Hormonal Regulation of Abrormal Growth in Plants

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Introduction

The varied abnormal growths in plants are characterised by extensive alterations and overgrowths due to the plant organ losing control over the growth potential of the affected area. Amongst the different types of abnormal growth, galls, hypertrophies, malformations and witches-brooms are worth mention. The various agents or conditions reported to act as incitants of abnormal growth in plants are: physical and chemical agents, genetic constitution, bacteria, viruses, fungi, insects, mites and nematodes. The abnormal growths are unique examples of complex interactions and mutual adaptation between the host and the pathogen. As a result of an attack on a plant by a pathogen the normal growth hormone balance is disturbed which brings about the change in the growth habit of the host. Within the context of this volume the text will be confined to a consideration of some of the important abnormal growths in plants and their hormonal regulation.

Genetic Tumors

A few plants seem genetically endowed with the capacity to produce diseased growth of one kind or another. No external agents are concerned with the spontaneous occurrence of such growths in plants. They include a non-parasitic burr-knot on apple trees (Swingle, 1925), spontaneous tomor in B–21 line of sweet clover, *Melilotus alba* (Littau and Black, 1952), and the tumors in a number of hybrids of *Nicotiana*, *Brassica*, *Bryophyllum*, *Lilium* and *Lycopersicum*. The literature on genetic tumors has been reviewed by Smith (1972) and Bayer (1982).

Hybrid tissues from the tumor forming *N. sauveolus* × *N. langsdorffii* and *N. debneyi-tabacum* × *N. longiflora* grow well on a minimal nutrient medium without auxin and cytokinin, while their non-tumorous parents require addition of phytohormones for continued growth (Schaeffer and

Smith, 1963; Ahuja and Hagen, 1966a, b). The growth hormone autonomy of gentic tumors indicates that tumor cells synthesize growth substances for their growth in medium lacking these (Cheng, 1972). The tumor formation in plants may be dependent on increased hormone levels (Bayer, 1977). *N. glauca* × *N. langsdorffii* hybrids possessed higher free-auxin contents, a more effective enzyme system for conversion of tryptophan to indole-3-acetic acid (IAA) and a higher free tryptophan content than either of the parents (Kehr and Smith, 1954). Bayer (1965) reported the highest levels of IAA in *N. glauca* × *N. langsdorffii* hybrids, lower in *N. glauca* and lowest in *N. langsdorffii.* Three auxin inhibitors were also present in the hybride and *N. langsdorffii* and only two in *N. glauca.* It was subsequently shown by the same author that large tumors (GL and GGLL) contained higher auxin level than the parent and the triploid (GLL) (Bayer, 1967). Amongst *N. longiflora*, *N. debneyi* and their tumor-producing hybrid, higher levels of auxin were recorded in potentially tumorous combination than the parents and the non-tumorous segregents (Bayer and Ahuja, 1968). Large amounts of scopoletin in tumor-prone hybrids (*N.glauca* × *N.langsdorffii*) as compared to their parents were reported (Tso *et al.*, 1964). These compounds altered IAA-oxidation and could thus influence the level of IAA in the tumorous tissues.

The additional biosynthetic capacities for IAA in tumorous plants and tumor cells were confirmed by many workers (Cheng, 1972; Beauchesne, 1974; Liu *et al.*, 1978a). The enzymes tryptophan aminotransferase, indoleacetaldehyde oxidase and indoleacetaldehyde reductase were described as the gene products of significance in tumorous hybrids for the conversion of tryptophan to IAA. The rate of IAA and its conjugate synthesis were also higher in tumorous hybrids (Liu *et al.*, 1978b). Bound auxins upon hydrolysis were supposed to either provide the free-auxin for tumor cells or trigger endogenous IAA synthesis accompanied by tumorous growth.

Ames (1972) showed that cytokinins markedly accelerated the rate of tumorigenesis when applied on young tumor-prone seedling of *Nicotiana* amphidiploids. Gibberellic acid (GA) application suppressed the formation of tumors and teratomas in seedlings of *N. suaveolens* × *N. langsdorffii* amphidiploids (Ames, 1971). Ames (1976) subsequently reported a reduction in tumor formation in tobacco hybrids by cyclic -3′,5′-adenosine monophosphate (cAMP) which probably mimics the action of IAA. There was also no correlation between the onset of tumor formation in tobbacco hybrids and the level of cGMP (Ames *et al.*, 1980).

The genetic tumors are formed due to the abnormal growth hormone metabolism caused by the unusual genotype whose genes interact in controlling the increased phytohormone production.

Virus Tumors

The Black's wound tumor disease, a-non-self-limiting neoplastic disease, is incited by a virus, *Aureogenus magnivena* (Black, 1935, 1949). The virus is transmitted by the leaf hoppers, *Agalliopsis constricta* and *A. novella*. A few review articles on viral etiology and molecular biology of wound tumor are available (Braun and Stonier, 1958; Black, 1972, 1979, 1982; Reddy, 1977).

Root and stem tumors, leaf distortions and shoot thickened stems are the major symptoms of infected plants. The wounds are important in initiating tumors probably by secreting substances which help the virus to stimulate the growth of the infected plant (Black, 1957). There have been only a few studies of wound tumor tissue in aseptic culture and role of plant hormones in tumor formation. Tissue from three root tumors in *Rumex acetosa* were grown in culture (Nickell 1955; Black, 1957). The virus disappears after prolonged culture of tumor tissues. The virus-free tumor tissue shows continuous growth in the absence of growth hormones and may be grafted on the healthy plants (Streissle, 1971). The application of plant hormones to the stems of plants infected with wound tumor virus (WTV) resulted in the stimulation of tumor induction (Black and Lee, 1957). The tumor formation did not occur in similarly treated control plants without WTV. The hormonally stimulated tumors showed adventitious root in sweet clover stems, whereas the normal stem tumors do not show such root formation.

Mycoplasma Caused Abnormal Growths

The plant mycoplasma diseases are very poorly understood and until 1967 were considered to be virus induce. A number of plant diseases like yellow type, big bud, proliferations and witches-broom type are caused by mycoplasma-like organisms (MLO; Dio *et al.*, 1967). The MLO probably produces specific toxins that interfere with plant hormones and cause the characteristic proliferations and disease symptoms (Moramorosch, 1976). Increased accumulation of phenolics and their involvement in establishing higher IAA levels by inhibiting IAA-oxidase in *Sesamum* phyllody, a MLO caused disease, has been reported (Purohit *et al.*, 1979b). Probably as a result of imbalance in growth hormones, the transformation of normal flowers to phylloid, stimulation of axillary buds resulting in increased leaf size and proliferation of ovules occurs.

Crown-Gall

The autonomous tumor (crown-gall), induced in many dicotyledonous plants by the specific action of the bacterium, *Agrobacterium tumefaciens*, is probably the most intensively studied plant tumor disease because of its economic importance and its usage as a model for animal cancers. With recent advances in molecular biology it has been possible to show that crown gall disease involves transfer of DNA from bacteria to plant cells which has opened up the possibility of using this DNA for transfer of desired genes into the plant cells. The whole problem of crown-gall induction has been lucidly reviewed by many workers (Braun and Stonier, 1958; Braun, 1972, 1982; Butcher, 1973; Meins, 1974, 1982; Kahl and Schell, 1982).

The crown-gall is a non-self-limiting neoplastic disease. During the induction of crown-gall, DNA sequences from the oncogenic tumor inducing (Ti) plasmids (Van Larebeke *et al.*, 1975; Watson *et al.*, 1975) are transferred to normal plant cells (Schell *et al.*, 1979). These sequences, referred to as T-DNA are incorporated, maintained (Chilton *et al.*, 1977, 1980; Willmitzer *et al.*, 1980; Yadav *et al.*, 1980) and transcribed (Drummond *et al.*, 1977; Gurley *et al.*, 1979) in the transformed plant cells. The T-DNA is essential for the maintenance of the tumorous state (Depicker *et al.*, 1978; Holsters *et al.*, 1980) and for biosynthesis (Holsters *et al.*, 1980) of a group of unusual plant metabolites called opines (Petit *et al.*, 1978; Schell *et al.*, 1979).

The physiological autonomy exhibited by tumor cell reflects an abnormal growth hormone metabolism (Braun and Stonier, 1958; Meins, 1974, 1982; Lippincot and Lippincot, 1976; Weiler and Spanier, 1981). The transformation of normal cells to tumor cells is accompanied by the capacity of the former in the absence of the inciting bacterium to grow in culture on basal medium without exogenous auxin and cytokinin (Braun, 1958). On the other hand, normal plant cells generally require these growth substances. The growth hormone auotnomy of crown-gall cells is apparently a result of the production of auxin and cytokinin by these cells (Braun, 1958; Miller, 1974; Pengelly and Meins, 1977; Nakajima *et al.*, 1979; Enist, 1980; Scott *et al.*, 1980; Weiler and Spanier, 1981; Amasino and Miller, 1982). Liu *et al.* (1982) reported the presence of *Agrobacterium* Ti plasmid IAA gene (iaaP) at 20.9 kilobases to the left of the T-DNA. The iaap gene is necessary for virulence and IAA production in crown-gall. It is further supported from the fact that *A. tumefaciens* cells themselves elaborate abnormally high amount of IAA and that the production of IAA is controlled by Ti plasmid (Liu and Kado, 1979).

Whereas the crown-gall disease is relatively well studied at the genetic level, the physiological events associated with and required for initiation and maintanance of unorganised growth of the infected tissues still remain largely obscure (Weiler and Spanier, 1981).

Fasciation and False Broomrape Diseases

The fasciation disease in a number of dicotyledonous seedlings is caused by *Corynebacterium fascians*. The shoots become more in number fleshy and misshapen (Roussaux, 1965). The infected seedlings show many notes with outgrowths of one or more lateral buds. $6-\Delta^2$ -isopentenylaminopurine (2iP) (Klämlet *et al.*, 1966; Helgeson and Leonard, 1966) and cis-zeatin (Scarbrough *et al.*, 1973) have been isolated from the culture filtrates of the bacteria. Application of 2iP, kinetin or benzyladenine to the lateral buds caused diseased symptoms (Sachs and Thimann, 1964; Douce and Roussaux, 1969). However, relatively high amounts of exogenous kinetin are required to cause abnormal growth. It seems that bacteria in some way stimulate the host cells to produce cytokinins and the amount of cytokinin produced by bacteria is of secondary importance in disease initiation.

Several investigation have suggested possible causes of false broomrape root disorder including viruses, bacteria and other organisms. Valleau (1954) first considered that *C. fascians* caused false broomrape of root, but was unable to induce the disease by infecting the plants with the bacteria. Symptoms of the disorder are white succulent outgrowths which first appear as tumor-like protuberances on the roots (Lucas, 1965). The disorder can be induced by the application of very high concentration of cytokinin (Hamilton *et al.*, 1972, Jenkins *et al.*, 1973) or by applying extracts from tobacco plants with false broomrape. On the other hand disease can be prevented by treating debudded plants with auxin.

Fungi Caused Abnormal Growths

Fungi often incite growth disturbances and the resulting symptoms show an excess or imbalance of growrh hormones. A few reviews on this problem have been written by Show (1963), Thrower (1965), Scott (1972), Sequeira (1963, 1973) and Ingram (1973).

Green-ear disease of pearl-millet

Green-ear of pearl millet (*Pennisetum typhoides*) is caused by *Sclerospora graminicola*. The flower proliferation of normal ear results into a shooty mass called green-ear (Butler, 1907). In severe infections, ear-head formation is suppressed, mother axis is expanded and stunted, developing into leafy shoots. The effect of obligate parasite on the hormonal control of host tissue growth and dual cultures for host-parasite interaction have been the main subject of study in this disease. The callus tissue was obtained from hypocotyls of *P. typhoides* on modified white's basal medium fortified with growth hormones and aseptically infected with *S. graminicola* (Tiwari and Arya, 1969). The sporulation and axenic growth of *S. graminicola* were achieved and the fungus survived two transfers in the absence of the host tissue.

The diseased tissue showed substantial synthesis of phenols and auxins. The accumulation of phenols prevented auxins from being oxidized by IAA-oxidase resulting in abnormal growth of the ear-head (Shekhawat and Arya, 1979). Increased accumulation of aromatic amino acids-phenylalanine, tryptophan and tyrosine (Shekhawat and Arya, 1980) and higher synthesis of IAA (Arya *et al.*, 1981) were found associated with the diseased ear-head.

Clubroot

Plasmodiophora brassicae causes clubroot disease of crucifers and during its vegetative part of the life cycle takes the form of multinucteale plasmodia inhibiting the cytoplasm of the cells of the host root (Ingram and Tommerup, 1972). The host cells and the adjacent cells without fungus show continued cell divison and enlargement, indicating a disturbance of the growth hormone balance (Ingram, 1973; Dekhuijzen, 1976, 1980). The clubroots do not only contain higher cytokinin

(Dekhuijzen and Overeem, 1971) but also much higher auxin levels (Raa, 1971; Butcher *et al.*, 1974) as compared to healthy turnips. In contrast to normal turnip tissue, the infected tissue could grow in the absence of an exogenous supply of both cytokinin and auxin (Ingram, 1969; Dekhuijzen and Overeem, 1971). However, the infected callus showed optium growth in the medium containing auxin. Three cytokinin-active compounds were reported in the extracts of infected callus grown on cytokinin-free medium (Reddy and Williams, 1970). Further, once the callus lost the pathogen it could no longer grow on growth-hormone-free medium suggesting that the plasmodia could not transform the host cells for growth autonomy. Recently, Rausch *et al.* (1981) reported that after inoculation with *P. brassicae*, the infected roots and hypocotyls exhibited higher nitrilase activity. This increase in nitrilase activity was correlated with higher levels of indole-3-acetonitrile (IAN) and IAA.

Rust galls

Cadar apple rust gall on cedar is induced by *Gymonsporangium* spp. The pine stem galls are formed by the infection of *Cronartium fusiforme*. The gall tissue of pine possessed nearly 10 times greater cytokinin activity than the normal stem wood (Rowan, 1970). The studies on rust galls are mainly confined to growth factor requirements of both parasite and host, independently and together. The dual cultures of *C. fusiforme* and *Pinus elliottii* (Walkinshaw *et al.*, 1965) as well as axenic cultures of the fungus (Scott, 1972) on well defined media have been established. The fungus, *C. ribicola*, which causes white pine blisters on *P. monicola* could be grown for a limited time on a defined medium which supported the growth of the tissue cultures of the pine. The growth of both healthy and white-pine-blister-rust infected tissue was more at higher concentrations of auxins in the medium (Harvey and Grasham, 1969; Harvey *et al.*, 1971). The growth of these tissues could not be influenced by kinetin alone or in conjunction with an auxin, but the combination enhanced the longevity of both the host and the parasite. GA up to 1 ppm in the medium did not affect growth of the host tissue, but inhibited the growth of the fungus.

Other abnormal symptoms

Smut galls on corn (*Zea mays*) are produced by *Ustilago maydis*. *Sempervivum tectorum* (houseleek) infected with *Endophyllum sempervivi* and *Euphorbia cyparissias* infected by *Uromyces pisi* show abnormal growths. In the latter, extreme elongation of unbranched stems, thick and short leaves which fall off prematurely, and atrophied flowers are produced. The diseased tissue growth was accompanied by an increase in IAA levels (Pilet, 1960). In the hypertrophied growths of *Capsella bursapastoris* infected by *Albugo candida* and *Peronospora parasitica*, higher IAA and IAN were reported by Kiermeyer (1958). However, contradictory results were published by Srivastava *et al.* (1962).

Insect-and Mite-Galls

Among the most interesting types of growth abnormalities found in plants are those that result from the activity of certain gall forming insects or mites. The developmental morphology, organization and functioning of insect-gall tissues has been reviewed (Mani, 1964; Braun, 1969; Rohfritsch, 1971a; Rohfritsch and Shorthouse, 1982). However, the physiology and biochemistry of insect-gall formation in plants has received attention only in the recent past (Arya *et al.*, 1975; Purohit *et al.*, 1980; Tandon and Arya, 1982).

A large number of insects and mites stimulate the tissue of the host plants to initiate galls or cecidia. These galls may by simple, involving a single organ, or compound where two or more plant organs are concerned in the production of galls. An interspecific association between the plant and the arthopod is established during gall formation. It is believed that the latter provides the stimulus for gall formation, but the identification and mode of action of stimulus is poorly understoood.

Although it is difficult to culture complex insect-galls, mary-galls, especially from woody plants, have been successfully cultured. The insect-gall tissues grew best on media supplemented with coconut milk or coconut milk, 2,4-dichlorophenoxyacetic acid (2,4-D) and α-naphthyleneacatic acid (NAA) (Hildebrandt, 1965). The *Phylloxera* gall and normal grape stem tissues grew on the basis mineral salt medium with NAA and kinetin (Pilet *et al.*, 1960; Arya *et al.*, 1962). *In vitro* cultured gall tissues are considered to have similar growth hormone requirements as do their corresponding. Normal tissue (Rohfritsch, 1971b).

In contrast to non-self-limiting tumor tissues in plants with etiological factors like bacteria, viruses and genetic constitutions, insect-and mite-incited galls are reported to be self-limiting and continued presence of insect or mite is considered essential for gall tissue growth (Braun, 1969). However, Tandon *et al.* (1976) reported that galls incited by a plant mite, *Eriophyes cernuus*, on *Zizyphus jujuba* possess both transplantable and tumefacient properties. The galls were induced aseptically on stem segments cultured on auxin and kinetin-free, modified Murashige and Skoog's medium by (i) gall callus graft, (ii) gall tissue extract and, (iii) incorporation of NAA into the medium. The gall tissues isolated from *Zizyphus* stem galls showed hyperauxinity and auxin-kinetin autotrophy and converted more auxin from DL-tryptophan incorporated in the auxin–free culture medium (Tandon and Arya, 1980a). An integral association of growth regulators with the activity of α-amylase and sugar accumulation was found in both normal and gall tissues in culture (Tandon and Arya, 1979). The gall tissues contained more total carbohydrates and reducing sugars than the normal. The former exhibited higher α-amylase activity in cultures grown on media containing 2,4–D and IAA separately. While the gall tissue showed a marked decline in IAA-oxidase and peroxidase activities, the polyphenol oxidase and o-dihydroxyphenols increased with the growth of the gall tissue (Tandon and Arya, 1982). A differential response of normal and gall tissues to different growth regulators such as NAA, IAA, 2,4-D, DL–tryptophan, GA and cycloheximide incorporation separately into MS medium, in terms of the said enzyme activities was established. A general tendency towards a decrease in polyphenol oxidase and an increase in peroxidase and IAA-oxidase activities was observed in the gall tissue. Tandon and Arya (1980, b, c) reported unregulated synthesis of auxin protectors (o–dihydroxyphenols) that are possibly responsible for hyperauxinity and growth autonomy of the *Zizyphus* gall tissue. Three auxin protectors were isolated from the gall tissues having molecular weights exceeding 200,000 daltons, approximately 10,000 daltons and 2,000 daltons. These substances prevent IAA destruction by inducing a lag period in the oxidation of IAA and thus causing hyperauxinity and abnormal growth of the gall tissue.

The roles of oxidative enzymes and phenolics in hyperauxinity and abnormal growth in many mite-incited galls such as on *Achyranthes aspera* (Shekhawat *et al.*, 1978), *Ficus mysorensis* (Bhansali *et al.*, 1978), *Prosopis cineraria* (Purohit *et al.*, 1979a), *Cordia myxa* (Ramawat *et al.*, 1979), *Cinnamomum tamala* (Tandon and Joshi, 1983), *Leea indica* (Joshi and Tandon, 1984) and *Camelia sinensis* and *Elaeocarpus lancifolius* (Joshi *et al.*, 1985) have been established.

Root-Knot Galls

As a result of infection by larvae of nematodes like *Meloidogyne* spp., root gall formation occurs in many plants. Inside the root, the larvae grow and the surrounding root tissue exhibits redifferentiation. Due to hypertrophy accompanied by repeated nuclear divisions an incorporation of neighbouring cells a large, thick-walled multinucleate syncytium develops (Bird, 1973). During infection of host cells by *Meloidogyne incognita* and other nematodes, the endogenous levels of plant hormones change. Increased auxin levels in *Meloidogyne* galls have been reported by many workers. Indole compounds and growth promoting activity in extracts of root-knot gall, but not in healthy roots, was recorded by Balasubramaniam and Rangaswami (1962). On the other hand, Viglierchio and Yu (1968) extracted a number of indole compounds from infective larvae and egg masses of *M. haple*, *M. javanica* and *M. incognita*. A methyl ester of IAA was also reported from *Ditylenchus dipsaci* (Culter and Krusberg, 1968). Varghese and Kumari (1970) suggested that the changes in the auxin-kinin ratio in roots of *Solanum melongena* infected with *M. incognita acrita* may be responsible for the acceleration of the phyisological mechanism in the disease initiation. Krupasagar and Barker (1966) detected a higher activity of cytokinin in tobacco roots infected with *M. incognita* than the uninfected ones.

Tissue culture studies have provided an idea of the role of the plant hormones in nematode and host cell associations. The nematodes increased the growth of excised tobacco-pith tissue and induce typical syncytia only when auxin and cytokinin were incorporated into the medium. The nematodes, did not supply the growth hormones (Sandstedt and Schuster, 1966a). The nematodes, IAA or 2,3,5-triiodobenzoic acid (TIBA) were applied aseptically on peeled tobacco stem segments cultured *in vitro* by Sandstedt and Schuster (1966b). *M. incognita* induced tissue proliferation in a manner analogous to that of TIBA, by causing infected tissues to retain and use endogenous auxin that would otherwise be transported way. The response of resistant variety of tomato was changed and it became susceptible by the exogenous application of benzyladenine, kinetin, zeatin or 2iP on the seedlings (Dropkin *et al.*, 1969). This change was not brought about by IAA or GA.

There is some evidence, therefore, that change in the concentration of growth regulator occurs following infection with nematodes and these in turn cause the diseased growth.

Conclusions

Most of the abnormal growths in plants show phytohormone autonomy and increased production of growth hormones, particularly auxin and cytokinin, by the diseased cells. In some cases exogenous application of growth hormones on infected plants stimulates disease induction. The molecular basis of tumorigenesis at genetic level is well documented in crown-gall tumors but the regulation of physiological events of initiation and maintenance of abnormal growth requires more attention. The transplantable and tumefacient properties and growth autonomy reported in *Zizyphus* gall tissues incited by a plant mite has given new dimensions to the problem of tumor formation in plants incited by insects and mites. The crucial research needs to be directed towards understanding the molecular mechanism of insect-and mite-incited tumor formation. Although plant abnormal growths have been a subject of study for many years, a comprehensive approach to the problem is still warranted more so with the advances in molecular biology and biochemistry that have revolutionized the modern science in the last one decade or so.

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