

Chapter 13

EFFECT OF HERBICIDES ON GROWTH OF COTTON AND ASSOCIATED CROPS

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INTRODUCTION

Herbicides are an essential part of cotton culture in the United States where labor is relatively expensive. This may not be the case in parts of the world where labor is abundant and relatively inexpensive. For the most part, herbicides used in accordance with state recommendations and labeling do not adversely affect cotton growth or production. However, with improper use such as overdose or uneven application or adverse weather which slows crop growth, herbicides may cause crop injury. A combination of the above circumstances can destroy a crop. Herbicide residues in soil from previous crops, and drift of non-target herbicides also can seriously injure cotton. In most cases, adverse effects from herbicide use are compensated for by reduced competition from weeds.

Surveys were conducted in 1962, 1965 and 1968 to determine how much herbicide persistence created a problem for various crops (Danielson *et al.*, 1965; Danielson *et al.*, 1968; Jansen *et al.*, 1972). In 1962, nine states indicated herbicide residues were a problem for cotton producers and five states were not affected by herbicide residues. All three western states—Arizona, California and New Mexico—indicated a problem. In 1965, eight states had troublesome herbicide residues in cotton and nine states were not troubled with herbicide residues. In 1968, the survey was more comprehensive and five percent, or 500,000 acres,

of the 10 million acres of cotton were affected by herbicide residues. However, this was a decrease from 1965 because seven states had a problem and 11 did not.

This chapter will provide information about potential herbicide injury: (a) from herbicides used in cotton on growth and yield of the crop, (b) when cotton is exposed to residues of persistent herbicides used in prior crops, and (c) from drift of nontarget herbicides to cotton. In addition, the effect of cotton herbicides on subsequent crops, and response of cotton cultivars to herbicides will be discussed. Finally, the roll of genetic engineering and biotechnology on production of cotton and other crops will be presented.

EFFECT OF COTTON HERBICIDES ON COTTON GROWTH AND YIELD

There are four major visual herbicide injury symptoms in cotton: stunting, chlorosis, necrosis and malformation (Cudney, 1986). Stunting or lack of growth can be caused by trifluralin (Treflan®) or pendimethalin (Prowl®) residues in soil that restrict root growth and development; top growth can be reduced by drift from some postemergence herbicides. Chlorosis (yellowing or whitening of foliage) results from herbicides such as diuron (Karmex®, others) and prometryn (Caparol®) that restrict photosynthesis. Residues of atrazine (AAtrex®, others) or propazine (Propazine, Milogard®) in soil can cause this type of injury. Necrosis or death of plant tissue is usually preceded by severe chlorosis, and occurs only from severe overdose or herbicide carry-over in soil. An exception to this generality would be drift of a non-target contact herbicide such as paraquat (Gramoxone®).

Non-lethal doses of contact herbicides may cause dead spots or flecking on leaves. Malformations of top growth of cotton are usually caused by drift of hormone type herbicides. Malformation can be twisting or bending of stems as well as unusual or abnormal growth of leaves. Dicamba (Banvel®), 2,4-D and MCPA have caused the most damage, but chlorsulfuron (Glean®), metsulfuron (Ally®) or glyphosate (Roundup®) which are not considered hormone herbicides can cause malformations to cotton plants. Common herbicide injury symptoms to cotton are summarized in Table 1.

Although not discussed here, injury symptoms caused by herbicides can be confused easily with problems caused by diseases, poor plant nutrition and toxic gasses in the air.

In 1984, there were 21 herbicides registered for weed control in cotton (Chandler, 1984). Of this group the following were marketed in 1988:

alachlor (Lasso [®])	linuron (Lorox, [®] Lunuron)
bensulide (Prefar [®])	methazole (Probe [®])
cyanazine (Bladex [®])	MSMA
dalapon (Dowpon [®])	norflurazon (Zorial [®])
DCPA (Dacthal [®])	oryzalin (Surflan [®])
diuron (Karmex [®])	paraquat (Gramoxone [®])
EPTC (Eptam, [®] Eradicane, [®] Short Stop, [®] Genep [®])	pendimethalin (Prowl [®])
fluometuron (Cotoran [®])	prometryn (Caparol [®])
glyphosate (Roundup [®])	trifluralin (Treflan [®])

Since 1988, the following have been added to this list:

metolachlor (Dual [®])	oxyfluorfen (Goal [®])
fluazifop-P (Fusilade 2000 [®])	sethoxydim (Poast [®])

Injury symptoms caused by herbicides most commonly used in cotton are discussed below.

DINITROANILINES (TRIFLURALIN, PENDIMETHALIN AND ORYZALIN)

Trifluralin (Treflan[®]) and pendimethalin (Prowl[®]) can severely stunt cotton-when incorporated too deeply into the soil. Oryzalin (Surflan[®]) must be sprayed preemergence to avoid injury. The effect is especially pronounced if cold wet weather follows planting. Growth of roots may be slowed; roots become stunted and swollen; and development of root hairs and secondary roots can be inhibited or prevented (Hicks and Fletchal, 1964; Holstun, 1957; Miller *et al.*, 1964; Mitchell and Bourland, 1986; Oliver and Frans, 1965). These symptoms were more pronounced when a fungicide and systemic insecticide were added to the soil with trifluralin (Schweizer and Ranney, 1965; Skroach *et al.*, 1986) (Figure 1).¹ Hardpans in the soil can cause injury symptoms to cotton roots very similar to those from dinitroaniline herbicides. Hardpans are most likely to occur in sandy soils that are wet and compacted by tractor traffic before and during planting. The worse injury develops if sandy soil is dry for several weeks immediately after planting.

S-TRIAZINES (PROMETRYN AND CYANAZINE)

Prometryn and cyanazine interfere with photosynthesis by inhibiting electron transport in the Hill reaction. This in turn reduces normal chlorophyll production and usually results in a yellow interveinal chlorosis. However, under some conditions, veinal chlorosis occurs. Severe injury results in stunting and death. This injury is most likely to occur on sandy soils with low organic matter and high pH

¹All of the figures in this chapter are placed as a group toward the end of the chapter between the text and Literature Cited section.

Table 1. Herbicide injury symptoms on cotton (Monaco *et al.*, 1986).

Herbicides	Injury symptom ¹ category	Injury symptoms
Trifluralin (Treflan [®]) Pendimethalin (Prowl [®]) Oryzalin (Surflan [®])	3	Thickened hypocotyls and tap roots having reduced lateral roots and root hairs resulting in stunted plants.
Diuron (Karmex [®]) Linuron (Lorox [®])	1a	Veinal chlorosis (yellow) and stunting. Occasionally interveinal chlorosis.
MSMA (Many) DSMA (Many)	4	Red stems and leaf petioles, abnormal bolls and stunting.
Norflurazon (Zoriat [®])	1b	Veinal chlorosis (white) and stunting.
Alachlor (Lasso [®]) Metolachlor (Dual [®])	3	Stunted with rolled leaves that do not open normally.
Paraquat (Gramoxone [®])	4	Drift can cause leaf flecking to complete destruction of leaves.
Chlorsulfuron (Glean [®]) Chlorimuron (Classic [®]) Metsulfuron (Ally [®]) Imazaquin (Scepter [®])	3	Stunting, mild chlorosis of leaves, and reddish purple stems.
Atrazine (AATrex [®]) Simazine (Princep [®]) Propazine (Milogard [®]) Fluometuron (Cotoran [®]) Prometryn (Caparol [®])	1a	Interveinal chlorosis (yellow) and stunting. Occasionally veinal chlorosis.
Fomesafen (Relfex [®])	4	Stunted seedlings with yellow leaves. Dead leaves turn brown or black as if burned.
2,4-D and other phenoxy herbicides Dicamba (Banvel [®]) Picloram (Tordon [®])	2	Stunted with crinkled, strapped, elongated and malformed leaves.

¹Injury symptom category: 1a = Photosystem II (photosynthetic) inhibitors; 1b = carotenoid (pigment) inhibitors; 2 = growth regulator herbicide; 3 = cell division inhibitors; 4 = contact (membrane destruction) herbicides.

(Black, 1985; Brian, 1964). Interveinal chlorosis can be caused by iron deficiency and is impossible to distinguish from herbicide injury (Figure 2).

It may be possible to distinguish injury by the weeds or volunteer crops that are controlled. For example, prometryn (Caparol®) would severely injure corn but cyanazine (Bladex®) would not.

UREAS (DIURON, FLUOMETURON AND LINURON)

These herbicides also inhibit the Hill reaction in photosynthesis. However, the symptoms on cotton plants vary. With diuron (Karmex®) and linuron (Lorox®), leaf veins become yellow and tissue between veins stays green. Fluometuron (Cotoran®), on the other hand, causes yellow interveinal chlorosis similar to the s-triazines, prometryn and cyanazine. This symptom can occur following an overdose in the soil or following postemergence directed sprays. However, on occasions, interveinal chlorosis has been observed (Figure 3).

ARSENICALS (MSMA AND DSMA)

MSMA and DSMA are used widely for controlling johnsongrass, annual grasses, cocklebur and nutsedge in cotton. Sprays should be directed at the base of cotton plants but frequently are applied over-the-top to the crop. Typical injury symptoms from MSMA and DSMA are stunting and red discoloration of cotton stems and petioles. In most cases, both stunting and discoloration disappear as the season progresses. With severe injury from overdose, bolls can be abnormally shaped reducing both turnout and yield. Injury symptoms are most likely to develop from topical sprays at early square (Lucas, 1964; Guthrie, 1986; Wiese and Hudspeth, 1968).

PYRIDAZINONES (NORFLURAZON)

Norflurazon (Zorial®) is a carotenoid or pigment inhibitor. These pigments are associated with protection of chlorophyll. Injured cotton has whitish to pale yellow leaf veins and green interveinal tissue. Chlorotic areas may have pink or red coloration around the edges (Figure 4) (Monaco *et al.*, 1986). In corn and sorghum, leaves become white, and if severe enough, leaf tissue dies along the edges. Under low rainfall and high pH soils common in semi-arid areas, norflurazon persists a long time and white leaves may show up on sensitive crops for several years after the herbicide is used. Severely affected seedlings may be devoid of chlorophyll and severely stunted.

CHLOROACETAMIDES (ALACHLOR AND METOLACHLOR)

These herbicides, Lasso® and Dual®, interfere with cell division, cell elongation and protein synthesis. Under cool wet conditions and when rain or irrigation comes shortly after planting, cotton plants may be severely stunted and have rolled or poorly shaped leaves. Also, root growth may be inhibited, leaving plants

subject to diseases. With severe injury, germination can be inhibited or stopped (Monaco *et al.*, 1986).

GLYPHOSATE

Although cotton is moderately tolerant, glyphosate cannot be sprayed safely on top of the crop. The herbicide is registered for use on weeds before the crop is planted, or applied with ropewick or roller applicators for weeds that extend above the crop (Keeley *et al.*, 1984a; Keeley *et al.*, 1984b). Also, glyphosate has been used extensively for spot-treating perennial weeds in cotton. Severe stunting may result if the herbicide contacts cotton plants. Most serious injury will occur when glyphosate is sprayed over-the-top of cotton at 1.5 to 3 pounds per acre. These rates delay maturity and decrease boll size enough to reduce yield (Banks and Santelmann, 1977). In another study, glyphosate sprayed over-the-top of cotton reduced blooming, fruit set and yield (Frans *et al.*, 1982). Repeated treatments at 0.5 pound per acre almost eliminated growth and yield of cotton. At normal use rates, once glyphosate contacts soil it is no longer toxic to cotton.

PARAQUAT

Paraquat (Gramoxone®, others) is used to control weeds prior to planting and for desiccating cotton to speed harvest. The herbicide is absorbed by green plant tissue; it is nonselective and fast acting—produces wilting, tissue darkening and necrosis within a few hours after treatment depending on environmental conditions. Symptoms from spray drift are necrotic spots often referred to as leaf flecking. Paraquat is a divalent cation that is reduced to an unstable form in the plant's photosynthetic system. The unstable products interact with plant parts triggering a series of events leading to rapid breakdown of cell membranes. Young actively growing plants are extremely sensitive to injury. Paraquat is adsorbed by clay and organic matter in soil; consequently, soil residues are not a problem except in very sandy soils with low organic matter (Monaco *et al.*, 1986).

CYCLOHEXENONES (PHENOXAPROP AND SETHOXYDIM) AND ARYLOXYPHENOXY ALKANOIC ACIDS (FLUAZIFOP-P)

These herbicides (Poast® and Fusilade 2000®) are postemergence growth regulators that are sprayed on plant foliage. Injury to cotton has not been observed at rates of application up to 10 pounds per acre. These herbicides are very toxic at 0.5 pounds per acre or less to both annual and perennial grass weeds that infest cotton or other broadleaved crops. Injury symptoms on treated grasses include reddening of stems and leaves. This is followed by necrosis and gradual death of the plant (Figure 5). In large plants that have a well defined stem, the top internode is the first part of the plant to die. Leaves or heads of affected plants can be pulled out easily because of the dead node.

EFFECT ON COTTON OF HERBICIDE CARRYOVER FROM OTHER CROPS

When cotton is grown in rotation with other crops, persistent herbicides used in previous crops may cause serious injury to cotton. In general, herbicide persistence varies with location in the Cotton Belt, and consequently recropping intervals vary accordingly (Table 2). Herbicides persist longest under dry conditions, cool temperatures and in soils with high pH. Herbicide persistence is the longest in the Southwest and West, intermediate in the Mid-South, and least in the southeastern part of the Cotton Belt. The most common offenders are chlor-sulfuron (Glean[®]) and metsulfuron (Ally[®]) used in wheat; atrazine (AAtrex[®], others) and propazine (Milogard[®] and others) used in sorghum or corn; and metribuzin (Sencor[®], Lexone[®]), chlorimuron (Classic[®]), imazaquin (Scepter[®]) and fomesafen (Reflex[®]) used in soybeans. The more herbicide applied, the longer period of persistence in soil.

Table 2. Recropping intervals for cotton after using herbicides in crops rotated with cotton.

Crop	Herbicide	Rate (LB/A)	Recropping interval		
			Southwest	Mid-South	Southwest/West
			(Months)		
Corn	Atrazine	3	12	12	12
Sorghum	Propazine	3	12	12	12-18
Soybeans	Metribuzin	0.75	8	8	12
	Chlorimuron	0.016	9	9	NR ²
	Imazaquin	0.125	18	18	NR
	Fomesafen	0.19	10	10	NR
	Chlorsulfuron ¹	0.024	NR	14	14-26
Wheat	Metsulfuron	0.004	NR	NR	34

¹On soils with pH less than 7.9.

²NR = Not registered in that area.

A bioassay is the safest and cheapest way to determine if residue in soil will injure a crop. The easiest and quickest bioassay can be done in a greenhouse or a south window. About twenty samples of soil should be taken to a 6-inch depth over the field in question and carefully mixed. Soil from another field known to be free of herbicide residues should be taken to the same depth, mixed and used for a check or untreated control. At least three pots of soil from each of the two fields should be planted to the crop in question. Crop injury will be observable in three to six weeks.

With sulfonylurea herbicides, planting the crop in the field is the only effective bioassay because greenhouse bioassays are not as sensitive as field bioassays.

The amount of herbicide that will injure a susceptible crop varies with soil type

as well as crop. It takes less herbicide to injure a crop in a coarse textured soil than in a fine textured soil.

Soil can be sent to a laboratory for a chemical analysis. However, the amount of herbicide, usually given in parts per million (ppm), does not indicate if a crop can be grown safely. The ppm reading from the analyses can be related to pounds per acre by assuming the top three inches of soil weights one million pounds and all of the herbicide is in that soil layer. Then a ppm reading is equal to pounds per acre. If six inches of soil is sampled, dividing the ppm reading by two will give pounds per acre because two million pounds of soil were sampled. Very little research has been done to relate herbicide concentration (ppm) in soils and injury to various crops.

SULFONYLUREAS (CHLORIMURON, CHLORSULFURON AND METSULFURON)

Sulfonylurea herbicides produce toxic effects by inhibiting cell division and growth (Anonymous, 1983). The primary mode of action is through inhibition of valine and isoleucine biosynthesis (Ray, 1984) (Figure 6). This leads to stunting, mild chlorosis, purpling or anthocyanin expression and eventually death.

Chlorimuron (Classic[®]) is used postemergence in soybeans at 0.008 to 0.012 pounds per acre for control of common cocklebur, pigweed, yellow nutsedge, smartweed and annual morningglories. It can be applied anytime from soybean emergence to sixty days before harvest. A second application may be made two to three weeks after the first, but total amount applied may not exceed 0.016 pounds per acre. Recropping intervals vary with soil pH and area of the country. Cotton can be planted nine months after application of chlorimuron anywhere if soil pH is 6.8 or less. In the Southeast and Mid-South, cotton can be planted nine months after application if soil pH ranges from 6.9 to 7.5. The interval is eighteen months in Tennessee and southern Missouri.²

Chlorsulfuron (Glean[®]) is registered in many states—including the cotton producing states of Arkansas, California, Louisiana, New Mexico, Oklahoma and Texas—for use in small grains and fallow land. In these states, the maximum use rate is 0.024 pounds per acre on soils having a pH of 7.9 or less.³ No more than one application of this amount can be made to a crop or fallow period.

In Arkansas, Louisiana, eastern Oklahoma and eastern Texas, the rotation interval is fourteen months regardless of rate of application, but can be extended longer until there is at least 25 inches of precipitation. In western Oklahoma and central Texas, maximum rate of application is only 0.016 pounds per acre in order to have a 14-month rotational interval before planting cotton. In western Texas, a field bioassay must be used prior to planting cotton.

Metsulfuron (Ally[®]) is used in small grains and may persist in soil for more than one year at high enough concentrations to injure cotton. Metsulfuron is regis-

²Classic[®] label, E. I. duPont De Nemours & Co., Willmington, DE 19898.

³Glean[®] label, E. I. duPont De Nemours & Co., Willmington, DE 19898.

tered at 0.004 pounds per acre in several states, but the list includes only New Mexico and Texas that produce cotton. No specific crop rotation guidelines are established for cotton, but a minimum crop rotation interval of thirty-four months is suggested on the label.⁴ A field bioassay should be used the year before planting cotton to determine if residues are no longer injurious to cotton. A field bioassay can be conducted by planting strips of cotton across the field perpendicular to the way it was sprayed.

IMIDAZOLINONE (IMAZAQUIN)

Imazaquin (Scepter[®]) is a broad spectrum herbicide for soybeans that can be applied preplant incorporated, preemergence or postemergence for control of most common broadleaf and grass weeds. It is registered for eastern, central and southern United States, and is used in a variety of rotations with soybeans and cotton. Imazaquin kills plants by inhibiting production of the amino acids valine, leucine and isoleucine. Consequently, symptoms of injury are very similar to those caused by sulfonylurea herbicides. Maximum application rate is 0.125 pounds of active ingredient per acre for all times of treatment. Small grains and rice may be planted four months after application, but cotton planting must be delayed for eighteen months.⁵

S-TRIAZINES (ATRAZINE, SIMAZINE AND PROPAZINE)

Atrazine (AAtrex[®], others), simazine (Princep[®], others), and propazine (Milogard[®], others) are marketed under many trade names, and have been used for over twenty-five years. Residues of these herbicides can be a problem when cotton is grown in rotation with corn or sorghum. Injury from s-triazine herbicides is typified by chlorosis (yellowing) along the edge of leaves followed by necrosis. With more serious injury, yellowing occurs between leaf veins (Figure 7). This is in contrast to veinal chlorosis caused by the urea herbicide, diuron (Monaco *et al.*, 1986).

Rotational crop restrictions vary for the three herbicides. Atrazine treated fields can be rotated only to corn or sorghum the year following application. Corn is the only crop that can be planted the year following use of simazine. With propazine, recropping restrictions vary with location. In Arkansas, Louisiana, Texas Gulf Coast, Texas Blacklands and the Southeast, fields treated with propazine can be planted to cotton, soybeans or corn twelve months after treatment. In Oklahoma, New Mexico and West Texas, treated fields can be planted to cotton if no more than 1.0 pound per acre was used the previous year.⁶

BENZOIC AND PHENOXYACETIC ACID (DICAMBA, 2,4-D)

Safe use of dicamba (Banvel[®]) or 2,4-D for controlling weeds just before plant-

⁴Ally[®] label, E. I. duPont De Nemours & Co., Wilmington, DE 19898.

⁵Scepter[®] label, American Cyanamid Company, Wayne, NJ 07470.

⁶Milogard[®], Princep[®], and AAtrex[®] labels, CIBA-GEIGY Corporation, Greensboro, NC 27419.

ing cotton in no-tillage cropping systems varies with location. In the dry parts of the West and Southwest, dicamba should not be used in the spring prior to planting cotton otherwise seedlings may be killed. Applications of 2,4-D may be made until six weeks before planting; however, if it does not rain during this time, serious injury may result. In the more humid areas of the Cotton Belt, such as the Mississippi Delta, dicamba at 0.25 or 2,4-D at 2.0 pounds per acre could be sprayed on stale seedbeds sixteen days prior to seeding cotton without reducing yields. Cotton planted three or nine days after spraying was seriously injured by the herbicides (Baker, 1988).

FOMESAFEN

Fomesafen (Reflex[®]) is a postemergence herbicide for soybeans that controls most broadleaf weeds at 0.19 pounds per acre or less. Some perennial grass weeds are suppressed at this rate of application. The product is registered for the cotton producing states along the Atlantic seaboard west to Missouri, Arkansas, Louisiana and southeastern Texas.⁷ When fomesafen residues in soil cause injury to susceptible plants, seedlings are stunted, leaves turn yellow, and plants eventually may die. Dead leaves turn dark brown or black and appear burned. Small grains are very tolerant of fomesafen and may be replanted on treated fields four months after application. Cotton, corn, peanuts and rice may be replanted after ten months.

EFFECT OF SPRAY DRIFT ON COTTON

PHENOXY AND OTHER HORMONE-LIKE HERBICIDES

The phenoxy and other hormone-like herbicides such as picloram (Tordon[®]), dicamba (Banvel[®]), triclopyr (Garlon[®]) and clopyralid (Lontrel[®]) are extremely important for control of weeds and brush on grazing lands. However, these herbicides will cause injury if allowed to drift from sprayed areas to cotton fields.

2,4-D—Ergle and Dunlap (1949) showed that greenhouse-grown cotton (Stoneville 2B) was sensitive to low rates of the sodium salt of 2,4-D. Plant height and the number of main-stalk nodes decreased in proportion to the amount of 2,4-D applied. The 2,4-D inhibited development of fruiting branches and reduced leaf weight. The two highest concentrations suppressed flower production, boll set and seed yield. Fiber weight, maturity and tensile strength also were reduced by low rates of 2,4-D. The authors concluded that, if evenly dispersed, 0.06 pounds of 2,4-D could cause serious damage to every cotton plant in 35 acres.

It was recognized early that drift from 2,4-D could cause serious injury to cotton (Brown *et al.*, 1948; Dunlap, 1948; Ergle and Dunlap, 1949) (Figure 8). Arle (1954) reported injury to cotton also could occur from volatilization of 2,4-D ester, use of 2,4-D contaminated sprayers, 2,4-D carried in irrigation water,

⁷Reflex[®] 2LC label, ICI Americas Inc., Willmington, DE 19897.

smoke from burning weeds and brush treated with 2,4-D and reuse of 2,4-D containers.

In Mississippi, Carns and Goodman (1956) indicated that applications of 0.001 pounds per acre of 2,4-D at the seedling stage delayed cotton maturity, but had little effect on seed yield. Application of 0.01 pounds per acre delayed maturity and reduced yield. At the squaring stage, 0.001 pounds per acre had no clear-cut effect on yield or earliness, but 0.01 pounds per acre reduced yield. At flowering, applications of 0.001 and 0.01 pounds per acre had little effect, but 0.1 pounds per acre reduced both yield and earliness. The boll stage was most resistant to 2,4-D.

In Arizona, Arle (1954) found that seed yields from 'Acala 44' cotton were reduced when spray applications of the amine salt of 2,4-D were made at low rates on June 7 (a few squares on each plant) and picked on September 30. However, at later harvests the trend was reversed and yield from treated areas was consistently higher than from untreated areas. Application made on July 7 (numerous squares and blooms) caused greater yield reduction at the first and second picking than June treatments, but late-season recovery on December 28 yielded about 350 percent over the control. The August 7 treatment had little or no adverse effect on quantity of cotton picked. Arle's experiments also indicated that cotton could tolerate quantities of up to 1.0 pound per acre of 2,4-D in irrigation water without adversely affecting yields.

2,4-D Versus Other Phenoxy's—Most research indicates that 2,4-D is more phytotoxic to cotton than other phenoxy's (Behrens *et al.*, 1955; Goodman, 1953; Miller *et al.*, 1963; Porter *et al.*, 1959; Watson, 1955). Goodman (1953) indicated that 2,4-D reduced yield of 'Coker 100W' cotton more than 2,4,5-T. Goodman *et al.* (1955) found that, at equal rates, 2,4-D reduced yield of cotton ten times more than 2,4,5-T. MCPA caused more leaf modification in cotton than 2,4,5-T, but yield response was similar. Cotton exposed to 2,4-D generally was most sensitive at the seedling and square stages than at flowering or boll stages. Cotton response to MCPA and 2,4,5-T was similar to 2,4-D except magnitude of injury was less. The butyl ester of silvex was more injurious than the butyl ester of 2,4,5-T. Dichlorprop had little or no effect on cotton yield. Watson (1955), using 'Coker 100W' cotton, generally agreed with Goodman (1953) and Goodman *et al.* (1955) except that 2,4-D and MCPA were about equal in reducing yields of cotton. Watson (1955) indicated the amine salt of silvex was more injurious than the amine salt of 2,4,5-T, and that the sodium salt and ester of silvex were more injurious than the amine salt.

At four locations in Texas, Behrens *et al.* (1955) found that 2,4-D caused more leaf malformation in cotton than 2,4,5-T and MCPA. Silvex resulted in no appreciable leaf malformations. Similarly, 2,4-D caused greatest reduction in cotton yield followed by 2,4,5-T and MCPA with silvex causing the least reduction in yield.

Porter *et al.* (1959) evaluated low rates of several herbicides at four stages of cotton growth. The 8- to 10-leaf stage was most sensitive to 2,4-D when measured by yield reduction. Silvex and 2,4,5-T were nonspecific in yield reduction relative to stage of cotton growth. Rates of 2,4-D as low as 0.0001 pound per acre did not stimulate yield of cotton as reported by other investigators. Miller *et al.* (1963) reported cotton seed yields were most drastically reduced by 0.01 and 0.1 pound per acre of 2,4-D applied during the flowering and fruit-setting stages. Seed quality was reduced by 2,4-D treatment and fiber quality was reduced by foliar application of 2,4-D at 0.1 pound per acre.

Other Hormone Herbicides—Recently Smith and Wiese (1972) compared 2,4-D to dicamba (Banvel®), picloram (Tordon®), bromoxynil (Buctril®) and 2,3,6-TBA. Order of damage to cotton was 2,4-D ester > 2,4-D amine >> dicamba > MCPA > picloram >> bromoxynil >> 2,3,6-TBA. Sprays of 2,4-D, dicamba or MCPA at 0.1 pound per acre reduced lint yields from 20 to 97 percent. Yield losses were most severe when cotton was sprayed before blooming. However, lint quality (micronaire and length) was not affected by these herbicides.

Patterson and Buchanan (1981) found that commercial formulations of 2,4,5-T might contain trace quantities of 2,4-D. Experiments were conducted to determine if the 2,4-D contaminant was sufficient to cause greater injury to cotton than purified 2,4,5-T applied alone. Commercial formulations of 2,4,5-T with small quantities of added 2,4-D caused slightly more visual injury to cotton than purified 2,4,5-T. However, there were no differences in cotton seed yield among materials. The only treatment that decreased cotton yield was 0.1 pound per acre of 2,4-D ester.

Bovey *et al.* (1968) found that 'Blightmaster' cotton could be planted in a tropical soil two months after applications of the potassium salt of picloram at 6.0 pounds per acre, the butyl ester of 2,4-D + 2,4,5-T at 12 pounds per acre each, or a 2:2:1 mixture of the isooctyl esters of 2,4-D + 2,4,5-T + picloram at a total of 15 pounds per acre. 'Tancot' cotton seedlings were injured by foliar sprays of 2,4,5-T, triclopyr (Garlon®) and clopyralid (Lontrel®) at 0.03 pound per acre in the greenhouse (Bovey and Meyer, 1981). No new growth occurred when cotton was treated with 0.125 or 0.5 pound per acre of 2,4,5-T or triclopyr. Clopyralid was less injurious to cotton than triclopyr and 2,4,5-T and only slight leaf malformations occurred at 0.03 pound per acre or less. Since clopyralid has shown excellent control of honey mesquite in Texas, damage from spray drift of this herbicide should be minimal.

PHYSIOLOGICAL EFFECTS OF 2,4-D AND PICLORAM

2,4-D—All seed from bolls (one to 15 days old) set prior to treatment with 0.0000036 or 0.000036 ounce (0.1 or 1.0 mg) of 2,4-D per plant produced seedlings which exhibited symptoms of 2,4-D injury in 60-day old 'Stoneville 2B' cotton (McIlrath *et al.*, 1951). Some seedlings from seed produced in bolls initi-

ated eight weeks after application of 0.0000035 to 0.00035 ounce (0.1 to 10 mg) 2,4-D per plant exhibited 2,4-D injury, but seed formed fourteen weeks after application did not produce malformed seedlings. Plants treated with 0.00000035 or 0.0000012 ounce (0.01 or 0.04 mg) of 2,4-D at seedling or floral primordia stages showed no transmission of the stimulus into seed embryos, but seed embryos formed in bolls initiated five weeks after anthesis showed significant 2,4-D injury. McIlrath and Ergle (1953) showed that 2,4-D symptoms persisted in 'Stoneville 2B' and 'Marie Galante' cottons up to six months. The 2,4-D was extracted eighty days after application from plants showing leaf malformation characteristic of 2,4-D. However, Morgan and Hall (1963) indicated that cotton decarboxylated the side chain of 2,4-D several times faster than sorghum and in young leaves and bolls slowly converted 2,4-D to a chromatographically different material. After cotton recovered from 2,4-D treatment, it could not be detected in subsequent vegetative or reproductive growth.

Picloram—Picloram increased the soluble protein concentration of cotton when applied at 1 ppb into the roots of seedlings (Baur *et al.*, 1970). At 100 ppb picloram, soluble protein tended to be reduced but was not different than for untreated plants.

RICE HERBICIDES

In certain areas, cotton is grown adjacent to rice. The potential for drift or accidental overspray of rice herbicides on cotton can be significant.

The most widely used rice herbicide is propanil (Stam[®], Stampede[®]). Smith *et al.* (1977) demonstrated that low rates of propanil were most injurious to small cotton. Propanil at 0.5 pound per acre or above resulted in significant yield reductions when applied to 2- to 4-inch tall or 10- to 22-inch tall cotton. Generally, those plants not killed produced new leaves and near-normal yields, but often maturity was delayed.

Eastin (1975)^{*} concluded that propanil was the most injurious of several rice herbicides evaluated on 3- to 5-leaf cotton. In addition, molinate (Ordram[®]) at 3.0 pounds per acre and oxadiazon (Ronstar[®]) at 0.8 pound per acre reduced cotton yield. Combinations of molinate, thiobencarb (Bolero[®]), and oxadiazon with Stam[®] also resulted in yield reductions comparable to using Stam[®] alone. However, propanil was the chemical causing the majority of injury.

Hurst (1982, 1984) and Smith *et al.* (1977) concluded that leaf injury did not always result in reduced yield because plants recovered from initial injury. Hurst (1982) also concluded that injury was greater during years of cold or wet conditions which reduced seedling vigor, and made the plants more sensitive to herbicides. The order of phytotoxicity to cotton was propanil (Stam[®], Stampede[®]) > bifenox (Modown[®]) > oxadiazon (Ronstar[®]) > acifluorfen (Tackle[®], Blazer[®]) > butachlor (Machete[®]) = thiobencarb (Bolero[®], Saturn[®]) (Hurst, 1982). When ap-

^{*}Eastin, E. F. 1975. Unpublished data.

plied to 5- to 8-node cotton, propanil was the only herbicide that caused significant injury at lower than recommended rates for rice.

In response to problems of cotton adjacent to rice fields, Helms *et al.* (1987) determined the effect of propanil and thiobencarb when applied preemergence to the cotton. Although propanil is a contact herbicide with little preemergence activity, cotton grown in contaminated soil was injured and yield reduced. Addition of aldicarb (Temik®) or disulfoton (Di-syston®), in-furrow cotton insecticides, increased herbicide injury. When applied preemergence, addition of thiobencarb to propanil resulted in more cotton injury than from propanil alone. Simulated drift of 0.1 and 0.01 times the normal rate of application to rice resulted in significant yield reductions when applied to cotton in the cotyledon or 4-leaf stages of growth. The available information indicates that extreme care should be taken when applying rice herbicides, particularly those containing propanil, near cotton fields.

SOYBEAN HERBICIDES

Careless use of mixtures of bentazon (Basagran®) or acifluorfen (Blazer®, Tackle®) can result in drift damage to cotton. Injury is characterized by blotchy yellow areas on leaves that were hit by the spray (Figure 9). Fortunately, this injury does not cause serious reduction in yield of cotton.

EFFECT OF COTTON HERBICIDES ON OTHER CROPS

Herbicides used in cotton can injure other crops by spray drift from adjacent areas or by persisting in the soil. Of herbicides used in cotton production, glyphosate (Roundup®) and paraquat (Gramoxone®, others) are most likely to drift to adjoining crops such as winter small grains. Drift of either herbicide must be prevented or serious damage may result. Yates *et al.* (1978) observed drift of glyphosate over 300 feet downwind from a ground rig using conventional nozzles. Drift from sethoxydim (Poast®) or fluazifop-P (Fusilade 2000®) may injure grass crops. These herbicides are toxic to susceptible plants immediately after spraying, but do not persist and affect subsequent crops (Buhler and Burnside, 1984; Chernicky and Slife, 1986).

TRIFLURALIN AND PENDIMETHALIN

Trifluralin (Treflan®) and pendimethalin (Prowl®) applied either at layby or preplant incorporated can injure sorghum planted before May 15 of the following year in West Texas (Abernathy and Keeling, 1979; Wiese *et al.*, 1969). If more than recommended rates were applied at layby, sorghum planted after the middle of May was injured. There is not a carryover problem with these herbicides in areas with more precipitation than West Texas. Layby applications of these herbicides may seriously reduce stand of sugarbeets (Warner *et al.*, 1987).

FLUOMETURON

Fluometuron (Cotoran[®]) persists in the soil longer than either trifluralin (Treflan[®]) or pendimethalin (Prowl[®]), and can injure soybeans at very low rates (Bode and McWhorter, 1977). Postemergence sprays also can reduce yield of soybeans when sprayed during the mid-bloom stage. The effect of three cotton herbicide programs on yield of subsequent crops of wheat, hairy vetch, corn, sorghum, rice, soybeans and cucumber was determined on three soil types (Rogers *et al.*, 1986). Herbicide systems were either: (a) none, (b) fluometuron (Cotoran[®]) applied preemergence followed by a mixture of MSMA and fluometuron applied twice postemergence, and (c) trifluralin (Treflan[®]) preplant incorporated, fluometuron preemergence, two sprays of fluometuron plus MSMA postemergence, followed by a directed spray of linuron (Lorox[®]). Herbicide injury to wheat and hairy vetch planted immediately after cotton harvest was most pronounced on Sharkey silty clay but was less on Dundee silt loam and Loring silt loam soils. The intensive herbicide program (c) was most injurious. The following year, corn and grain sorghum suffered the least damage while rice, soybeans and cucumbers were injured most. Fluometuron residues in the soil was the chief cause of injury.

In most areas, crops other than cotton cannot be planted until six months after the last application of fluometuron. No more than three applications of fluometuron should be made per season. In Arizona, California and New Mexico, cotton, corn and grain sorghum can be planted the next crop season but other crops should not be planted for one year after the last application.⁹

NORFLURAZON

Norflurazon (Zorlan[®]) persists in soil for more than one year in dry areas and is not registered for the entire Cotton Belt. There are rotational crop restrictions in certain states. Holt (1986) indicates norflurazon has an initial half-life of thirty to sixty days, but a second half-life is usually six to twelve months. Speed of breakdown is affected by soil type, incorporation method, moisture and temperature. These results were from fields that had been treated for three years at recommended rates and incorporated prior to planting. Norflurazon persisted longest in soil with high silt content. The year following the third annual application of norflurazon at 1.25 pound per acre, there was less than 0.62 pound per acre in the soil. Seedlings of corn and sorghum planted the year following the last application were bleached after eight weeks but by twelve weeks were normal (Figure 10).

In a similar study, norflurazon at 1.5 and 3 pounds per acre was sprayed annually for three years and incorporated with tandem disking on a Bosket silt loam soil in Mississippi (Hurst and Tupper, 1981). A greenhouse bioassay indicated enough norflurazon present in the soil two months after cotton harvest to injure wheat and soybeans.

⁹Cotoran[®] label, CIBA-GEIGY Corporation, Greensboro, NC 27419.

PROMETRYN AND METOLACHLOR

Prometryn (Caparol®) and metolachlor (Dual®) do not persist in the soil for more than a few weeks and are registered on many crops.¹⁰ If these herbicides are used on cotton and the crop fails, other labeled crops can be replanted. Cabbage, okra, onions, peas, red beets and sweet corn may be double-cropped immediately after cotton harvest following prometryn applied preplant or preemergence. In California and Arizona, spring-seeded crops and vegetables may be planted after April 1. Barley, oats, rye or wheat may be planted 4.5 months after treatment with metolachlor. Most other crops can be planted the next spring.

TOLERANCE OF COTTON CULTIVARS TO HERBICIDES

GLYPHOSATE

During the late 1970s, researchers tried glyphosate (Roundup®) as a post-emergence topical herbicide in cotton and soybeans (Banks and Santelmann, 1977; Wills, 1978). In order to increase cotton tolerance to glyphosate, Jordan and Bridge (1979) evaluated resistance of 405 genotypes to sprays of 0.75, 1.5 and 3.0 pounds per acre during early bloom. There was considerable difference in tolerance, and six of the most resistant lines were sprayed the next year at 0.5 and 0.75 pound per acre. None of the treated lines yielded as much as untreated comparisons; however, genotypes 'DES 04-11' and 'DES 04-606' yielded more than untreated controls when glyphosate at 0.5 pound per acre was directed at the base of the cotton plants. Although there was considerable tolerance of some cotton lines to glyphosate, it was not safe to spray the many varieties that are planted. Because the crop is quite tolerant, glyphosate can be used in ropewick applicators, recirculating sprayers or for spot treatments to control perennial weeds in cotton (Keeley *et al.*, 1984a, 1984b). Using a ropewick applicator to control johnsongrass markedly increased yield of cotton compared to cultivating and hoeing (Keeley *et al.*, 1984b). When mixed with the defoliant DEF®, glyphosate has been used to control regrowth prior to harvest (Cathy and Barry, 1977).

2,4-D

Differential tolerance of cotton cultivars to 2,4-D was noted when drift from neighboring fields caused injury in a yield trial (Regier *et al.*, 1986). Under these conditions, 'Paymaster 145' cotton did not show injury symptoms and outyielded eleven other cultivars. Yield of the twelve cultivars was negatively correlated with an injury rating where 1 was no leaf injury, and 5 was when all leaves had 2,4-D injury symptoms. 'McNair 307' and 'G&P 3774' cotton were injured most by the 2,4-D drift. In an effort to explain the tolerance of 'Paymaster 145', two studies were conducted to determine if leaf hairiness affected resistance to 2,4-D

¹⁰Caparol® and Dual® labels, CIBA-GEIGY Corporation, Greensboro, NC 27419.

(Dilbeck *et al.*, 1987). In the first study, lines used were 'Paymaster 145' along with 'Texas marker- 1' isolines 'pilose' and 'smooth leaf'. A single spray of 2,4-D at 0.5 pound per acre was allowed to drift across the three lines. In a second study, five lines were sprayed with 2,4-D at 0.001, 0.01, and 0.05 pound per acre. Visual ratings and lint yield indicated an advantage to leaf hairiness, but 'Paymaster 145' yielded most, showing presence of an additional genetic mechanism for tolerance to 2,4-D.

PROPAZINE AND ATRAZINE

Atrazine (AAtrex®, others) and propazine (Milogard®, others) were sprayed preemergence at 0.5 pound per acre on forty-eight cotton cultivars in a sandy clay loam soil (Abernathy *et al.*, 1979). Injury over two years averaged from 8 to 80 percent for atrazine and 3 to 53 percent for propazine. 'Paymaster 303', 'Stripper 31 A', 'Auburn M', 'Acala B-3030' and 'Acala 1517-70' were most tolerant to atrazine, and 'Stripper N' was most susceptible. The most tolerant cultivars to propazine were 'Auburn M', 'Acala 3080', 'Paymaster 303', 'Coker 5110' and 'Coker 312'. The most sensitive were 'Paymaster 110', 'Deltapine SR-2' and 'Blightmaster A-5'. In another trial, lint yield of 'Paymaster 303' was not reduced by preemergence sprays of propazine at 1.0 pound per acre while lint yield of 'Paymaster 101A' was reduced from 371 pounds per acre on untreated areas to 112 pounds per acre where the herbicide was applied.

TRIFLURALIN

Twenty cotton cultivars were evaluated for tolerance to trifluralin (Treflan®) (Mesri, 1980; Bourland *et al.*, 1981). Laboratory evaluation with germination paper or cups of sand treated with trifluralin indicated 'McNair 235' and 'DES 24' had some tolerance. Later field evaluations at one, two and three times labeled rates of trifluralin showed that 'Tancot CAMD-E', 'Stoneville 603', 'Stoneville 825' and 'McNair 235' cottons had the highest tolerance.

BIOTECHNOLOGY AND GENETIC ENGINEERING

Biotechnology and genetic engineering are two areas of research that have emerged and grown rapidly during the last fifteen years. Biotechnology is a general term that deals with application of biological and engineering techniques to microorganisms, plants and animals. Genetic engineering relates to the part of biotechnology where genetic components of organisms are altered by human intervention. Traditional plant breeding by crossing lines among species that hybridize, as well as more modern techniques where genes or chromosomes are moved from one organism to another are part of genetic engineering. Genetic engineering has been used for over one hundred years to select and create plants and animals with desired characteristics.

The laws of inheritance were discovered by Mendel (1866) when he made crosses, observed and interpreted the definite pattern of flower color in the offspring of peas. About three years after Mendel's work on the laws of inheritance, Miescher (1869) discovered nucleic acids, which later were proved to be the building blocks of heredity. These two discoveries remained unused for decades.

Mendel's principles of inheritance were not rediscovered until the early part of the 20th century when Bateson *et al.* (1905) again reported additional cases of the interaction of factors or genes. Shortly thereafter a number of individuals contributed to the theory that genes were located on chromosomes. The scattered bits of information were summarized and interpreted by Morgan *et al.* (1915) in "The Mechanism of Mendelian Heredity". Other developments that led to modern hybrid plants were published by Shull (1908, 1909). He demonstrated that a genetically heterogeneous crop like corn could be selfed and resolved into a large number of genetically uniform and stable inbred lines. Jones (1918) took this information a step farther and proposed a system of double-crossing the selfed lines of corn. He demonstrated hybrid vigor and thereby laid the foundation for practical production of high yielding hybrids of corn and other crops.

The key to genetic engineering in the modern sense was discovered by Watson and Crick (1953) who described the double helix structure of deoxyribonucleic acid (DNA). DNA in cells acts like a magnetic tape and directs each phase of development, be it a single cell or a highly specialized plant or animal. The long double strands of spiraling thread-like molecules of DNA are located in chromosomes in the cell nuclei of all plants and animals. The term "gene" is given to the region of the DNA molecule that gives rise to a particular genetic characteristic. Genes control synthesis of specific protein molecules within the DNA which are comprised of different combinations of the four amino acids: adenine, thiamine, cytosine and guanine. Combinations of the four amino acids hooked together in long sequences constitute the almost unlimited number of genetic codes that control development of organisms.

PLANT CELL AND TISSUE CULTURE

Regeneration of plant cells or tissue cultures is a part of biotechnology. Using many different techniques, a variety of plants can be grown from single cells or tissues (Legates, 1986). This technique has the potential of producing a large number of genetically identical plants from another plant having rare or desirable characters such as superior growth or disease resistance. The best method currently developed involves placing tissue into a container of culture medium made up of plant hormones, auxin and cytokinin along with organic and inorganic nutrients necessary for growth. The cells divide and form a mass of cells called callus. When callus cells are transferred to a special regeneration medium, they differentiate into roots and shoots and, in some instances, develop into whole plants. Detailed methods of doing this are provided by Camper (1986).

RECOMBINANT DNA TECHNOLOGY

The technique called recombinant DNA technology is relatively new. It involves removing or adding genes in an individual species. The purpose of this technique is to transfer a desirable characteristic from one organism to another that cannot be crossed by conventional plant breeding methods. This sounds simple, but is very complicated. First, it is difficult to identify a specific gene in a chromosome that controls a particular characteristic. Once this is done, however, it is even more complicated to transfer a gene to another plant or animal.

Incorporating genes from one species to another has been accomplished by using restrictive enzymes that cut DNA strands at a specific sequence of amino acids. This technique makes it possible to isolate specific genes. Restrictive enzymes also are used to incorporate the isolated gene into the receptor organism. Scores of restriction enzymes have been isolated that are capable of cutting DNA at specific points.

PRACTICAL APPLICATIONS OF BIOTECHNOLOGY

Several applications of biotechnology and genetic engineering have been used to modify plants. The ability to make a toxin has been transferred from a bacterium that does not normally occur in the root zone to a bacterium that grows on or near plant roots. This new modified bacterium produced the toxin which inhibited cutworms feeding on roots of young plants.

Resistance to the herbicide imazapyr (Arsenal®) has been transferred into lines of corn. Similarly, resistance to bromoxynil (Buctril®), a postemergence herbicide used in small grains and corn, has been transferred into tomatoes and tobacco (Arthur, 1987). A gene that is tolerant to glyphosate (Roundup®) was isolated from a soil bacterium and transferred into *Agrobacterium tumefaciens* that causes crown-gall disease in certain plants. The leaves of poplar trees were infected with the new bacterium containing the gene for glyphosate tolerance. This moved the desirable gene into cells of the poplar leaves. Then the bacterium was killed, and poplar shoots were regenerated by tissue culture from these leaves. This resulted in poplar trees that could be sprayed with glyphosate giving foresters a new herbicide for poplar nurseries. Similar resistance to glyphosate has been genetically engineered into petunia and tobacco plants (Camper, 1986). Tissue and cell culture techniques also have been used to study herbicide resistance and metabolism.

Prospects for future improvement in plants have scientists in industry and academic institutions excited (Fraley, *et al.*, 1984; Sundquist *et al.*, 1982; Widholm, 1984; Wild, 1984). Transferring a single gene between varieties or lines of plants that normally cross could be more rapid by molecular techniques than by conventional plant breeding. A trait could be transferred into a new variety without six to eight backcrosses normally used to transfer desirable characteristics from one variety to another.

The ability to produce multicellular plants from a single cell or protoplast of-

fers many possibilities for plant improvement. The most exciting possibility is using recombinant DNA to improve economically important plant species by transferring genes from one species to another that do not normally hybridize. For example, corn and sorghum will not cross, but transferring certain traits such as disease resistance or drought tolerance would be highly desirable. Wild or exotic species could be more readily utilized in breeding programs by introducing whole sets of new genes or genomes. Also, with new molecular techniques, it may be possible to modify a gene from one species before inserting it into another. Resistance to pests or pesticides, for the most part, is controlled by single genes that may not be found in crop plants. Creating cultivars of cotton and other crops that are resistant to certain pests or pesticides should be relatively easy because of the simple inheritance.

Another exciting possibility that will be more complicated is transferring genes for nitrogen fixation from microorganisms into non-leguminous plants such as cotton. The increased supply of nitrogen for crop growth would be especially beneficial in Third World countries where nitrogen fertilizer is not readily available or is too expensive. Transferring characteristics such as stress tolerance, quality and yield which are controlled by numerous genes or groups of genes will be difficult to accomplish.

Biotechnology and genetic engineering combined with conventional plant breeding will hasten production of new and improved varieties of cotton and other crop plants. It is most likely that in the next ten to twenty years, cotton seed will be available that is resistant to many common diseases and insects. In addition, cultivars will have tolerance to specific herbicides. Weed problems in cotton may be eliminated by over-the-top sprays of such herbicides as glyphosate, paraquat or imazapyr. Farther in the future, cotton cultivars may release toxins into the soil that eliminate weeds.

SUMMARY

Herbicides are an essential part of cotton culture and, if used according to label instructions, neither cotton or crops grown in rotation will be injured. When cotton is injured by misuse or poor application of herbicides, yields may be maintained or increased because of decreased competition from weeds. However, a combination of adverse circumstances can result in serious injury to cotton and other crops grown in rotation.

Stunting, necrosis, chlorosis and malformation are the four major herbicide injury symptoms in cotton. Overdose or misuse of trifluralin (Treflan®) or pendimethalin (Prowl®) can cause stunting because root growth is restricted. This injury occurs when the herbicides are incorporated into beds several inches below where cotton seed are planted. This problem is most pronounced when dry weather follows planting. Compacted soils, as well as herbicides, may restrict

root development. In many instances, damage to roots caused by soil compaction has been blamed on trifluralin or pendimethalin.

Stunting or malformed growth can be caused by drift of 2,4-D or similar compounds. Injury to cotton from drift of 2,4-D and similar herbicides will be a problem as long as cotton is grown near wheat and sorghum crops where 2,4-D is the most economical herbicide available. Also, in areas where cotton is grown near brush-infested rangeland, some damage from drift will continue to be a problem. However, since 2,4,5-T was banned, drift damage decreased because new herbicides for brush are less toxic to cotton. Chlorosis or yellowing of leaves is caused by herbicides that inhibit photosynthesis. This includes diuron (Karmex[®]), prometryn (Caparol[®]), fluometuron (Cotoran[®]), linuron (Lorox[®]), atrazine (AAtrex[®], others) and propazine (Milogard[®], others). Norflurazon (Zorial[®]) inhibits pigment formation and causes cotton and other plants to turn white.

Atrazine, propazine, chlorsulfuron (Glean[®]) and metsulfuron (Ally[®]) are persistent herbicides used in other crops that can injure subsequent crops of cotton. Herbicide persistence in soil has been a problem to cotton producers on five percent or about 500,000 acres. This problem is most severe in the driest part of the country, including the states of Texas, New Mexico, Arizona and California. Herbicide persistence in soil is increased when dry weather occurs the year before cotton is planted. Herbicides used in cotton that may affect subsequent crops are trifluralin, pendimethalin, fluometuron and norflurazon. Sorghum is most frequently injured by trifluralin and pendimethalin. This injury can be prevented by planting after May 15. Fluometuron and norflurazon are not registered in parts of the West and Southwest because of potential for injuring crops following cotton.

Certain cotton cultivars and lines have tolerance to glyphosate, 2,4-D, propazine, atrazine and trifluralin. Biotechnology including genetic engineering offers the promise of greatly increasing production of cotton and other crops. Present advances in biotechnology include development of corn and tobacco that can tolerate specific herbicides. Using a combination of conventional crop breeding and biotechnology, cotton cultivars will be developed that are resistant to many diseases and insects as well as specific herbicides such as glyphosate.

A



B



Figure 1. Injury to cotton (upper) and sorghum (lower) roots caused by trifluralin (Treflan®). In the cotton deformed roots on outside and normal on inside. (Courtesy of J. R. Abernathy, Texas Agricultural Experiment Station and L. R. Oliver, University of Arkansas.)



A



B

Figure 2. Interveneal chlorosis: (upper) caused by a poorly directed application of cyanazine (Bladex®) mixed with DSMA, and (lower) iron deficiency in cotton. (Courtesy of C. T. Bryson, USDA, ARS and J. R. Abernathy, Texas Agricultural Experiment Station.)



Figure 3. Veinal chlorosis can be caused by overdose of diuron (Karmex®) and under some circumstances by prometryn (Caparol®) which normally causes interveinal chlorosis. (Courtesy J. R. Abernathy, Texas Agricultural Experiment Station.)



Figure 5. Red johnsongrass leaves caused by a spray of fluazifop (Fusilade®).

A



B

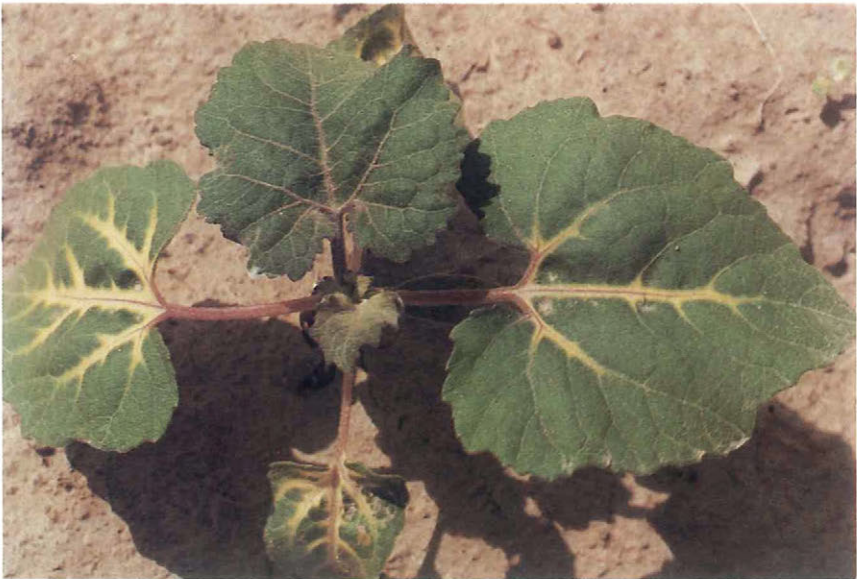


Figure 4. Veinal chlorosis (white) caused by norflurazon (Zorial[®]) on cotton (upper) and common cocklebur (lower). (Courtesy of C. T. Bryson, USDA, ARS and L. R. Oliver, University of Arkansas.)

A



B

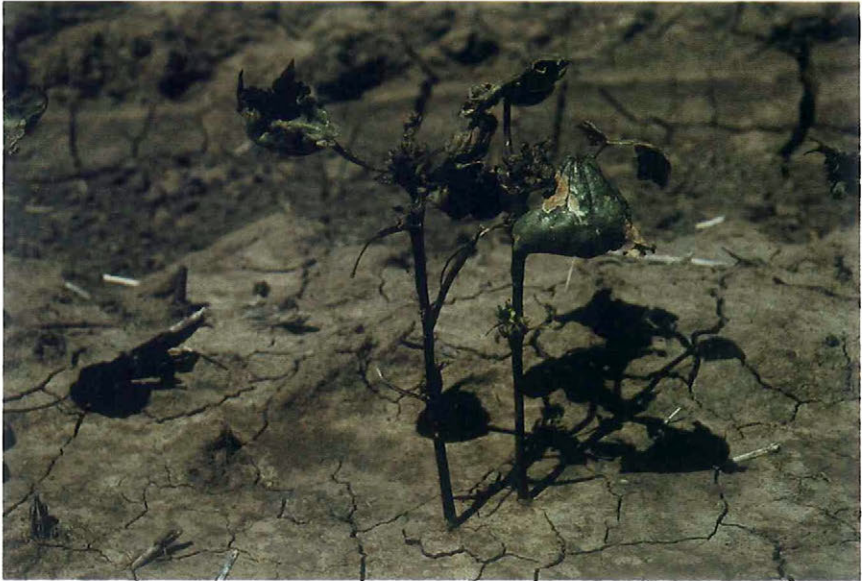


Figure 6. Sulfonylurea injury to cotton: (upper) metsulfuron (Ally®) 0.005 pounds per acre preplant incorporated and (lower) chlorsulfuron (Glean®) 0.001 pound per acre postemergence.



Figure 7. Interveinal chlorosis on cotton caused by preplant incorporated application of atrazine (AAtrex[®], others) at 1 pound per acre.

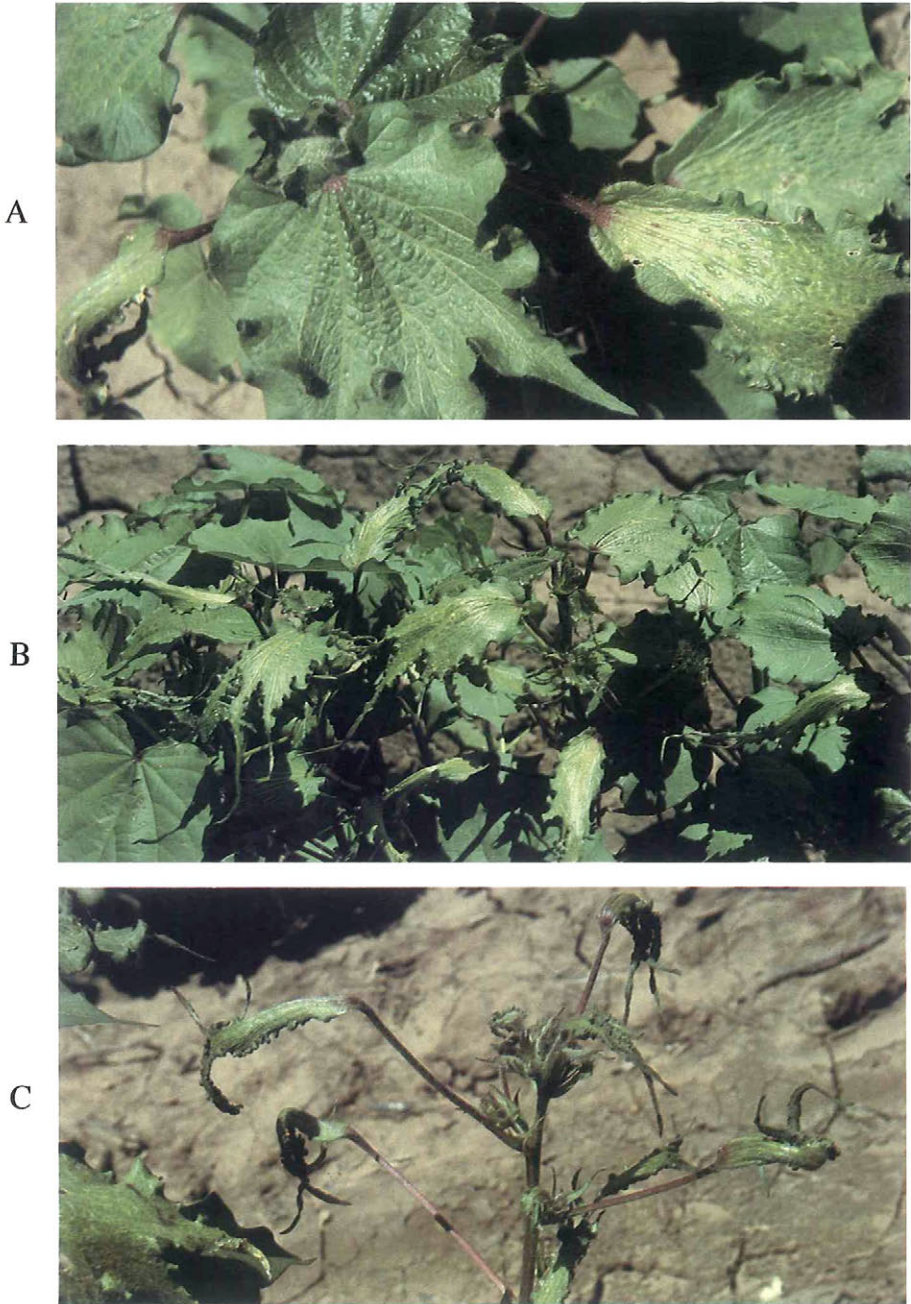


Figure 8. Effect of 2,4-D drift on cotton leaves. Upper, middle and lower: light, moderate and severe damage, respectively.



Figure 9. Leaf chlorosis in cotton caused by drift from bentazon (Basagran®).
(Courtesy of J. R. Abernathy, Texas Agricultural Experiment Station.)



Figure 10. Norflurazon (Zorial®) injury to corn. (Courtesy of L. R. Oliver, University of Arkansas.)

LITERATURE CITED

- Abernathy, J. R. and J. W. Keeling. 1979. Efficacy and rotational crop response to levels and dates of dinitroaniline herbicide applications. *Weed Sci.* 27:312-317.
- Abernathy, J. R., J. W. Keeling, and L. L. Ray. 1979. Cotton cultivar response to propazine and atrazine. *Agron. J.* 73:929-931.
- Anonymous. 1983. Glean technical bulletin. No. 43612. E.I. duPont de Nemours and Co. Willmington, Delaware. 12 pp.
- Arle, H. F. 1954. The sensitivity of Acala 44 cotton to 2,4-D. *Proc. West. Weed Control Conf.* 14: 20-25.
- Arthur, T. E. 1987. Crop biotechnology. *Ag Consultant.* 43:(5)6-8.
- Baker, R. S. 1988. Response of cotton planted after application of hormone herbicides on stale seedbeds. *Abstr. Weed Sci. Soc. Am.* Page 10.
- Banks, P. A. and P. W. Santelman. 1977. Glyphosate as a postemergence treatment for johnsongrass control in cotton and soybeans. *Agron. J.* 69:579-582.
- Bateson, W., E. R. Saunders, and R. C. Punnett. 1905. Experimental studies in the physiology of heredity. *Reports to Eval. Comm. Royal Soc.* 2:1-131.
- Baur, J. R., R. W. Bovey, and C. R. Benedict. 1970. Effect of picloram on growth and protein levels in herbaceous plants. *Agron. J.* 62:627-630.
- Behrens, R., W. C. Hall and C. E. Fisher. 1955. Field responses of cotton to four phenoxy-type herbicides. *Proc. South. Weed Conf.* 8:72-75.
- Black, C. C., Jr. 1985. Effects of herbicides on photosynthesis. Pages 1-36 in S. O. Duke ed. Weed Physiology. Vol. II. Herbicide Physiology CRC Press, Inc. Boca Raton, Florida.
- Bode, L. E. and C. G. McWhorter. 1977. Toxicity of MSMA, fluometuron and propanil to soybeans. *Weed Sci.* 25:101-105.
- Bourland, F. M., S. Mesri, and B. White. 1981. Differential tolerance of cotton cultivars to trifluralin. 1981 *Proc. Beltwide Cotton Prod. Res. Conf.* Pages 103-106.
- Bovey, R. W., F. R. Miller, and J. Diaz-Colon. 1968. Growth of crops in soils after herbicidal treatments for brush control in the tropics. *Agron. J.* 60:678-679.
- Bovey, R. W. and R. E. Meyer. 1981. Effects of 2,4,5-T, triclopyr, and 3,6-dichloropicolinic acid on crop seedlings. *Weed Sci.* 29:256-261.
- Brian, R. C. 1964. A classification of herbicides and types of toxicity. Pages 1-38 in L. J. Audus ed. The Physiology and Biochemistry of Herbicides. Academic Press, New York.
- Brown, C. A., Q. L. Hodleman and E. S. Hagood. 1948. Injuries to cotton by 2,4-D. *Bull.* 426. Louisiana Agric. Exp. Stn. 12 pp.
- Buhler, D. D. and O. C. Burnside. 1984. Herbicidal activity of fluzifop-butyl, haloxyfop-methyl and sethoxydim in soil. *Weed Sci.* 32:824-831.
- Camper, N. D. 1986. Herbicide studies with plant tissue and cell cultures. Pages 385-398 in N. D. Camper ed. Research Methods in Weed Science. Southern Weed Science Society. Champaign, Illinois.
- Carns, H. R. and V. H. Goodman. 1956. Responses of cotton to 2,4-D. *Bull.* 541. Mississippi Agric. Exp. Stn. 15 pp.
- Cathey, G. W. and H. R. Barry. 1977. Evaluation of glyphosate a harvest aid chemical in cotton. *Agron. J.* 69:11-14.
- Chandler, J. M. 1984. Cotton protection practices in the USA and world. Section D: Weeds. Pages 330-365 in R. J. Kohel and C. F. Lewis eds. Cotton. Agronomy Monograph No. 24. American Society Agronomy. Madison, Wisconsin.
- Chernicky, J. P. and F. W. Slife. 1986. Effects of sublethal concentrations of bentazon, fluzifop, haloxyfop and sethoxydim on corn. *Weed Sci.* 34:171-174.
- Cudney, D. W. 1986. Herbicide symptoms in agronomic crops. *Proc. 38th Annual California Weed Conf.* 38:123-127.

- Danielson, L. L., W. B. Ennis, Jr., D. L. Klingman, W. C. Shaw, and F. L. Timmons. 1965. A survey of extent and cost of weed control and specific weed problems. Agric. Res. Serv., Fed. Ext. Serv., U. S. Dep. Agric., 34-23-1, Washington, DC. 78 pp.
- Danielson, L. L., W. B. Ennis, Jr., J. T. Holstun, Jr., L. L. Jansen, D. L. Klingman, and F. L. Timmons. 1968. Extent and cost of weed control with herbicides and an evaluation of important weeds 1965. Agric. Res. Serv., U. S. Dep. Agric., ARS 34-102. 85 pp.
- Dilbeck, R. E., J. E. Quisenberry, A. F. Wiese and C. G. Regier. 1987. Comparison of hairy and smooth leaf phenotypes to drift and direct application of 2,4-dichlorophenoxy acid. 1987 Proc. Beltwide Cotton Prod. Res. Conf. Page 132.
- Dunlap, A. A. 1948. 2,4-D injury to cotton from airplane dusting of rice. Phytopathology. 38:638-644.
- Ergle, D. R. and A. A. Dunlap. 1949. Responses of cotton to 2,4-D. Bull. 713. Texas Agric. Exp. Stn. 18 pp.
- Fraley, B., S. Rogers, and R. Horsch. 1984. Expression of foreign genes in plants. Proc. South. Weed Sci. Soc. 37:13.
- Frans, R. E., M. E. Terhune and T. J. Cothren. 1982. Response of cotton to foliar applications of MSMA and glyphosate. Proc. Plant Growth Regulator Soc. Am. 9:112-113.
- Goodman, V. H. 1953. The yield and progeny seedling responses to treatment with 2,4-D, 2,4,5-T and M.C.P. Proc. South. Weed Conf. 6:47-56.
- Goodman, V. H., W. B. Ennis, Jr., and R. D. Palmer. 1955. Cotton responses to 2,4-D, 2,4,5-T, MCP and related growth regulators. Proc. South. Weed Conf. 8:76-81.
- Guthrie, D. S. 1986. Fruiting profile of cotton following overtop applications of fluometuron and MSMA. Proc. Beltwide Cotton Prod. Res. Conf. Page 252.
- Helms, R. S., T. N. Tripp, and J. S. McConnell. 1987. Simulated herbicide drift injury to cotton. Proc. South. Weed Sci. Soc. 40:68.
- Hicks, R. D. and O. H. Fletchall. 1964. Preplant incorporation studies in cotton weed control. Proc. South. Weed Conf. 17:157.
- Holstun, J. T., Jr. 1957. A preliminary study on the effects of weeds in cotton. Proc. South. Weed Conf. 10:30.
- Holt, T. J. 1986. Norflurazon rotational crop studies. Proc. Beltwide Cotton Prod. Res. Conf. Pages 252-253.
- Hurst, H. R. and G. R. Tupper. 1981. Nutsedge control in narrow-row cotton. 1981 Proc. Beltwide Cotton Prod. Res. Conf. Pages 165-168.
- Hurst, H. R. 1982. Cotton (*Gossypium hirsutum*) response to simulated drift from selected herbicides. Weed Sci. 30:311-315.
- Hurst, H. R. 1984. Cotton response to simulated drift. 1984 Proc. Beltwide Cotton Prod. Res. Conf. Pages 254-255.
- Jansen, L. L., L. L. Danielson, W. B. Ennis, Jr., P. A. Frank, J. T. Holstun, Jr., and D. L. Klingman. 1972. Extent and cost of weed control with herbicides and an evaluation of important weeds, 1968. Econ. Res. Serv., Ext. Serv., and Agric. Res. Serv., U. S. Dep. Agric., ARS 34-102. 227 pp.
- Jones, D. F. 1918. The effects of inbreeding and crossbreeding on development. Bull. 207. Connecticut Agric. Exp. Stn. Bull. Pages 5-100.
- Jordan, T. N. and R. R. Bridge. 1979. Tolerance of cotton to the herbicide glyphosate. Agron. J. 71:927-928.
- Keeley, P. E., R. J. Thullen, C. H. Carter, and J. H. Miller. 1984a. Control of johnsongrass in cotton with glyphosate. Weed Sci. 32:306-209.
- Keeley, P. E., C. H. Carter, R. J. Thullen, and J. H. Miller. 1984b. Comparison of ropewick applicators for control of johnsongrass in cotton with glyphosate. Weed Sci. 32:431-435.
- Legates, J. E. 1986. Genetic engineering in food and agriculture. Council of Agricultural Science and Technology. Report No. 110. Ames, Iowa. 47 pp.
- Lucas, R. E. 1964. Ansar 184, Disodium methanearsonate (DSMA) and related compounds as selected johnsongrass herbicides in cotton. Proc. South. Weed Conf. 17:62-64.

- McIlrath, W. J., D. R. Ergle, and A. A. Dunlap. 1951. Persistence of 2,4-D stimulus in cotton plants with reference to its transmission to the seed. *Bot. Gaz.* 112:511-518.
- McIlrath, W. J. and D. R. Ergle. 1953. Further evidence of persistence of the 2,4-D stimulus in cotton. *Plant Physiol.* 28:693-702.
- Miescher, F. 1869. (cited by Legates, J. E. 1986. Genetic engineering in food and agriculture. Council of Agricultural Science and Technology. Report No. 110. Ames, Iowa. 47 pp.)
- Mendel, G. 1866. Versuche Uber Pflanzenhybriden. *Verh. Naturf. ver. Brunn.* 4:3-47.
- Mesri, S. 1980. Differential effects of trifluralin on cotton cultivars. MS thesis, Mississippi State University. Mississippi State, Mississippi. 56 pp.
- Miller, J. H., H. M. Kempen, J. A. Wilkerson, and C. L. Foy. 1963. Response of cotton to 2,4-D and related phenoxy herbicides. *Tech. Bull.* 1289. U.S. Dep. Agric. 28 pp.
- Miller, J. H., B. Fisher, and A. H. Lange. 1964. Progress report on pre-plant herbicides for weed control in cotton. *California Agric.* 18:6-7.
- Mitchell, G. A. and F. M. Bourland. 1986. Effects of trifluralin and pendimethalin on cotton emergence and seedling characteristics. 1986 Proc. Beltwide Cotton Prod. Res. Conf. Pages 64-66.
- Monaco, T. J., A. R. Bonanno, and J. T. Baron. 1986. Herbicide injury: Diagnosis, causes, prevention and remedial action. Pages 399-428. in M. D. Camper, ed. Research Methods and Weed Science. Southern Weed Science Society. Champaign, Illinois.
- Morgan, P. W. and W. C. Hall. 1963. Metabolism of 2,4-D by cotton and grain sorghum. *Weeds.* 11:130-135.
- Morgan, T. H., A. H. Sturdevant, H. J. Muller, and C. B. Bridges. 1915. The Mechanism of Mendelian Heredity. Henry Holt and Co., New York.
- Oliver, L. R. and R. E. Frans. 1965. Influence of trifluralin rate and depth of incorporation on cotton and soybean lateral root development. *Proc. South. Weed Conf.* 18:85-91.
- Patterson, M. G. and G. A. Buchanan. 1981. Response of cotton to phenoxy herbicides. *Proc. South. Weed Sci. Soc.* 34:221.
- Porter, W. K., Jr., C. H. Thomas, and J. B. Baker. 1959. A three-year study of the effect of some phenoxy herbicides on cotton. *Weeds.* 7:341-348.
- Ray, T. B. 1984. The site of action of chlorsulfuron: inhibition of valine and isoleucine biosynthesis in plants. *Plant Physiol.* 75:827-831.
- Regier, C. G., R. E. Dilbeck, D. J. Undersander, and J. E. Quisenberry. 1986. Cotton resistance to 2,4-dichlorophenoxy acid spray drift. *Crop Sci.* 26:376-377.
- Rogers, C. B., R. Talbert, and R. Frans. 1986. Effect of cotton herbicide carryover on subsequent crops. *Weed Sci.* 34:756-760.
- Schweizer, E. E. and C. D. Ranney. 1965. Interactions of herbicides, a fungicide and a systemic insecticide in cotton. *Info. Sheet* 877. Mississippi Agric. Exp. Stn. 1 pp.
- Shull, G. H. 1908. The composition of a field of maize. *Am. Breed. Assoc. Rep.* 4:296-301.
- Shull, G. H. 1909. A pure line method of corn breeding. *Am. Breed. Assoc. Rep.* 5:51-59.
- Skroach, W. A., T. J. Sheets, H. D. Coble, M. A. Cohen, F. T. Corbin, W. M. Lewis, T. J. Monaco, D. E. Moreland, L. Thompson, J. B. Weber, and A. D. Worsham. 1986. Herbicide injury symptoms and diagnosis. AG-85. North Carolina Agric. Ext. Ser. 30 pp.
- Smith, D. T. and A. F. Wiese. 1972. Cotton response to low rates of 2,4-D and other herbicides. B-1120. *Texas Agric. Exp. Stn.* 8 pp.
- Smith, R. J., Jr., W. T. Flinchum and D. E. Seaman. 1977. Weed control in U. S. rice production. *Handbook* 497. U. S. Dept. Agric. 82 pp.
- Sundquist, W. B., K. M. Menz, and C. F. Neumeyer. 1982. A technology assessment of commercial corn production in the United States. *Bull.* 546. Minnesota Agric. Exp. Stn. 22 pp.
- Warner, J. R., S. R. Winter, and A. F. Wiese. 1987. Persistence of dinitroaniline herbicides and potential for injury to sugarbeets. *J. Am. Soc. Sugar Beet Technol.* 24:57-66.
- Watson, A. J. 1955. The response of cotton to low rates of 2,4-D, 2,4,5-T, MCP and silvex. *Proc. South. Weed Conf.* 8:82-86.

- Watson, J. D. and F. H. C. Crick. 1953. Molecular structure of nucleic acids. *Nature* 171:737-738.
- Widholm. 1984. Genetic engineering in weed science. *Abstr. Weed Sci. Soc. Am.* Page 120.
- Wiese, A. F. and E. B. Hudspeth, Jr. 1968. Effects of DSMA and MSMA on cotton yield and arsenic content of cotton seed. MP-877. *Texas Agric. Exp. Stn.* 4 pp.
- Wiese, A. F., E. W. Chenault, and E. B. Hudspeth, Jr. 1969. Incorporation of preplant herbicides for cotton. *Weed Sci.* 17:481-483.
- Wild, J. R. 1984. Genetic engineering impact on weed science. *Abstr. Weed Sci. Soc. Am.* Page 119.
- Wills, G. D. 1978. Factors affecting toxicity and translocation of glyphosate in cotton. *Weed Sci.* 26:509-513.
- Yates, W. E., N. B. Akesson, and D. E. Bayer. 1978. Drift of glyphosate sprays applied with aerial and ground equipment. *Weed Sci.* 26:597-604.