941) observed that the species of Gloeosporium and Colletotrichum infect the young banana fruit and that can remain in latent stage of infection for 5½ months. When the fruit attained maturity, the fungus resumed activity to produce typical anthracnose lesions in the ripened stage. This observation was further supported by Veitch (1941).

Jain (1950) noted that the fungus G. musarum could directly penetrate the immature banana fruit tissue. Chakravarty (1957) reported that G. musarum penetrates the cuticle of green banana and remains in inactive condition in sub cuticular region until fruit ripening. Later it resumes activity and produces typical anthracnose lesions.

Meredith (1960) reported that the primary source of inoculum for the fruit infection is from banana leaves

and the spores reach the surface of the fruit, germinate and form appressoria in the course of 72 hours. The appressoria were abundant on the inner surface of fruit near the finger stalk. Cardenosa-Barriga (1964) observed that the infection of immature fruit and flowers is by sundry wounds and stigma. Meredith (1964) reported that bananas grown in humid tropics will have latent infections of C. gloeosporioides in the peel at harvest. The infection by this fungus may take place at any time in the development of the fruit when free water on the surface of the fruit permits spore germination and penetration to take place. Murhead and Deveralls (1981) while working on the role of appressoria on banana fruit infection by C. musae, observed that the dark appressoria remain dormant on unripe fruit and during ripening it put forth the germination peg.

Although the pathogen generally infects the immature fruit in the field as latent infections, the greater development of fruit rot by C. gloeosporioides become evident in ripening room as final maturity is attained. Apart from this type of infection there are reports of getting infection after harvest through the injuries occurred during the harvest, transport and storage.

Wardlaw (1972) stated that this type of post harvest infection is mainly due to injuries occurred after the harvest.

Baker and Wardlaw (1937) and Baker (1938) reported that due to contamination occurred during transit and from packing sheds and ripening rooms, the anthracnose disease incidence will be aggravated. Green and Goos (1963) observed that the water used for washing the banana bunches after harvest is also a source of inoculum for fruit rot fungi including G. musarum and stated that this pathogen can enter the host tissues to a depth of five to seven mm and this deep seated infection will be difficult to eradicate by fungicidal treatment. Green (1966) stated that the propogules of the parasitic fungi causing banana fruit rot including G. musarum are abundant in the atmosphere and they fall on surface of harvested fruit and enter through the stomata or through the lenticels.

Meredith (1970) had given a detailed review on major banana diseases. In his review he stated that the banana fruit suffered varying amount of mechanical injury resulting in varying degree of fruit rot caused by many fungi including G. musae. Later Meredith (1971) further confirmed the above findings.

The factors which influence the infection process of the anthracnose pathogen include the climatic conditions, presence of injuries on fruit, state of maturity and

chemical constituents of the fruit.

Agati (1922) reported that wet weather is a very important factor for development of the symptoms in the field and damp conditions for development of symptoms during storage. During rainy days, the inoculum developed rapidly resulting in high percentage of field infections. Park (1930) also stated that in wet weather, plantain bunches were often affected by Gloeosporium fruit rot which makes its appearance soon after the fruit has set.

Since C. gloeosporioides is a weak parasite, the importance of wounding had been stressed by several workers for initiating infection. Tero (1922) stated that the fungus cannot enter the fully matured or partly matured green fruit without injury under field conditions even on most susceptible varieties. Park (1930) also observed that the fungus is capable of causing the disease only after wounding the fruit.

Simmonds and Mitchell (1940) had taken the ability of the fungus to infect injured and uninjured fruit into consideration for varietal resistance and susceptibility. They observed that the varietal difference in susceptibility cannot be judged if the fruit are inoculated after the injury but there is a variation in the susceptibility when the uninjured fruit were inoculated. On this basis they stated that the Veimama variety is less susceptible or more resistant than the Cavendish varieties.

The state of maturity of the fruit and its level of chemical constituents are the most important factors governing the infection. Toro (1922) stated that the completely matured fruit when placed under proper conditions were easily infected whether injured or not when compared to immature or partly green fruit. Wardlaw (1931) also reported that fungal penetration remained very limited in less matured fruit until normal saccharification has reached. Deighton (1935) also proved that G. musarum readily infected the ripe fruit when inoculations were carried out in vitro on half grown and ripe fruit.

The infection by the pathogen is high in fully mature and ripe stages, than in immature green fruit. The reason attributed to this phenomenon is mainly due to changes in chemical constituents of banana fruit in different stages of maturity and ripening. Several workers worked in this line viz., Belevel (1932); Leonard and Barnell (1939); Wardlaw and Leonard (1940); Barnell and Barnell (1945); Loesecke (1950); Yang and Ho (1952); Goldstein and Swain (1963); Simmonds (1966); Lodh et al. (1971) and Singh et al. (1980).

Belevel (1932) reported that there were two distinct periods in the growth of the fruit. The first period was that of starch reserve in the course of which the fruit always low in soluble sugar fixed its starchy

reserve at the expense of the reducing sugars. The next period was one of maturation and here soluble sugars were formed from part of starch which after hydrolysis yields invert sugars. Leonard and Barnell (1939) stated that during the development of the banana bunch, sugars remained at a very low concentration while starch rapidly accumulated.

Wardlaw and Leonard (1940) have studied the carbohydrate changes taking place during ripening. The predominant carbohydrate of green banana is starch, which is very largely replaced by sucrose, glucose and fructose during ripening. Loesecke (1950) also observed that the most important and conspicuous change in the maturation of the banana is the conversion of starch to sugars. Young and Ho (1958) studied the changes in carbohydrate metabolism and respiratory mechanism during the course of ripening which indicated the existence of a transition stage between maturation and senescence in which there is a marked and sudden rise of respiration accompanied by physiological and chemical changes. Starch is converted into various sugars like sucrose, glucose and fructose.

Simmonds (1966) summarised the earlier works and reported that sugars were present in green fruit only in very small amount averaging about one to two per cent of the fresh pulp and they increased to 15 to 20 per cent at ripening. Starch disappears concurrently dropping from about 20 per cent in the green fruit to about one to two

per cent in the ripe fruit, it being higher in the ripe culinary bananas (about six per cent) than in dessert bananas. Lodh *et al.* (1971) observed that the total sugars were low until 100 days after shooting of the bunches and increased markedly after harvesting in Dwarf Cavendish banana pulp and starch concentration declined during ripening. Based on the biochemical studies on the developing and ripening bananas, Singh *et al.* (1980) stated that a linear increase in starch content occurs from immature to mature stage. It was reduced to 40 per cent in ripening which indicates a rapid change in carbohydrate metabolism during ripening.

Barnell and Barnell (1945) estimated a tannin fraction in banana fruit. They found that tannin is responsible for the astringency of the unripe fruit. They also observed that this fraction reduced in the ripe fruit to about one fifth of its value in green hard fruit of banana. This tannin content was three to five times more in the peel than in the pulp and also fell sharply during ripening. Goldstein and Swain (1963) have presented preliminary evidences to show that the loss of astringency took place during ripening of bananas due to polymerisation of polyphenols.

Many workers have attempted to find out the resistance and susceptibility of banana against anthracnose disease on the basis of chemical constituents of fruits. Most of them correlated this aspect with sugar and tannin. Agati (1922) stated that the sweet varieties readily succumbed to disease

whereas low sweet varieties showed a slight degree of resistance. This finding was supported by Toro (1922) who found the fruit of Dwarf Cavendish bananas to be more susceptible. He correlated this observation with changes in physical and chemical composition of peel including sugar content and softening of tissues. The extent of infection therefore appears to depend on the degree of saccharification reached. Wardlaw (1931) reported that fungal penetration remained very limited in less mature fruit until normal saccharification is obtained. Damodaran and Ramakrishnan (1964) found that the reducing sugar content of the fruit is positively correlated with incubation period and diameter of lesions caused by G. musarum infection. Thakur (1969) noted in different banana varieties that a higher percentage of total sugars in the peel increased susceptibility to G. musarum whereas low sugar content decreased the susceptibility.

Cook and Taubenhaus (1911) found that the germination of conidia of G. musarum was cent per cent in 0.6 per cent tannin solution in the course of 24 hours, but failed to germinate in higher concentrations. Chakravarty (1957) reported that the green skin of juice of banana fruit has an inhibitory effect on the germination of conidia of G. musarum, possibly due to the presence of tannin which disappears to some extent as the fruit ripens. Green and Morales (1967) observed that tannins from green banana

fruit latex inhibited the activity of β-amylase produced by G. musarum resulting no spread of the fungus. Reghunathan et al. (1966) reported that resistant leaves of banana to G. musarum infection possessed more phenols than the tissues of the susceptible varieties. However, Toro (1922) attempted to correlate the tannin content of the peel with resistance to infection by G. musarum but obtained negative results. He, therefore concluded that resistance and susceptibility are probably correlated with anatomical characters.

Sugars and tannin were correlated with fruit diseases caused by G. cingulata in other fruit crops also.

Sitterly and Shay (1960) observed that lack of critical concentration of sugars such as sucrose or fructose is involved in immature apple (Pyrus malus L) fruit resistance to fungi including G. cingulata. High amounts of sugar were prerequisite for fungal growth in development of apple fruit rot including G. gloeosporioides. They also showed that tannin content in apple imparts resistance to infection in immature fruit only by virtue of its relatively high concentration as related to the low available carbohydrate concentration. Stretch and Cappellani (1965) reported that in high bush blue berry fruit, decay by G. cingulata was inhibited in green immature fruit due to lower concentrations of sugar. They concluded that

the fungus require certain amounts of sugars in the fruit to produce the enzymes for degrading the tissues.

Mohanraj et al. (1972) observed that the susceptible tissues of leaves of various grape vine (Vitis vinifera L) varieties to the anthracnose disease (Gloeosporium ampelophagum Sacc) contained more sugars. They also pointed out the anthracnose disease to be a high sugar disease.

Materials and Methods

MATERIALS AND METHODS

Location of the experiment

The laboratory studies connected with the 'varietal screening of banana against anthracnose disease' were conducted at the Department of Plant Pathology, College of Horticulture, Vellanikkara, Trichur. The field studies were conducted at the Banana Research Station, Kannara, Trichur.

Materials used

Twenty-five banana varieties were grown under uniform conditions of growth in the field as per the package of practice recommendations (Anon, 1978). The banana fruit of twenty-five varieties at different stages were detached and brought to laboratory for conducting inoculation studies. The list of varieties is presented in Table 1. Each variety was inoculated at different stages of maturity, namely, immediately after the female phase, one-fourth maturity, half maturity, three-fourth maturity and full maturity. The stages of maturity were determined based on the number of days the bunch takes to mature fully after shooting.

Isolation and purification of fungus

The fungus causing the anthracnose disease of banana was isolated from the infected parts of the banana fruit of the variety, Robusta using standard isolation techniques as described by Riker and Riker (1936). Pure culture of the pathogen was maintained in the potato dextrose agar medium and used for *in vitro* studies.

All the microscopical studies of the pathogen - measurements and drawings were done using Leitz Orthoplan research microscope and its drawing apparatus under the maximum possible magnification. All the taxonomical descriptions of the pathogen have been done either from the infected fruit or from the pure culture maintained in potato dextrose agar medium in room temperature.

Artificial inoculation experiments

The fruit were disinfected with mercuric chloride solution (1:1000) and rinsed with three changes of sterile water and then smeared with 95 per cent ethyl alcohol. Inoculations were carried out both with pinprick injuries and without pinprick injuries on surface of the fruit. Pricks were made on the fruit with a sterile sharp needle. The spore suspensions were made in sterile water. The density of the suspension was adjusted in such a way that the spore density was eight to ten spores in a low power field of the microscope in all the inoculations.

The fruit were then inoculated by placing a drop of spore suspension at one-fourth and three-fourth length of the fruit. The inoculated fruit were kept under the bell jar and humidity was maintained by placing sterile wet cotton wool in the side and top of the bell jar and by spraying sterile water once in 24 hours. Four replications were maintained. Sufficient number of control fruit were also kept. The length and breadth of the lesions were recorded at an interval of forty-eight hours.

The percentage intensity of the disease was worked out from the formula.

Percentage intensity of the disease =

$$\frac{\text{Area of the lesion}}{\text{Total area of the fruit}} \times 100$$

For the purpose of calculating percentage of infection, the area of the lesion on a particular day after inoculation was used for all the varieties throughout the experiment. The inoculated fruit were kept for ten days for observation except in the case of full maturity where the inoculated fruit were kept only for eight days as the fruit started complete decaying after eight days.

Symptomatology

The symptom development on banana fruit of 25 varieties mentioned in Table 1 was studied in field itself. The

post harvest symptoms were also studied.

Symptom development after artificial inoculation *in vitro* was also studied in detail from initial stage onwards.

Screening of banana varieties against the disease

For separating the relative susceptibility and resistance of banana varieties, the following criteria (Score card) have been used. The mean percentage of disease intensity of all the stages of fruit development has been taken into consideration and classified as follows:

Above 25%	-	Highly susceptible
Above 15 upto 25%	-	Susceptible
Above 10 upto 15%	-	Moderately susceptible
Above 5 upto 10%	-	Moderately resistant
Above 2 upto 5%	-	Resistant
Below 2%	-	Highly resistant

Biochemical studies

The biochemical constituents of banana fruit namely reducing sugar, total sugar, starch, crude fibre, crude protein and tannin were estimated at different stages of fruit development viz., immediately after female phase, one-fourth maturity, half maturity, three-fourth maturity

and full maturity. The values are expressed as percentages on moisture free basis. At each stage, four replications were taken for analysis. The fruits were dried in hot air oven at 85°C, powdered well and then analysed.

Reducing sugar

The reducing sugars of the sample using Fehling A and B solutions was determined as per the method described in AOAC (1960).

Total sugars

The total sugars were estimated after inversion of the sample at room temperature and using Fehling A and B solutions as per the method given by AOAC (1960).

Starch

The total carbohydrate content of the sample was first estimated using Fehling solutions A and B as described in AOAC (1960). From total carbohydrate content, the total sugar content was subtracted to get the starch content.

Crude fibre

The fat free material of the samples was dried in oven at 105°C and then ignited in a muffle furnace at

600°C to get crude fibre content and was expressed as percentage on moisture free basis as mentioned by AOAC (1960).

Crude protein

Total nitrogen percentage of the sample was estimated using Kjeldahl digestion and distillation method and was multiplied by the factor 6.25 to get the crude protein content as mentioned by AOAC (1960).

Tannin

Colorimetric method of tannin estimation using Follin-Denis reagent as described by Ranganna (1977) was made use of and the value was expressed as percentage.

Statistical analysis

All the data obtained were analysed statistically by using completely randomised design. All the data were transformed to angles by using inverse sin transformation given by $Q = \text{Sin}^{-1} \frac{1}{\sqrt{P}}$ Where Q is the angle corresponding to per cent P. Wherever there were large number of zero values, the data had been transformed by using $\sqrt{x + 1}$ transformation.

The relative susceptibility and resistance of different varieties of banana were analysed in all the

four stages of development of fruit and to know comparative relative susceptibility and resistance of the different varieties, pooled analysis of different stages of the fruit had been made.

Coefficients of correlation between percentages of disease intensity and each of chemical constituents under study were worked out.

Table 1. Banana varieties used for screening against Anthracnose disease

Sl.no.	Name of variety
1.	Robusta
2.	Dwarf Cavendish
3.	Palayankodan
4.	Njali poovan
5.	Gros Michel
6.	Koduppilla kunnan
7.	Poocha kunnan
8.	Adakka kunnan
9.	Red banana
10.	Pisang lilin
11.	Chinia
12.	Matti
13.	Bodles Altafort
14.	Kanchikela
15.	Nendra padaththi
16.	Boodida bontha batheea
17.	Hybrid sawai
18.	Peyan
19.	Kapok
20.	Pisang mas
21.	Vennaettu mannan
22.	Klue teparod
23.	Jurmani kunthali
24.	Nendran
25.	Zanzibar

RESULTS

1. Symptomatology

The disease has been observed in the field and in the harvested fruit. The general symptom in both the cases is that of typical anthracnose.

1.1. Field symptoms

The pathogen was found to attack the young immature banana fruits in the field. The infection usually takes place at the distal end of the banana fruit and spreads further downwards. Rarely, the infection was also noticed on the skin of the banana fruit where the initial symptoms were chlorotic specks, shortly becoming black circular necrotic area. The necrotic areas increased in size and later became sunken and coalesced forming larger patches. In severe infections the fruit may be entirely covered with dark blemishes. Under humid condition, on the surface of the black spots the characteristic bright salmon coloured conidial masses appeared. The infected fruits became blackened, shrivelled and mummified.

Usually the attack by the pathogen was restricted to a few hands only but in certain cases it affected the whole bunch also (Plate I & II).

Plate I. Symptoms of anthracnose disease on immature banana fruit in the field (partially infected bunch)

Plate II. Symptoms of anthracnose disease on immature banana fruit in the field (fully infected bunch)

1.2. Post harvest symptoms

Symptoms appeared as small irregularly round, brown coloured spots contrasting with yellowing skin. These brown spots enlarged quickly and became dark brown to black in colour. These spots coalesced together and large patches were formed and this completely covered the fruit within a few days (Plate XII). In a moist atmosphere, the affected area was covered with orange to salmon pink conidial masses and on drying these affected areas were very rough and the pulp of the fruit at this stage exhibited a soft rot symptom.

Symptom development on artificial inoculation

On artificial inoculation, the symptoms at three-fourth and full maturity were the same as described in post harvest symptom development. In one-fourth and half maturity, the infection was mainly due to injury. The development of initial symptom was very slow. The infected area became irregularly dark black coloured spots. These spots further enlarged all around and the development of the spots was very slow when compared to the full and three-fourth maturity stages. In advanced cases, most of the spots were sunken with a rough surface.

2. Morphology and cultural characters

When sections of the infected fruit were examined,

Plate III. Symptoms of anthracnose disease on fully matured fruit after harvest

fructification of the pathogen was observed on the necrotic area. The pathogen was isolated, purified and monosporic cultures were made as described in materials and methods.

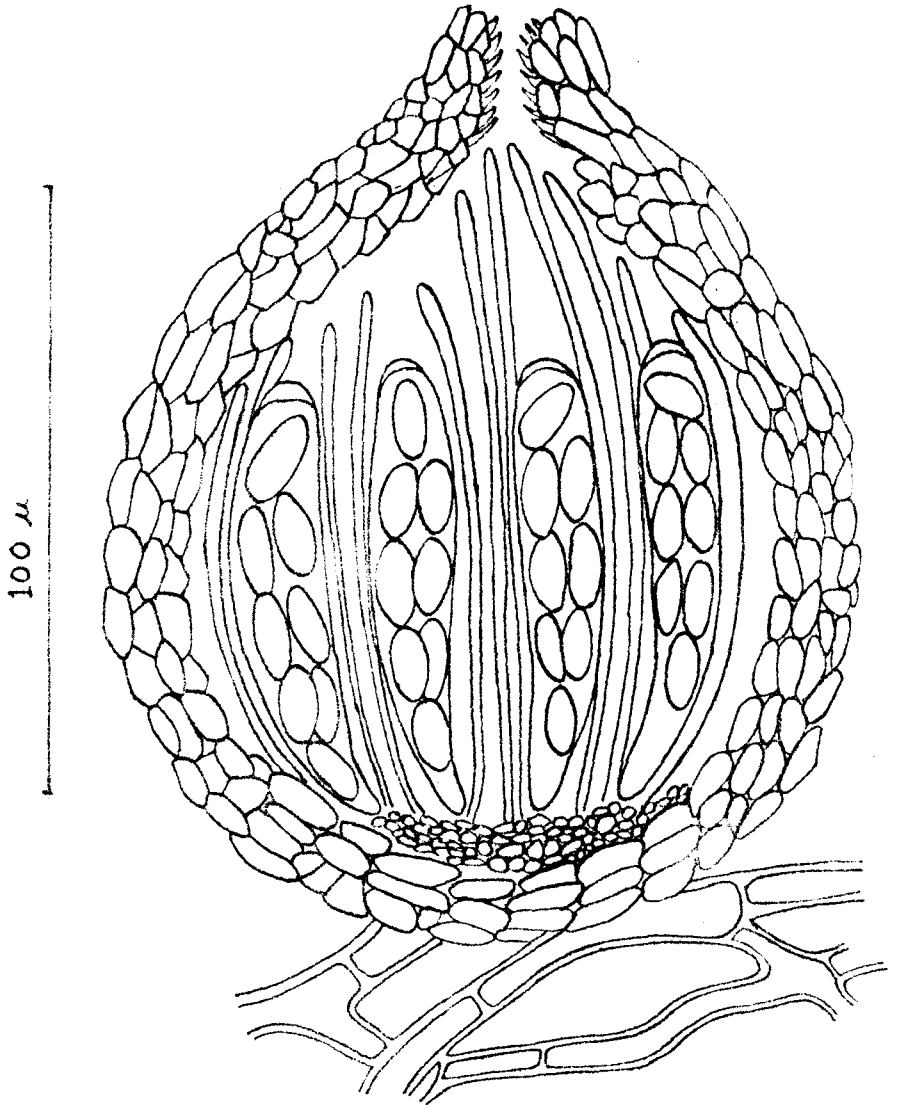
On potato dextrose agar, the growth was luxuriant with extensive mycelial development. At first, the colonies were olive grey with white, young mycelial growth at the edges. Later the entire colony became dark. The aerial mycelia formed felted mat, often with diurnal zonations.

Perithecia in young cultures were rare but abundant in fifteen day old cultures. The ascocarp was aggregated, globose, obpyriform, dark brown to black 90-370 /u diameter; wall upto six cell thick with pseudoparenchymatic cells, ostiolate, slightly papillate and circular, often with a lining of paraphyses inside the ostiolar canal (Figure 1).

Numerous, well matured perithecia were observed in twenty day old cultures. Asci were formed from the hyaline, pseudoparenchymatic cells at the base of the perithecium. They were unitunicate, eight spored, clavate to cylindrical, thickened at the apex and narrowed at the base, 40 - 80 /u long, 7 - 14 /u broad, interspersed with paraphysis (Figure 2B). Ascospores are biserially arranged, oval to cylindrical, some times slightly curved, unicellular, faintly coloured in old cultures, 9 - 21 /u long and 4 - 8 /u broad (Figure 2A).

FIG 1

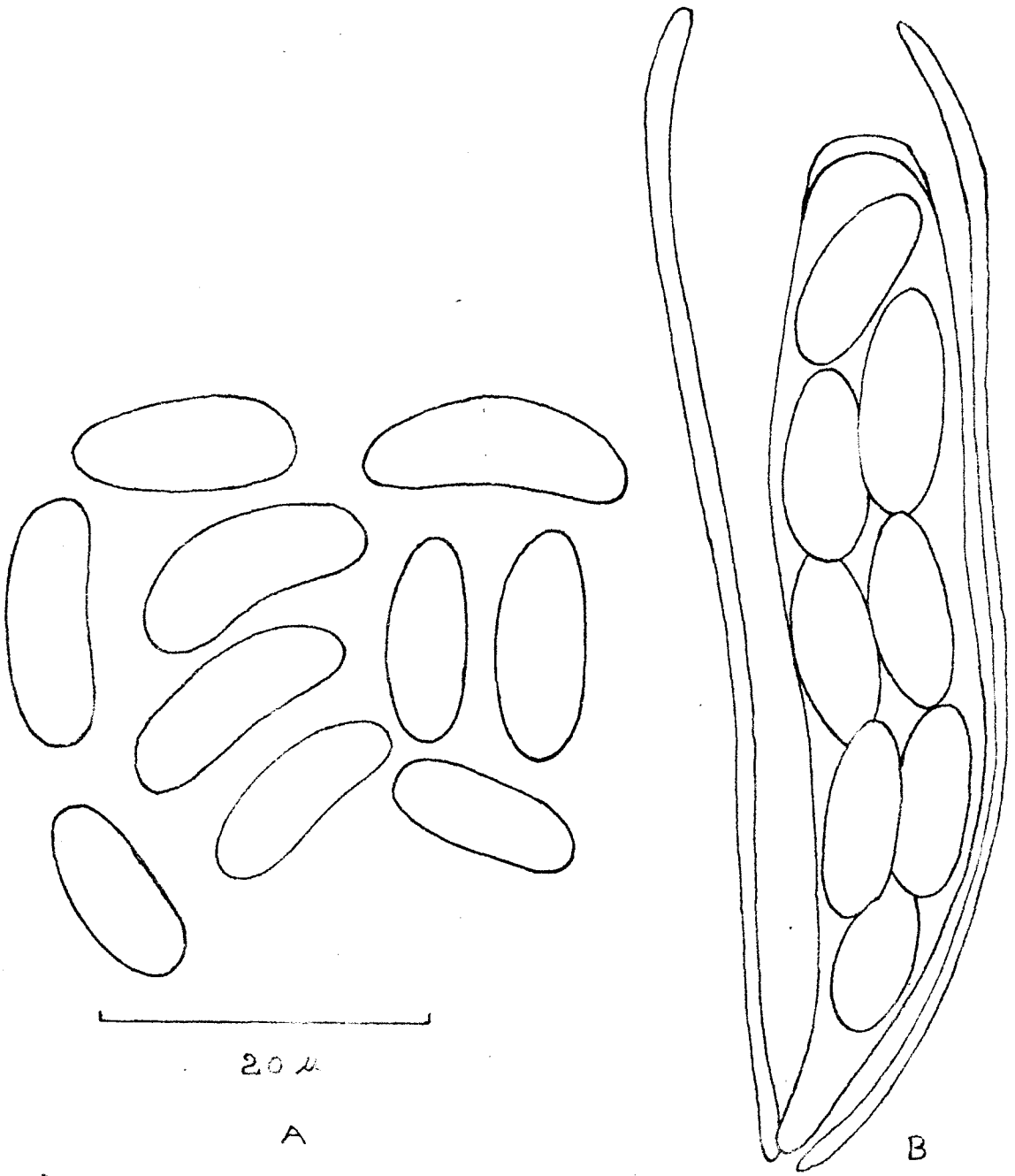
Glomerella cingulata, the perfect stage of **Colletotrichum gloeosporioides**



T.S. of perithecium

FIG 2

Glomerella cingulata, the perfect stage of Colletotrichum gloeosporioides



A) Ascospores

B) Ascus with paraphysis

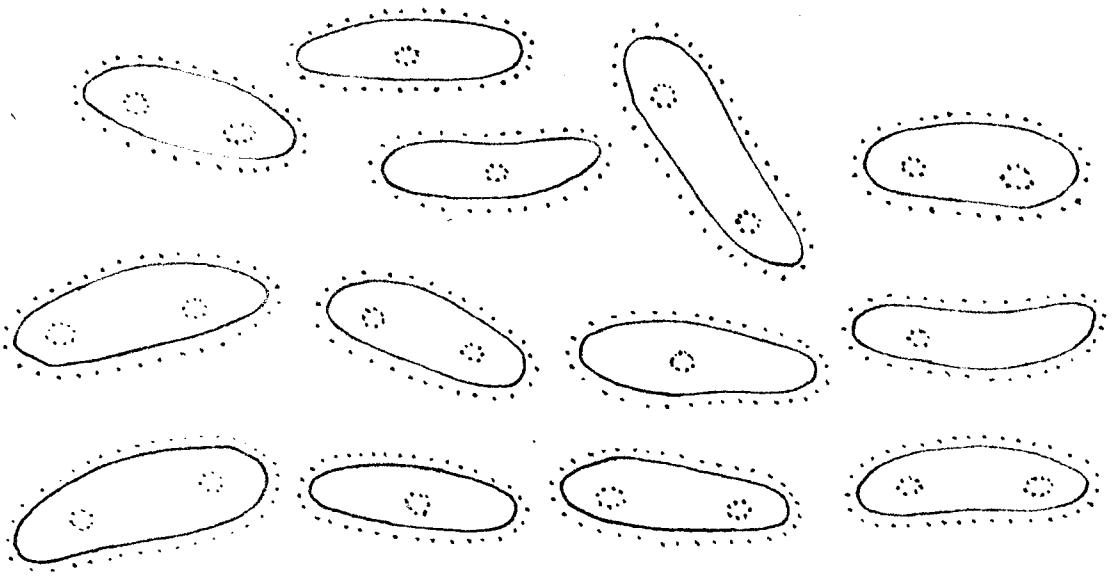
A good number of circular, longitudinal, irregular acervuli were observed on infected fruits and in cultures but setose acervuli were not observed. In cultures, salmon pink coloured spore masses were observed on acervuli beneath the olive green hyphal mat. Each acervulus measured 40 - 120 /u across.

Conidiogenous cells were phialidic, hyaline and fasciculate in acervuli. Solitary phialids on loose hyphae were very common in slide cultures. These were aseptate, cylindrical to obclavate and 8 - 14 /u x 3 - 4 /u in acervuli. Solitary phialids on loose hyphae measured upto 50 /u long (Figure 3 B). Conidia were enteroblastic, produced on the apex of phialidic conidiogenous cells, hyaline, cylindrical, oblong to elliptical with both ends obtuse, base truncate, unicellular, smooth walled with mucilaginous coating, 1 - 2 gutilate, usually 10.5 - 17.5 x 4.5 - 7.0 /u often becoming hyaline or faintly brown and uniseptate prior to germination (Figure 3A).

In the slide cultures, appressoria were abundant both in vegetative mycelia and on the apices of short hyphae of the germinating spores. They were formed as simple, expanded ends of hyphae which were separated by transverse septa. The whole structure was cut off and slowly thickened. The appressoria from the vegetative hyphae were larger and darker in colour when compared to appressoria formed from

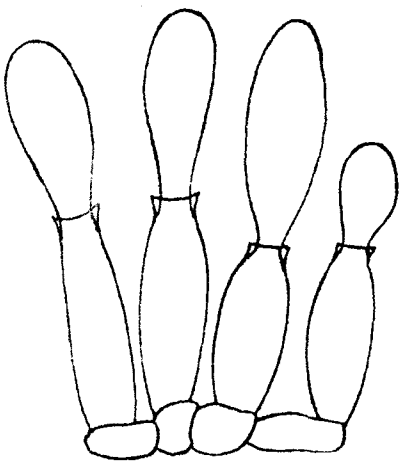
FIG 3

Colletotrichum gloeosporioides

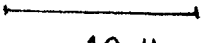


A

20 μ



B



10 μ

A) Conidia

B) Developing conidia and conidiogenous cell

germ tubes. The mature appressoria were generally terminal, very rarely intercalary, aseptate, irregular with lobes, with one or more germ pores, wall smooth, thick, dark coloured and measured 12 - 25 /u x 8 - 18 /u. Appressoria from the germinating conidia were smaller, elephant skin coloured and measured 6 - 12 /u x 3 - 8 /u (Fig. 4A and B).

3. Screening of banana varieties (in vitro) against the anthracnose disease caused by C. gloeosporioides

Twenty-five banana varieties were screened in vitro against C. gloeosporioides, the imperfect stage of C. cinculata as mentioned in materials and methods. The screening was carried out at five developmental stages of the banana fruit starting from immediately after female phase to full maturity. The inoculations were made on the fruit with and without injury. None of the fruit kept as control took infection in this experiment. The details of the inoculation experiments are given below.

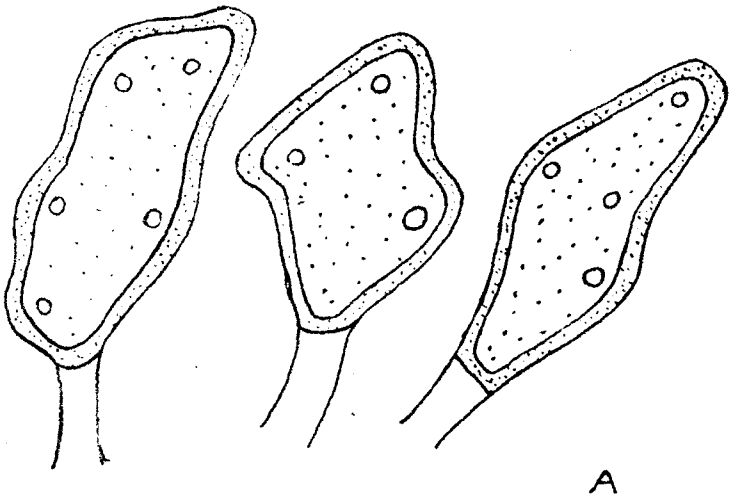
Inoculation with C. gloeosporioides on banana fruit

3.1. Immediately after the female phase

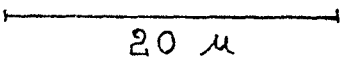
No symptom had developed on the fruit of all the varieties tested, when inoculated with the pathogen both on injured and uninjured fruit. The results indicated that none of the varieties tested during this stage was susceptible to the pathogen.

Fig 4.

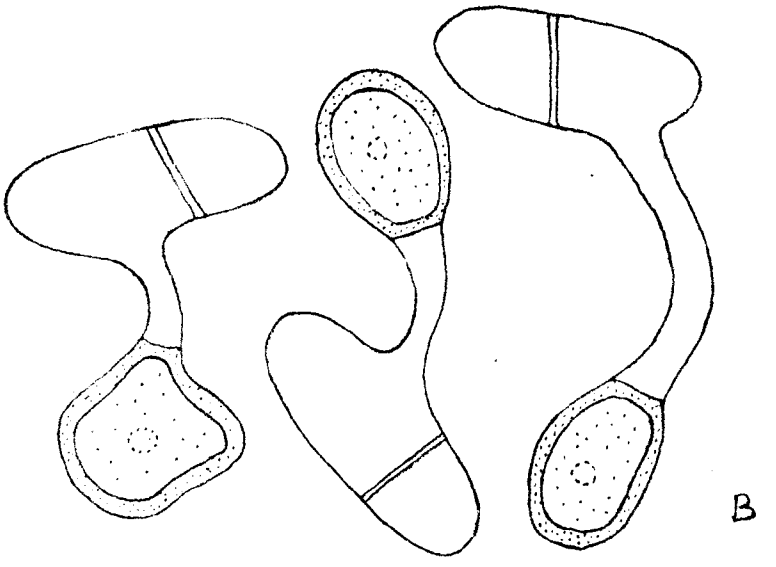
Colletotrichum gloeosporioides



A



20 μ



B

A) Appressoria of vegetative hyphae
B) Appressoria development of germinating conidia

3.2. One-fourth maturity

All the varieties tested at this stage showed a negative reaction when the pathogen was inoculated on unwounded fruit . However, twelve varieties expressed symptoms of the disease when inoculated on the wounded fruit . Even in this case, the symptom development was very slow and took minimum six days to get the visible necrotic area on the fruit . The percentage of infection on these twelve varieties varied from 0.03 per cent to 1.56 per cent. Among the twelve varieties which took infection, the maximum infection was observed in Koduppilla kunnan (1.56 per cent) followed by Poocha kunnan (0.85 per cent) and the minimum was observed in Bodles Altafort (0.03 per cent) followed by Red banana (0.05 per cent) (Table 2).

The statistical analysis revealed that there was significant difference among the varieties. When the varieties were ranked and arranged in descending order of susceptibility, Koduppilla kunnan was found to take maximum infection followed by Poocha kunnan, Adakka kunnan, Kapok, Chinia, Venneettu mannan and Hybrid sawai. The variety Pisang mas was found to be on par with Hybrid sawai. Other four varieties also took infection at this stage namely Dwarf Cavendish, Zanzibar, Red banana and Bodles Altafort.

Table 2. Disease intensity of banana varieties at one-fourth maturity

Sl. no.	Variety	Per cent intensity of disease (injured)
1.	Robusta	0.00 (1.00)
2.	Dwarf Cavendish	0.19 (1.09)
3.	Palayankodan	0.00 (1.00)
4.	Njali poovan	0.00 (1.00)
5.	Gros Michel	0.00 (1.00)
6.	Koduppilla kunnan	1.56 (1.60)
7.	Pooche kunnan	0.85 (1.36)
8.	Adakka kunnan	0.79 (1.34)
9.	Red banana	0.05 (1.02)
10.	Pisang lilin	0.00 (1.00)
11.	Chinia	0.43 (1.20)
12.	Matti	0.00 (1.00)
13.	Bodles Altafort	0.03 (1.01)
14.	Kanchikela	0.00 (1.00)
15.	Nendra padaththi	0.00 (1.00)
16.	Boodida bontha bathees	0.00 (1.00)
17.	Hybrid sewai	0.34 (1.16)
18.	Poyan	0.00 (1.00)
19.	Kapok	0.47 (1.21)
20.	Pisang mas	0.34 (1.16)
21.	Vennesttu mannan	0.40 (1.18)
22.	Klue teparod	0.00 (1.00)
23.	Jurmani kunthali	0.00 (1.00)
24.	Nendran	0.00 (1.00)
25.	Zanzibar	0.10 (1.05)

(Figures given in parenthesis are the $\sqrt{x + 1}$ transformed values)

CD (0.05) between varieties = 0.0099

Conclusions:

Comparison of varieties

6 7 8 19 11 21 17 20 2 25 9 13

1 3 4 5 10 12 14 15 16 18 22 23 24

3.3. Half maturity stage

When the pathogen was inoculated on the fruit at this stage, the infection was observed only on the injured fruit. However, among the varieties tried with injury, only fourteen varieties have taken infection and the rest eleven varieties were found to be free from the disease.

Among the susceptible fourteen varieties, the range of infection varied from 0.05 per cent to 1.22 per cent. Here, the maximum infection was observed on Poocha kunnan (1.22 per cent) followed by Koduppilla kunnan (0.97 per cent) and the minimum was on Zanzibar (0.05 per cent) followed by Nendra padaththi (0.06 per cent).

There was significant difference among the varieties which were susceptible at this stage. The maximum infection was noticed on Poocha kunnan followed by Koduppilla kunnan, Adakka kunnan, Venneettu mannan and Kapok. The variety Red banana was on par with Kapok. Variety Chinia was on par with Red banana while Gros Michel was on par with Chinia. Variety Pisang mas and Dwarf Cavendish were on par followed by Hybrid sawai. Bodles Altafort was on par with Nendra padaththi while Zanzibar was on par with Nendra padaththi (Table 3).

3.4. Three-fourth maturity

At this stage, the varieties tested had shown different degrees of infection when inoculated with and without injury.

Table 3. Disease intensity of banana varieties at half maturity

Sl. no.	Variety	Per cent intensity of disease (injured)
1.	Robusta	0.00 (1.00)
2.	Dwarf Cavendish	0.31 (1.14)
3.	Palayankodan	0.00 (1.00)
4.	Njali poovan	0.00 (1.00)
5.	Gros Michel	0.46 (1.21)
6.	Koduppilla kunnan	0.97 (1.40)
7.	Poocha kunnan	1.22 (1.48)
8.	Adakka kunnan	0.80 (1.34)
9.	Red banana	0.56 (1.24)
10.	Pisang lilin	0.00 (1.00)
11.	Chinia	0.50 (1.22)
12.	Matti	0.00 (1.00)
13.	Bodles Altafort	0.10 (1.05)
14.	Kanchikela	0.00 (1.00)
15.	Nendra padaththi	0.06 (1.03)
16.	Boodida bontha bathees	0.00 (1.00)
17.	Hybrid sawai	0.31 (1.14)
18.	Poyan	0.00 (1.00)
19.	Kapok	0.57 (1.25)
20.	Pisang mas	0.34 (1.16)
21.	Vennaettu mannan	0.67 (1.29)
22.	Klue teparod	0.00 (1.00)
23.	Jumani kunthali	0.00 (1.00)
24.	Nendran	0.00 (1.00)
25.	Zanzibar	0.05 (1.02)

(Figures given in parenthesis are the $\sqrt{x+1}$ transformed values)

CD (0.05) between varieties = 0.0227

Conclusion:

Comparison of varieties

7 6 8 21 19 9 11 5 20 2 17 13 15 25

1 3 4 10 12 14 16 18 22 23 24

In the case of injured fruit, the symptom expression was a bit early and took four days while the uninjured fruit took six days to exhibit the symptoms.

When the fruit were inoculated without injury, ten varieties viz., Robusta, Palayankodan, Kanchikela, Nendra padaththi, Boodida bantha bathees, Peyan, Kapok, Pisang mas, Klue teparod and Jurmani kunthali did not take infection. Out of the fifteen varieties which took infection, Dwarf Cavendish, Njali poovan, Gros Michel, Pisang lilin and Matti showed cent per cent infection after ten days of inoculation. Other ten varieties having very low infection rates namely Bodles Altafort, Hybrid sawai, Red banana, Zanzibar, Koduppilla kunnan, Vanneettu mannan, Adakka kunnan, Chinia, Poocha kunnan and Nendran were found to be less susceptible and infection rating ranged from 0.27 to 5.92 per cent.

When the fruit were injured and inoculated at this stage, only two varieties viz., Palayankodan and Jurmani kunthali did not take any infection. All other varieties showed different degrees of infection. Of these, six varieties viz., Robusta, Dwarf Cavendish, Njali poovan, Gros Michel, Pisang lilin and Matti were found to be highly susceptible and showed cent per cent infection within ten days. The varieties, Hybrid sawai and Red banana were found to be moderately susceptible with values 20.21 and 13.04 per cent respectively. Other eleven

varieties namely, Pisang mas, Zanzibar, Kapok, Poocha kunnan, Klue teparod, Nendran, Venneettu mannan, Chinia, Adakka kunnan, Koduppilla kunnan and Bodles Altafort were found to be less susceptible and infection ranged from 3.06 to 9.93 per cent. The varieties Nendra padaththi, Boodida bontha batheea, Kanchikela and Peyan showed high degrees of resistance and infection percentages recorded were 0.11, 0.15, 0.17 and 0.22 respectively, even after ten days of inoculation (Table 4).

The statistical analysis revealed that there was significant difference within the treatment receiving inoculation without injury. The varieties were ranked and arranged in descending order of susceptibility. The varieties Dwarf Cavendish, Njali poovan, Gros Michel, Pisang lilin and Matti are highly susceptible than other varieties tested and they showed cent per cent infection. The varieties Nendran and Poocha kunnan were also found to be susceptible to a lesser degree and they were on par. The variety Chinia was also found to be on par with Poocha kunnan followed by Adakka kunnan which was on par with Venneettu mannan. Venneettu mannan was on par with Koduppilla kunnan. The varieties Zanzibar, Red banana, Hybrid sawai and Bodles Altafort were found to be in almost same range. These were the least susceptible ones and were on par with each other. The varieties Robusta, Palayankodan, Kanchikela, Nendra padaththi, Boodida bontha batheea, Peyan, Kapok, Pisang mas, Klue teparod and

Table 4. Disease intensity of banana varieties at three-fourth maturity

Sl. No.		Per cent intensity of the disease		Mean
		Uninjured	Injured	
1.	Robusta	0.00(0.57)	100.00(90.00)	50.00(45.00)
2.	Dwarf Cavendish	100.00(90.00)	100.00(90.00)	100.00(90.00)
3.	Palayankodan	0.00(0.57)	0.00(0.57)	0.00(0.57)
4.	Njali poovan	100.00(90.00)	100.00(90.00)	100.00(90.00)
5.	Gros Michel	100.00(90.00)	100.00(90.00)	100.00(90.00)
6.	Koduppilla kunnan	3.78(11.24)	9.69(18.15)	6.70(15.00)
7.	Poocha kunnan	5.27(13.31)	5.66(13.81)	5.47(13.56)
8.	Adakka kunnan	5.02(12.92)	9.52(17.95)	7.27(15.68)
9.	Red banana	2.80(9.63)	13.04(21.13)	7.92(16.32)
10.	Pisang lilin	100.00(90.00)	100.00(90.00)	100.00(90.00)
11.	Chinia	5.22(13.18)	9.48(17.95)	7.35(15.79)
12.	Matti	100.00(90.00)	100.00(90.00)	100.00(90.00)
13.	Bodles Altafort	0.27(2.99)	9.93(18.34)	5.10(13.05)
14.	Kanchikela	0.00(0.57)	0.17(2.36)	0.09(1.72)
15.	Nendra padaththi	0.00(0.57)	0.11(1.91)	0.06(1.40)
16.	Boodida bontha batheea	0.00(0.57)	0.15(2.22)	0.08(1.62)
17.	Hybrid sawai	2.65(9.46)	20.21(26.71)	11.43(19.73)
18.	Peyan	0.00(0.57)	0.22(2.69)	0.11(1.91)
19.	Kapok	0.00(0.57)	5.22(13.18)	2.61(9.28)
20.	Pisang mas	0.00(0.57)	3.06(10.14)	1.53(7.04)
21.	Vennoettu nannan	4.29(11.97)	8.39(16.85)	6.34(14.54)
22.	Klue teparod	0.00(0.57)	7.52(15.89)	3.76(11.24)
23.	Jurmani kunthali	0.00(0.57)	0.00(0.57)	0.00(0.57)
24.	Nendran	5.92(14.06)	7.83(16.22)	6.88(15.23)
25.	Zanzibar	2.83(9.63)	3.86(11.39)	3.85(11.39)

(Figures given in parenthesis are the angular transformed values)

CD (0.05) between varieties = 10.478

between varieties / within treatment = 14.827

Contd.....

Table 4. Contd.....

Conclusion :

Comparison of varieties

2 4 5 10 12 1 17 9 11 8 24 6 21 7 13 25 22 19 20 18 14 16 15 3 23

Comparison of varieties within treatments (uninjured)

2 4 5 10 12 24 7 11 8 21 6 25 9 17 13 1 3 14 15 16 18 19 20 22 23

Comparison of varieties within treatments (injured)

1 2 4 5 10 12 17 9 13 8 6 11 21 24 22 7 19 25 20 18 14 16 15 3 23

Jurmani kunthali did not take infection in this stage but they were found to be on par with each other.

When the injured fruit were inoculated there was significant difference among the varieties. The varieties are arranged in descending order of susceptibility. Here six varieties viz., Robusta, Dwarf Cavendish, Njali poovan, Gros Michel, Pisang lilin and Matti showed cent per cent infection after ten days of inoculation. All other varieties have shown different degrees of infection and the varieties Peyan, Kanchikela, Boodida bontha bathees, Nendra padaththi were having high degrees of resistance and the varieties Palayankodan and Jurmani kunthali were found to be free from the disease at this stage.

After considering the two treatments in combination with and without injury, statistical analysis showed high significant difference among the varieties. The varieties were arranged in descending order of susceptibility. The varieties Dwarf Cavendish, Njali poovan, Gros Michel, Pisang lilin and Matti were found to be highly susceptible than other varieties as far as the disease was concerned followed by the varieties Robusta and Hybrid sawai. Hybrid sawai was found to be on par with Red banana while Red banana was on par with Chinia as far as the disease is concerned. The varieties Adakka kunnan, Nendran, Koduppilla kunnan, Vannaettu mannan, Poocha kunnan and

Bodles Altafort were found to be less susceptible when compared to the former ones. The varieties Zanzibar, Klue teparod, Kapok, Pisang mas showed good resistance while the varieties Peyan, Kanchikela, Boodida bontha bathees, Nendra padaththi exhibited high degree of resistance.

The varieties Palayankodan and Jurmani kunthali were completely free from the disease at three-fourth maturity stage and the order of susceptibility was as follows: Dwarf Cavendish, Njali poovan, Gros Michel, Pisang lilin, Matti, Robusta, Hybrid sawai, Red banana, Chinia, Adakka kunnan, Nendran, Koduppilla kunnan, Venneettu mannan, Poocha kunnan, Bodles Altafort, Zanzibar, Klue teparod, Kapok, Pisang mas, Peyan, Kanchikela, Boodida bontha bathees and Nendra padaththi.

3.5. Full maturity

In the case of injured fruit, the symptom expression was a bit early and took three days while the uninjured fruit took four days.

At full maturity stage all the twenty-five varieties had shown cent per cent infection after eight days of inoculation both in the case of injured and uninjured fruit (Plate IVa,b and c), but there was much variation among the varieties when observations were taken after six days of inoculation (Table 5).

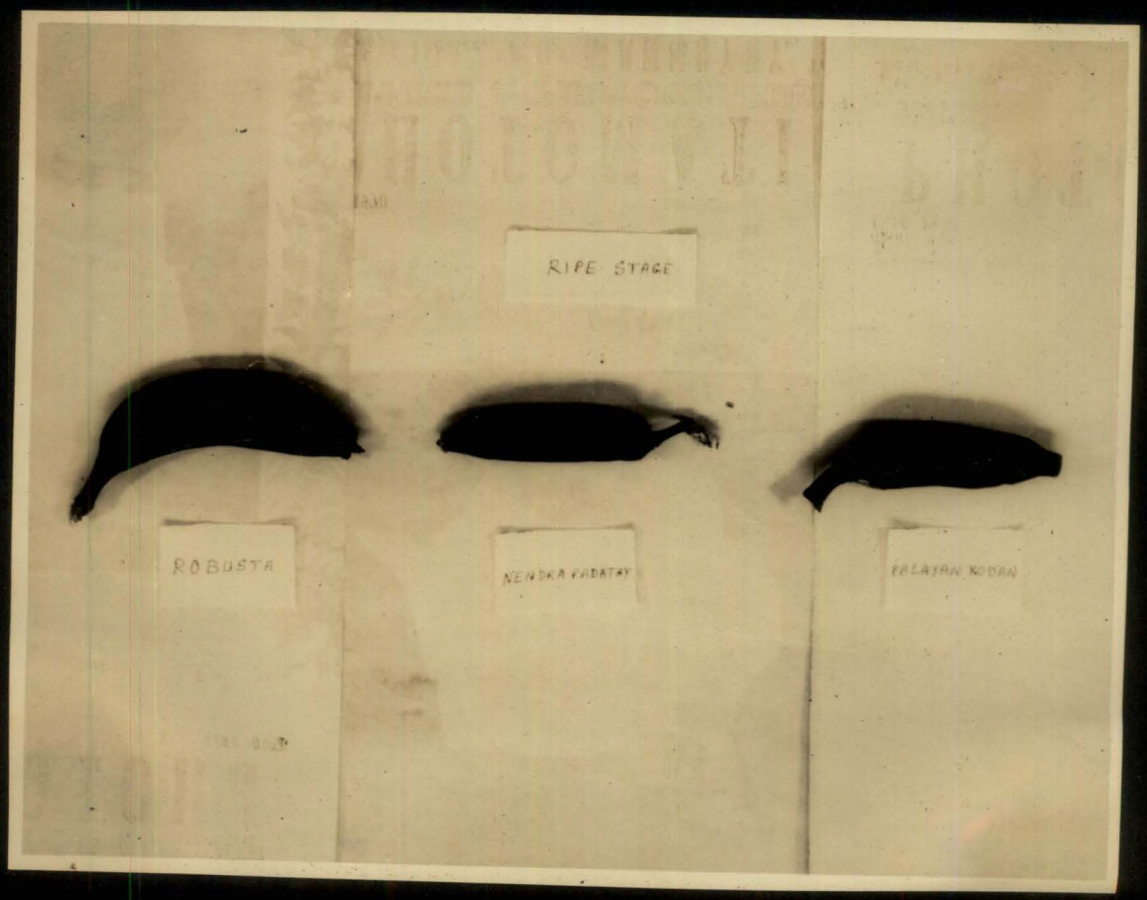


Plate IV a) Three varieties showing cent per cent disease intensity after eight days of inoculation at full maturity stage (After eight days the fruit were completely ripened)

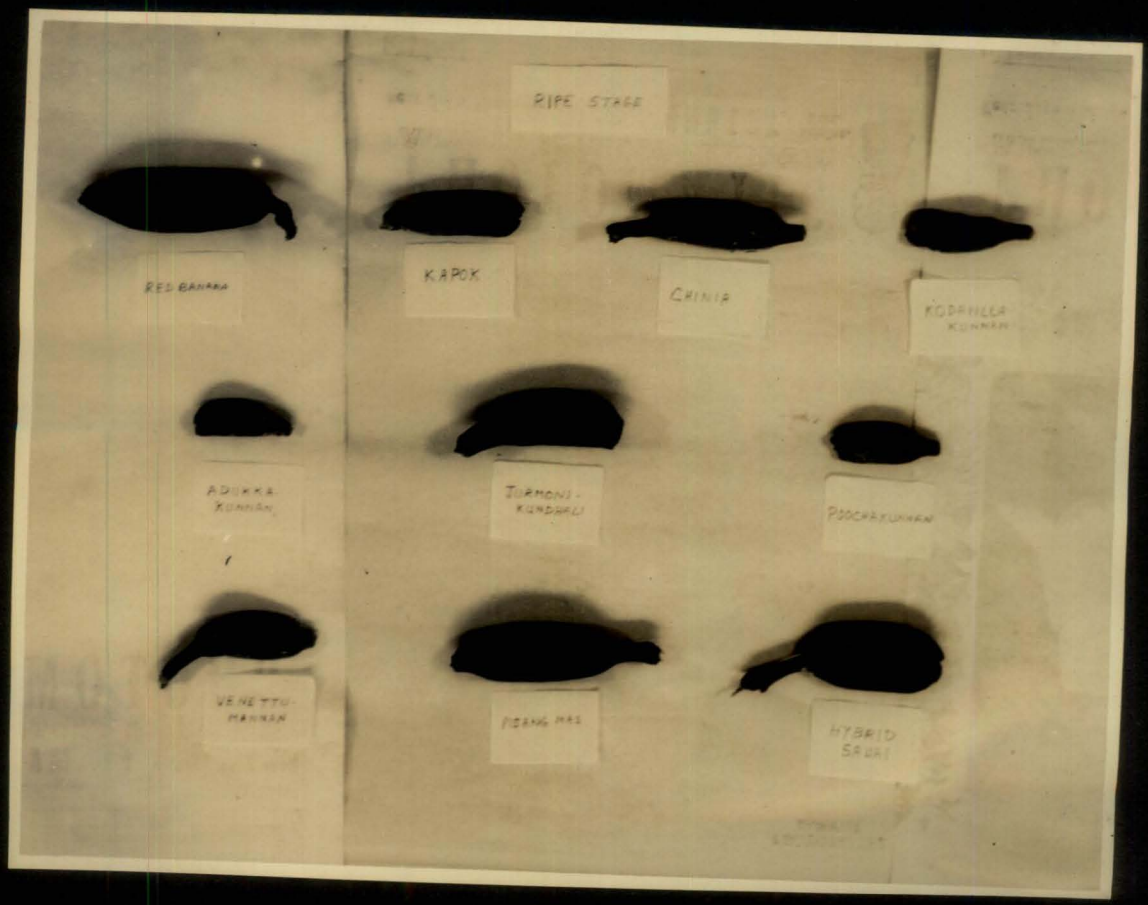


Plate XV b) Ten varieties showing cent per cent disease intensity after eight days of inoculation at full maturity stage (after eight days the fruit were completely ripened)

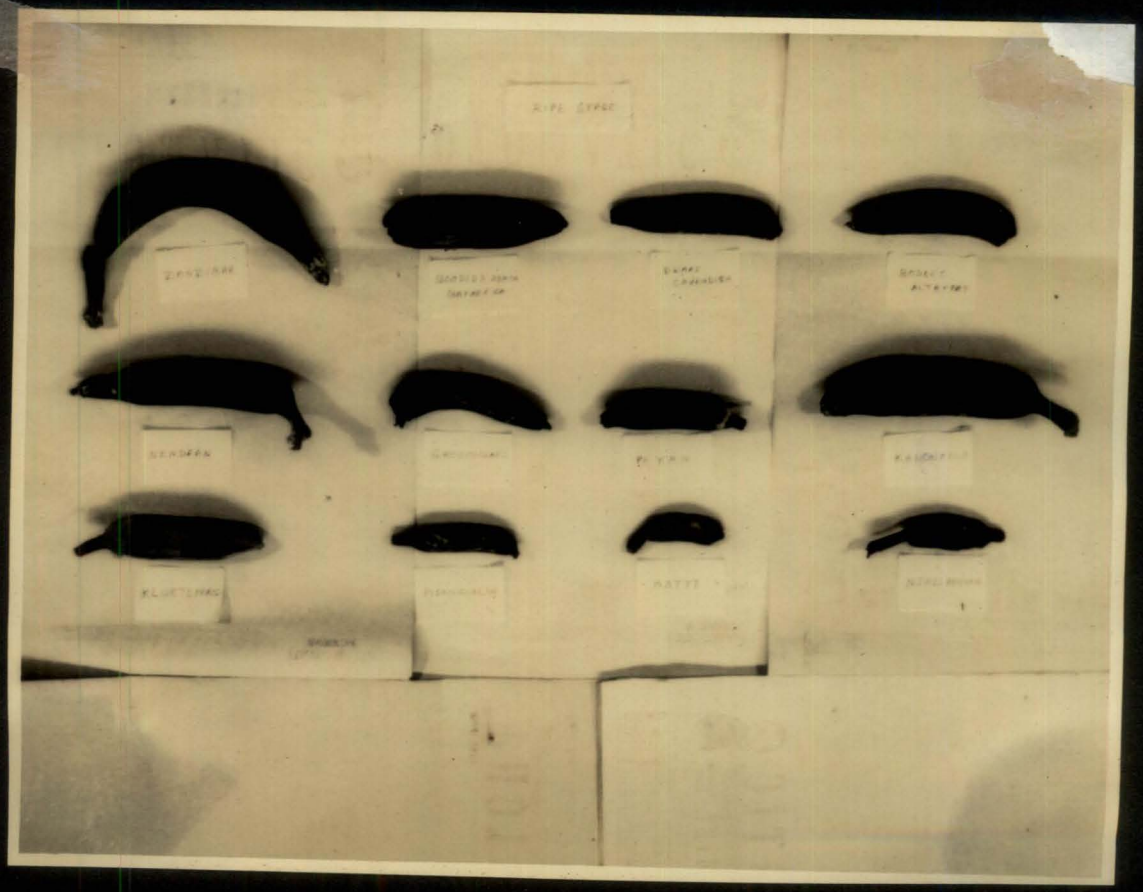


Plate IV c) Twelve varieties showing cent per cent infection after eight days of inoculation at full maturity stage (After eight days the fruit were completely ripened)

Table 5. Disease intensity of banana varieties at full maturity

Sl. no.	Variety	Per cent intensity of the disease		Mean
		Uninjured	Injured	
1.	Robusta	3.51(10.78)	21.27(27.49)	12.39(20.62)
2.	Dwarf Cavendish	2.36(8.91)	14.13(22.06)	8.25(16.74)
3.	Palayankodan	1.30(6.55)	2.32(8.72)	1.81(7.71)
4.	Njali poovan	42.91(40.92)	69.94(56.37)	56.43(48.68)
5.	Gros Michel	6.40(14.65)	9.36(17.85)	7.88(16.32)
6.	Koduppilla kunnan	5.38(13.44)	6.86(15.23)	6.12(14.30)
7.	Poocha kunnan	6.78(15.12)	11.17(19.55)	8.96(17.46)
8.	Adakka kunnan	3.46(10.78)	3.84(11.24)	3.65(11.09)
9.	Red banana	7.18(15.56)	8.72(17.15)	7.95(16.43)
10.	Pisang lilin	5.64(13.69)	13.45(21.56)	9.54(17.95)
11.	Chinia	3.42(10.63)	6.77(15.12)	5.09(13.05)
12.	Matti	7.50(15.89)	9.09(17.56)	8.30(16.74)
13.	Bodles Altafort	7.70(16.11)	14.89(22.71)	11.30(19.64)
14.	Kanchikela	3.86(11.39)	5.67(13.81)	4.77(12.66)
15.	Nendra padaththi	0.62(4.44)	2.41(8.91)	1.52(7.03)
16.	Boodida bontha bathees	0.58(4.44)	5.07(13.05)	2.83(9.83)
17.	Hybrid sawai	3.19(10.36)	4.36(12.11)	3.78(11.24)
18.	Payan	4.21(11.83)	5.11(13.05)	4.66(12.52)
19.	Kapok	2.26(8.72)	2.84(9.63)	2.55(9.28)
20.	Pisang mas	2.62(9.28)	4.16(11.83)	3.39(10.63)
21.	Venneettu nannan	4.88(12.79)	9.01(17.46)	6.95(15.34)
22.	Klue taparod	4.38(12.11)	11.82(20.09)	8.10(16.54)
23.	Jurmani kunthali	1.97(8.13)	2.87(9.80)	2.42(8.91)
24.	Nendran	2.17(8.53)	10.70(19.09)	6.43(14.65)
25.	Zanzibar	2.84(9.63)	5.69(13.81)	4.27(11.97)

(Figures given in parenthesis are the angular transformed values)

CD (0.05) between varieties = 12.453

between varieties I
within treatment I = 17.612

Contd.....

Table 5. Contd.....

Conclusions

Comparison of varieties

Comparison of varieties within treatments (uninjured)

Comparison of varieties within treatments (injured)

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In the case of inoculation without injury, the percentage of infection varied from 0.58 to 42.91 per cent six days after inoculation. The maximum was found in Njali poovan and the minimum was found in Soodida bontha bathees. Except Njali poovan, all others showed less than ten per cent infection. The varieties Koduppilla kunnan, Pisang lilin, Gros Michel, Poocha kunnan, Red banana, Matti and Bodles Altafort were found to be moderately susceptible and the infection ranged from 5.38 to 7.70 per cent. The varieties Nendran, Kapok, Dwarf Cavendish, Pisang mas, Zanzibar, Hybrid sawai, Chinia, Adakka kunnan, Robusta, Kanchikela, Peyan, Klue teparod, Vanneettu mannan were found to be less susceptible and the infection rates ranged from 2.17 to 4.88 per cent. However, the remaining four varieties viz., Soodida bontha bathees, Nendra padaththi, Palayankodan and Jurmani kunthali showed high degrees of resistance and their infection percentage were 0.58, 0.62, 1.30 and 1.97 respectively.

When the fruit were injured and inoculated at this stage, maximum infection was shown by Njali poovan (69.94 per cent) and the minimum was shown by Palayankodan after six days of inoculation (2.32 per cent). Njali poovan was found to be highly susceptible. The varieties Nendran, Poocha kunnan, Klue teparod, Pisang lilin, Dwarf Cavendish, Bodles Altafort and Robusta showed moderate susceptibility and the infection rates ranged from

10.7 per cent to 21.27 per cent. Other thirteen varieties proved to be less susceptible and their infection rating varied from 3.84 to 9.36 per cent. They were Adakka kunnan, Pisang mas, Hybrid sawai, Peyan, Boodida bontha bathees, Zanzibar, Kanchikela, Chinia, Koduppilla kunnan, Red banana, Vennecttu mannan, Matti and Gros Michel. The varieties Jurmani kunthali, Kapok, Nendra padaththi and Palayankodan exhibited high degrees of resistance and infection percentages recorded were 2.87, 2.84, 2.41 and 2.32 respectively.

The varieties were ranked and arranged in descending order of susceptibility. The statistical analysis revealed that significantly high infection was observed in Njali poovan and was found to be highly susceptible when uninjured fruit were inoculated. The varieties Bodles Altafort, Matti, Red banana, Poocha kunnan, Gros Michel, Pisang lilin, Koduppilla kunnan were found to be moderately susceptible, but they were found to be on par with each other and also on par with less susceptible varieties viz., Vennecttu mannan, Klue taparod, Peyan, Kanchikela, Robusta, Adakka kunnan, Chinia, Hybrid sawai, Zanzibar, Pisang mas, Dwarf Cavendish, Kapok and Nendran. However, the resistant varieties viz., Jurmani kunthali, Palayankodan, Nendra padaththi and Boodida bontha bathees were found to be on par with each other and also with less susceptible varieties.

When the injured fruit were inoculated again significantly high infection was shown by Njali poovan. The varieties were ranked and arranged in descending order of susceptibility. The varieties Robusta, Bodles Altafort, Dwarf Cavendish, Pisang lilin, Klue teparod, Poocha kunnan and Nendran were found to be moderately susceptible but they were on par with each other and also with less susceptible varieties like Gros Michel, Matti, Vannaettu mannan, Red banana, Koduppilla kunnan, Chinia, Kanchikela, Zanzibar, Boodida bontha bathees, Peyan, Hybrid sawai, Pisang mas and Adakka kunnan which were again on par with each other. The resistant varieties, Jumani kunthali, Kapok, Nendra padaththi and Palayankodan were also found to be on par with each other and also with less susceptible varieties.

After considering the two treatments (both injured and uninjured in combination), statistical analysis showed that the variety Njali poovan showed significantly high infection and thus found to be highly susceptible as far as the disease is concerned. The varieties Robusta, Bodles Altafort, Pisang lilin, Poocha kunnan, Dwarf Cavendish, Matti and Klue teparod were on par with each other and they were found to be moderately susceptible. The varieties Red banana, Gros Michel, Vannaettu mannan, Nendran, Koduppilla kunnan, Chinia, Kanchikela, Peyan, Zanzibar, Hybrid sawai, Adakka kunnan, Pisang mas were on par with

each other and they were found to be less susceptible. All other varieties viz., Soodida bontha bathees, Kapok, Jurmani kunthali, Palayankodan and Nendra padaththi were also found to be on par with each other and showed only very less disease intensity thus exhibiting moderately high degree of resistance.

The data regarding the screening of the banana varieties against the anthracnose pathogen, in all the stages of development of fruit taken together have been analysed and the results are given in Table 6.

The statistical analysis clearly showed that there were significant differences in the relative susceptibility of the varieties against the disease. Among the varieties tested, Nendra padaththi and Palayankodan showed high resistance to the disease with lowest disease intensity percentages of 0.401 and 0.423 respectively. The variety Jurmani kunthali (0.605 per cent) was found to be on par with the variety Palayankodan. The varieties Soodida bontha bathees (0.726 per cent), Peyan (1.192 per cent), Kanchikela (1.214 per cent), Pisang mas (1.315 per cent) and Kapok (1.436 per cent) were also found to be highly resistant and they were on par with each other. The varieties Zanzibar (2.048 per cent), Adakka kunnan (2.331 per cent), Klue taparod (2.964 per cent), Chinia (3.231 per cent), Nendran (3.327 per cent), Venneettu mannan (3.469 per cent), Koduppilla kunnan

Table 6. Disease intensity of banana varieties, pooled analysis

Sl. no.	Variety	Per cent disease intensity				
		Replications				Mean
		I	II	III	IV	
1.	Robusta	15.615(4.076)	15.515(4.064)	15.606(4.075)	15.660(4.082)	15.599(4.074)
2.	Dwarf Cavendish	27.096(5.301)	27.193(5.310)	27.118(5.303)	27.086(5.300)	27.123(5.304)
3.	Palayankodan	0.509 (1.228)	0.470 (1.212)	0.339(1.157)	0.374(1.172)	0.423(1.192)
4.	Njali poovan	50.000(7.141)	35.655(6.054)	36.289(6.106)	34.483(5.957)	39.107(6.315)
5.	Gros Michel	27.033(5.295)	27.029(5.294)	27.050(5.296)	27.003(5.292)	27.028(5.294)
6.	Koduppilla kunnan	3.011(2.003)	3.768(2.184)	3.606(2.146)	3.725(2.174)	3.528(2.127)
7.	Poocha kunnan	3.744(2.178)	4.035(2.244)	3.985(2.233)	3.899(2.213)	3.916(2.217)
8.	Adakka kunnan	2.753(1.937)	3.086(2.021)	2.945(1.986)	2.939(1.985)	2.931(1.982)
9.	Red banana	4.030(2.243)	4.030(2.243)	4.011(2.239)	4.110(2.261)	4.045(2.247)
10.	Pisang lilin	27.361(5.326)	27.393(5.329)	27.378(5.327)	27.412(5.330)	27.356(5.328)
11.	Chinia	3.210(2.052)	3.214(2.053)	3.224(2.055)	3.276(2.068)	3.231(2.057)
12.	Matti	27.335(5.323)	26.823(5.275)	27.120(5.303)	27.033(5.295)	27.077(5.299)
13.	Bodles Altafort	4.100(2.258)	4.108(2.260)	4.088(2.256)	4.170(2.273)	4.116(2.261)
14.	Kanchikela	1.213(1.489)	1.210(1.487)	1.211(1.487)	1.222(1.489)	1.214(1.488)
15.	Nendra padaththi	0.394(1.181)	0.409(1.189)	0.395(1.181)	0.404(1.185)	0.401(1.183)
16.	Boodida bontha bathees	0.831(1.353)	0.675(1.254)	0.697(1.303)	0.701(1.304)	0.726(1.313)
17.	Hybrid sawai	3.853(2.203)	3.935(2.221)	3.823(2.196)	3.926(2.219)	3.884(2.209)
18.	Peyan	1.205(1.423)	1.645(1.626)	1.013(1.419)	1.087(1.445)	1.192(1.478)
19.	Kapok	1.523(1.588)	1.406(1.555)	1.394(1.547)	1.421(1.556)	1.436(1.562)
20.	Pisang mas	1.260(1.503)	1.420(1.556)	1.256(1.502)	1.325(1.528)	1.315(1.522)
21.	Vennoettu manman	3.128(2.032)	2.876(1.969)	4.161(2.272)	3.713(2.171)	3.469(2.111)
22.	Klue taparod	2.962(1.990)	2.988(1.997)	2.921(1.980)	2.987(1.997)	2.964(1.991)
23.	Jumani kunthali	0.662(1.289)	0.546(1.243)	0.611(1.269)	0.601(1.265)	0.605(1.267)
24.	Nendran	0.813(2.077)	3.337(2.083)	3.300(2.074)	3.360(2.088)	3.327(2.081)
25.	Zanzibar	2.020(1.738)	2.049(1.431)	2.041(2.010)	2.083(1.756)	2.048(1.734)

(Figures given in parenthesis are the $\sqrt{X+1}$ transformed values)

CD (0.05) between varieties = 0.1758

Conclusion :

Comparison of varieties

4 10 2 12 5 1 13 9 7 17 6 21 24 11 22 8 25 19 20 14 18 16 23 3 15

(3.528 per cent), Hybrid sawai (3.884 per cent), Pooche kunnan (3.916 percent), Red banana (4.045 per cent) and Bodles Altafort (4.116 per cent) were found to be resistant to the disease and they were on par with each other except Zanzibar.

The variety Robusta with 15.599 per cent disease intensity was found to be susceptible to the disease while the varieties Gros Michel (27.028 per cent), Matti (27.077 per cent), Dwarf Cavendish (27.123 per cent), Pisang lilin (27.356 per cent) and Njali poovan (39.107 per cent) were found to be highly susceptible to the disease. They were on par with each other except Njali poovan which showed the maximum disease intensity.

4. The chemical constituents of fruit of banana varieties at different stages of development

The major chemical constituents of the banana fruit of twenty-five varieties at five different stages were analysed as mentioned in materials and methods. The major chemical constituents estimated were reducing sugars, total sugar, starch, crude fibre, crude protein and tannin. The results are given below:

4.1. Reducing sugars

The minimum content of reducing sugars was found during the female phase and that too was varying in different

varieties. At this state, the maximum sugar content was observed in Dwarf Cavendish (1.412 per cent) and minimum was in Zanzibar (0.540 per cent). The reducing sugar content of all varieties increased from female phase to full maturity state (Table 7).

The reducing sugar has been slightly increased at one-fourth maturity of the fruit when compared to the female phase. The maximum reducing sugar content was observed in Robusta (1.643 per cent) and the minimum was in Zanzibar (0.634 per cent).

At half maturity slight increase of reducing sugar was observed when compared to one-fourth maturity. Here also Robusta maintained the highest percentage of reducing sugar (1.955 per cent) and minimum was observed in Zanzibar (0.607 per cent).

At three-fourth maturity also there was increase in reducing sugar content compared to half maturity. However, it was found to vary in different varieties. As in the case of former stages maximum and minimum contents of reducing sugars were observed in Robusta (2.658 per cent) and Zanzibar (0.808 per cent) respectively.

Slight increase of reducing sugar content was also observed at full maturity stage over three-fourth maturity. Here also Robusta and Zanzibar recorded maximum and minimum values (2.935 and 1.282 per cent) respectively.

Table 7. Reducing sugars at different stages of fruit maturity, per cent.

Sl. no.	Variety	Immediately after female phase	One-fourth maturity	Half maturity	Three-fourth maturity	Full maturity
1.	Robusta	1.363	1.643	1.955	2.658	2.935
2.	Dwarf Cavendish	1.412	1.593	1.841	2.300	2.799
3.	Palayankodan	1.329	1.576	1.840	1.940	2.895
4.	Njali poovan	1.348	1.541	1.806	2.408	2.696
5.	Gros Michel	1.260	1.480	1.751	2.286	2.608
6.	Koduppilla kunnan	1.342	1.489	1.676	1.978	2.552
7.	Poocha kunnan	1.271	1.401	1.632	1.840	2.339
8.	Adakka kunnan	1.215	1.372	1.591	1.906	2.241
9.	Red banana	1.192	1.335	1.563	1.824	2.387
10.	Pisang lilin	1.213	1.390	1.619	1.942	2.611
11.	Chinia	1.238	1.445	1.586	1.909	2.801
12.	Matti	1.216	1.404	1.577	1.935	2.473
13.	Bodles Altafort	1.188	1.327	1.454	2.335	2.535
14.	Kanchikela	1.149	1.299	1.421	1.821	2.662
15.	Nendra padaththi	1.338	1.520	1.676	1.908	2.535
16.	Soodida bontha hathees	1.253	1.473	1.682	1.962	2.496
17.	Hybrid sawai	1.193	1.375	1.546	1.868	2.453
18.	Peyan	1.322	1.513	1.638	1.886	2.494
19.	Kapok	1.123	1.220	1.526	2.139	2.571
20.	Pisang mas	0.944	1.169	1.440	2.273	2.678
21.	Vennoettu nannan	1.138	1.446	1.628	2.034	2.535
22.	Klueteparod	1.330	1.529	1.761	1.971	2.180
23.	Jurmani kunthali	0.728	0.935	1.196	1.563	2.059
24.	Nendran	0.547	0.653	0.708	0.841	1.338
25.	Zanzibar	0.540	0.634	0.687	0.808	1.282

CD (0.05) between varieties = 0.0983

Contd.....

Table 7. Contd.....

Conclusion :

Comparison of varieties at

(1) immediately after female phase

2 1 4 6 15 22 3 18 7 5 16 11 12 8 10 17 9 13 14 21 19 20 21 24 25

(2) one-fourth maturity

1 2 3 4 22 15 18 7 6 5 16 21 11 12 10 17 8 9 13 14 19 20 23 24 25

(3) half maturity

1 2 3 4 22 5 16 6 15 19 7 21 10 8 11 12 9 17 19 13 20 14 23 24 25

(4) three-fourth maturity

1 4 13 2 5 20 19 21 6 22 16 10 3 12 11 15 8 18 17 7 9 14 23 24 25

(5) full maturity

1 3 11 2 4 20 14 10 5 19 6 13 15 21 16 18 12 17 9 7 8 22 23 24 25

Statistical analysis showed that there was significant difference among the varieties with regard to reducing sugar content in all the five stages. The varieties in all the five stages are arranged in descending order with respect to their reducing sugar content. The results are presented in Table 7.

4.2. Total sugars

During female phase, minimum content of total sugar was noticed and that too varied in different varieties. At this phase, maximum total sugar was observed in Koduppilla kunnan (1.659 per cent) while minimum was in Nendran (1.101 per cent). Total sugars increased steadily from female phase to full maturity phase in all the varieties (Table 8).

Total sugar content increased as the fruit reached one-fourth maturity. The maximum and minimum amounts were seen in Peyan (2.100 per cent) and Zanzibar (1.271 per cent) respectively.

At half maturity stage, slight increase of total sugars was observed further. Here Palayankodan maintained highest amount (2.308 per cent) and Nendran maintained lowest amount (1.512 per cent).

During three-fourth maturity there was increase in total sugar content compared to former phase and maximum was observed in Dwarf Cavendish (3.447 per cent) and minimum was in Nendran (1.768 per cent).

Table 8. Total sugar at different stages of fruit maturity, per cent

Sl. no.	Variety	Immediately after female phase	One-fourth maturity	Half maturity	Three-fourth maturity	Full maturity
1.	Robusta	1.549	1.849	2.083	3.254	3.556
2.	Dwarf Cavendish	1.645	1.925	2.170	3.447	3.807
3.	Palayankodan	1.598	1.911	2.308	3.042	3.691
4.	Njali poovan	1.566	1.848	2.189	3.034	3.477
5.	Gros Michel	1.519	1.828	2.266	2.950	3.519
6.	Koduppilla kunnan	1.659	1.957	2.094	3.276	3.741
7.	Poocha kunnan	1.634	1.907	2.152	2.929	3.559
8.	Adakka kunnan	1.615	1.966	2.032	3.122	3.496
9.	Red banana	1.582	1.911	2.196	2.946	3.521
10.	Pisang lilin	1.551	1.843	2.079	2.756	3.303
11.	Chinia	1.617	1.909	2.096	2.759	3.360
12.	Matti	1.533	1.844	2.191	2.941	3.457
13.	Bodles Altafort	1.463	1.980	2.106	2.776	3.267
14.	Kanchikela	1.556	1.868	2.120	2.545	3.187
15.	Nendra padaththi	1.642	1.901	2.061	2.657	3.298
16.	Boodida bontha bathees	1.586	1.929	2.285	2.515	2.971
17.	Hybrid sawai	1.658	2.024	2.203	2.664	2.944
18.	Peyan	1.642	2.100	2.299	2.768	3.216
19.	Kapok	1.541	1.939	2.202	2.880	3.162
20.	Pisang mas	1.617	1.979	2.128	3.146	3.275
21.	Venneettu mannan	1.573	1.933	2.019	2.426	3.155
22.	Klue teparod	1.472	1.870	2.081	2.485	2.945
23.	Jurmani kunthali	1.446	1.865	1.997	2.475	3.066
24.	Nendran	3.101	1.307	1.512	1.768	2.127
25.	Zanzibar	1.162	1.271	1.548	1.854	2.237

CD (0.05) between varieties = 0.0626

Contd.....

Table 8. Contd.....

Conclusion :

Comparison of varieties at

(1) immediately after female phase

6 17 2 15 18 7 11 20 8 3 16 9 21 4 14 10 1 19 12 5 22 13 23 25 24

(2) one-fourth maturity

18 17 13 20 8 6 19 21 16 2 3 9 11 7 15 22 14 23 1 4 12 10 5 24 25

(3) half maturity

3 18 16 5 17 19 9 12 4 2 7 20 14 13 11 6 1 22 10 15 8 21 23 25 24

(4) three-fourth maturity

2 6 1 20 8 3 4 5 9 12 7 19 18 13 11 10 17 15 14 16 22 23 21 25 24

(5) full maturity

2 6 3 7 1 9 5 8 4 12 11 10 15 20 13 18 14 19 21 23 16 17 22 25 24

Total sugars further increased at full maturity and here also Dwarf Cavendish maintained highest percentage (3.807 per cent) and minimum was observed in Nendran (2.127 per cent).

Statistical analysis revealed that there was significant difference among the varieties in all the five stages of fruit development with respect to total sugar content. The minimum content of total sugars was observed in female phase in all the varieties and maximum was found at full maturity. The varieties in all the five developmental stages are arranged in descending order with regard to their total sugar content and presented in Table 8.

4.3. Starch

At female phase, highest starch content was observed in Pisang mas (1.568 per cent) and lowest was in Peyan (1.003 per cent). However, it varied with different varieties. The starch content increased from female phase to full maturity in all the stages of fruit development (Table 9).

At one-fourth maturity there was increase in starch content and maximum was seen in Nendran (6.071 per cent) and minimum was in Palayankodan (2.725 per cent).

During half maturity, again the starch content increased and maximum was maintained in Zanzibar (20.826 per cent) and the minimum was in Chinia (7.266 per cent).

Table 9. Starch at different stages of fruit maturity, per cent

Sl. no.	Variety	Immediately after female phase	One-fourth maturity	Half maturity	Three-fourth maturity	Full maturity
1.	Robusta	1.236	3.205	11.244	13.675	20.077
2.	Dwarf Cavendish	1.119	2.967	10.970	12.973	18.951
3.	Palayankodan	1.152	2.725	11.374	13.504	19.285
4.	Njali poovan	1.279	3.245	9.905	14.244	20.448
5.	Gros Michel	1.356	2.879	9.746	14.437	20.539
6.	Koduppilla kunnan	1.181	3.238	7.872	11.463	18.802
7.	Poocha kunnan	1.231	3.843	7.609	11.323	19.629
8.	Adakka kunnan	1.321	3.213	7.619	11.918	20.634
9.	Red banana	1.357	3.321	7.659	13.275	19.789
10.	Pisang lilin	1.242	2.959	8.369	11.695	20.482
11.	Chinia	1.277	3.072	7.266	12.496	18.120
12.	Matti	1.205	3.082	7.469	13.890	19.559
13.	Bodles Altafort	1.157	2.897	7.328	13.189	18.508
14.	Kanchikela	1.248	2.975	7.715	13.811	20.339
15.	Nendra padaththi	1.248	3.085	7.619	12.399	17.185
16.	Boodida bontha bathees	1.228	3.799	7.112	17.439	21.072
17.	Hybrid sawai	1.098	4.762	10.372	18.611	25.015
18.	Peyan	1.003	4.319	12.083	23.450	28.538
19.	Kapok	1.561	4.783	10.898	22.048	29.338
20.	Pisang mas	1.568	4.705	11.135	23.420	28.414
21.	Vennoettu mannan	1.304	4.818	11.097	18.577	28.410
22.	Klue teparod	1.439	3.911	11.921	18.113	30.384
23.	Jurmani kunthali	1.343	3.764	14.896	18.927	29.386
24.	Nendran	1.518	6.071	20.090	28.041	31.380
25.	Zansibar	1.549	5.968	20.826	27.679	33.215

At three-fourth maturity stage, Nendran (28.041 per cent) contained highest amount of starch and Poovha Nunnan (11.323 per cent) contained the lowest amount.

During full maturity stage, starch reached highest level in all the varieties and maximum was noticed in Zanzibar (33.215 per cent) and minimum was observed in Nendra padaththi (17.185 per cent).

As the starch content in all the varieties at different stages of fruit development did not show any correlation with the percentage disease intensity, the statistical analysis of the data was not carried out.

4.4. Crude fibre

The crude fibre content was present in very low amounts at female phase. However, it varied with different varieties. At female phase, maximum crude fibre was observed in Palayankodan (4.319 per cent) and the minimum was in Matti (1.526 per cent). The crude fibre content increased from female phase to full maturity in all the stages of fruit development (Table 10).

At one-fourth maturity, there was increase in crude fibre content and maximum was seen in Jumani kunthali (7.478 per cent) and minimum was in Hybrid sawai (4.883 per cent).

Table 10. Crude fibre at different stages of fruit maturity, per cent

Sl. no.	Variety	Immediately after female phase	One-fourth maturity	Half maturity	Three-fourth maturity	Full maturity
1.	Robusta	3.593	6.593	10.167	12.345	16.053
2.	Dwarf Cavendish	3.389	6.805	9.957	12.769	16.561
3.	Palayankodan	4.319	7.120	9.235	12.943	16.575
4.	Njali poovan	3.468	6.494	9.506	12.150	16.037
5.	Gros Michel	2.876	6.032	9.478	12.417	16.601
6.	Koduppilla kunnan	3.092	6.163	9.434	11.599	15.761
7.	Pooche kunnan	2.967	6.415	9.711	12.037	16.445
8.	Adakka kunnan	2.846	6.249	9.991	13.329	17.679
9.	Red banana	3.296	6.471	9.201	12.337	17.942
10.	Pisang lilin	3.396	6.878	8.369	12.270	16.687
11.	Chinia	1.951	4.978	8.386	12.350	18.795
12.	Matti	1.526	5.461	8.993	11.686	18.625
13.	Bodles Altafort	2.700	5.545	9.638	11.380	17.379
14.	Kanchikela	2.317	5.285	7.990	10.648	15.129
15.	Nendra padaththi	2.945	6.474	9.802	11.075	19.967
16.	Boodida bontha batheea	2.392	5.168	8.626	12.727	17.570
17.	Hybrid sawai	1.809	4.883	8.025	10.368	18.748
18.	Peyan	2.974	5.417	9.968	13.387	19.939
19.	Kapok	3.496	6.448	9.465	11.525	18.099
20.	Pisang mas	2.096	5.634	8.150	10.119	17.267
21.	Vennaettu nannan	3.282	6.520	9.381	11.133	17.970
22.	Klue teparod	2.924	5.977	8.288	13.297	19.362
23.	Jurmani kunthali	3.752	7.478	10.255	12.364	17.669
24.	Nendran	2.412	6.726	9.455	12.159	18.890
25.	Zanzibar	2.633	7.010	9.659	11.994	18.772

During half maturity stage, there was again increase in crude fibre content and Jurmani kunthali (10.255 per cent) maintained the highest amount and Kanchikela (7.990 per cent) maintained the lowest.

At three-fourth maturity, crude fibre further increased and maximum was observed in Peyan (13.387 per cent) and minimum was in Pisang mas (10.119 per cent).

During full maturity, the crude fibre content reached the highest level in all the varieties and Nendra padaththi contained the maximum (19.967 per cent) and Kanchikela the minimum (15.129 per cent).

As the crude fibre content in all the varieties at different stages of maturity did not show any correlation with percentage disease intensity, the data were not analysed statistically.

4.5. Crude protein

The maximum crude protein content was noticed in female phase but it was found to vary among the different varieties. At this phase, the highest crude protein was noticed in Soodida bontha bathees (3.838 per cent) and minimum was in Gros Michel (2.948 per cent). Crude protein content steadily decreased from female phase to full maturity phase in all the varieties (Table 11).

Table 11. Crude protein at different stages of fruit maturity, per cent

Sl. no.	Variety	Immediately after female phase	One-fourth maturity	Half maturity	Three-fourth maturity	Full maturity
1.	Robusta	3.085	2.866	2.238	1.971	1.525
2.	Dwarf Cavendish	3.034	2.889	2.318	1.908	1.599
3.	Palayankodan	3.377	3.038	2.507	2.067	1.669
4.	Njali poovan	2.951	2.708	2.481	2.116	1.577
5.	Gros Michel	2.948	2.701	2.468	2.153	1.589
6.	Koduppilla kunnan	3.301	2.777	2.425	2.003	1.637
7.	Poocha kunnan	3.148	2.895	2.277	2.072	1.713
8.	Adakka kunnan	3.213	2.837	2.461	1.974	1.739
9.	Red banana	3.576	2.965	2.393	2.048	1.769
10.	Pisang lilin	3.599	3.028	2.386	1.887	1.813
11.	Chinie	3.290	2.708	2.314	1.988	1.514
12.	Matti	3.477	2.783	2.385	1.600	1.403
13.	Bodles Altafort	3.607	3.249	2.648	2.121	1.559
14.	Kanchikela	3.439	3.095	2.567	2.009	1.661
15.	Nendra padaththi	3.489	3.134	2.742	2.135	1.578
16.	Boodida bontha batheas	3.838	3.391	2.911	2.534	2.158
17.	Hybrid sawai	3.806	3.230	2.921	2.227	2.152
18.	Peyan	3.389	3.118	2.774	2.351	2.081
19.	Kapok	3.311	2.919	2.526	2.033	1.724
20.	Pisang mas	3.232	2.869	2.544	1.808	1.325
21.	Vennaettu nannan	3.193	2.923	2.448	1.869	1.426
22.	Klue teparod	3.645	3.550	2.742	2.141	1.813
23.	Jurmani kunthali	3.385	2.979	2.617	1.845	1.317
24.	Nendran	3.282	2.872	2.463	2.027	1.495
25.	Zanzibar	3.455	3.002	2.695	2.222	1.665

CD (0.05) between variety = 0.2003

(Contd.....)

Table 11. Contd.....

Conclusion :

Comparison of varieties at

(1) immediately after female phase

16 17 22 13 10 9 15 12 25 14 18 23 3 19 6 11 24 20 8 21 7 1 2 4 5

(2) one-fourth maturity

22 16 13 17 15 18 14 3 10 25 23 9 21 19 7 2 24 20 1 8 12 6 4 11 5

(3) half maturity

17 16 18 15 22 25 13 23 14 20 19 3 4 5 24 8 21 6 9 10 12 2 11 7 1

(4) three-fourth maturity

16 18 17 25 5 22 15 13 4 7 3 9 19 24 14 6 11 8 1 2 10 21 23 20 12

(5) full maturity

16 17 18 10 22 9 8 19 7 3 25 14 16 2 5 15 4 13 1 11 24 21 12 21 23

Crude protein content was decreased slightly in one-fourth phase when compared to former phase. The maximum crude protein was present in Klue taparod (3.550 per cent) and minimum was in Gros Michel (2.701 per cent).

At half maturity, the crude protein content further reduced and highest amount was found in Hybrid sawai (2.921 per cent) and minimum was in Robusta (2.238 per cent).

Again crude protein content decreased in three-fourth maturity when compared to half maturity. The maximum and minimum percentages of crude protein were observed in Koodida bontha bathees (2.534 per cent) and Matti (1.600 per cent) respectively.

At full maturity phase highest amount of crude protein was observed in Koodida bontha bathees (2.158 per cent) and minimum was found in Jurmani kunthali (1.317 per cent).

The statistical analysis revealed that there was significant difference among the varieties with respect to crude protein content. The maximum amount was observed in female phase in all the varieties and minimum was found at full maturity. The varieties are arranged in descending order in all the five developmental stages with respect to their crude protein contents and presented in Table 11.

4.6. Tannin

Maximum tannin content was observed at female phase and was found to vary with different varieties. At this

phase, highest amount of tannin was noticed in Palayankodan (2.697 per cent) and the minimum was in Red banana (1.820 per cent). The tannin content steadily reduced in all varieties from female phase to full maturity stage (Table 12).

The tannin content was slightly reduced at one-fourth maturity when compared to the female phase. Palayankodan maintained maximum amount (2.532 per cent) and minimum was seen in Pisang mas (1.594 per cent).

At half maturity, tannin was further reduced and here also maximum was observed in Palayankodan (2.233 per cent) and minimum was in Vannesttu mannan (1.293 per cent).

Tannin again reduced at three-fourth maturity and Palayankodan maintained highest amount (1.916 per cent) and Dwarf Cavendish showed minimum amount (0.749 per cent).

At full maturity stage, the tannin content further decreased compared to the former stage. The maximum tannin was present in Matti (0.989 per cent) and minimum was seen in Dwarf Cavendish (0.396 per cent).

Statistical analysis showed that there was significant difference among the varieties with respect to tannin content in all the five developmental stages of the fruit. The maximum tannin content was observed in female phase and minimum was found in full maturity stage. The varieties in all the five developmental stages of the fruit are arranged in descending order with respect to their tannin content and presented in Table 12.

Table 12. Tannin at different stages of fruit maturity, per cent

Sl. no.	Variety	Immediately after female phase	One-fourth maturity	Half maturity	Three-fourth maturity	Full maturity
1.	Robusta	2.311	2.032	1.864	1.746	0.869
2.	Dwarf Cavendish	2.146	1.968	1.765	0.749	0.596
3.	Palayankodan	2.697	2.532	2.233	1.916	0.845
4.	Njali poovan	2.155	1.986	1.939	0.977	0.869
5.	Gros Michel	2.206	1.928	1.627	0.871	0.737
6.	Koduppilla kunnan	2.035	1.754	1.541	1.167	0.753
7.	Poovha kunnan	2.049	1.763	1.586	0.974	0.748
8.	Adakka kunnan	2.128	1.723	1.352	1.005	0.698
9.	Red banana	1.820	1.665	1.338	0.859	0.623
10.	Pisang lilin	2.319	2.094	1.849	0.966	0.787
11.	Chinia	1.932	1.721	1.310	0.976	0.765
12.	Matti	2.216	1.936	1.944	1.189	0.989
13.	Bodles Altafort	1.977	1.730	1.472	1.179	0.988
14.	Kanchikelu	2.335	2.040	1.964	1.825	0.876
15.	Nendra padaththi	2.218	1.962	1.615	1.841	0.833
16.	Boodida buntha buthees	2.327	2.035	1.893	1.738	0.977
17.	Hybrid sawai	2.046	1.701	1.564	1.180	0.861
18.	Peyan	2.456	2.160	1.945	1.701	0.826
19.	Kapok	2.048	1.798	1.625	1.303	0.963
20.	Pisang mas	1.837	1.594	1.480	1.273	0.946
21.	Vennettu mannan	1.954	1.618	1.293	1.166	0.905
22.	Klue taparod	2.344	2.024	1.991	1.613	0.854
23.	Juxmani kunthali	2.651	2.453	2.105	1.890	0.979
24.	Nendran	2.262	1.992	1.819	0.971	0.795
25.	Zanzibar	2.172	1.983	1.593	0.994	0.787

CD (0.05) between varieties = 0.0606

Contd.....

Table 12. Contd.....

Conclusion :

Comparison of varieties at

(1) immediately after female phase

3 23 18 22 14 16 10 1 24 15 12 5 25 4 2 8 7 19 17 6 13 21 11 20 9

(2) one-fourth maturity

3 23 18 10 14 16 1 22 24 4 25 2 15 12 5 19 7 6 13 8 11 17 9 21 20

(3) half maturity

3 23 22 14 18 12 4 16 1 10 24 2 5 19 15 25 7 17 6 20 13 8 9 11 21

(4) three-fourth maturity

3 23 15 14 1 16 18 22 19 20 17 13 12 6 21 8 25 4 7 24 11 10 5 9 2

(5) full maturity

12 13 23 16 19 21 14 1 4 17 22 20 3 15 18 24 25 10 11 6 7 5 8 9 2

At ripe stage, the reducing sugar content and total sugar content reached highest level in all the varieties tested. But starch and crude fibre contents, though increased from immediately after female phase to full maturity, fell down sharply at ripe stage. However, tannin and crude protein contents were minimum in all varieties at ripe stage. The results are presented in Table 13.

Influence of chemical constituents of banana fruit on infection and intensity of disease

The major chemical constituents were found to have a lot of variation from the female phase to ripening stage. The reducing sugars and total sugars steadily increased from female phase to ripe stage. The starch and crude fibre were having an increasing tendency till the full maturity and during ripening, these two components showed sudden decrease. The tannin and crude protein have shown a steady decrease from female phase to ripening stage.

The infection pattern of C. gloeosporioides in different stages of development of fruit has shown much variation. At the stage immediately after female phase, the pathogen did not infect the fruit even after giving an injury indicating immunity to disease at this stage. At one-fourth maturity and half maturity stages, the pathogen

Table 13. Major chemical constituents at ripe stage of different banana varieties, per cent

Sl. no.	Variety	Reducing sugars	Total sugar	Starch	Crude fibre	Crude protein	Tannin
1.	Robusta	6.930	16.414	0.763	2.176	1.265	0.532
2.	Dwarf Cavendish	6.903	16.210	0.992	2.004	1.242	0.505
3.	Palayankodan	13.312	17.039	2.131	1.701	1.342	0.829
4.	Njali poovan	7.454	17.098	1.457	1.579	1.345	0.707
5.	Gros Michel	9.420	18.580	1.950	0.809	1.343	0.610
6.	Koduppilla kunnan	10.626	13.155	1.243	1.579	1.260	0.683
7.	Poocha kunnan	9.708	12.418	0.887	1.709	1.127	0.639
8.	Adakka kunnan	9.443	11.767	1.832	1.454	1.149	0.501
9.	Red banana	7.771	9.797	1.985	1.287	1.028	0.533
10.	Pisang lilin	6.282	11.185	1.243	1.034	0.953	0.409
11.	China	6.255	18.077	1.598	0.809	0.927	0.670
12.	Matti	7.575	13.478	1.578	1.402	0.981	0.751
13.	Bodles Altafort	7.122	12.684	1.595	0.922	1.173	0.753
14.	Kanchikela	8.784	13.332	1.390	1.029	1.022	0.498
15.	Nendra padaththi	6.108	14.546	1.375	1.215	1.099	0.571
16.	Boodida bontha bathees	8.423	13.429	2.587	0.915	1.408	0.653
17.	Hybrid sawai	8.974	15.243	3.542	1.053	1.503	0.496
18.	Peyan	6.062	16.811	3.219	1.252	1.315	0.662
19.	Kapok	7.672	13.505	4.746	0.811	0.879	0.637
20.	Pisang mas	6.648	13.261	5.491	1.373	1.029	0.566
21.	Vennaettu mannan	8.242	13.538	3.521	0.767	1.001	0.741
22.	Klue teparod	8.966	14.845	4.560	1.261	1.167	0.729
23.	Jurmani kunthali	5.816	12.947	2.033	1.345	1.049	0.717
24.	Nendran	6.679	11.472	4.485	1.046	1.348	0.862
25.	Zanzibar	5.605	10.826	5.869	0.966	1.228	0.789

could enter the host tissues only after giving pin prick injuries and that too in few varieties only, showing that these banana fruit were resistant to the fungus (Tables 2 & 3). When the fruit became more mature and reached three-fourth maturity most of the varieties were found to be more susceptible. At this stage, fifteen varieties have taken infection when uninjured and all varieties except Palayankodan and Jurmani kunthali have taken infection when injured (Table 4). When the fruit became fully mature, all the varieties were found to be highly susceptible and even without injury the pathogen could infect all the varieties (Table 5).

In general, younger fruit were more resistant to the pathogen and when the fruit are more matured the susceptibility increased.

In younger stages, the tannin and crude protein were maximum and these two constituents decreased towards maturity. On the other hand, reducing sugars, total sugars, crude fibre and starch increased till full maturity. This clearly indicates that when the tannin and crude protein contents were high, the resistance of the fruit against the pathogen was very high. When the reducing sugars, total sugars, crude fibre and starch increased in all the stages of fruit development till maturity, the susceptibility also increased. Thus when these chemical constituents namely reducing sugars,

total sugars, starch and crude fibre were more, the fruit became more susceptible to anthracnose disease.

As far as the disease intensity is concerned, from one-fourth maturity to full maturity there is an increasing tendency. The reducing sugar, total sugar, starch and crude fibre were found to increase from immediately after female phase to full maturity in all varieties tested. But tannin and crude protein were found to decrease till full maturity (Tables 7, 8, 9, 10, 11 and 12).

The statistical analysis of the data showed that there was positive correlation between intensity of disease with reducing sugars and total sugars. Even though the starch and crude fibre contents increased they did not show any significant correlation with the disease intensity (Tables 14, 15 and 16).

The crude protein and tannin were found to decrease towards the maturity and there was an increase in disease intensity from one-fourth to full maturity. Statistical analysis of the data clearly indicated that there was a negative correlation between crude protein and tannin content of the fruit and disease intensity (Tables 14, 15 and 16) (Figures 5, 6 and 7).

The coefficients of correlation between percentage intensity of infection (when injured) and major chemical constituents at one-fourth maturity were 0.0908, 0.2289, -0.0299, -0.0621, -0.3329 and -0.4952 for reducing sugars,

Table 14. Relationship between chemical constituents (x) and per cent disease intensity (y) at one-fourth maturity (injured)

Chemical constituents	Correlation coefficient(r)	Regression equation y = a + bx
Reducing sugar	0.0908	-
Total sugar	0.2289	-
Starch	-0.0299	-
Crude fibre	-0.0621	-
Crude protein	-0.3329	-
Tannin	-0.4952*	y = 2.4422 - 1.1518 x

* Significant at 5 per cent level

Table 15. Relationship between chemical constituents (x) and per cent disease intensity (y) at half maturity (injured)

Chemical constituents	Correlation coefficient (r)	Regression equation y = a + bx
Reducing sugar	0.1572	-
Total sugar	0.1180	-
Starch	-0.3322	-
Crude fibre	0.1311	-
Crude protein	-0.4052*	y = 4.8454 - 1.8088 x
Tannin	-0.6758**	y = 1.8462 - 0.9189 x

* Significant at 5 per cent level

** Significant at 1 per cent level

Table 16. Relationship between chemical constituents (x) and per cent disease intensity (y) at three-fourth maturity

Chemical constituents	Nature of inoculation	
	injured	uninjured
	Correlation coefficient (r)	Correlation coefficient (r)
Reducing sugar	0.4559*	0.2813
Total sugar	0.4238*	0.3123
Starch	-0.3481	-0.3040
Crude fibre	0.1433	0.1429
Crude protein	-0.3317	-0.3152
Tannin	-0.3740	-0.4878*

* Significant at 5 per cent level

FIG 5. Relationship between tannin per cent (x) and per cent disease intensity (y) at one-fourth maturity (injured)

$r = -0.4952^{**}$

$y = 2.4422 - 1.1518x$

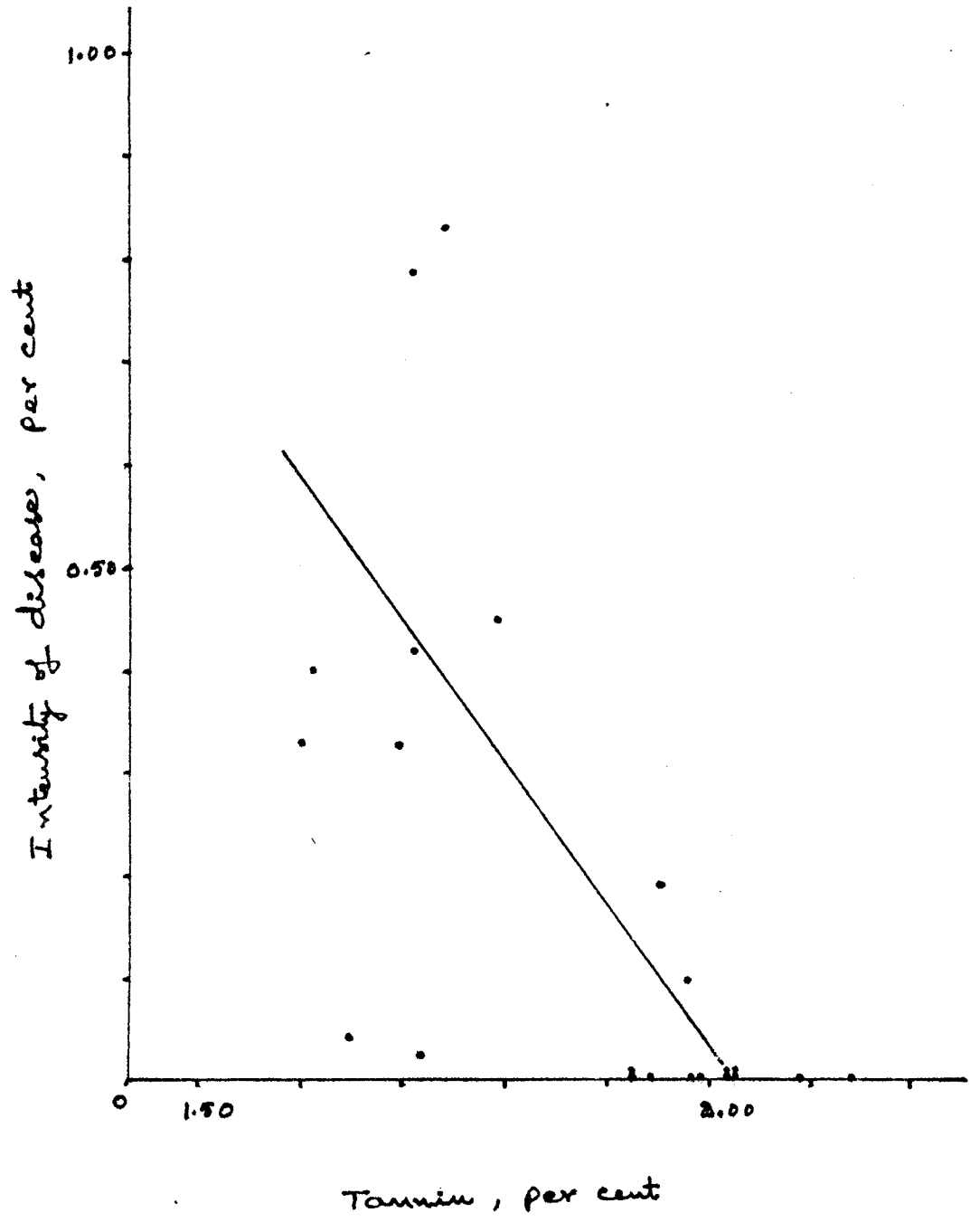


FIG 6. Relationship between Crude protein per cent (x) and per cent disease intensity at half maturity (inoculated)

$r = -0.4052^{**}$

$y = 4.8454 - 1.8068x$

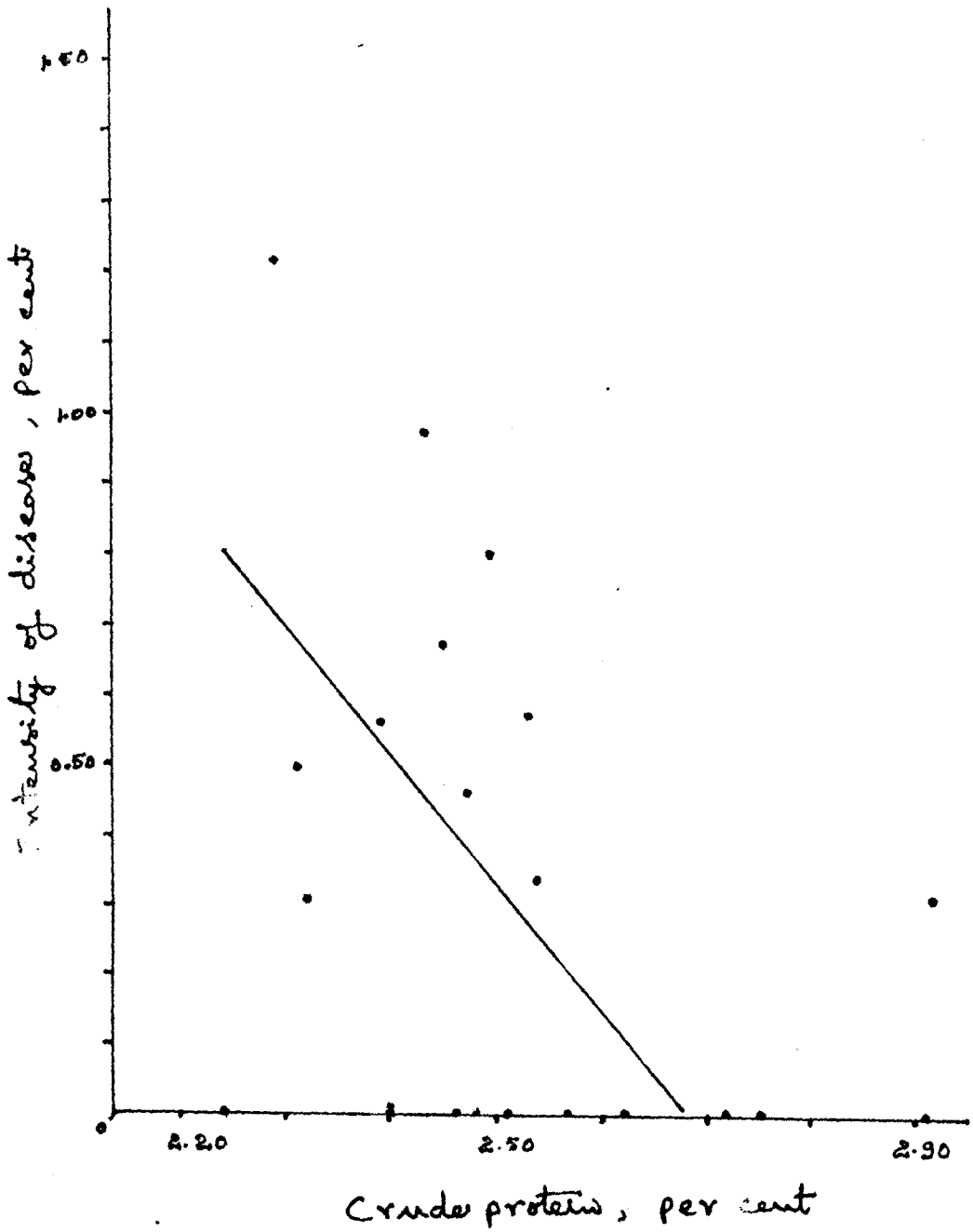
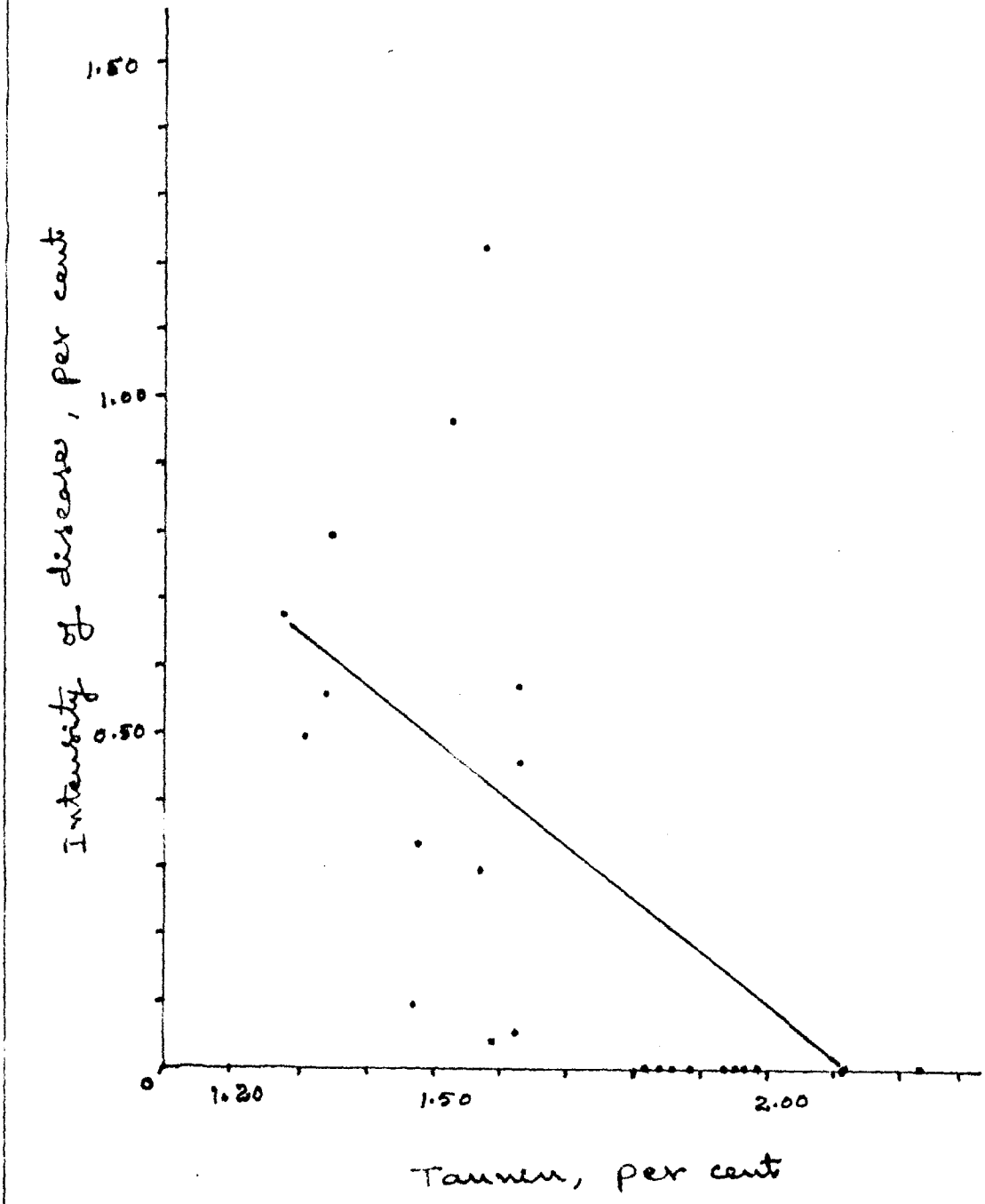


FIG 7. Relationship between tannin per cent (x) and per cent disease intensity (y) at half maturity (ungrafted)

$r = -0.6758^{**}$

$y = 1.0462 - 0.3188x$



total sugar, starch, crude fibre, crude protein and tannin respectively. All these values were not significant at five per cent level except for tannin.

The coefficients of correlation between percentage intensity of disease (when injured) and major chemical constituents at half maturity stage were 0.1572, 0.1180, -0.3322, 0.1311, -0.4052 and -0.6758 for reducing sugars, total sugar, starch, crude fibre, crude protein and tannin respectively. All these values were not significant at five per cent level except for tannin and crude protein.

At three-fourth maturity stage, the coefficients of correlation between percentage disease intensity and major chemical constituents were 0.4559, 0.4238, -0.3481, 0.1433, -0.3317 and -0.3740 when injured and 0.2813, 0.3123, -0.3040, 0.1429, -0.3152 and -0.4878 (when injured) for reducing sugar, total sugar, starch, crude fibre, crude protein and tannin respectively. All these values were not significant at five per cent level except for reducing sugar, and total sugars (when injured) and tannin (uninjured).

At full maturity stage, the correlation coefficients between percentage intensity of the disease and major chemical constituents were 0.1624, 0.1611, -0.1775, -0.3375, -0.1359 and 0.0172 when injured and 0.1481, 0.1897, -0.1744, -0.2927, -0.1007 and 0.0421 for reducing sugars, total sugar, starch, crude fibre, crude protein and tannin respectively.

All these values were found to be not significant at five per cent level.

Thus reducing sugars and total sugars were major chemical constituents responsible for susceptibility of the disease and crude protein and tannin were responsible for resistance to the disease.

Discussion

DISCUSSION

The anthracnose disease of banana caused by C. *gloeosporioides*, the imperfect stage of C. *cingulata* is mainly considered as an important post harvest disease of banana. However, this disease can also cause damage to the crop in the field. In the present investigation an attempt has been made to study the disease *in vivo* and *in vitro*.

In the field, the pathogen was found to attack immature banana fruit and leaves. However, during the present study no leaf infection was observed. The symptom expression was seen on young immature banana fruit and infection usually occurred on the distal end of the fruits and later spread downwards. If the infection was noticed on the skin of the banana fruit, the initial symptoms were chlorotic specks, shortly becoming necrotic areas which further developed and increased in size. Later, these areas became depressed, coalesced forming large patches. In severe infections, under humid conditions, the conidial masses appeared on necrotic areas. The infected fruit became blackened, shrivelled and mummified. Generally, the spread of the disease was restricted to few hands but rarely whole bunches also. The present findings agree with the description given by earlier workers (Cobb, 1906; Dastur, 1916; Agati, 1922; Park, 1930 and 1933; Chona, 1933; Roy and Sharma, 1952; Damodaran and Ramakrishnan, 1958).

The infected banana fruits generally exhibited conspicuous symptoms after the harvest. Here the symptoms appeared as small, irregularly round, brown coloured spots contrasting with the yellowing skin. This indicates that the symptoms will be more pronounced during the ripening stage. At this stage these spots enlarged very quickly, ultimately becoming dark brown to black in colour. These spots coalesced together forming large patches and covered the fruit within a few days. The activity of the pathogen was more and the production of large masses of acervuli was observed on infected fruits. Several workers (Wardlaw and Mc Guire, 1931; Wardlaw, 1935 and Meredith, 1960) have described the post harvest symptoms of banana fruit when attacked by this pathogen. Present observation confirms the earlier findings.

Desmazieres and Montagne (Ann. Sci. Nat. 3:12:235, 1849) created the genus Gloeosporium to accommodate straight spored acervulus fungi which are not having any setae or appendages on the acervuli (Sacc, 1884). Later on, lot of alteration, modification and segregation of the genus occurred. The modern concept of the genus was established on experimental basis by Shear and Wood (1913) resulting merger of large number of Gloeosporium to the earlier created genus Colletotrichum Corda. (Sturm's Deutschl. Kr. F Part 3; III; 41, 1837). A wide range of hosts belonging to many families of higher plants has been reported to be

infected by different species of Colletotrichum and Gloeosporium (Ibrahimov, 1951; and Morgan, 1956). A large number of Colletotrichum and Gloeosporium species till then distinguished by slight and inconsistant morphological characters and supposed fixity of host relationships were made synonymous with G. gloeosporioides Penz (Sacc), the conidial phase of G. cingulata (Stonem) Spauld and Shrenk.

The above workers also found that in different isolates of G. gloeosporioides a straight spored species, there is a great variation in size, shape, setae, appressoria and morphology of the fructification. The inoculation experiments also showed most of the isolates would produce characteristic symptom on the other hosts and further, there is evidence for strain differentiation among the isolates which were examined.

Thus, a broad concept of these two particular genera came into being, with the morphological characters in artificial cultures, natural infection and cross inoculation tests on a wide range of hosts determining the limits of variation. Arx (1957a) finally undertook a comprehensive taxonomical studies in which he expanded the synonymy of G. cingulata, the conidial form of G. gloeosporioides into 600 names which included about 100 species of Gloeosporium.

When Cooke and Massee gave a new specification for the fungus they studied on banana fruit, G. musarum (Sacc, 189 a very scanty description was given. Further studies on

this fungus were conducted by Krüger (1913), Agati (1922), Toro (1922), Ashby (1931) and Wardlaw (1935). But none of the workers has emphasised on taxonomical position of this pathogen. Krüger (1913) observed the ascospores of this pathogen, but he did not identify the perfect stage. Ashby (1931) had given a detailed morphology of ascigerous stage of G. musarum and identified as G. cingulata, but he did not rearrange the imperfect stage. Wardlaw (1935) also observed the ascomycetous stage. There also, no taxonomical rearrangement of the imperfect stage was made. Lastly Arx (1957b) undoubtfully named the imperfect stage of the G. cingulata as C. gloeosporioides and no other imperfect stage was observed for this ascomycetous fungus. Perusal of literature showed that even after making G. musarum as a synonym of C. gloeosporioides, it is known in two different names, G. musarum and C. musae.

In the present study, the imperfect and perfect stages description of this pathogen fully agreed with the description given by Krüger (1913), Agati (1922), Toro (1922), Ashby (1931), Wardlaw (1935), Jain (1950), Damodaran and Ramakrishnan (1958). Thus it is very reasonable and realistic to name the pathogen as C. gloeosporioides, the imperfect stage of G. cingulata.

The strain variation proposed by Toro (1922) and Ashby (1931) on the basis of slight variation in cultural characters and dimensions of the spores cannot be accepted for a pathogen like C. gloeosporioides which is having more than 1,000 host range. Only after a detailed study of severity of pathogenecity, host ranges and variations in the above aspects if any one can propose strains and forms. Till such a detailed study is made, there is no justification making this pathogen into different strains.

Twenty-five banana varieties were screened in vitro against the anthracnose disease caused by C. gloeosporioides. As the pathogen is considered to be weak, it enters the host tissues mainly through injury (Toro, 1922; Park, 1930; Simmonds and Mitchell, 1940; Meredith, 1960). Therefore, two types of inoculation methods namely with injury and without injury were adopted. The infection was more in the case of injured fruit in all the cases when compared to the uninjured ones. In the early stages of fruit development (Tables 2 and 3), the infection was noticed only after giving injury except in the case of female phase. Without injury, no infection was observed on fruit at one-fourth and half maturity stages. At three-fourth maturity stage, all the varieties took infection when injury was made except Palayankodan and Jurmani kunthali. But ten varieties namely Robusta, Palayankodan, Kanchikela, Nendra padaththi, Boodida bontha batheea, Peyan, Kapok, Pisang mas,

Klua teparod and Jurmani kunthali have not taken infection when injury was not given. At full maturity stage, the pathogen infected all the varieties even without any injury but in injured fruit , the symptom expression was very fast. These results clearly indicated that when there was an injury, the infection was too high and without injury only highly susceptible varieties were infected. These findings confirm the earlier observations that infection is mainly due to fruit injury (Toro, 1922; Park, 1930; Simmonds and Mitchell, 1940; Meredith, 1960; Cardemosa - Barriga, 1964; Meredith, 1970, Wardlaw, 1972). However, the varieties which were harvested at full maturity stage were infested even without injury coinciding with the earlier reports (Toro, 1922; and Deighton, 1935).

There was no infection of this pathogen during the female phase even after injury was made on the fruit. This is perhaps due to the chemical constituents of the fruit during that stage. In general, in all the varieties tested, the crude protein and tannin contents were high and reducing sugars, total sugars, starch and crude fibre were very low (Tables 7, 8, 9, 10, 11 and 12). The variation and proportion of chemical constituents of the fruit at this stage might have attributed to the adverse condition for penetration and development by this pathogen.

During one-fourth phase only twelve varieties got infected after injuring the fruit at the time of inoculation (Table 2). The low symptom expression was observed only after six days of inoculation. This indicates that six days after harvest of the fruit, some biochemical changes probably have occurred facilitating favourable conditions for development of the pathogen (Tables 7, 8, 9, 10, 11 and 12).

At half maturity, only injured fruits took infection and the number of varieties infected also increased to fourteen (Table 3). At this stage also, visible symptoms on the inoculated fruits were observed only after six days of inoculation. The reason attributable for low and delayed infection may be the same as in one-fourth maturity. Wardlaw (1931) stated that in less matured fruits, the penetration and development of the pathogen remained very limited and the present study confirms this observation.

During three-fourth maturity, fifteen varieties, got infected without any injury and twenty-three varieties were infected when injury was made (Table 4). In case of injured fruits, the symptom expression was noticed four days after inoculation while uninjured fruits took infection after six days. During this stage, crude protein and tannin contents decreased while the contents of sugars, starch and crude fibre increased.

These might have attributed for the increased infection. However, two varieties, Palayankodan and Jurmani kunthali did not show any infection even after injury was made. Crude protein and tannin contents of these two varieties were high when compared to the highly susceptible variety Dwarf Cavendish and sugars were found to be low (Tables, 7, 8, 11 and 12). The symptom expression steadily increased upto eight days and after eight days, the increase of disease intensity was very rapid ie., by that time fruit started ripening, reducing and total sugars increased considerably and starch content declined. The crude fibre, crude protein and tannin reached the minimum amounts during ripening of the fruit (Table 13). It is probable that due to these bio-chemical changes occurred during the ripening, the pathogen is able to grow very rapidly thereby increasing the intensity of disease considerably.

At full maturity, both the injured and uninjured fruit of all 25 varieties have shown varying degrees of infection and the symptom expression was noticed only after three days in the injured fruit and four days after inoculation in case of uninjured fruit. Later on the symptom expression was very rapid in all the varieties. This may be due to the fact that, by this time, the fruit are almost ripened. The reducing sugars and total sugars were considerably increased at the expense of starch reserves. The contents of tannin

crude protein and crude fibre decreased to the minimum which favoured the rapid growth of the pathogen resulting in quick expansion of symptoms on the ripened fruit (Table 13). After eight days of inoculation, 100 per cent infection was observed on all the 25 varieties both in case of injured and uninjured fruit. But there was much variation among the varieties, when observations were taken after six days of inoculation. To get a comparable susceptibility/tolerance limit during this stage, the observations were restricted on sixth day and statistical analysis was carried out. Here, out of 25 varieties tested, Njali poovan was found to be the most susceptible variety and the varieties Palayankodan and Nendra padaththi were the least susceptible ones.

All the 25 varieties tested had shown varying degree of susceptibility during the different stages of development. During early developmental stages ie., one-fourth and half maturity stages, the varieties Koduppilla kunnan and Poocha kunnan were found to be most susceptible ones and the varieties Robusta, Palayankodan, Njali poovan, Pisang lilin, Matti, Kanchikela, Boodida bontha bathees, Peyan, Klue teperod, Jurmani kunthali and Nendran were the highly resistant ones. But these varieties did not show that trend during further developmental stages. However, at three-fourth maturity, the varieties Dwarf Cavendish and

Njali poovan were the most susceptible ones and the varieties Nendra padaththi, Palayankodan and Jurmani kunthali were the least susceptible ones (Table 4). At full maturity stage almost the same trend was observed as that of three-fourth maturity and the varieties Njali poovan, Robusta and Sodlea Altafort were found to be highly susceptible and least susceptible varieties were the same as that of three-fourth maturity (Table 5).

When all the developmental stages of the fruit were taken into consideration, all the 25 varieties were found to be susceptible with varying degrees of disease intensity. The variety Njali poovan was found to be highly susceptible, followed by Pisang lilin, Dwarf Cavendish, Matti and Gros Michel. The varieties Nendra padaththi and Palayankodan showed high resistance to the disease with lowest disease intensity percentages of 0.401 and 0.423 respectively. The variety Jurmani kunthali can also be categorized along with these two varieties with 0.605 per cent disease intensity. The chemical constituents of these varieties are presented in Tables 7, 8, 9, 10, 11, 12 and 13. The varieties Soodida bontha bathees, Peyan, Kanchikela, Pisang mas and Kapok were also found to be highly resistant having less than two per cent disease intensity. The varieties Zanzibar, Adakka kunnan, Klue taparod, Chinia, Nendran, Venneettu mannan, Koduppilla kunnan,

Hybrid sawai, Poocha kumnan, Red banana and Bodles Altafort were found to be resistant and their disease intensities were in the range of two to five per cent. None of the varieties tested was found to be moderately resistant (5 to 10 per cent disease intensity) or moderately susceptible (10 to 15 per cent disease intensity). The variety Robusta was found to be susceptible showing disease intensity in the range of 15 to 25 per cent. The highly susceptible varieties showed more than 25 per cent disease intensity. They were Gros Michel (27.028 per cent), Matti (27.077 per cent), Dwarf Cavendish (27.123 per cent), Pisang lilin (27.356 per cent) and Njali poovan (39.107 per cent). Perusal of data on chemical constituents of these varieties revealed that there is very little variation among them (Tables, 7, 8, 9, 10, 11, 12 and 13).

The reducing sugar content of the fruit was found to be minimum in all the twenty-five varieties (0.540 to 1.412 per cent) during the female phase but they increased during further development stages. However the increase was very slow. At full maturity stage, the reducing sugar content was observed to be ranging from 1.282 to 2.935 per cent only. The total sugars also showed the same tendency from female phase to ripening stage. However, the starch content during the female phase was very low in all the 25 varieties (1.003 to 1.568 per cent) and there was a steady increase upto

full mature stage. The maximum starch was obtained during full mature stage in all the 25 varieties which ranged from 17.185 to 33.215 per cent. In the early developmental stages, the increase was low but during the later stages, the starch content increased markedly. This finding fully supports the investigation of Lodh et al. (1977) who worked on Dwarf Cavendish banana. When the fruits started ripening, most of the starch got hydrolysed resulting in sudden increase of reducing sugars and total sugars. The starch content of these 25 varieties was depleted to low amounts during ripening (varied from 0.763 to 5.869 per cent). This has resulted in marked increase of both reducing sugars (varied from 5.605 to 13.312 per cent) and total sugars (varied from 10.826 to 18.580 per cent). This kind of phenomenon was observed by several early workers who reported that matured fruits will have maximum starch and during ripening it was converted to different sugars (Belevel, 1932; Leonard and Barnell, 1933; Loesecke, 1950; Young and Ho, 1958; Simmonds, 1966 and Singh et al. 1980). The present investigation fully supports the earlier findings.

More sugar content was observed during ripening stage in sweet varieties like Robusta (16.414 per cent), Dwarf Cavendish (16.210 per cent), Palayankodan (17.039 per cent), Njali poovan (17.098 per cent), Gros Michel (18.580 per cent) and Chinia (18.077 per cent).

The starch content in these varieties was depleted to very low amounts during this period ie., 0.763 per cent in Robusta, 0.992 per cent in Dwarf Cavendish, 2.131 per cent in Palayankodan, 1.457 per cent in Njali poovan, 1.950 per cent in Gros Michel and 1.598 per cent in Chinia. The culinary varieties were found to have high starch content during full maturity stage. Here also the starch content depleted during ripening stage which was found to be little higher than the sweet varieties. They were Kapok (4.746 per cent), Pisang mas (5.491 per cent), Klue teparod (4.560 per cent), Nendran (4.485 per cent) and Zanzibar (5.869 per cent). All other varieties showed less than four per cent starch during ripening. It is very interesting to note that all culinary and dual purpose varieties contained high starch at full maturity and only a part of it was hydrolysed. This finding is supporting the observations of Simmonds (1966) who stated that hydrolysis of starch is more in dessert bananas than in culinary bananas during the ripening of the fruit.

The status of tannin during early stages was found to be high and it gradually reduced as the maturity advanced and it was minimum during ripening stage. During the female phase the tannin content of 25 varieties ranged from 1.820 per cent to 2.697 per cent and at full maturity, it varied from 0.596 per cent to

0.989 per cent. Further, the tannin content reduced during ripening time and it varied from 0.409 per cent to 0.862 per cent. The astringency character of unripe fruit is due to fairly large content of tannin. The softness of fruits after ripening is due to low content of tannin. Loss of astringency occurring during ripening is due to polymerisation of polyphenols. The reduction of polyphenols about 1/5th of its value from the green hard fruit to the soft ripening fruit was observed by few earlier workers (Barnell and Barnell, 1945; Goldstein and Swain, 1963). The present study fully supports their findings.

The crude protein content was more in all the varieties during early stages of development. During female phase, the crude protein content in different varieties ranged from 2.948 to 3.838 per cent. Till ripening steady decrease was obtained and reached minimum during ripening which varied from 0.879 to 1.503 per cent. Perusal of the available literature showed that the importance of crude protein for the quality of the banana fruit or its relation to anthracnose disease resistance was not investigated by earlier workers. In the present investigation, an attempt has been made to study the crude protein status of fruit at different stages of development.

The status of crude fibre was just reverse compared to that of crude protein. The minimum crude fibre was observed in female phase which ranged from 1.526 to 4.319 per cent. There was a steady increase till the ripening stage where the status of crude fibre was found to be maximum in all the 25 varieties analysed. When the fruit started ripening, the crude fibre content decreased considerably and it was too low in ripened fruit which ranged from 0.767 to 2.176 per cent. A perusal of literature did not reveal the status of this chemical constituent during developmental stages of banana fruit and its importance in the anthracnose disease incidence.

There are good number of reports stating that the chemical constituents of fruit and leaves have a direct relationship to infection and intensity of development of disease caused by C. gloeosporioides, the imperfect stage of C. cinculata. Results of the present investigation also revealed that the chemical constituents of banana fruit have high influence on the anthracnose disease incidence and intensity. Among the chemical constituents tested, reducing sugar, total sugar, crude protein and tannin have profound influence on the disease intensity. The reducing sugar and total sugar have clearly showed a positive correlation as far as the disease is concerned. But the correlation coefficients were found to be significant only during three-fourth

maturity (Table 16). During the early developmental stages of banana fruit, the sugars were present in very low amounts and the disease intensity was also very low in the early stages. The pathogen did not infect the fruit in many varieties. More over even if pathogen gets entry the development of symptom was limited in these stages of fruit development. At three-fourth maturity the coefficients of corfelation between percentage disease intensity and reducing sugars and total sugar (injured) were found to be 0.4559 and 0.4238 respectively (Table 16).

The delay of the development of symptoms on fruit in earlier stages may be due to the fact that the carbohydrates were present in very small amounts. During ripening, hydrolysis of starch occurred leading to formation of sugars and it was found to be essential for good symptom expression. When the fruit started ripening, the symptom expression was rapid in all the 25 varieties tested and within eight days after inoculation, cent per cent infection was noticed in most of the varieties. By that time, almost all the starch content present in fruit was converted into sugars and maximum sugar content was observed at ripening stage. The earlier workers (Agati, 1922; Toro, 1922; Wardlew, 1931; Damodaran and Ramakrishnan, 1964 and Thakur, 1969) also observed the same trend and stated that the pathogen is more active only when the sugar contents of the fruit are more.

The influence of high sugar content on infection by *C. gingulata* was also observed on other fruits like apple by Sitterly and Shay (1960) and High bush blue berry fruit by Stretch and Cappellani (1965). They reported that high infection by *C. gingulata* occurs when the sugar content is high in these fruits. The influence of sugar in leaves of Grape vine on anthracnose disease intensity was observed by Mohanraj *et al* (1972). They found that high sugar is responsible for high disease intensity due to infection by *C. gloeosporioides* (*Gloeosporium ampelophagum*).

The importance of polyphenols in the disease resistance and susceptibility is a well established factor in plant pathology. High polyphenol contents always give resistance to the plants especially against fungal pathogens (Wood, 1967). In banana fruit the tannin content was found to be very high during early stages of development and steadily decreased during final maturity and ripening. The tannin content has a negative correlation with the disease intensity in the banana fruit. Significant correlation was obtained mostly in early stages of fruit development when the tannin content was very high (Tables 14 and 15) (Figure 5 and 7). The correlation coefficients for tannin at one-fourth maturity and half maturity (injured) were found to be -0.4952 and -0.6758 respectively. The corresponding value at three-fourth maturity (uninjured) was -0.4878.

At full maturity and ripening stages, the tannin content was very low and at these stages, fruit were found

to be more susceptible and development of symptom was very rapid. The findings of earlier workers (Cook and Taubenhaus, 1911, Chakravarty, 1957; Green and Morales, 1967; Raghunathan et al. 1966)also showed that high tannin content adversely affected the infection and spread of anthracnose disease on banana.

However Toro (1922) did not get any correlation between tannin content of peel of banana fruit and infection by C. musarum. High status of tannin was observed in immature apple fruit and at this stage, the pathogen C.gloeosporioides could not infect the fruit (Sitterly and Shay, 1960). Similarly, Cheeran (1974) observed that on tender leaves of Tectona grandis having high content of polyphenols during that stage, the pathogen C. gloeosporioides could not infect. Even if the pathogen infects, the symptom expression is observed only when leaves are matured indicating that the pathogen will be active only when tannin content is low. The present study fully supports the earlier works (Cook and Taibenhaus, 1911; Chakravarty, 1957; Green and Morales, 1967; Raghunathan et al. 1966; Sitterly and Shay, 1960; and Cheeran, 1974).

In the present study, the crude protein content of different banana varieties in various stages was found to have some influence on infection and disease intensity. In the present investigation it has established a negative correlation and was significant only in early stage of maturity (Table 15)(Fig. 6). The co-efficient of

correlation for crude protein at half maturity stage was found to be -0.4052. The available literature did not reveal any influence of crude protein content of banana fruit on the infection of C. gloeosporioides.

Although the chemical constituents like reducing sugars and total sugars have a positive correlation with the disease intensity and the tannin and crude protein contents have a negative correlation against anthracnose disease, the sum total of all these chemical constituents and their relations and ratios will have more response for the susceptibility and resistance characters. One factor alone cannot be taken for determining the relative susceptibility or resistance. Apart from the chemical constituents, the anatomical characters of fruit may also have their own role in infection and disease intensity. Among the responsible factors on the influence of disease, the chemical constituents play only a part. Therefore, it is worth-while to explore the role of anatomical characters of fruit along with chemical constituents to find out the factors influencing the infection and intensity of anthracnose disease.

Summary

SUMMARY

1. The study entitled "Varietal screening of banana against anthracnose disease" was conducted at the College of Horticulture, Vellanikkara during 1981-83. The field studies were conducted at the Banana Research Station, Kannara. The laboratory studies were conducted at the Department of Plant Pathology, College of Horticulture, Vellanikkara, Trichur.

2. The detailed symptomatology of the disease has been studied *in vivo* and *in vitro*.

3. The pathogen was found to attack the young immature fruit in the field. The infection usually takes place at the distal end of the banana fruit and spreads downwards. The initial symptom was chlorotic speck, shortly becoming black circular necrotic area. In advanced stages, the infected fruit became blackened, shrivelled and mummified. Usually the attack of the pathogen is restricted to few hands but in certain cases it affected the whole bunch.

4. After the harvest, symptoms appeared as small, irregular brown coloured spots contrasting with yellow skin, which later became dark brown to black in colour. These spots enlarged quickly and coalesced and large patches were formed. The entire fruit became black in colour in course of time.

5. None of the varieties has taken infection at immediately after female phase. Only injured fruit

took infection during one-fourth and half maturity stages. At three-fourth and full maturity stages, the pathogen took infection without injury also.

6. At one-fourth and half maturity stages, the development of the symptom was very slow when inoculated artificially.

7. The symptom expression at three-fourth and full maturity stages was very fast and same as in the case of post harvest symptoms, when tested *in vitro*.

8. The causal agent of the disease was found to be *Colletotrichum gloeosporioides* Penz (Sacc), the imperfect stage of *Glomerella cingulata* (Stonem) Spauld and Shrenk.

9. On PDA, the growth of the pathogen was luxuriant and large number of acervuli were formed. The conidiogenous cells were found to be phialidic, hyaline and solitary phialids on loose hyphae were common in slide cultures. Conidia are enteroblastic, straight spored and are produced on apex of conidiogenous cells.

10. Numerous perithecia were found in old cultures. The asci are unitunicate, eight spored, clavate to cylindrical, thickened at the apex and narrowed at the base. Ascospores are hyaline, biserially arranged, oval to cylindrical and unicellular.

11. Twenty-five varieties of banana fruit were screened *in vitro* in different stages of development against anthracnose disease.

12. The varieties showed different degrees of susceptibility at various developmental stages of the fruit. The pooled analysis of the data showed that the variety Nendra padaththi followed by Palayankodan, Jurmoni kanthali, Boodida bontha bathees, Peyan, Kanchikela, Pisang mas and Kapok were found to be highly resistant.

13. The varieties Zanzibar, Adakka kunnan, Klue taparod, Chinia, Nendran, Venneettu nannan, Koduppilla kunnan, Hybrid sawai, Pooche kunnan, Red banana and Bodles Altafort were found to be resistant to the disease.

14. The variety Robusta was found to be susceptible. The varieties Njali poovan, Pisang lilin, Dwarf Cavendish, Matti and Gros Michel were found to be highly susceptible.

15. The major chemical constituents of banana fruit viz., reducing sugars, total sugars, starch, crude fibre, crude protein and tannin at different stages of twenty-five varieties were analysed.

16. Minimum content of reducing sugar was found during female phase and maximum during ripening stage. At female phase, the maximum reducing sugar content was observed in Dwarf Cavendish (1.412 per cent) and minimum was in Zanzibar (0.540 per cent). During ripening stage, the highest sugar was present in Palayankodan (13.312 per cent) and lowest was in Zanzibar (5.605 per cent).

17. The total sugar content was minimum at female phase and maximum was observed at ripening stage. At female phase, Koduppilla kunnan contained maximum total sugar (1.659 per cent) and minimum was observed in Nendran (1.101 per cent). During ripening, highest total sugar content was present in Gros Michel (18.580 per cent) and lowest was found in Zanzibar (10.826 per cent).

18. The starch content increased steadily from female phase and reached maximum level at full maturity and it declined at ripening phase. During full maturity stage, maximum starch content was observed in Zanzibar (33.215 per cent) and minimum was in Nendra padaththi (17.185 per cent).

19. The crude fibre content increased steadily from female phase and reached highest amount at full maturity stage, but it declined at ripening stage. The maximum content of crude fibre was observed in Nendra padaththi (19.967 per cent) and least was in Kanchikela (15.129 per cent) at full maturity stage.

20. Highest crude protein was noticed at female phase and minimum was observed at ripening stage. At female phase, maximum crude protein was observed in Koodida bontha bathees (3.838 per cent) and lowest were in Gros Michel (2.948 per cent). During ripening Hybrid sawai contained maximum crude protein (1.503 per cent) while Kapok contained minimum (0.879 per cent).

21. Maximum content of tannin was observed in female phase and minimum was seen in ripening phase. At female phase, tannin was highest in Palayankodan (2.532 per cent) and minimum was in Pisang mas (1.594 per cent). During ripening maximum tannin was observed in Nendran (0.862 per cent) and minimum was in Pisang lilin (0.409 per cent).

22. The biochemical constituents of fruit are having high influence on infection and spread of the disease.

23. When the fruit contained maximum tannin content and minimum reducing sugars and total sugar, the pathogen did not infect the fruits without injury. At ripening stage, maximum symptom development was observed due to high content of sugars.

24. Reducing sugars and total sugar have a positive correlation with the disease intensity. The tannin and crude protein have a negative correlation with the disease intensity.

25. The sum total of all the chemical constituents and their ratios and relations will have more response for susceptibility or resistance to the disease.

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* Original not seen

VARIETAL SCREENING OF BANANA AGAINST ANTHRACNOSE DISEASE

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ABSTRACT OF A THESIS

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ABSTRACT

Laboratory and field studies of the "Varietal screening of banana against anthracnose disease" were conducted at the College of Horticulture, Vellanikkara and at Banana Research Station, Kannara respectively during 1981-1983.

In the field, the infection started at the distal end of the banana fruit and in course of time the infected fruit became blackened, shrivelled and mummified. After harvest, the symptoms appeared as small brown spots which enlarged quickly and coalesced forming larger patches. The affected areas were covered with orenge to salmon pink coloured conidial masses.

The detailed morphological studies of the fungus proved that the anthracnose disease of banana is caused by Colletotrichum gloeosporioides Cooke and Massee, the imperfect stage of Glomerella cingulata (Stonem) Spauld and Shrenk.

Twenty-five varieties of banana fruit were screened in vitro at different stages of development against anthracnose disease. The varieties showed different degrees of susceptibility at various developmental stages of the fruit. The pooled analysis of the data showed that the variety Nendra padaththi followed by Palayankodan, Jurmani kunthali, Poovlida bontha bathees, Poyan, Kanchikela, Pisang mas and Kapok were found to be highly resistant. The varieties Zanzibar, Adakka kunnan, Klue teparod, Chinia,

Nendran, Vennoettu mannan, Koduppilia kunnan, Hybrid sawai, Poocha kunnan, Red banana and Bodles Altafort were found to be resistant to the disease. The variety Robusta was found to be susceptible. The varieties Njalipoovan, Pisang lilin, Dwarf Cavendish, Matti and Gros Michelware found to be highly susceptible.

The major chemical constituents of banana fruit viz., reducing sugars, total sugars, starch, crude fibre, crude protein and tannin at different developmental stages of twenty-five varieties were analysed. The reducing sugars and total sugar were found to increase steadily from immediately after female phase to ripened stage in all the varieties. The starch and crude fibre contents, though increased steadily upto full maturity, declined sharply at the ripening stage. The crude protein and tannin contents were maximum at immediately after female phase but steadily decreased and were minimum at ripening phase.

There was a significant positive correlation between reducing sugars, total sugars and per cent disease intensity at three-fourth maturity. High sugars were responsible for susceptibility to the disease. A significant negative correlation was obtained between crude protein and per cent disease intensity at half maturity. A significant negative correlation was also obtained between tannin and per cent disease intensity at one-fourth and half maturity stages. High crude protein and high tannin contents were responsible for resistance to the disease.