Alternaria
Biology, Plant Diseases
and Metabolites

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ALTERNARIA-BLIGHT OF COTTON: EPIDEMIOLOGY AND TRANSMISSION

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1. INTRODUCTION

Alternaria-blight of cotton is a relatively unresearched disease that is predominant in many cotton growing regions of the world. As yet, Alternaria-blight has not had an economical impact on cotton regions of the western hemisphere. This chapter will reveal and discuss the variables and questions related to the epidemiology and transmission of this cotton disease.

2. DISEASE AGENTS AND GEOGRAPHICAL DISTRIBUTION OF THE DISEASE

At least two species of Alternaria are capable of producing symptoms commonly known as "Alternaria-blight of cotton". A. macrospora is considered the main causal agent in high quality cotton plants (Gossypium barbadense cv. Pima, and its many derivatives) (1), while A. alternata is the disease causal agent of Asian cotton (G. abroreum) and the upland cotton plants (G. hirsutum) (2, 3). The disease has a wide distribution, covering cotton growing areas throughout the world (4).

3. DESCRIPTION OF DISEASE SYMPTOMS

Symptoms of Alternaria-blight of cotton differ slightly, depending on the pathogen species present in the plant, the cotton cultivar, and the severity of disease development.

On G. barbadense plants, symptoms typically begin as small necrotic spots (1-2 mm in diameter), surrounded by conspicuous purplish halo up to 2 mm wide. Later, the spots can expand to 1 cm in diameter or even up to 2-3 cm in severe infections. The center of the spot becomes grey or light brown, and cracked. As the spots enlarge, they form a series of concentric markings within the border of the spots which are typical to Alternaria diseases. These markings are more clearly defined on the dorsal side of the leaf on G. hirsutum, but lack the purple rings (Fig. 1 A). The necroses appear much lighter in color than those which develop on
Fig. 1. Symptoms of Alternaria-blight on cotton leaves.
B- A. alternata on G. hirsutum cv. Acala.
Left- initial symptoms; right- mature symptoms
**G. barbadense** plants (Fig. 1 B). However, in both cotton species, a marked yellow chlorotic halo surrounding the necrosis is common in mature leaves (2, 4, 5, 6).

In the beginning, lesions on the veins of leaves are reddish-purple, elongated, and slightly sunken in the center. With development, the color changes to brown, and a small defined depression appears in its center.

Stem or twig lesions, caused by both pathogens, begin as small dark-brown semi-circular sunken spots which rapidly develop into a canker. The infected tissues split longitudinally or crack into small fragments. Finally, the stem or the twig breaks off at the canker. The entire plant rarely dies (5, 7, 8).

Lesions can appear everywhere on the plant: on leaves, buds, flowers, peduncles, and to a small extent, on bolls. However, the pathogen seldom penetrates the bolls. Initially, spotting occurs at the seedling stage on the cotyledons and immediately afterwards, on the emerging first leaves. Visible infections which occur late in the season when the cotton plant has already developed into a small bush, appear on the shaded lower leaves. These leaves then turn yellow and shed when infection is heavy. Under environmental conditions which predispose the plant to infection (discussed later), the spots increase in size and number, and also appear in the upper canopy (2, 4, 6).

When the disease becomes acute, the leaves shed. In severe occurrences, the entire plant is stripped of its foliage and only the leaves on the top of the plant remain green. However, new leaves generally emerge to replace the shed ones. This phenomenon gives the erroneous impression that the plant is healthy. Defoliation of infected leaves is the most marked symptom of **Alternaria**-blight disease.

Unusual disease symptoms, restricted to very severe infections, are: (i) plant death (8), and (ii) spots take on a black sooty appearance due to massive sporulation by the fungus. Once this stage of the disease is reached, the plant rapidly defoliates (6).

At the beginning of the growing season, it is common to isolate **A. macrospora** from the surfaces of leaves, stems, twigs, flowers, bolls and peduncles of field grown plants. In one survey, over 80% of all samples contained propagules of the pathogen. At the end of the growing season, seeds in open bolls were also highly infested with the pathogen (over 90% of all seeds) (9).
4. DISEASE EPIDEMIOLOGY

The epidemiology of Alternaria-blight in cotton is only fragmentally known. Research in this area began in the 80's and since then, only a few contributions have been published, most of which concentrated on environmental factors affecting disease development. This section will therefore describe the known facts, from the onset of the disease on the cotyledon until reduced yields from infected plants are apparent.

4.1 Inoculum origin

Although cotton plants are perennials, common agricultural practices are based on a 5-7 month season. Thus, there is a substantial period of time when there are no cotton plants in the entire growth area. This is especially important for the survival of A. macrospora for which cotton is its sole known host.

Cotton cotyledons are infected by Alternaria-blight very early in the new season. The cause of this infection can originate from several sources as summarized in table 1.

<table>
<thead>
<tr>
<th>Source</th>
<th>Importance of initiating disease</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infested seeds</td>
<td>Probably a main source</td>
<td>9, 11, 12</td>
</tr>
<tr>
<td>Plant debris</td>
<td>Marginal</td>
<td>9, 31</td>
</tr>
<tr>
<td>Secondary host plants</td>
<td>Probably a main source, however, only one host is as yet known.</td>
<td>14, 36</td>
</tr>
<tr>
<td>Post-season plants</td>
<td>Marginal in intensive cultivated area, main source in less developed agriculture.</td>
<td>9</td>
</tr>
</tbody>
</table>

The findings from these sources and their relative importance are described later in this chapter.

4.2 Transmission of the pathogens from infected leaves to seeds and then to seedlings

Cotton seeds can become infected with an inoculum of 100 or more spores; the resulting seedlings grow up diseased. Susceptibility of seedlings differs with the species of cotton. While G. barbadense cv. Pima is very susceptible to A. macrospora, G. hirsutum cv. Acala is entirely resistant (Fig. 2).
Fig. 2. Infection of cotton seedlings with *A. macrospora* originating from various concentrations of inoculum applied to seeds. Regression was performed at inoculum concentrations of 100 spores and above. Graf is printed with courtesy and permission from *Journal of Phytopathology*.

Seedlings can be infected at the cotyledon stage or soon after, producing a substantial epidemic early in the season. Although the boll surface can be heavily spotted with *Alternaria*-blight symptoms, the fungus is unable to penetrate the boll to the seed site while it is green and tightly closed. The main timing of seed infestation is after the natural opening of the bolls when the fibers become exposed to the field environment containing airborne spores of *Alternaria* (Fig. 3; 9, 10).

In late-season seed infestation, only the seed surface becomes contaminated with *Alternaria* spores. The seeds at this stage have a hard coat which the pathogen is unable to penetrate (9). One contradictory research claimed internal seed infestation for *G. barbadense* cv. Pima (11).

4.2.1 *Transfer of A. macrospora from cotton seed to seedling*. In a detailed study, the transfer of *A. macrospora* from the seed to the developing seedling was recorded. Artificially inoculated plants were used for this study since electron microscope observation in the field is impractical. After germination of the pathogen spores, mycelium moved to the upper parts of the seedling. This is an event which can occur in at least two ways: (i) Internally, by growth of the pathogen mycelium inside the germinating seed, concomitantly with seedling
development; (ii)Externally, by growth of the mycelium on the outer surfaces of the plant. The study of Bashan and Levanony (12) showed that immediately after seed inoculation with low concentrations of A. macrospora spores, the spores were rarely observed within the cavities of the seeds. Twenty four hours later, mycelium, spreading from sporadic spores, was revealed at the base of the emerging stem. Thirty six hours after inoculation, the mycelium mass increased, and after forty eight hours, covered most of the germinating seed surface. Although the seed surface was entirely covered with a thick layer of mycelium, this did not prevent germination of the cotton seed. At that time, the mycelium penetrated the internal seedling tissue. Shortly afterwards, the fungus began to sporulate mainly on the cotyledon stem. One hundred hours after inoculation, only a few spores were formed on the cotyledon leaves. At the cotyledon stage, there was no difference in the degree of infection between susceptible and resistant cotton species. Leaves of both species produced less spores (per area) at this stage than the stems of the same seedlings. This could be explained by the relatively low levels of phenols present in the stems, as compared to the leaves (Table 2; 13).

Fig. 3. Contamination of cotton seeds (cv. Pima) with A. macrospora inoculated at different stages of boll development. Columns followed by a different letter differ significantly at P<0.05. □ non-inoculated; □□ inoculated at closed boll stage;□ inoculated at open boll stage;□□ inoculation of buds. Modified data printed with courtesy and permission from Journal of Phytopathology.
TABLE 2

Phenol content of stem and leaves of healthy susceptible and resistant cotton seedling.

<table>
<thead>
<tr>
<th>Plant part</th>
<th>Cotton type (R-resistant S-susceptible)*</th>
<th>Phenol content (µg/g fresh weight)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cotyledon leaves</td>
<td>R</td>
<td>140 a(^b)</td>
</tr>
<tr>
<td></td>
<td>S</td>
<td>120 a</td>
</tr>
<tr>
<td>Cotyledon stems</td>
<td>R</td>
<td>72 b</td>
</tr>
<tr>
<td></td>
<td>S</td>
<td>41 c</td>
</tr>
</tbody>
</table>

*Resistant *G. hirsutum* cv. Acala
Susceptible *G. barbadense* cv. Pima

b Numbers followed by a different letter differ significantly at P ≤ 0.05.

No external mycelium was observed on the cotyledons during disease development. The relative resistance of the leaves was only temporary. With time, the leaves of the susceptible species (*G. barbadense*) were infected, while the leaves of resistant species (*G. hirsutum*) continued to show small amounts of both internal mycelium and negligible surface sporulation.

Six days after inoculation there was an intensive sporulation on the cotyledon leaves of susceptible plants (> 2200 spores/cm² leaf area), whereas only light sporulation was observed on the cotyledon leaves of resistant plants (300 spores/cm² leaf area). At the same time, (i) secondary external infections in susceptible species developed from the internal mycelium of infected stems and, (ii) spores were scarce on the surface of the necrotic area, whereas, the tissue surrounding the necrosis contained large numbers of spores (12). In conclusion, in both cotton species, *A. macrospora* moved from the seeds to the plant foliage within a week after seed infection.

Light microscopy and scanning electron microscopy of the leaf colonization process by *A. alternata* suggest that leaf colonization may be the outcome of either the growth of the mycelium in the intercellular spaces of leaves without internal sporulation, or the germination of spores on leaf surface. Penetration of superficial mycelium into the tissue occurred through the stomata. Removal of surface spores revealed compartmentalization; sporulation occurred ultimately on the leaf surface and mycelium was concentrated inside the leaf tissue (14). In this respect, the
effect of \textit{A. alternata} on cotton leaves differs from that of \textit{A. macrospora}.

The data presented so far on both \textit{A. macrospora} and \textit{A. alternata} suggest that both fungi have the potential to be transferred from infected seeds to seedlings. Thus, diseased seedlings, found early in the season, may originate from infested seeds.

4.3 Development of \textit{Alternaria}-blight on cotyledons

Cotyledons are the most susceptible plant organ to be attacked by \textit{A. macrospora} (9). In the field, cotyledons support the early stages of the disease. Under controlled environments, the disease develops much faster in cotyledons than in the leaves. Susceptibility of the cotyledons to infection by \textit{A. macrospora} increases with age. However, shedding of the infected cotyledons is determined more by their physiological age than by the extent of their infection (15). It is still unknown why, physiologically, the cotyledons of both cotton species are more susceptible than the leaves, and what kind of ecological advantage \textit{A. macrospora} gains by infecting the cotyledons of \textit{G. hirsutum} (a resistant plant species).

4.4 Shedding of plant organs

Shedding appears to be the principle effect of \textit{Alternaria} infection. Highly infected plant organs such as leaves, flowers and young bolls shed frequently, leaving the plant with only slightly infected organs.

Rarely will heavy infection strip the plant of its leaves. However, infected flowers nearly always shed (9). Usually, new leaves are continuously emerging on the plant which completely camouflage the damage. This new growth gives the infected plant a healthy, dark green appearance (9, 16). Leaf shedding is probably the main manifestation of the disease and the main cause of yield reduction. Therefore, the assessment of a disease by measuring the proportion of infected leaves can be misleading for \textit{Alternaria}-blight of cotton (16).

The rate of leaf shedding has been correlated with yield loss, disease severity, and the quantity of \textit{A. macrospora} populations (16). Yet, leaves tended to shed only when the lesions covered 1-3 % of the entire leaf area. Pre-mature defoliation was also affected by the location of the leaf on the plant. The lower canopy leaves, which are usually less infected, shed more
frequently than the young canopy leaves which are more infected.

Although the effect of leaf shedding has been measured on well nourished plants, other studies suggest that pre-mature defoliation in the field only develops in conjunction with potassium deficiency (6, 7). Shedding as a result of infection occurred only when the leaves were already exhibiting discoloration symptoms of potassium deficiency (turning from green to a bronze color). Although potassium deficiency can initiate pre-mature defoliation by itself, defoliation occurred earlier and was more pronounced when the plants suffered from both potassium deficiency and Alternaria symptoms. Thus, a practical cure which delayed or reduced defoliation was the application of potassium fertilizer (6).

Mechanical flower removal prevented both the development of potassium deficiency symptoms and Alternaria-blight development. This treatment provided indirect evidence that discoloration and shedding are primarily due to potassium deficiency and not due to Alternaria symptoms. The developing bolls become a strong sink for potassium which is diverted from the expanding leaves at the top of the plant. Furthermore, plants without potassium deficiency symptoms also lacked Alternaria symptoms, despite the close proximity of these plants to highly infected plants (6). Thus, the appearance of potassium deficiency symptoms are probably an indicator and/or a basic prerequisite for development of Alternaria-blight epidemic.

This conflicting evidence (from two different cotton regions, Israel and Zimbabwe in Africa), leaves open several fundamental questions: (i) How does leaf shedding cause damage? After all, the missing leaves are quickly replaced. Two possibilities exist to explain healthy looking plants which produce low yields. (a) The plant is replenishing shed leaves for survival and thus, robbing itself of fruit potential and, (b) the new growth is merely compensation for a metabolic damage which would reduce fruiting in any case. (ii) Why is potassium depleted from the upper canopy leaves, whereas the lower leaves usually shed? (iii) Why does severe defoliation occur in plants grown in soils known to have sufficient potassium, such as in Israel? (iv) Why do the lower, less-infected leaves shed, and not the highly-infected upper leaves?

In conclusion, one can say that pre-mature defoliation is phenotypically the most marked event associated with Alternaria-
blight disease. However, still unclear is the physiological cause and the role of the pathogen under field conditions. Perhaps the infection by the pathogen serves only as a trigger which enhances an already existing defoliation.

4.5 Environmental effects on disease development

Crucial factors which determine the extent of fungal and bacterial diseases are environmental in nature (i.e., temperature, relative humidity, condensation, rain, wind, injuries, and solar irradiation). These variables quantify both the symptoms and the area affected by the disease (17).

4.5.1 Effects of temperature and high relative humidity.

Early reports on Alternaria-blight mentioned the importance of frequent rains (18) and prolonged periods of high humidity (19) for the development of an epidemic. However, changes in free water availability on leaves and high relative humidity are not sufficient to explain epidemics of Alternaria-blight in relatively hot and dry areas such as Israel. There, the summers lack rain and cotton foliage is kept permanently dry by the use of drip irrigation directly to the soil. Moisture can only be found at night during the dew hours of most of the cotton growing season. Thus, perhaps a combination between favorable temperatures and short wet periods play a key role in the development of Alternaria-blight of cotton.

A. macrospora, whether in culture media or on cotton plants, develops well under a wide range of temperatures. However, the free-water requirements vary. For instance, cotyledons exhibited severe symptoms after exposure to 4 hours of free-water on the leaves and at temperatures ranging from 20° to 25°C. Leaves, on the other hand, required longer wet periods to become infected by the pathogen. While the optimal temperatures for infection for both leaves and cotyledons ranged between 20° to 30°C, and infection was inhibited above 35°C. Cotyledons could be infected at temperatures lower than 10°C, while leaves required temperatures higher than 10°C (15).

Elevated temperatures proved to be a limiting factor for Alternaria-blight infection and development. An increase in the ambient air temperature to 45°C resulted in 99-100 % reduction in the number of spots developed per unit area relative to plants infected at optimal temperatures. Plants exposed to these high temperatures immediately after inoculation produced significantly
fewer symptoms. Thus, the early pre-symptomatic stage of Alternaria-blight disease is highly susceptible to disruption by elevated temperatures. These high temperatures were also lethal to \textit{A. macrospora}. The spore germ-tube lysed and spore viability decreased (20).

The inhibition of \textit{A. macrospora} by high temperatures may explain the limited distribution of Alternaria-blight disease in the vast cotton areas of Arizona, where temperatures above 40°C are frequent during most of the growing season (20). Indirect evidence for this hypothesis was provided in the 1982–1984 cotton growth seasons. In these years, rainfall increased and high temperatures decreased in Arizona. Concomitantly, moderate to heavy epidemics of Alternaria-blight occurred in an area known to be only slightly affected in previous years (20).

From the data presented so far, it can be concluded that temperature and free-water are limiting factors in the development of Alternaria-blight, but they are not necessarily coupled. In cotton regions having some precipitation in the cotton season but suffering from high temperatures (such as Arizona and New Mexico in the southwest part of the USA), a high temperature is the limiting factor. In areas with moderate temperatures but lacking sufficient moisture like Israel, dew periods are probably the limiting factor.

The following hypothesis, though lacking physiological proof, can be suggested to explain the effect of temperature and wetness on the development of Alternaria-blight. When the ambient temperature is very high, the pathogen cannot compensate itself, and dies (20). When the temperature is optimal and the limiting factor is moisture, the leaves are unsuitable for infection. In essence, the cotyledons offer the pathogen a temporary refuge while it waits for more favorable moisture conditions to attack the leaves.

4.5.2 Effect of sun-light on disease development. Spore formation in cultures of \textit{A. macrospora} is light-dependent (21). When inoculated cotton plants were exposed to a light period between two dark wet periods, sporulation of \textit{A. macrospora} on leaves significantly increased, being greater on the cotyledons than on the leaves (22). Exposure of naturally-infected or artificially-infected cotton plants to sun-light, significantly increased the symptoms of \textit{A. alternata} in \textit{G. hirsutum} plants.
This effect was independent of plant age and could be induced under a range of temperatures and moisture (23). It has been proposed that sunlight acts as a catalyst for symptomless infections to become visible (23). However, this theory does not consider the known occurrence of symptomless *Alternaria* infections under normal growing conditions of daily sunlight.

5. SYMPTOMLESS INFECTIONS

As in many other foliar diseases (17), *A. macrospora* and *A. alternata* can produce symptomless infections (Table 3). These infections can be revealed by light and scanning electron microscopy. Detection is based on either the formation of the typical spores on the leaf surfaces or on conventional isolation of the respective pathogen on growth medium from apparently healthy tissues. Frequently, the pathogens produce miniature infections comprised of a few plant dead cells. These infections are invisible to the naked eye. However, under low magnification, sporulation of the respective fungi can be easily detected and identified. Why the symptoms do not develop into larger, visible ones is, as yet unknown, although the doubtful involvement of sunlight into their formation has been proposed (23).

As in other plant diseases, formation of symptomless plants is of extreme economical and scientific importance. Most field surveys for pest management are done by the naked eye. Then, according to the subjective judgement of the observer, the field is usually subjected to a chemical treatment. In this respect, a "hidden disease" is practically an non existing one and as such,

| TABLE 3 |
| Production of symptomless cotton plants after inoculation with *A. macrospora* and *A. alternata* |

<table>
<thead>
<tr>
<th>Cotton species</th>
<th><em>A. macrospora</em></th>
<th><em>A. alternata</em></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>with symptoms</td>
<td>symptomless</td>
</tr>
<tr>
<td>G. barbadense</td>
<td>82</td>
<td>14</td>
</tr>
<tr>
<td>G. hirsutum</td>
<td>2</td>
<td>22</td>
</tr>
</tbody>
</table>

" Mean of 5 independent samplings, from a total of 408 plants before the flowering stage in Bet Shean Valley in Israel, 1985."
is not being treated. When the symptoms are visible, or an epidemic has already developed, it is usually too late; we do not yet have a fungicide which can stop a heavy epidemic of Alternaria-blight. Scientifically, symptomless plants may mislead conclusions drawn from infection experiments.

6. EFFECT OF THE DISEASE ON COTTON YIELD

Alternaria-blight of cotton is considered to be of minor economical importance in some cotton production regions. In Arizona for example, where large acreages of susceptible G. barbadense (Pima) are grown annually and virulent strains of A. macrospora exist, the damage is severe only in years of relative low summer temperatures and higher rainfall. However, no numerical estimation of losses has been published in scientific publications. In Israel, estimation of yield-loss indicated that a reduction of 25 % was due to the disease (16). However, this evaluation was probably an underestimation since the yields were compared to plots which had been treated with a fungicide. Using a treated control plot, instead of an un-diseased plot, changes the result because fungicide treatment seldom eliminates the disease completely (Management of Cotton Growers of Israel, Annual reports published in hebrew).

7. ALTERNARIA-BLIGHT AS A POSSIBLE DISEASE COMPOSITE OF TWO PATHOGENS

Greenhouse and field experiments have shown that Alternaria-blight of cotton is caused by at least two Alternaria species, A. macrospora and A. alternata.

A. macrospora attacks mainly G. barbadense plants and only slightly attacks G. hirsutum plants. A. alternata is fully capable of infecting G. barbadense and G. hirsutum plants. A recent study demonstrated that many plants which exhibited Alternaria-blight symptoms were, in fact, infected by both pathogens. Dual leaf colonization occurred and the two pathogens were sharing the same cotton leaf (24).

In G. barbadense plants, A. macrospora initiated symptoms in 61 % of all inoculated plants and in only 3 % of G. hirsutum plants. The rest of the inoculated plants remained symptomless. Inoculation with A. alternata yielded visible symptoms in 85 % of G. barbadense plants and in 56 % of G. hirsutum plants. Dual
Fig. 4. Disease incidence on leaves of *G. barbadense* (A) and on leaves of *G. hirsutum* (B) after dual inoculation with various concentrations of *A. macrospora* and *A. alternata*. □□□□ - Percentage of plants exhibiting symptoms. Original graphs were printed in the Canadian Journal of Botany (24).
inoculations with sub-optimal concentrations of each pathogen yielded maximal symptoms formation in 90–100 % of all inoculated plants in both cotton species (Fig. 4). This may indicate a synergism between the two pathogens (24). In commercial fields in Israel the ratio of A. macrospora/A. alternata lesions was 1:4 in G. barbadense cv. Pima and 1:19 in G. hirsutum cv. Acala (25). Thus, a new definition of Alternaria-blight disease might be proposed: a cotton disease composed of at least two Alternaria species.

Another disease composite responsible for leaf blight of cotton is the possible association between A. tenuis and Cercospora gossypina. Research in this disease composite, detected exclusively in the USA more than 20 years ago, has not been pursued any further. On one hand, a single report stated that this disease composite caused a severe, pre-mature defoliation and die-back of the main stem terminal and uppermost fruiting branches. A cessation of boll set was apparent, which resulted in loss of yield and fiber quality. A characteristic smokey-blue color was produced on lesions in which both fungi sporulated together (26). On the other hand, two other reports claimed that Alternaria-Cercospora leaf spot association occurred only on weakened plants already suffering from either lack of moisture, mineral (N, K) deficiency, or following infection by an aggressive pathogen such as Xanthomonas campestris pv. malvacearum. Furthermore, the "disease" causing organisms were non-aggressive pathogens. Thus, they concluded that this association is, at best, a minor disease and that any stress on cotton plants at the time of heavy fruit load increased susceptibility to secondary minor pathogens (27, 28).

In conclusion, the idea that Alternaria-blight disease is, in fact, a disease composite in which a synergism between two pathogens exists, is a new avenue in this field which deserves further attention.

8. SUSCEPTIBILITY AND RESISTANCE TO ALTERNARIA-BLIGHT

Most cotton species grown in the world are susceptible to A. macrospora. However, the predominant species grown in the USA, G. hirsutum is considered highly resistant. The high quality extra-long fiber cotton (G. barbadense cv. Pima) is extremely susceptible. These differences in susceptibility were attributed to the oxidative metabolism of the plant and in particular to
polyphenoloxidase activity (13). Recently, cotton prices in the world trade are relatively low, especially for low quality fibers. Thus, the high-value Pima cotton has become increasingly favored by growers in Israel and the USA. This tendency increases the danger of Alternaria-blight of cotton in areas that have been relatively unaffected in the past (2, 8).

Criteria for the evaluation of susceptibility and resistance to Alternaria-blight have varied greatly, as is common for many other plant diseases. Of several criteria tested recently, lesion diameter was found to be the most accurate and useful criterion for evaluating host susceptibility (2).

Although most G. hirsutum cultivars are not normally susceptible to severe infection by A. macrospora, they may become predisposed to infection by stress factors such as nutrient balance (potassium deficiency in particular), water stress (6, 27), or nematode attack (29). Early maturing cultivars were the first to show potassium deficiency symptoms and the first to become infected with A. macrospora (6).

Although much research has been done on the breeding of resistant cotton cultivars against many pests including Alternaria-blight, very little has been published in international scientific literature. The bulk of literature on this aspect can be found in reports of Regional Experimental Stations and in local agricultural journals. Since these sources are not available world-wide, they were not cited in this chapter.

9. FIELD DISPERSAL OF A. macrospora AND A. alternata

Inoculum dispersal is of the utmost importance for the biology of any plant disease agents. Many phytopathogens are remarkably well adapted to passive dispersal by wind, seeds, insects, vegetatively propagated plants, irrigation water, agricultural tools, machinery, and other means. The dispersal mechanisms are highly dependent on environmental conditions, but not equally efficient. The importance of each means of transmission varies according to the prevailing conditions in the field, i.e., differences in plant cultivation techniques, sanitary conditions, weed control, monoculture and the growing season (30). Therefore, several means, acting simultaneously, may be necessary to ensure successful dispersal of the disease pathogen.
The transmission of pathogenic agents is inefficient if their compatible hosts are far apart. Therefore, since many pathogenic fungi usually find their host by accidental encounter, they must produce a large number of dispersal units.

The cultivation of one crop over a large area, such as cotton in Israel (the main summer crop), enables A. macrospora to disperse in the fields via agricultural machinery, tools, insects, animals, aircraft or water. The dispersal mechanisms and their relative importance are summarized in table 4.

TABLE 4

Dispersal vectors for A. macrospora and A. alternata pathogenic to cotton.

<table>
<thead>
<tr>
<th>Vector</th>
<th>A. macrospora</th>
<th>A. alternata</th>
<th>Vector importance</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wind</td>
<td>+</td>
<td>+</td>
<td>major</td>
<td>10,14,32</td>
</tr>
<tr>
<td>Insects</td>
<td>+</td>
<td>+</td>
<td>major</td>
<td>33,34,35</td>
</tr>
<tr>
<td>Mites</td>
<td>+</td>
<td>-</td>
<td>minor</td>
<td>33</td>
</tr>
<tr>
<td>Mammals</td>
<td>+</td>
<td>-</td>
<td>minor</td>
<td>33</td>
</tr>
<tr>
<td>Reptiles</td>
<td>+</td>
<td>-</td>
<td>minor</td>
<td>33</td>
</tr>
<tr>
<td>Birds</td>
<td>+</td>
<td>-</td>
<td>minor but to long distances</td>
<td>33</td>
</tr>
<tr>
<td>Agricultural machinery</td>
<td>+</td>
<td>-</td>
<td>major</td>
<td>33</td>
</tr>
<tr>
<td>Agricultural tools</td>
<td>+</td>
<td>-</td>
<td>minor</td>
<td>33</td>
</tr>
<tr>
<td>Labors</td>
<td>+</td>
<td>-</td>
<td>minor</td>
<td>33</td>
</tr>
<tr>
<td>Aircraft</td>
<td>+</td>
<td>-</td>
<td>minor but to long distances</td>
<td>33</td>
</tr>
<tr>
<td>Adhering soil particles</td>
<td>+</td>
<td>-</td>
<td>major</td>
<td>33</td>
</tr>
<tr>
<td>Water sources</td>
<td>+</td>
<td>-</td>
<td>major</td>
<td>33</td>
</tr>
<tr>
<td>Seed lots</td>
<td>+</td>
<td>+</td>
<td>major</td>
<td>9,11</td>
</tr>
<tr>
<td>Post season plants</td>
<td>+</td>
<td>-</td>
<td>minor</td>
<td>9</td>
</tr>
<tr>
<td>Plant debris</td>
<td>+</td>
<td>-</td>
<td>minor</td>
<td>9,31</td>
</tr>
<tr>
<td>Alternative host</td>
<td>-</td>
<td>+</td>
<td>major</td>
<td>24,36</td>
</tr>
</tbody>
</table>

+ Data for this pathogen exists
- No data

9.1 Dispersal of A. macrospora and A. alternata by the wind

Inocula of Alternaria-blight pathogens moves primarily by wind, regardless of whether the source is diseased cotton plants, secondary host plants or plant debris left on the soil surface at the end of the season (10, 14, 31, 32). No detailed study on air
dispersal of *A. macrospora* is available. However, a current study on the air-borne phase of *A. alternata* revealed that wind is probably the main vehicle responsible for cross-field and inter-field transmission of this pathogen (10).

The direction of the rows and age of the plants have a crucial effect on the wind dispersal of *A. alternata* air-borne spores. Monitoring of Alternaria-blight infected plants in a commercial plot and after creating three artificial disease sources, resulted in three local epidemics downwind, when the cotton plants were at their first stages of growth (Fig. 5 A, hatched areas). Disease incidence further than 7 meters from the disease sources was small (Fig. 5 A). A nearby cotton plot, upwind of the disease sources, was only scarcely diseased. On the other hand, monitoring disease incidence in mature plants, exposed to similar disease sources, showed a different pattern; the disease foci was scattered randomly in the plot. The foci developed without an apparent relationship to the artificial disease sources (Fig. 5 B, hatched areas). In addition to the disease foci, randomly scattered infected plants were located within the plot (Fig. 5 B and symbols). Two weeks after the field was exposed to the presence of *A. alternata*-infected plants, concomitantly with disease development, the number of air-borne spores of *A. alternata* was high for either young or mature cotton plants (Fig. 5 C).

When the cotton rows in the field were parallel to the average wind direction, local epidemics which originated from the disease foci developed downwind (Fig. 6 A, hatched areas). The infected areas remained constant and had not increased during the growing season. At the same time, disease incidence in the rest of the plot was low (Fig. 6 A).

Air-borne spore counts from traps located within in the plot showed a permanent low level of *A. alternata* spores prevailing naturally in the field. However, when an epidemic developed, the number of air-borne spores increased dramatically (Fig. 6 B). In contrast, spore traps which were even slightly to the side of the disease foci contained significantly fewer spores than traps located directly downwind (Fig. 6 B).

A precise air dispersal measurement of *A. alternata* showed that spore transfer via air currents is only for short distances, although air-borne spores could be detected as far as 20 m from the inoculum source. At 6 meters from the diseased plants, the number of air-borne spores decreased sharply to 50% (Fig. 7).
Fig. 5. The effect of wind and row direction in the field on the spread of Alternaria-blight disease and on A. alternata air-borne spores which originated from diseased plants two weeks after inoculation. A - young cotton plants; B - mature cotton plants. 
- area which contained more than three mature cotton plant/m row; - site containing two diseased plants; - site of a single diseased plant; - location of spore traps; - location of the artificial disease source. C - number of A. alternata air-borne spores; - 5 hours after placing inoculated plants in the field and - 14 days later. Columns followed by different letter differ significantly at P ≤ 0.05. Graph is printed with the courtesy and permission of Journal of Phytopathology.
Fig. 6. Spread of Alternaria-blight of cotton from naturally-infected disease foci as affected by wind and row direction four weeks after exposure of infected plants (A). □ - area which contained more than three diseased plants per row; ● - site containing two diseased plants; ■ - site for a single diseased plant; ◊ - location of spore traps; ▲ - site of the original naturally infected plants. B - number of A. alternata air-borne spores; □ - 5 hours after exposure of the field to the diseased plants (covered previously with a plastic sheet); ■ - 4 weeks later. Columns followed by a different letter differ significantly at P ≤ 0.05. Graph is printed with courtesy and permission of Journal of Phytopathology.
Fig. 7. Transfer of *A. alternata* spores via air from diseased plants in the greenhouse. ● - *A. alternata* in the presence of diseased plants; ○ - *A. alternata* spores in the absence of diseased plants. Bars represent SE. Graph is printed with courtesy and permission of Journal of Phytopathology.

Fig. 8. Evaluation of *A. alternata* air-borne spores trapped during the cotton season in relation to wind direction. Columns followed by a different letter differ significantly at P< 0.05. Graph is printed with courtesy and permission of Journal of Phytopathology.

The local wind direction also greatly affected dispersal of *A. alternata* spores within the field. A continuous survey for air-borne spores in Bet-Shean Valley in Israel (which has a permanent strong west wind for few hours a day due to its topographical
structure) showed that spore traps exposed to the west wind collected more spores of *A. alternata* than traps facing other directions (Fig. 8).

![Graph showing Alternaria spore distribution over a year](image)

**Fig. 9.** Distribution pattern of *A. alternata* air-borne spores above cotton fields in Bet-Shean Valley, Israel during 1986 and 1987. ● - data from sampling in 1986; ▲ - data from sampling in 1987. Graph is printed with courtesy and permission of Journal of Phytopathology.

*A. alternata* is always present in the cotton field in low numbers. In a recent study (10), a bimodal spore distribution pattern was recorded; one peak between April and June and the other in September–October. These peaks corresponded to the two epidemics of Alternaria-blight recorded each year in Bet-Shean Valley (Fig. 9).

In conclusion, it can be postulated that (i) viable *A. alternata* spores are air-borne above infected cotton plants throughout the growing season, and (ii) increases in their numbers are correlated with the epidemic occurring in the field. Spore dispersal is greatly affected by the local wind, the disease source, and the direction of the rows. However, this dissemination
is local, within the field borders, and restricted to short distances.

9.2 DISPERAL OF *A. macrospora* BY DIFFERENT ORGANISMS

Air currents are not solely responsible for inoculum movement. Insects have also been associated with the transfer of pathogens to cotton. Insects (particularly those which are cotton pests by themselves), and mites were found to carry the highest number of *A. macrospora* propagules (Table 5; 33). In the laboratory, adult cotton boll weevils transfer inoculum of *A. tenuis* from pure culture to healthy bolls (34). Conidia of *Alternaria* remained viable after passage through the alimentary canal of this insect (35). Since this is a common insect pest of cotton fields, this ability makes it an efficient vector of disease propagules. It seems that members of any insect group that commonly visit diseased and healthy plants can acts as a vector for *A. macrospora*. In addition, the great abundance of non specific insect visitors that forage for nectar and pollen in the cotton fields are extremely important in the dispersal of *A. macrospora*. In many semi-arid areas, cotton plants are the main flowering plants of the dry season. Even the carnivorous Oriental Hornet carried *A. macrospora* propagules on their bodies in the cotton field (Table 5).

In conclusion, since insects are the most prevalent organisms in cotton fields, and since they are heavily contaminated with *Alternaria* spore, they serve as one of the major vectors in spore dissemination. However, since most of them are localized within a growth region, their contribution to spore dissemination is local.

Cotton fields in dry areas provide the main green habitat for small mammals whose fur is contaminated with fungal spores. Many mammals trapped in the field (mice, rats, rabbits) and even farm workers, were capable of transmitting *A. macrospora* propagules, though less efficiently than insects. Birds of several types were found to have a lower capability for transmitting *A. macrospora* propagules than insects, but they did so for longer distances. Two reptile species (agama and chamaeleon) had the lowest transmission capability, at only a few propagules per animal (Table 5; 33).
TABLE 5

Possible dispersal of Alternaria macrospora propagules by different motile organisms (according to 33).

<table>
<thead>
<tr>
<th>Common name</th>
<th>Scientific name</th>
<th>Mean no. of propagules/sample</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MAMMALS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouse</td>
<td>Mus musculus</td>
<td>43</td>
</tr>
<tr>
<td>Rat</td>
<td>Rattus sp.</td>
<td>78</td>
</tr>
<tr>
<td>Rabbit</td>
<td>Lepus syriacus</td>
<td>25</td>
</tr>
<tr>
<td>Human (skin of hand)</td>
<td>Homo sapiens</td>
<td>240</td>
</tr>
<tr>
<td><strong>REPTILES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Agama</td>
<td>Agama stillio</td>
<td>6</td>
</tr>
<tr>
<td>Chamaeleon</td>
<td>Chamaeleo chamaeleo</td>
<td>13</td>
</tr>
<tr>
<td><strong>BIRDS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>House sparrow</td>
<td>Passer domesticus biblicus</td>
<td>8</td>
</tr>
<tr>
<td>Common bulbul</td>
<td>Pycnonotus capensis vallembrosa</td>
<td>19</td>
</tr>
<tr>
<td><strong>INSECTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cotton leafworm</td>
<td>Spodoptera littoralis</td>
<td></td>
</tr>
<tr>
<td>larvae</td>
<td></td>
<td>425</td>
</tr>
<tr>
<td>butterflies</td>
<td></td>
<td>146</td>
</tr>
<tr>
<td>Tobacco whitefly</td>
<td>Bemisia tabaci</td>
<td>223</td>
</tr>
<tr>
<td>Ants</td>
<td>Acantholepis frauenfeldi bipartita</td>
<td>27</td>
</tr>
<tr>
<td>Oriental hornet</td>
<td>Vespa orientalis</td>
<td>13</td>
</tr>
<tr>
<td>Spiny bollworm</td>
<td>Earias insulana</td>
<td>360</td>
</tr>
<tr>
<td><strong>MITE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red mite</td>
<td>Tetranychus telarius</td>
<td>710</td>
</tr>
</tbody>
</table>

9.3 Dispersal of A. macrospora through agricultural machinery, tools and light aircraft

High levels of A. macrospora propagules were found adhering to the plastic, rubber, or the metal body of almost every tool and agricultural machinery that passed through or touched the plant foliage (Table 6; 33). Even infrequent cars passing between the fields became contaminated by A. macrospora propagules. Agricultural spray aircraft and ultra-light sport aircraft, which flew regularly at a low level above the fields, also carried A. macrospora propagules on their surfaces (Table 6; 33).
9.4 Dispersal of *A. macrospora* by soil particles adhering to agricultural machinery and tools

Since agricultural machinery is usually not washed or disinfected during the growing season, soil particles, and especially mud, are very common on almost all machinery. Analysis of the mud adhering to tools frequently employed in the field such as tractor wheels, labor boots, cultivators, plows, or even the wheels of cars, showed the presence of *A. macrospora* (Table 6; 33).

**TABLE 6**

Possible dispersal of *A. macrospora* propagules by agricultural machinery, tools, soil particles adhering to machinery and by light aircraft (According to 33).

<table>
<thead>
<tr>
<th>Tool or machinery</th>
<th>Mean no. of propagules/sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plow</td>
<td>$2.80 \times 10^4$</td>
</tr>
<tr>
<td>Cultivator</td>
<td>$2.23 \times 10^2$</td>
</tr>
<tr>
<td>Towed motor sprayer</td>
<td>$2.70 \times 10^4$</td>
</tr>
<tr>
<td>Tractor wheels (rubber)</td>
<td>$7.12 \times 10^2$</td>
</tr>
<tr>
<td>Wheels of the sampling car</td>
<td>$8.76 \times 10^2$</td>
</tr>
<tr>
<td>Labor shoes</td>
<td>$3.37 \times 10^2$</td>
</tr>
<tr>
<td>Labor clothes</td>
<td>$6.06 \times 10^2$</td>
</tr>
<tr>
<td>Automatic cotton harvester</td>
<td>$3.60 \times 10^2$</td>
</tr>
<tr>
<td>Hammer</td>
<td>1 to 5</td>
</tr>
<tr>
<td>Wrenches</td>
<td>9</td>
</tr>
<tr>
<td>Agricultural spray aircraft</td>
<td>$2.73 \times 10^2$</td>
</tr>
<tr>
<td>Sport aircraft</td>
<td>$1.02 \times 10^2$</td>
</tr>
</tbody>
</table>

9.5 Dispersal of *A. macrospora* by water sources

Water sources can serve as a vehicle for transferring *A. macrospora*. All drainage water from sprinkle irrigation contained pathogenic propagules. In one cotton growing region, other water sources such as flow water in open ditches, oxidation sewage water pools, natural swamps, a river and a lake, all contained infectious propagules of this fungus. The fungal population was relatively low in flowing water sources and massive in still water (Table 7).

In conclusion, the dispersal study by Bashan (33) shows that almost every animal, insect, mite, tool, agricultural machinery, or person passing through an infested cotton field can disseminate *A. macrospora*. People working in the field or even light aircraft and birds flying low above the field are contaminated with, and can transfer *A. macrospora*. These latter vehicles are of great importance, despite the low amount of inoculum that they carry,
because they can transfer the disease agent for long distances. Soil can act as a dispersal vehicle by adhering to everything which passes through it. Thus, even random visitors can become A. macrospora vectors. In addition to these biotic vectors and abiotic vehicles, irrigation water may be one of the most important disease vehicles in arid cultivation areas, because water is recycled or transferred over very long distances. For example, irrigation water pumped from the Sea of Galilee, located in the center of cotton growing region of Northern Israel, is transferred to the Negev region in Southern Israel, a distance of several hundred km. There, the water is used in cotton fields without any specific fungal control treatment. All the above mentioned factors contribute to the dissemination of A. macrospora, but the most predominant vectors are cotton specific insect pests which are usually more contaminated with the pathogens propagules.

One main question remains unanswered: How do virulent propagules initiate disease in a field that they have randomly reached? This depends on many environmental, biological, genetic and agricultural factors discussed earlier in this chapter and are largely unknown with regard to A. macrospora and A. alternata.

10. OVER-SEASON TRANSFER OF ALTERNARIA-BLIGHT

10.1 Transfer in seeds

Seed infestation by A. macrospora in the field occurs only when the boll is naturally open at the end of the growing season. The physiological growth stage at which the boll surface is infected has no effect on seed infestation. When the entire plant is visibly diseased, seeds are also infested. But, at the end of the growing season, seeds become infested even if in many cases the bolls themselves are not infected. However, there is no proof that
this infestation can create an epidemic in the next season seven months later, despite the fact that the pathogen has the ability to survive for prolonged periods under dry conditions.

Internally infected bolls (by artificial inoculation) were usually degenerative when compared to non-infected bolls. A high percentage of them did not open naturally at the end of the growing season (9). The phenomenon of boll degeneration was also observed in commercial cotton fields, but at very low percentage (< 0.5% of all surface infected bolls monitored over several growing seasons) (9).

10.1.1 Presence of *A. macrospora* in commercial seed stocks. In a limited survey, the pathogen was found contaminating commercial seed lots at relatively low rates [200 - 300 propagules/250 g seeds, (9)]. However, more information is required on the level of infection of *A. macrospora* in seed stocks.

Although *A. macrospora* can passively contaminate seeds at the end of the growing season, it appears that seed contamination is a major source for the over-season transfer of *Alternaria* propagules. The chemical treatments used by the seed industry (sulfuric acid + a fungicide) probably do not eliminate all of the pathogen propagules from the seed surface. The remaining few propagules on the seed surface, together with the possible internal seed infection, might initiate a disease in limited numbers of plants early in the season. However, it is unlikely that the massive infection at the cotyledon growth stage originated only from this inoculum source. Only infection, combined with an efficient vector such as wind or insect, may create a disease focus in the field. At the present time, research into the possibility of a combination between over-season survival and dispersal mechanisms has not been done for *Alternaria*-blight of cotton.

10.2 Transfer of *A. macrospora* in plant debris and in post-season plants

Post-season plants are a common phenomenon in many cotton areas in less developed countries. The harvested plants are left in the field until the new crop season. Then, they are cut and the field is plowed. In intensive cotton cultivation areas, the fields are usually deep-plowed in the end of the growing season for control of dormant pest insects. Thus, in these areas, post-season plants are hardly found. Only a few plants per field which have escaped plowing can be located usually near the irrigation system
Fig. 10 (A). Survival of *A. macrospora* in bolls and stems from post-season plants and dead plants either buried or located on the soil surface. (B) Percentage of seed infestation in these plants. □ post season plants; □□□ buried plants; □□ plants on soil surface.

of the field.

In a recent study, infested plant debris placed in a field caused an epidemic which started earlier and spread faster than usual. Over-wintering of *A. macrospora* in the plant debris was related to the degradation of the debris: debris that was less decomposed caused more infection. An increase in the soil moisture in which the debris was buried, reduced the survival of *A. macrospora*. In cases where the debris was not in close proximity
to the germinating cotton seedlings, the pathogen had not spread from the debris to the seedling. Only upon physical contact between the buried debris and the seed, did the seedling become infected. In addition, when the plant debris was placed on the soil surface, the new crop was highly infected due to sporulation of *A. macrospora* after dew periods (31).

Populations of *A. macrospora* survived on the surface of post-season cotton plants (Fig. 10 A), and seeds in the open bolls of these plants were contaminated with *A. macrospora* propagules (Fig. 10 B). The detection of the infectious propagules in these plants could be done throughout the winter season. Isolates originating from these post-season plants were highly infective to *S. barbadense* cotyledons growing nearby in the following growth season (9).

Although survival of *A. macrospora* in plant debris and post-season plants has been demonstrated, this appears likely to have only a marginal role in field re-infestation. In Israel, for example, after the cotton fields are extensively plowed at the end of the season, survival of the pathogen in buried plant debris is minimal. In addition, it is rare to find non-degraded debris on the soil surface in the middle of the rainy season (which is out-of-season for cotton). Therefore, it can be concluded that the significance of post-season plants and plant debris in the re-infestation of the field can be confined only to areas of less developed agricultural techniques.

10.3 Transfer of *A. alternata* by wild beet carrier plants

All of the previously mentioned causes can not adequately explain severe early-season epidemics over large cultivation areas for two reasons: seed are produced by the seed industry from Alternaria-free fields and they are subjected to surface disinfection. This process destroys most of the seed surface microflora which are the major seed contaminants, leaving only few internal *Alternaria* propagules intact. Post-season plants, left over after the growing season, are very rare in intensive growing areas, and the dispersal of *A. macrospora* and *A. alternata* via air currents in the field is restricted to short distances. Therefore, it is unlikely that the frequent severe epidemics originate from these sources. It is possible that common weeds bordering cotton fields may serve as over-season carriers for the pathogens. Such a weed was identified in Israel as common wild beet. Strains of
\textit{A. alternata}, isolated from infected wild beet plants, were capable of infecting cotton plants (36). Several cotton fields, adjacent to \textit{A. alternata}-infected wild beet plants, were also infected by \textit{A. alternata} early in the growing season. The wild beet grows profusely, and since the natural growth period of these plants connected the end of one cotton season with the beginning of the next one, they may serve as over-season hosts for the \textit{A. alternata} pathogen (24).

11. \textbf{\textit{A. macrospora} as mycoherbicide}

Spurred anoda is an important weed in cotton cultivation which is both difficult and expensive to control with conventional herbicides. An isolate of \textit{A. macrospora}, with host specificity to anoda, was capable of efficiently controlling this weed and at the same time was completely un-aggressive towards cotton plants (8, 37, 38, 39). Pathogenic isolates of \textit{A. macrospora}, which were only pathogenic to cotton, were harmless to anoda plants at any inoculum concentration. The disease initiated by \textit{A. macrospora} on Spurred anoda was very destructive, causing death of the anoda plants, especially when \textit{A. macrospora} inoculum was combined with \textit{Fusarium lateritium} (a pathogen of several weeds). The synergetic interaction between these two pathogens can be exploited to make both pathogens more effective as potential mycoherbicides (40).

Using a destructive pathogen of cotton as a bio-control agent for a weed which grows with cotton cultivation presents a potential hazard, even if this isolate is, at the moment, harmless to cotton. Aggressive mutants of this isolate (which is identical to pathogenic \textit{A. macrospora} of cotton) may spontaneously evolve with intensive application. If they retain their lethality, they will severely threaten cotton cultivation. Thus, the resistance of cotton varieties to the anoda isolate needs to be firmly established before the use of this pathogen can be implemented as a bio-control agent of Spurred anoda.

12. CONCLUDING REMARKS

Alternaria-blight of cotton is one of the least studied plant diseases, despite the fact that it has caused agricultural damage for more than 50 years (19, 41).

Most of the ecological, pathological and physiological aspects of this disease are unknown. Remaining unclear, are basic
questions such as: (i) the identification of the causal agents of this disease, (ii) how the damage to the plant occurs and (iii) the extent of economical damage. As yet, two different Alternaria species are identified as causal agents and it is still vague as to which one is the primary agent and/or secondary pathogens.

This disease is considered a minor disease in the cotton growing areas of the USA, yet it remains a major disease in other areas such as Israel and Africa. It has not been established why such light occurrences have been detected in the USA, especially when highly virulent strains, a susceptible host, and a vast range of environmental conditions favorable for infection exist in many American cotton growing regions. As a result of its unimportance in the USA, no resources are available to study this disease. Thus, most of the available studies concentrate on descriptions of factors affecting disease development and transmission. Except for phenol involvement in the resistance mechanisms of the plant (13), no other physiological mechanisms relating to the attack of the plant is known. Furthermore, most of the current knowledge of the disease was produced by isolated research groups with no confirmation from research groups elsewhere. Most of the studies on dissemination of the pathogens were done by Israeli groups. These studies focused on dry semi-arid conditions, and it is still unknown how the pathogen is being transmitted in cotton growing regions having summer rains.

In conclusion, the current international notion is that this disease does not deserves substantial research resources and attention. However, since cotton is a main cash crop in many areas in the world, and more and more growers are annually increasing the cultivation of Pima cotton (which is extremely susceptible), it seems that severe epidemics are inevitable.

ACKNOWLEDGEMENTS

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REFERENCES


