

Tactics for Maintaining Plant Health of Cotton

L. S. BIRD, Professor
Department of Plant Pathology
and Microbiology
Texas Agricultural Experiment
Station, College Station

Cotton (*Gossypium hirsutum* L.) is a dicotyledonous taprooted perennial that, under domestication, has been bred to perform so it can be managed as an annual. The main stem has buds at each node that develop into fruiting and vegetative branches. Vegetative branches may produce either fruiting branches or additional vegetative branches. Sympodia of fruiting branches terminate in a leaf and two buds; one bud produces the next sympodium and the other may initiate another fruiting branch. Fruiting branches usually have three or four sympodia, but 8–10 are not unusual (1,4). Healthy plants, i.e., those undamaged by plant pathogens and insects, behave as annuals and unhealthy ones behave more as perennials. Because the annual habit is required today for efficient crop management, plant health is crucial.

Cotton plants damaged by pathogens may recover if the root is not killed. Plants adjacent to dead plants may compensate for yield loss by growing larger. Plants recovering from damage show delayed maturity, and adjacent compensating plants are larger and also mature late. Delayed maturity often increases the cost of controlling insects, and product quality (seed and fiber) is reduced. Uniformly spaced plants of the same size and with bolls maturing simultaneously are necessary to ensure efficiency in cultivation; in spatial use of moisture, nutrients, and sunlight; in controlling insects; in preparing for harvesting; in machine harvesting; and in obtaining uniform product quality. This can be achieved with 40,000–60,000 healthy plants per acre.

Crop management components

Reliable tactics that assure total plant health are required to prevent losses caused directly by disease and indirectly by disruptive plant behavior leading to

inefficiency and costly problems in management (2,3). Strategies for plant health in cotton relate to land preparation, pesticides, fertilization, irrigation, cropping sequences, crop residues, weeds serving as alternate hosts, host resistance, and host morphological traits. Ideally, farmers should have the option of using any or all of the crop management components. Some farmers do have a broad choice, but others have situational and economic constraints. For example, in many arid areas under compulsory irrigation, cotton must always be planted near available water. This often dictates planting fields continuously to cotton, especially when other adapted income-producing crops are not available. In other instances, the cost of moldboard plowing may prohibit the desired practice of turning under crop residues (2,3,5).

For a number of years, cotton farmers have been fortunate in being able to choose among varieties with resistance to several major pathogens, and advances during the past 10 years have increased the number of pathogens included. Eighty cotton varieties are planted to 1% or more of the acreage in the United States. Sixty-four varieties are planted in Texas, 37 in Oklahoma, 33 in New Mexico, and as few as 10 each in California and South Carolina. The problem is that fewer than 25 varieties have resistance to three diseases and no more than 15 have resistance to four or more diseases. Constraints imposed by fiber quality and adaptation limit the use of many varieties to relatively small areas of the cotton belt, and farmers in many cotton-growing regions cannot plant varieties with resistance to some of the major diseases (1).

Constraints placed on crop management by economics and concern for the environment dictate that host-plant resistance be the key component in strategies for maintaining plant health. Future strategies must incorporate components that complement and add to host resistance in a manner to achieve realistic results. Strategies must include the use of resistance so long as a meaningful level is available in an adapted variety (1,2).

The major diseases

Seedling disease. Seedling disease is handled as a complex that may be caused primarily by *Rhizoctonia solani* Kühn, *Pythium* spp., and *Thielaviopsis basicola* (Berk. & Br.) Ferraris, singly or

combined. Symptoms include seed rot, preemergence and postemergence damping-off, and root damage of surviving seedlings. Control strategies must involve preventing the various types of damage regardless of the pathogen or pathogens involved (3,5,6).

High-quality planting seed, i.e., with seed coat resistance to mold and reduced rate of germination after 7 days at 18 C, help prevent seedling disease. Such seed and their seedlings perform well in early-season plantings at 15–18 C, temperatures that are suboptimal for the soilborne pathogens. An escape from pathogens is provided while the crop is getting off to an early-season start, a desirable practice in all cotton-producing regions. Because seed lose these traits when exposed to moisture and high temperatures, high-quality seed can be produced only where relative humidities are low during the boll maturation and harvest period. Many seed companies produce planting seed in areas with such favorable conditions (e.g., the Trans-Pecos area of Texas, New Mexico, and Arizona), but this unfortunately is not always the case. When seed of reduced or unknown quality must be used, planting should be delayed until soil temperatures are high enough (20–22 C) to maximize the chance of success (1,3,5).

A high percentage of planting seed in the United States is treated with acid to remove the seed coat fuzz. The delinted seed are cleaned and gravity-graded to eliminate damaged and immature seed, then coated with protectant fungicides, one of which is usually a systemic. Such seed permit precision planting and play a significant role in controlling seedling disease.

Soil fungicides applied at planting and mixed into the covering soil provide added protection where seedling disease is a chronic problem. The practice is used in many regions of the cotton belt.

Varieties bred for seed quality preservation, reduced-temperature performance, and intermediate resistance to seedling pathogens are available for use in the Southwest. These improved varieties add reliability to strategies for controlling seedling disease (1,3).

Applying the appropriate strategy components (judicious use of different quality levels of seed, processed-treated seed, in-covering soil fungicides, and improved varieties) controls the seedling disease complex in most regions and under most circumstances. Controlling the pathogens that damage seed and/or seedlings and preserving optimal levels of

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certain seed and seedling traits helps assure good yield, earliness, and efficiency in combating other soil-inhabiting pathogens (seed and seedling traits explain 60–95% of the variability in yield, earliness, seedling damage, and host resistance to *Verticillium* wilt and *Phymatotrichum* root rot [3]).

Bacterial blight. *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye, the cause of bacterial blight, overwinters in infected and undecomposed dry plant debris and also survives on seed coat fuzz and inside the seed. Initial infection sites develop on cotyledons, from the seed, and by transmission in water from infected debris. Epidemics occur when rain or any form of moving water becomes contaminated and spreads the bacteria from the initial sites to other plants in a field.

Control involves burying infected debris and using pathogen-free planting seed. Debris decomposes naturally in high-rainfall areas, but in the arid Southwest and West, debris must be turned under and the land irrigated. Acid-delinting of planting seed in the United States assures no transmission in seed fuzz, but internal infestation remains a source of initial infection. Because internal infestation cannot be eliminated without seriously damaging the seed, disease-free seed and resistant varieties must be planted. States in which only a few varieties are planted may succeed in providing pathogen-free seed, but states

where many varieties are planted generally rely on resistant varieties. In the Southwest, 20 highly resistant and 10 partially resistant varieties are being used, accounting for about 43% of the acreage in Texas, 37% in Oklahoma, and 65% in New Mexico (1,3,5,6).

Fusarium wilt/root-knot nematode complex. Either *Fusarium oxysporum* f. sp. *vasinfectum* (Atk.) Snyder & Hans. or *Meloidogyne incognita* (Kofoid & White) Chitwood can damage cotton, and together they can destroy it. Control involves using resistant varieties and reducing populations of both soil-inhabiting pathogens. The wilt fungus is transmitted in seed, so seed produced in infested fields must be avoided. Varieties planted must have resistance to both the wilt fungus and the root-knot nematode. Since 1950, progressive improvement has been achieved in developing varieties with increasing levels of resistance to nematodes. Even so, use of cropping sequences that alternate nonhost crops with cotton is wise, and nematicides are required at times to keep nematode populations from becoming unusually high (1–3,5,6).

Verticillium wilt. *Verticillium albo-atrum* Reinke & Berth., the fungus causing *Verticillium* wilt, survives as microsclerotia in soil and in undecomposed plant debris. Control relies on the use of resistant varieties coupled with practices that reduce the inoculum potential. These include applying

nitrogen at a rate less than that required for optimum yield, maintaining a balanced nitrogen-potassium nutrition, providing for good water drainage, reducing by one-third the amount of irrigation water that would maximize yield in the absence of disease, maintaining soil temperature at about 30 C, using cropping sequences with nonhost crops, controlling taprooted host weeds, and using plant populations of 60,000–70,000 per acre. Desirable agronomic varieties with good resistance to *Verticillium* wilt are available for all areas of the cotton belt. The use of a resistant variety and several inoculum-reducing management practices prevent serious economic loss in most cases (1–3,5,6).

Phymatotrichum root rot. The causative fungus, *Phymatotrichum omnivorum* (Shear) Dug., is native to the southwestern and western regions of the United States and to Mexico in highly calcareous and alkaline soils. Root rot occurs in definable areas in fields where the fungus survives as sclerotia. Control centers on reducing sclerotial production and survival.

Application and burial by moldboard plowing of barnyard manure in infested areas have always controlled root rot. Simulation of the manure effect by growing legume crops (usually in winter) and turning the green residue under along with phosphate fertilizer also is effective. Cropping sequences with monocotyledonous crops and burial of their

residue have been used to reduce disease incidence (3,5); sequences in which two or more resistant crops, such as corn and small grains, are rotated with cotton are more effective than no rotation or use of corn only (3,5). Moldboard plowing and chiseling soil to depths of 10–12 in. to disrupt sclerotial formation and survival are beneficial.

The pathogen is most active at soil temperatures of about 27 C, which usually occur 90 days after planting. Varieties that set and mature 50–60% of their bolls within this period, even though plants are killed, help minimize yield losses. Tamcot MAR-2 and MAR-4 varieties with resistance usually have only one-half to two-thirds the number of dead plants as susceptible varieties and are available in the Southwest. Resistant varieties are also fast-maturing. Practices that reduce inoculum potential used in conjunction with fast-maturing resistant varieties add a new dimension to strategies for controlling root rot (2,3,5,6).

Boll rot. A number of fungi, bacteria, and yeasts cause boll rot. Some, including *X. c. pv. malvacearum* and *Glomerella gossypii* Edg., are primary pathogens. Many gain entrance via plant traits, e.g., boll sutures and nectaries, and wounds caused by insects and primary pathogens. Conditions that prolong periods of wetness and high humidities within the plant canopy and at the boll base promote serious boll rot.

Control is based on practices that reduce inoculum densities and promote

dryness within the leaf canopy and at the boll base. Sanitation to prevent seed transmission and survival on infected plant debris reduces the inoculum. Control of bacterial blight and boll-damaging insects reduces rot caused by saprophytic fungal invaders. Strategies that promote ventilation and rapid drying within the leaf canopy include minimal use of nitrogen to reduce plant size and leafiness and skip-row plantings in patterns to maximize the number of outside rows. Inner canopy movement of air and sunlight penetration are greater with plants that have okra-shaped leaves (indented margins to the extent the lobes appear almost as leaflets). Frego bract is long and narrow compared with the normal broad-base bract and permits rapid drying around the boll base, where three nectaries are normally located and serve as ports of entry. Thus, three plant traits aid in reducing boll rot. Commercial varieties with okra-shaped leaves and the nectariless character are available (2,3,5,6).

Other diseases. These include south-western cotton rust, fungal leaf spots, and diseases caused by viruses.

Summing up

In most situations, serious losses can be prevented by planting resistant varieties and using such inoculum-reducing strategies as cropping sequences, burial and destruction of crop residues, sanitation, and judicious nutrition and water management. Sometimes, however, adapted resistant varieties are not

available, continuous cropping is necessary, or burying residue is too costly. Research is continuing on developing more varieties resistant to major diseases and new biological control approaches to reducing inoculum potentials. These will help remove some management and economic constraints and will provide more extensive and reliable control of cotton diseases.

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