Auxin-type growth-regulating herbicides encompass all chemicals that have a physiological action resembling that of indole-3-acetic acid (Woodford et al., 1958). The most conspicuous among such herbicides is the group represented by substituted phenoxy acids. They have a hormone-like activity in view of the fact that at low dosage they cause growth responses in regions distant from the point of application. Like the naturally-occurring plant auxins they affect germination, growth, development, morphogenesis, cambial activity, dedifferentiation, regeneration, root initiation, phototropic and geotropic responses, abscission, fruiting, etc. According to Ashton and Crafts (1973), these chemicals produce epinasty, cessation of growth, tumour formation and secondary root initiation. Yet, IAA is not a herbicide, presumably because the endogenous enzyme indole-acetic acid oxidase rapidly destroys or inactivates it in most plant tissues soon after application (Galston, 1956). The reason for this marked difference between one of these important naturally-occurring auxins and synthetic auxins (phenoxy compounds) is of great significance being reflected in abnormal growth induced by the latter.
The largest tonnage of the organic herbicides used throughout the world at present consists of the substituted phenoxy acids, viz. 2,4-D, 2,4,5-T, 2,4,5-TP, MCPA and their derivatives. These are widely and selectively used for the control of broad-leaf weeds and 2,4,5-T in particular as arboricide. These are used in form of mother acids, as a salt or as an ester. Woodford (1957) has suggested that the change in relative toxicity which arises from minor modifications to the aliphatic side-chain are probably due to alteration in physical properties of the molecule and this has a differential effect on penetration, retention and transport within the plant.

The growth abnormalities induced by these herbicides result from endogenous hormonal imbalance. It has been established that this effect of 2,4-D can be prevented completely by the simultaneous application of IAA and that under certain conditions 2,4-D alone reduces endogenous IAA content. This has led Weintraub (1953) to postulate that the vital action of 2,4-D lies in its interference with natural auxins in the plant. Furthermore, these herbicides interact with numerous enzyme systems (Woodford et al., 1958). As kinins cause orderly cell division, it has been proposed that an auxin-kinin imbalance results in the abnormally induced growth from disorganised cell division (Overbeek, 1964). It has been
opined that 2,4-D and relatives kill plants like cancer. Increased ethylene production by 2,4-D application has led Holmes and Abeles (1968) to infer that some of the responses of soybean-cessation of growth of seedling, tissue swelling, increase in RNA, DNA and protein in subapical hypocotyl tissue results from increased ethylene production. Abeles (1968) has concluded that ethylene do not account for the herbicidal activity of 2,4-D, since it did not kill his test plants. Rather ethylene might play a role in the proliferation growth. Hanson and Slife (1969) have also studied ethylene production by 2,4-D with particular interest. The phenoxy compounds are known to modify nucleic-acid metabolism in plants (Hanson and Slife, 1969) and cause cytological aberrations (Kum Kum and Sharma, 1976; Grant, 1979). Grant (1979) has shown that 2,4,5-T produces chromosomal aberration including bridges and micronuclei, cell enlargement, lengthens the duration of mitotic cycle, prolongs DNA synthesis, induces mitosis, C-mitosis, polyploidy, etc. Aberg and Eliasson (1978) have stated that the lethal effect of phenoxy acids on plants in low doses is related to their auxinic properties in physiological and biochemical aspects. The phytotoxicity is assumed to be a result of disturbance of normal regulatory mechanism of endogenous auxins. At higher concentrations, phenoxy acids may have other effects on plant tissues, such as uncoupling of oxidative
phosphorylation and destruction of membranes. Shevchenko et al. (1978) have pointed out that phenolic substances produced in plants under the influence of chloro-phenoxy-acetic acids play a significant role in phytotoxicity.

The narrow-leaf weeds are resistant, in general, to the phenoxy compounds. Zemskaya et al. (1977) are of the opinion that detoxification of 2,4-D is accompanied in oat, wheat, maize and barley by binding with cell-wall proteins, cytoplasmic protein and non-protein compounds. This prevents translocation in newly-forming organs which is a significant factor in the high resistance of cereals to the herbicide. Tomkins and Grant (1978), while studying survival of 75 weed species exposed to selective (auxin-type) and non-selective herbicides, have found that polyploid forms are more resistant.

The halo-aliphatic acids have occupied a place in agriculture. The grass-killing properties of sodium trichlorate (TCA) have been observed by McCall and Zahnley (1949). The herbicidal action of chloroacetic acid has been described by Zimmerman et al. (1951). Dalapon, obtained by substituting one chlorine atom of TCA by a methyl group, gave a new dimension to weed control after its introduction by Dow chemical Company in 1953. Although other chlorinated aliphatics have herbicidal properties, they have not come into common usage. These compounds act in grasses in much the same way that 2,4-D
do in broad-leaf plants. Dalapon has proved its superiority over TCA in that it is more freely translocated in the phloem and xylem and can be applied to either soil or foliage. TCA, on the other hand, is a soil-acting herbicide. Both compounds are particularly effective against grasses, but they also control certain broad-leaf weeds. TCA is registered for use in a limited number of crops. However, Dalapon is recorded for use over 25 crops (Ashton and Crafts, 1973).

These compounds inhibit plant growth, induce leaf chlorosis and produce leaf-necrosis at higher concentrations. Toxicity may be partly due to the ability of Dalapon and TCA to precipitate or effect conformational changes in protein (Redemann and Hamaker, 1957) and also interfere with lipid metabolism of leaves and a reduction in the uptake of phosphate ion into corn-seedling roots (Ingle and Rogers, 1961). They also stated that Dalapon interferes with utilisation of metabolic energy and does not affect either rates of respiration or photosynthesis. It is difficult to relate the herbicidal activity to any specific biochemical reaction(s).

Dalapon mainly interferes with the meristematic activity and arrests mitotic activity (Prasad and Blackman, 1964) and causes cytological abnormalities (Gooch and Erbe, 1967). Thomas and Murray (1978) have determined the responses of Cynodon
dactylon to treatment with Dalapon and deduced no relation­ship between tolerance to Dalapon and levels of ploidy.

Maleic hydrazide (MH) is an unique herbicide being introduced by Schoene and Hoffmann (1949). Currier et al. (1950) have first announced the selective inhibition and eventually killing of grasses by MH. This herbicide is somewhat uniform in action in diverse weed species (Naylor, 1950). MH appears to act like anti-auxins. This gained support from the work of Leopold and Klein (1952) who have shown that MH strongly inhibits auxin (IAA) action in the split pea-epicotyl test, but this inhibition is completely relieved by high IAA concentration, the main criterion of a competitive inhibition. In case of Avena coleoptile, no such anti-auxin action could be obtained (Johnson and Greulach, 1953). It is believed that the anti-auxin action can be due to its promotion of IAA oxidation in plant tissues (Andreae and Andreae, 1953), although no evidence has been forwarded from direct assay of reduction of native auxin level by MH in plant tissues (Kulescha, 1955; Audus and Thresch, 1956). Suda (1960) has indicated that MH specifically blocks biochemical processes catalysed by heavy metals which have bearing on auxin action. On the other hand, MH hinders the action of GA in dwarf pea and its action is released by application of GA. Thus, it can be an anti-gibbe-relic acid (Brian and Hemming, 1957). Moreover, as a non-
selective grass herbicide, MH gives promise of being the long-sought toxicant to complement 2,4-D for general weed control (Ashton and Crafts, 1973). This herbicide is absorbed by plant foliage as well as roots and transported easily by vascular channels. The site of action is usually the active areas of growth, where it hampers cell division. Toxicity is manifested in the inhibition of growth and development, induction of dormancy and reduction in flowering (Naylor and Davis, 1950; Ashton and Crafts, 1973).

Seed Germination

Seed germination is one of the most fundamental phases in the life cycle of a higher plant. Evenari (1957) has distinguished four phases of germination, viz. (i) the imbibition phase, (ii) the activation phase, (iii) phase of mitosis and (iv) radicle protrusion phase. Each of these phases is controlled by a distinct series of biochemical reactions which, in turn, can be influenced in different ways by either external factors or by application of growth regulators. One of the growth regulators is auxin.

The influence of auxin on seed germination has been studied since 1936 when Davis et al. have found that IAA and other indole derivatives inhibit germination and subsequent
growth of oat, mustard and cress seeds. The normal germination of many seeds can be affected by soaking them in low concentration of 2,4-D (Hamner et al., 1945). Mitchell and Marth (1945) have contended that the use of 2,4-D and other growth substances as herbicides has directed attention towards the inhibitory effect of these compounds on germination of seeds and growth of plants in soils contaminated with them. Allard et al. (1946) have pointed out that, in general, 2,4-D inhibits germination, decreases the growth of seedlings and causes abnormalities in seedlings of twenty-two broad-leaf and cereal species tried. Inhibitory influence on germination and persistence in active form in soil (Nutman et al., 1945) served the basis for Anderson and Ahlgren (1947) and Anderson and Wolf (1947) to discover the utility of 2,4-D as a pre-emergent weed-killer. This ushered in a new phase of weed control.

Auxins have rarely been known to have any stimulatory action on seed germination and 2,4-D and related herbicides have likewise generally been noted to be inhibitory rather than stimulatory. But there are some exceptions. These exceptions have emerged from experiments conducted with very low concentrations of the regulators. External application of regulators at optimum concentrations accelerate the translocation and accumulation of hormones of seeds which are suboptimal
to such an extent as to realise either impracticable or slow proceeding germination process to perfection. Seeds, when soaked in very low concentrations of auxin herbicides, invoke germination responses more promising than untreated seeds. The regulators probably accelerate reactions taking place during germination. Hsuch and Lou (1947) have maintained that 2,4-D accelerates germination at low concentrations. Chatterjee (1960), by way of presoaking treatment with certain concentrations, has been able to exhibit enhanced germination with IBA, IAA, Ascorbic acid, NAA, IPA and PAA. But he has failed to induce stimulatory influences of 2,4-D. The concentrations used by him in the case of 2,4-D are probably supraoptimal. Rojas-Garciduena and Kommedahl (1960) have reported an increase in germination of *Amaranthus retroflexus* when seeds had been soaked in 2,4-D amine at these concentrations and timings: 25 and 50 ppm for 1 hr, 5, 25 and 50 ppm for 5 hr, 5 and 25 ppm for 20-30 hr. With 500 ppm, stimulation is never obtained. Later, Rojas-Garciduenas et al. (1962) have again increased germination of *A. retroflexus* (from 43% to 85%) by exposing the seeds to 2,4-D solution at 1 mg/litre concentration continuously. MCPA has no significant stimulation at low concentration. Arai and colleagues (Arai et al., 1967; Chisaka et al., 1967a,b) have recorded the influence of MCPA allyl ester, 2,4-D acetonitrile and \(\alpha\)-(2,4-D) propionitrile in increasing germination of *Echinochloa crusgalli* after soaking for 2 days.
at 100-1000 mg/litre. The sodium salts of MCPA are effectless. Milyi (1972) has also succeeded in accelerating the germination of *A. retroflexus* and *Setaria lutescens* with 2,4-D. Biswas and Williams (1973) have claimed an increase in the germination of seeds of *Amaranthus retroflexus*, *Chenopodium album*, *Agropyron repens* and *Sorghum halepense* while studying the effect of 15 herbicides of common use at very low concentrations. Parker (1976) has made a valuable review of stimulatory effects of herbicides on seed germination. IAA increases germination of *Portulaca oleracea*, *Corchorus olitorius* and *Sida alba* (Rizk et al., 1978). Taylorson (1978), while examining the response of ethylene to seed germination of ten grasses and thirty-three broad-leaf weeds, has identified 9 species showing promotion of which *Portulaca oleracea*, *Chenopodium album* and several amaranths including *Amaranthus retroflexus* are most affected.

Agriculturists are more interested in the toxic influence of herbicides, since they have emphasised their use as pre-emergence (germination inhibitor) or as post-emergence herbicides (toxicant and seedling-growth retardant). Used at somewhat higher dosage, when selected as a pre-emergence herbicide the application is directed through water (De Datta *et al.*, 1971). The selectivity shown by herbicides between the broad-leaf and grass species is the most important cri-
Lee and Tee (1977) have emphasised chemical control to supplement cultural and mechanical methods of weed control. He has prescribed herbicides as pre-emergence and post-emergence weed-killers on the basis of selectivity for crops grown on peats.

The success of pre-emergence treatment depends largely on the presence of a high concentration of the herbicide in the upper 1 cm of soil. This is where most annual weed seeds germinate. Also, there must be a relatively low concentration of the herbicide in the zone where the crop seeds germinate, unless the crop seeds have unusual tolerance to the chemical. If the viable weed seeds in the surface of the soil are killed, the surface may remain weed-free long after the chemical has disappeared. This is because many weed seeds will not germinate if buried deeply in the soil (Klingman, 1961). The key use of herbicides to eradicate obnoxious weeds (grasses, broadleaf weeds and sedges) is the time of application. Many rice farmers in the Asian tropics are reluctant to use chemicals to control weeds until they see the weeds emerge in the crop fields. When it happens, it is too late to use herbicide to control the weeds. Thus, De Datta (1972) has recommended the use of 2,4-D and MCPA after transplanting when the weeds had not germinated but the rice seedlings were vigorous. Hammerton (1972) has demonstrated that 2,4-D markedly suppressed
the germination of broad-leaf weeds and Cyperus rotundus, the
effect on the latter being more pronounced with higher rates
of application. Nicholls et al. (1973) have conducted pre-
emergence and post-emergence application of seeds and seed-
lings respectively of pasture legumes and grasses in Hawaii.
Botton (1973) has obtained the germination inhibition of
Ocimum gratissimum seeds by 2,4-D. While advocating the effi-
cacy of herbicides on the retardation of germination, growth
and establishment, Purohit et al. (1978) have admitted that
even after leaching herbicides (2,4-D, Planavin, Bladex, Prefix,
etc.) they remain active to affect the said process of wheat
and gram. Bakale and Kolhe (1979) have proved that 2,4-D is
successful in the elimination of Tephrosia purpurea as pre-
emergent (germination inhibitor) as well as post-emergent
(lethal to seedling) weed-killer. Certain varieties of Sorghum
(NK 233 & NK 222) displayed sufficient tolerance to warrant
further investigation into the use of 2,4-D for pre-emergence
treatment on these cultivars (Marshall and Nel, 1979).

In brief, it can be said that the function of soil-
acting pre-emergence herbicide is to prevent or retard the
growth of weed seedlings. They or other herbicides may be
inhibitory in one or more of the phases of seedling growth:

(i) early germination, i.e. emergence of radicle;
(ii) seedling establishment at the expense of endosperm or cotyledonary reserve;

(iii) growth of seedlings after reserves are exhausted (Cartwright, 1976).

While examining the toxic effects on early seedling growth and germination, several workers (Hammer et al., 1945; Taylor, 1946; Allard et al., 1946; Blair, 1951; Behrens and Morton, 1960; Johanson and Muzik, 1961; Mohan Ram and Satsangi, 1963; Eshel and Warren, 1967; Khosla, 1969; Chang and Foy, 1971; Kratky and Warren, 1971; Wu and Kozlowski, 1972; Bakale and Dnyansagar, 1974; Mehta and Dubey, 1975; Bakale and Kolhe, 1979) followed with seeds and seedlings of various plants the inhibitory action of 2,4-D and/or related chlorophenoxy herbicides on the linear growth of shoot or root or both. In general, the elongation of radicle is more sensitive than the shoot growth. Even 0.01 parts/10^6 MCPA markedly inhibits the growth of cress roots (Yates, 1964). Khosla (1969) has explained that when seedlings of Achyranthes aspera, Cassia tora and Ruellia tuberosa are treated for 72 hr with different concentrations of 2,4-D, there is a marked inhibition of linear growth of radicle, hypocotyl and lateral roots. There is a marked swelling of radicles and hypocotyls of A. aspera and C. tora seedlings. With higher concentrations, cotyledons do not come out of the seed coat. These symptoms of toxicity
responsible for seedling mortality are also secured by the aforesaid workers while studying the toxicity of herbicides. In general, auxin herbicides are strongly detrimental to seedling establishment, dicots being more susceptible than monocots but many monocots are also sensitive, e.g. a corn-germination test is thought to be very sensitive to 2,4-D and related compounds (Cartwright, 1976).

Woodford et al. (1958) have aptly remarked that herbicides of chloro-substituted acetic acid groups (mono, di- and trichloroacetic acid; 2,2-dichloropropionic acid or Dalapon, etc.) are more toxic to monocotyledons and are in consequence used mainly for eradication of graminaceous weeds. Rod (1976), while studying the effectiveness of eleven different weedicides on germination of 13 weed species, has noticed that TCA causes hypertrophy of hypocotyl in majority cases and in Vicia hirsuta causes root hypertrophy. Chandra Singh and Rao (1976) have expressed the view that TCA at intermediate dosages do not kill germinating seeds and the seedlings develop typical formative effects including tubular leaves, inhibited growth, multiple tillering, deformed inflorescence, etc. Seed germination may be inhibited at higher dosages. MH is not a germination inhibitor normally, while it is able to inhibit seedling growth when applied to the soil (Schoen and Hoffmann, 1949).

When on a tissue, where most of the cells are in a state
of division, is penetrated by growth regulators at concentra-
tions modifying cell division, morphological irregularities
result. Fusion of primordia in very young apices is undoubted-
ly a consequence of disturbed growth correlations. Disturbed
growth in length can cause cells to increase in transverse
direction and in this way a fusion of primordia may occur.
Fasciation can arise from the fusion of several growing points
or from change in the shape of one growing point. In the first
case, they are called "connation" and in the second case "true
fasciation" (Gorter, 1965). This effect is quite common after
either soaking viable seeds or sowing them in soils impreg-
nated with auxin herbicides. At concentrations of 25 or 100
ppm 2,4,5-T maintained in direct contact with seeds and
recently germinated seedlings of Pinus resinosa cause abnormal
growth and development of seedlings. Responses of 2,4,5-T
include the inhibition of root elongation, proliferation,
expansion and collapse of parenchyma cells in stem, root and
cotyledon, formation of callus tissue, inhibition of forma-
tion of primary-needle primordia and their expansion, as
well as distortion of primary-needles and their fusion to
cotyledons (Wu and Kozlowski, 1972). Connation of cotyledons
of Eranthis hiemalis seedlings induced by 2,4-D has been
shown by Haccius and Trompeter (1960). The resulting struc-
tures show transitions from a flat to a cup-shaped organ.
GDBG and EPTC cause fusion of cotyledons of Pinus resinosa
(Sasaki et al., 1968). As already discussed, the inhibition of seedling growth is accompanied by formation of large tumour-like structures above and below or at the root-stem transition region. These tumours result from the production of a large number of lateral root initials, a simultaneous inhibition of their elongation and dedifferentiation of mature cells (Van Andel et al., 1976).

The normal orientation of plant organs is generally determined by external stimuli. The direction of orientation, as based on the direction of the stimulus, is called tropic response. Oriented growth responses induced by external stimuli like light and gravity are termed phototropism and geotropism respectively. These responses are the ultimate manifestations of actions of and interactions among the endogenous growth regulator(s). Some growth-regulating compounds and herbicides are able to disturb these normal reactions. Morphactins, naptalam and certain benzoic acids have the common property of interfering with geotropic and phototropic curvatures. The loss of geotropic responses of root has been reported by Counts (1961) following treatment of cotton seeds with amitrol and naptalam. The substituted benzoic acid (an auxin herbicide - 2,3,6-TBA) inhibits tropic curvatures in rye grass and TIBA having anti-auxin properties abolish normal geotropic responses of rape and rye grass.
roots (Jones et al., 1954). Van der Beek (1959) has proclaimed that 2,3,6-trichlorobenzoic acid or 2,6-dichlorobenzoic acid abolish the normal vertical position of shoots of oats, barley and cucumber. Other plant species respond similarly to these acids. Of the two widely studied growth regulators, 2,4-dichlorophenoxyacetic acid and 2,3,6-trichlorobenzoic acid, only the latter is capable of interference with geotropic response. The anti-tropistic activity is generally inversely correlated with auxin activity (Van der Beek, 1959; Schrank, 1960), although 2,3,6-TEA is a strong auxin (Bently, 1950) and a relatively potent inhibitor of tropic curvature. Brumfield (1955) has admitted that unlike auxin-IAA (this inhibited growth only), 2,4,6-trichlorophenoxyacetic acid greatly modifies geotropic response along with the growth inhibition in Phleum pratense. Even pretreatment with 2,4,6-TCPA inhibits curvatures induced by ultra-violet light. It has been theorised that high concentration of growth regulator 2,4-D has the capacity to change the geotropic behaviour of rice coleoptiles (Das and Roy, 1959). At 10 and 100 ppm, coleoptiles behave like roots. The growth of roots is checked at these concentrations. Agaronyman and Darbinyan (1976) have registered the influence of Dalapon on the geotropic response of common reed. Dalapon also disrupts the negative geotropism of the shoot.
Morphogenic Response

Growth and development of a plant are functions of genes and are thus the characteristics for a species. The normal orientation of a plant is determined by the shape, size and physical traits of its organs and their relative attachment in terms of function and location. Normal differentiation is, therefore, liable to be modified by external factors.

Development results from division, extension and differentiation of cells. In a very young embryo, cell division occurs almost in all parts; in later stages of development, this is restricted to the meristematic tissue only. In the various meristems, the division proceeds according to a pattern with a striking regularity in space and time. It is this regularity which is responsible for the characteristic development of an organ.

Many herbicides affect these tissues and alter the normal appearance of the plant consequently (Kiermayer, 1964). Of course, so far morphogenesis is concerned, the type of response depends on the species of the plant, the stage of development, the tissues involved, the kind of herbicide used and on environmental conditions (Van Andel et al., 1976). The differential susceptibility of various organs and tissues to a herbicide results in serious disturbance of the correla-
tions among them. In this connection, special reference should also be made to the response of permanent tissues to herbicides. There are several instances where such tissues are induced to become meristematic by a phenomenon called dedifferentiation. Etiolated soybean hypocotyls, when sprinkled with 2,4-D or IAA, the activity of apical meristem ceases and cells in sub-apical zone undergoes isodiametric elongation and dedifferentiation (Mikityuk et al., 1977).

Beal (1944) has coined the term "telemorphic effect" for the morphogenic responses induced or incited at considerable distance from the point of application of the herbicide. These telemorphic effects are reflected in fasciation of the shoot above and below the ground, leaves, flowers, inflorescence, fruits and roots. Either one or more organs may simultaneously fasciate (White, 1948; Gorter, 1965). These various parts of a plant exert influence on the development of each other. The most spectacular example of this "correlation" phenomenon is the dominating influence of apical meristem on subadjacent buds. This is called 'apical dominance', as a result of which the apical bud, when in active form, suppresses the growth of subadjacent axillary buds. If the apical bud is removed mechanically or killed or metabolically retarded, axillary buds begin to grow rapidly thereby alter the normal orientation. Wardlaw (1953), while describing the effects of
2,3,5-tri-iodobenzoic acid on tomato and 2,3,6-trichlorobenzoic acid on beans, has come across unusual configuration and organisation. Wayne et al. (1953) have also obtained stimulation in the production of vegetative laterals. Formation of axillary branches is accelerated by 2,4-D application on jute plants (Mitra and Singh, 1972). Growth regulators, viz. IAA, 2,4-D and MH, when sprayed singly on the 30-day old plants of *Arachis hypogea*, *Carthamus tinctorius* and *Linum usitatissimum* increase branching significantly (Singh and Singh, 1975). MH is a unique herbicide, having striking effect on morphogenesis which is uniform from species to species unlike hormone-like herbicides. The loss of apical dominance by MH is followed by vigorous branching as shown by Naylor and Davis (1950) in several plants including cotton and by Moore (1950) in many species. MH also inhibits terminal growing point of sweet clover which results in a release of axillary buds leading to brooming effect (Foote and Himmelman, 1971).

Roots of both monocot and dicots are susceptible to auxin herbicides, usually at lower concentrations than are required to induce abnormalities in shoots (Cartwright, 1976). Inhibition of elongation and stunting of primary root by auxin herbicides (Hanson and Slife, 1961) are usually associated with the production of large number of lateral primordia which may develop into lateral roots (Beal, 1944; Taylor, 1946; Bond,
1948; Wilde, 1951; Kiermayer, 1964; Callahan and Engel, 1965; Whitworth and Muzik, 1967). The application of 2,3,6-trichlorobenzoic acid on beans has led to increased root formation (Wardlaw, 1953). This lateral root formation may be extremely prolific when they burst through the root, disrupting it completely. These roots are so densely emergent that they almost form a single sheet. Watson (1950) have cited cases where tumour formation has resulted from an increase in the number of lateral initials and simultaneous inhibition of their elongation within the structural limits of root. These structural abnormalities are coupled with functional impairment (Hanson and Slife, 1961). Increased root formation with restricted elongation or premature death of root tip may be due to a decrease of or loss in apical dominance as a result of growth inhibition of the root tip. This is also a correlation phenomenon (Van Andel et al., 1976) influenced directly by herbicides.

If a plant loses its whole root system, the base of the stem is capable of regenerating new roots. The regeneration process is strongly promoted by auxins. As a result, they are extensively used in the practice of rooting of cuttings since very early times (Harrison, 1937; Holz, 1957). Zimmerman and Hitchcock (1933), Kender et al. (1969) and Krishnamoorthy (1970) have got adventitious root formation from stem as a response
to treatment with ethylene. Wample and Reid (1979) have linked the role of ethylene and auxins to the formation of adventitious root formation in *Helianthus annuus*.

The dedifferentiation phenomenon in stem tissue is also manifested in tumour formation and wart-like protrusions on the free surface (Zonderwijk, 1959; Mitra and Singh, 1972). These proliferations are composed essentially of root-forming tissue but in extreme cases these tissues are so disorganised that fasciation is the usual consequence of development (Murray and Whiting, 1949). The formation of many still invisible root initials may cause a certain degree of brittleness of the stem (Rodgers, 1952).

One of the most severe effects of auxin herbicide is that they disturb development of leaves to such an extent that a variety of abnormal or even fantastic shapes come into existence. Zimmerman and Hitchcock (1942) have adduced the formative effects on leaves due to several phenoxy compounds on tobacco, tomato and other plants. The effect of herbicides may be apparent only in leaves whose primordia exist at the time of application, as in *Phaseolus vulgaris* (Watson, 1948; Eames, 1949a). As shown by Gifford (1953), Mcllrath and Ergle (1953) and Mitra and Singh (1972), leaf primordia laid down after the time of treatment can also be affected. Gifford (1953) has described the changes occurring in the development of cotton
leaves following the administration of 2,4-D. Porter et al. (1959) have advanced the concept that 2,4-D, 2,4,5-T and 2,4,5-TP do not cause appreciable malformation to the mature leaves. Both 2,4-D and 2,4,5-T cause leaf malformation, while 2,4,5-TP do not. With 2,4,5-T, the amount required is higher than the amount of 2,4-D to realise abnormality. Gorter and Vander Zweep (1964), Ashton and Crafts (1973) and Van Andel et al. (1976) have sought morphogenetic changes in plants under the impact of auxin herbicides. Besides root and stem, they have depicted abnormalities of leaves — tubular and fasciated leaves by phenoxy herbicides, Dalapon, TCA, etc. Auxin herbicides grossly modify the pattern of division, expansion and differentiation of cells of leaf primordia and expanding leaves (Kiermayer, 1964; Cartwright, 1976). Leaves emerging, following the treatment, develop excessive vascular tissue, very compact mesophyll, less chlorophyll and fasciation (Hanson and Slife, 1961). The vulnerability of leaves to herbicides depends on the stage of development. Mature leaves show no formative effect, leaves in differentiating stages may be seriously deformed and leaves just emerging from shoot apex may be hardly recognisable. With a limited dosage, a plant may recover and again produce normal leaves (Sasaki and Kozlowski, 1967). In already expanded leaves, the effects of auxins are less conspicuous but some cells in the mesophyll may be stimu-
lated (Bradley et al., 1968). Mitra and Singh (1972) observed that in the two species of jute at the seedling stage (4-6 leaf stage) 5 ppm of the chemical is sufficient to cause the leaves to be very seriously malformed. Martin and Fletcher (1972) have investigated that with 2,3,6-TBA (10-50 μg/plant) and 2,4-D and MCOPA (at 1-5 μg/plant), after recovery from suppressed growth, the newly-formed leaves are abnormal in lettuce plants. Leaf shape is sometimes drastically changed; leaves of lettuce plants treated with MCOPA at an early stage are tongue-shaped or slipper-shaped (Arlt and Feyerabend, 1973).

The most typical phenomena produced is the fusion of leaves. The development of a leaf into cup-like or tube-like organ is very common. Haccius and Schneider (1958) have justified three fundamental types of funnel or cup-like leaves formed by marginal fusion, viz.

(i) Ascophylly: where margins of one leaf have fused with petioles developed to a certain degree;
(ii) Coleophylly: where stem is encircled entirely by leaf at an early stage;
(iii) Gamophylly: fusion of more than one leaf.

Cup formation in lettuce leaves by CDBC (Zink and Agamalian, 1965), 2,4-D and MCOPA (Martin and Fletcher, 1972) and MCOPA (Arlt and Feyerabend, 1973) have been produced. "Connation", resulting from fusion of primordia of two or
more organs at an early stage of development, has been a common effect of auxin herbicides. These herbicides and carbamate (Van Andel et al., 1976) stimulate growth in the transverse direction, together with stunting of longitudinal growth. MGPA and 2,4-D treated carrots have revealed a fantastic fusion of petioles to form a tubular stalk, carrying a tuft of leaves (Way, 1961).

Incomplete unrolling may cause changes in leaf shape. Such effects are to be seen in wheat or Beta by TCA and Dalapon (Arlt and Feyerabend, 1973). Narrowing of leaves by MH was figured by Moore (1950) in several plants. Naylor and Davis (1950) have received by MH the malformation of cotton leaves which are at the primordial stage. They are abnormal in size, shape and organisation.

Growth of mesophyll and vein is to some extent controlled by different mechanisms (Engelke et al., 1973). Obviously, mesophyll and vein development are to be affected to a different extent by herbicides. This results to a more drastic change in shape. Reduction of the amount of mesophyll by auxin leads to formation of narrow or even strap-shaped leaves as known in cotton by McIlrath (1955). In extreme cases, mesophyll may be completely absent. Fletcher (1968) has proved that picloram reduces the lamina of tomato leaves.
If the vein growth is more retarded than mesophyll, the latter projects between the veins and gives the leaf a 'crisped' appearance. Various crisping of leaves have been exemplified by Watson (1948) and Gifford (1953). This symptom of toxicity reminds one of the leaves of virus-diseased plants. It is not only caused by auxin-herbicides but also herbicides like Chlorpropham, Dalapon, TCA and diallate. Such crisping has been detected by Arlt and Feyerabend (1973). Besides a general inhibition of vein-growth, abnormal venation may also arise as a consequence of herbicidal treatment.

In many plants, the passage from vegetative to the reproductive phase requires a very special environment. The optimum conditions may include temperature, day or night lengths (i.e. photoinductive cycle) for specific duration. These factors induce a change in hormone make-up of the plant so as to cause a transition of vegetative bud to reproductive buds by certain histochemical changes. Treatment of seeds or plants with herbicides have shown to affect flowering either by induction (in pineapple by 2,4-D) or by retardation (in Phaseolus vulgaris by picloram). The effect of 2,4-D on flowering depends on the time of application. The herbicide does not intervene with flowering, if applied at an early stage and if the vegetative phase is long (Mitra and Singh, 1972). Reduction in the number of flowers or inflorescence
or the malformation of flowers by herbicides is not uncommon. White (1950) has given evidence of a delaying action on blossoming of vigorous Bristol-Black raspberries by MH. Naylor and Davis (1950), while investigating several effects of MH on plants, have stressed its striking effect on flowering. Klein and Leopold (1953) have declared that MH inhibits completely the formation of flower primordia in Wintex barley at concentrations as low as $4 \times 10^{-5}$ M. Photoperiodic induction of Biloxi soybean, a short day plant, is inhibited somewhat by MH but the treatment do not completely suppress the floral initiation. Foliar application of MH on chrysanthemum and peppermint inhibit flowering in terminal buds but not in laterals. They have brought forth the evidence that MH inhibits the production of flower primordia primarily through its inhibitory effect on growth, rather than by any specific action against photoperiodic mechanism.

Sharman (1978) has pictured 2,4-D induced abnormalities of morphogenesis in *Triticum aestivum*. Various abnormalities are reflected in branched heads, whorls of spikelets, superimposed pairs of spikelets ('banana twin' spikelets) and paired spikelets borne at same level (Yoked spikelets). Often more than one kind of abnormality is evident in a single head. These disturbances are temporary. Recovery to normal conditions ultimately results.
Interests in the herbicidal activity of phenoxyacetic acids and their physiological similarities with natural growth regulators envisage that a study of the responses produced may throw some light on the manner in which these compounds affect various plant tissues (Swanson, 1946). Again, their effect on the structure and function of conducting tissue is of much importance because the plant life is dependent upon normal activity of the vascular system (Barnes, 1950a).

The effect of auxin on cell division is stimulative. Although young meristematic cells are particularly reactive, cells that are mature and normally not capable of further division may be further induced to divide by auxin. Stimulation of cells leading to excessive formation of root initials by auxin has been first adjudicated by Kraus et al. (1930). In very low dosages, 2,4-D produces histological responses very similar to those induced by auxins (Tukey et al., 1945; Swanson, 1946) as seen in the activation of endodermal, phloem, ray and cambial cells. Unlike 2,4-D under the influence of IAA, the cambium is stimulated giving rise to derivatives recognisable as secondary xylem and phloem tissues.

Reports of histological changes (Beal, 1945; Tukey
et al., 1945; Swanson, 1946; Murray and Whiting, 1947, 1949; Eames, 1949b, 1950a; Rodgers, 1952; Sun, 1955; Rojas-Garciduenas and Kommedahl, 1958; Muni, 1959; Kiermayer, 1964; Gorter and vander Zweep, 1964; Cardenas et al., 1968; Rubin and Gritsaenko, 1968; Kiepal, 1970; Srivastava and Sharma, 1971; Mitra and Singh, 1972; Dnyansagar and Bakale, 1973, 1976; Cartwright, 1976; Bakale and Dnyansagar, 1977; Bakale and Hadke, 1978; Bakale and Kolhe, 1978, 1979; Deshmukh and Bakale, 1979) induced by auxin-herbicides which accomplish death at higher concentrations in susceptible species depict a promotion of cambial activity and secondary meristematic activity in vascular cylinder resulting in proliferative growth. This anomalous growth does not only result from vascular cambium but from dedifferentiation and rapid proliferation of cells in the phloem, endodermis and pericycle and rapid division accompanied by almost no or delayed differentiation of derived cells. Enlargement and disorganisation of cortical cells by ever-increasing centrifugal pressure and oriented by proliferative growth ruptures the epidermis. The tearing of epidermis creates an easy entrance for pathogens. This is an indirect or secondary effect which may cause death (Tukey et al., 1945; Gorter and vander Zweep, 1964; Ashton and Crafts, 1973). All the investigators in this connection have come to conclude that the proliferations in the stem are composed of essentially root-forming tissues, but these are
so disorganised that fasciated development has to result. The degree of proliferations have been shown to vary from slight stimulation resulting in the production of numerous individual roots to extreme fasciation where masses of tissues were hardly recognisable as functional elements of the root.

Roots of both dicot and monocot are susceptible to phenoxy herbicides, usually at concentrations lower than required to induce abnormalities in shoots. The inhibition of division and elongation of root-tip is accompanied by the stimulation of cell proliferation in the pericycle of sub-apical region, forming large number of greatly modified root primordia (Beal, 1944; Bond, 1948; Kiermayer, 1964; Wilde, 1951; Whitworth and Muzik, 1967).

Auxin herbicides also modify the pattern of division, expansion and differentiation of cells in leaf primordia and expanding leaves and cause vascular modification. The tissue from which mesophyll and palisade cells are derived is one of the prime victims of these herbicides. This tissue undergoes precocious and continuous division without normal differentiation. As a result, the reduced mesophyll is composed of closely packed cells forming a replacement tissue (Watson 1948; Eames, 1949a).

This cell-division activity of phenoxy herbicide is also common to benzoic acids and picloram; it is essentially
Crushing of phloem and eventually xylem and complete blocking of conducting tissue might serve as one of the most prominent contributors to death by disrupting the translocation of assimilatory materials (Tukey et al., 1945; Eames, 1950a; Muni, 1959; Kiermayer, 1964; Cardenas et al., 1968).

While studying the effect of herbicides on seedling anatomy of *Xanthium strumarium*, Dnyansagar and Bakale (1976) have noted that 1000 ppm MH in the hypocotyl causes disintegration of vascular elements and pith cells and extensive destruction of cortex and pith cells of radicle. It produces distortion in vascular elements and formation of protuberances from the abaxial surface of cotyledons. Bakale and Dnyansagar (1977) in the same species have found that MH do not produce any anatomical abnormality in the stem and leaf of the plant, whereas in the roots there is the disintegration of cortical and pith cells at many places.

Biochemical Response

Reduction in chlorophyll content of leaves is a very conspicuous effect of phenoxy herbicides as observed by Tukey et al. (1945) in bindweed; Behl et al. (1973) in *Eichhornia crassipes*; Nadakavukaren and McCracken (1977) in radish and
Mathur and Gorkhali (1978) in Galinsoga parviflora. Even after leaching herbicides from soil, a reduction in chlorophyll has been experienced with Prefix, 2,4-D, Bladex and Planvin by Purohit et al. (1977).

Toxic Effect

A striking and prompt response of shoots of many plants to auxin-type of herbicides including picloram is epinasty - a phenomenon of downward curvature of stems and leaves due to faster growth on the adaxial side of the organ (Kiermayer, 1964; Fisher et al., 1968; Ashton and Crafts, 1973; van Andel et al., 1976; Cartwright, 1976). Also, substituted benzoic acids (Minarik et al., 1951) and 2,6-substituted phenols are potent promoters of epinasty (Wain and Harper, 1967).

Epinasty is caused by tremendous enlargement of cells or cell complexes (Kiermayer, 1964). Morgan and Hall (1962) as well as Abeles (1968) have demonstrated the stimulation of ethylene production by 2,4-D. Possibly by affecting auxin distribution in that organ (Pratt and Goeschl, 1969), an increase in ethylene may result in epinasty (Kawase, 1974). Since bending is immediately visible within a few hours of auxin treatment, Cartwright (1976) has suggested that cell expansion and not cell division is involved. These curvatures may be temporary, but frequently they become fixed by anatomical
changes. Hypoplastic curvatures are also not uncommon (van Andel et al., 1976).

Epinasty is accompanied by other symptoms of toxicity like chlorosis, swelling, stem-splitting and reduction in plant growth leading to death following the treatment with auxin herbicides. These have been realised by Beal (1944) in *Lathyrus odoratus*; Hamner and Tukey (1946) in several shrubs, vines and trees; Rodgers (1952) in four strains of corn; Cardenas et al. (1968) in *Xanthium strumarium*; Saha (1972) in several dicot species; Martin and Fletcher (1972) in lettuce; Mitra and Singh (1972) in two species of *Corchorus*; Bakale and Hadke (1978) in *Psoralea corylifolia*; Bakale and Kolhe (1978) in *Solanum xanthocarpum*.

The study of the action of herbicides on reproductive organs of plants has received relatively less attention. This is not surprising, because the eradication of weeds has been emphasised either before emergence (De Datta, 1972) or at an early and active stage of growth before flowering (Hamner and Tukey, 1945; Weaver et al., 1946; Kumar and Solomon, 1952; Wayne et al., 1953; Botton, 1973; Linck, 1976). Reproductive structures and yield remain unaffected if the herbicides are applied at an early stage of growth (Rodgers, 1952; Mitra and Singh, 1972). An early paper reporting the
deleterious effect on reproductive structure is that of Tukey et al. (1945) in which bindweed has been shown to produce plasmolysed and disorganised pollen and stop flowering on account of treatment with 2,4-D. If fruit set is used for the evaluation of responses, early flowering stage is the readily affected period (Weaver et al., 1946; Johanson and Muzik, 1961). Friesen and Olsen (1953) have found two critical periods of injury following the application of 2,4-D on barley. The first is at the seedling stage and the second at the advanced boot stage just before spike emergence. Sterility is induced at the time when anthers and stigma are differentiating. But 2,4-D produces sterile pollen-grains with vacuolisation, plasma coagulation and emptiness (Rehm, 1952). Germination of seeds harvested from cotton plants damaged by 2,4-D is greatly affected (McIlrath et al., 1951). Verma and Bhardwaj (1959) have studied the effect of spraying 2,4-D on Carthamus oxyacantha when they are in early flowering stage. The production and development of seeds is almost immediately and completely arrested. The viability tests of seeds showed that not only the number and size of the seeds are greatly affected but also the viability of fully developed large size seeds is greatly impaired, being only 12.5% from treated as against 65.8% from untreated plants. Maun and Cavers (1969), using 2,4-D on Rumex crispus, have noted that the treatment 12 days before
anthesis prevented the development of viable seeds. If treated at anthesis, only 2% of seeds have minute embryos and none are viable. At seven days after flowering, 91% of the seeds have embryos but only 5-15% are viable. But 34 days after flowering, 2,4-D has no effect on the number of seeds or viability. The lethal effects of 2,4-D vapours to pollen-grains of brinjal have been investigated by Dubey and Mall (1972b); here loss of viability of pollen-grains is remarkable. Pollen germinability is reduced by Fenoprop, Acrolein and PCP (Dharurkar, 1974). Pollen fertility is slightly impaired by the treatment at tillering stage of barley by 2,4-D and germination energy is reduced especially in deformed ears but germination is unaffected (Khokholova, 1977). Khare (1978) has obtained reduction in pollen viability in pea with Amino-triazole and 2,4,5-T at concentration of 100-1000 ppm. Fawcett and Slife (1978) have observed that 2,4-D, when applied shortly before flowering at rates of 0.6 and 1.1 kg/ha, has reduced the seed production of Chenopodium album by 99% and 99%, Amaranthus retroflexus by 77% and 84% and Datura stramonium by 64% and 100% respectively. Dalapon at rates of 2.2 and 4.5 kg/ha has reduced the seed production of Setaria faberi by 100% and 100% and Datura stramonium by 100% and 91% respectively.

The halogen substituted aliphatic acids (Dalapon, TCA, etc.) inhibit plant growth, induce leaf chlorosis and influ-
ence formative effect especially at the shoot apex. Rapid
necrosis of leaves and contact injury may result from TCA
or high concentrations of Dalapon. Such injury will inhibit
their systemic transport by impairment of the symplastic
translocation system. Mayer (1957) has reported that TCA
inhibited the growth of both shoots and roots at higher con-
centration. Increased tillering with inhibited growth in
grasses is also a common response (Hilton et al., 1959;
Wilkinson, 1962). Dalapon is found to be inhibitory plant
growth in maize (Funderburk and Davis, 1960). Five times
more Dalapon is required for 50% inhibition of maize roots
compared to cucumber roots (Ingle and Rogers, 1961). Prasad
and Blackman (1964) have recorded that Dalapon interferes
with the meristematic activity and causes an arrest of mitosis
at prophase. Stem injury of Elodea caused by TCA is mani-
ested with damage to cells of epidermis, cortex and vascular
tissues and size of nuclei in meristematic cells is reduced
by 40% (Gooch and Erbe, 1967). Grasses are killed by Dalapon
in crops like pulse, brinjal, cabbage, mustard, radish and
potato which are resistant to it (Saha, 1972). The morpho-
logical symptoms caused by Dalapon in Cynodon dactylon begin
to appear 3-4 days after treatment and intensified with time.
The toxic symptoms are characterised by stunting effect
probably due to the malformation of terminal and axillary
buds. At higher concentrations, some plants die being seve-
rely scorched (Thomas and Murray, 1978).

MH has been known to produce striking effect on plant growth (Schoene and Hoffmann, 1949; Naylor and Davis, 1950; Currier et al., 1950; White, 1950; Moore, 1950; Klein and Leopold, 1953; Foote and Himmelman, 1971; Saha, 1972). Retardation of growth accompanied by burning or chlorosis of leaves and simultaneous accumulation of anthocyanin and suppression of flowering have been observed by most of the aforesaid authors. Naylor and Davis (1950) have regarded MH as responsible for the suppression of flowering and registered that 0.025% MH causes sterilisation of staminate inflorescence in maize when the plants were treated at the time of microsporogenesis.

Stimulatory Effect

Reports of stimulation of plant growth from sublethal doses of herbicides have appeared in literature. Increases in seed yield and in plant growth have occurred from the application of herbicides. The hypothesis that all poisons are stimulatory in small quantities was first introduced by Schulz (1888) and this was modified to become Arndt-Schulz rule (Luckey, 1959). Southam and Ehrlich (1943) have proposed the term "hormesis" to define a stimulatory concentration of any toxic substance on any organism. Another term
"hormoligosis" (from Greek 'hormo' meaning to excite and 'oligo' meaning small amounts) was proposed by Luckey (1959) to describe the phenomenon of stimulation of an organism under various conditions by minute quantities of stressing agent. Most substances which inhibit a biological system has the ability to stimulate that process at sufficiently low concentrations (Thimann, 1956). Webb (1966) has concluded that it is quite likely that enzyme inhibitors can stimulate metabolism. Thus, it is tempting to speculate that hormosis is a phenomenon common to all biological systems and that the herbicides are no exception in the improvement of plant growth. Two major solutions have eluded biologists. Firstly, they have not explained the general or individual mode of action for these very low doses of chemicals. Secondly, they have not been fully able to utilise these stimulatory effects (Ries, 1976).

A large number of reports have been received of increased growth and yield of crops attributed to herbicides. Taylor (1946) has been amongst the first to register an increase in soybean plant dry weight from low rates of 2,4-D in nutrient solution. Yield of sugarbeet (Ririe et al., 1952), potato (Payne et al., 1952), buckwheat (Wort, 1953) and cotton (Arle, 1954) have been increased by 2,4-D. Wort (1956, 1957a,b and c, 1962, 1966) has reported that foliar applica-
tion of 2,4-D and micronutrients increase the early growth and ultimate yields of several crop plants including beans, sugar beets, corn and potatoes. Wedding et al. (1956) have obtained a 35% increase in the yield of shelled green beans with an application of 10-20 parts/10^6 of 2,4-D applied as the ester form two weeks after emergence. Furthermore, the application of 5-20 parts/10^6 applied in different years and locations have resulted in increase up to 70% in some cases. Bringhurst et al. (1956) have shown that seasonal average increase of 12-18% in fruit yields have resulted from application of equal proportion of NOA (p-naphthoxyacetic acid) and PCPA (p-chlorophenoxy acetic acid) in concentrations of 50 ppm and 100 ppm on flowers and foliage of black berries. Leonard (1958) has recorded that aqueous solution of triethylamine salt of 2,4-D (150 ppm) slightly increases the fresh weight of bean plants 14 days after the application to seedlings. Wort (1959) has obtained increased yield of green beans as well as potatoes with foliar application of dusts and sprays with subtoxic levels of 2,4-D alone or in combination with iron. Later, Wort (1962) has got an improvement in the quality and yield of potatoes. Miller et al. (1962a) have indicated that very low concentrations increase the growth of young bean plants. Growth stimulation is further increased or preserved by the presence of either iron, an iron chelate or a chelating agent. Miller et al. (1962b) have
recommended an increased growth and yield of mature field beans with 0.5-1 parts/10^6 2,4-D, both with and without FeSO_4. This herbicide increases the size of beans, number of beans per pod and seed output. In a three-year study, Huffaker et al. (1967) have increased both yield and protein content of *Triticum aestivum* with a dust application of 13g/ha of 2,4-D mixed with micronutrients. A green-house investigation by Wiedman and Appleby (1972) have revealed the capacity of herbicides for increasing plant growth. Sixteen herbicides of different families have been utilised, out of which 11 can increase oat seedling shoot and root growth at relatively lower doses (1/100 or 1/1000 of the minimum inhibitory doses). Ries (1976) has made a valuable review of the subtoxic effect of herbicides on plants which provide information of biochemical basis for the stimulatory effect of subtoxic levels of herbicide. Fawcett and Slife (1978) have secured an increased seed production to 307 and 381% of the control by 0.6 and 1.1 kg/ha of 2,4-D respectively in *Setaria faberi*.

**Persistence Effect**

Information appears to be conflicting on the persistence of 2,4-D stimulus within the plant for an extended period of time after the application. Tullis and Davis (1950) have noted in *Stillingia sebifera* symptoms of 2,4-D injury
to reappear one year after the application, although they failed to establish the same in *Melia azadirachta*. Many instances are on record of orchards and vineyards treated during one season with formulation of 2,4-D for either weed control or prevention of premature abscission of fruits, to develop anomalous leaves, flowers and fruits in the following season (Tukey, 1950).

Pridham (1947) has established that injuries in plumular leaves appear when seedlings are grown from seeds of bean plants treated while pods are maturing. The work of Dunlap (1948) has indicated that the response of cotton plant persists for a considerable period of time under certain circumstances as shown by the fact that seeds taken from bolls, formed several weeks after treatment with 2,4-D produce malformed seedlings. Brown *et al.* (1948), on the other hand, have found no evidence of carryover of 2,4-D effect in seeds collected from cotton that exhibited heavy injury. Frequently, plants displaying delayed injury have received leaf injury during the formation of buds which did not expand until sometimes later (Watson, 1948). Application of growth regulators has a bearing on critical or sensitive periods of growth (Tukey, 1950). The effect of treatment on spring growth of many woody plants indicates that injury is done to the developing buds at the time of the treatment and is temporary. The action is evident only
later when buds develop. The dormant buds receiving treatment manifest the effects only after the resumption of growth. New tissues formed after treatment are not affected. There are conflicting reports of injury and persistence because of ignorance of the stage of bud development and in cotton where no effect has been noted, the treatment was probably given at a time when no bolls were developing (embryos being absent) or was so severe that bolls abscised (Games, 1950b). Investigations undertaken by McCrath et al. (1951) have registered that plants in later stages of development which show little or no effect from application of 2,4-D, produce seeds which are significantly affected. McCrath et al. (1951) have ascertained that 2,4-D stimulus continues over a considerable period of time in the cotton plant and is manifested by injury to seed embryos formed after application. They have opined that persistence is probably controlled in large measure by the extent of vegetative growth subsequent to the application. If the vegetative growth occurs for a period after the treatment, the stimulus may be exhausted in stimulating or inducing malformation of vegetative growth and does little or no harm to the embryo. Mitra and Singh (1972), working with two species of jute, have jumped to the conclusion that persistence of 2,4-D stimulus depends on the duration of vegetative phase of the life cycle and on the stage of the development of plants at the time of the treatment. Stimulus does not intervene with
the reproductive phase if the vegetative phase is long and if the chemical is applied at an early stage of development. Fawcett and Slife (1978) have noted that *Datura stramonium* seedlings grown out of seeds from 2,4-D treated plants show phenoxy-herbicide injury symptoms.

This mass of evidence with regard to the effects of herbicides may seem to be of little practical value but applications will follow later. Much of the evidence is fragmentary and there is need for a concerted attack on many facets of the weed-control problem. It is clear that each species reacts differently to each herbicide and there may be interactions between a variety of treatments and environmental factors.