CONTENTS

The tomatoes (above) are the same variety and the same age as those, shown below.

The deterioration and spoilage shown by the normal variety (above) is normal. The tomatoes, shown below have been genetically modified and stay fresher for longer. They have been given a gene which blocks the normal production of **ethene**, a plant hormone which promotes ripening.

CHAPTER

32

Plant Hormones

DEFINITION

Thimann (1948) designated the plant hormones by the term '*phytohormones*' in order to distinguish them from animal hormones. He defined a phytohormone as "*an organic compound produced naturally in higher plants, controlling growth or other physiological functions at a site remote from its place of production and active in minute amounts.*"

This definition includes a variety of compounds, besides those responsible for growth curvatures in organs like *Avena* coleoptile. For example, it embraces the hormones which induce flowering, wound-healing and also those vitamins which act as growth factors.

A definition of plant hormones with still wider scope has been given by Johannes van Overbeek (1950). According to him, the plant hormones are defined as "*organic compounds which regulate plant physiological process— regardless of whether these compounds are naturally occurring and/or synthetic ; stimulating and/or inhibitory ; local activators or substances which act at a distance from the place where they are formed.*"

The migratory nature of hormones has been specifically emphasized by Meirion Thomas (1956) who stated that "all hormones are migratory correlating substances or correlators which play an essential part in the integration of plant behaviour."

Three types of plant hormones are usually recognized. These are *auxins, gibberellins* and *cytokinins*. These were

discovered in the early decades of the twentieth century, in 1930's and in 1960's respectively. Naturally, the knowledge accumulated on auxins and gibberellins is far greater than that gathered for cytokinins.

AUXINS

Definition

Kögl and Haagen-Smit (1931) introduced the term 'auxin' (*auxein*^{G} = to grow or to increase) for designating those plant hormones which are specially concerned with cell enlargement or the growth of the the shoots. An auxin may, thus, be defined as "an organic substance which promotes growth (*i.e.*, irreversible increase in growth) along the longitudinal axis when applied in low concentrations to shoots of the plants freed as far as practicable from their own inherit growth-promoting substances. Auxins may, and generally do, have other properties but this one is critical" (Thimann, 1948). This definition is nowadays widely accepted among plant physiologists.

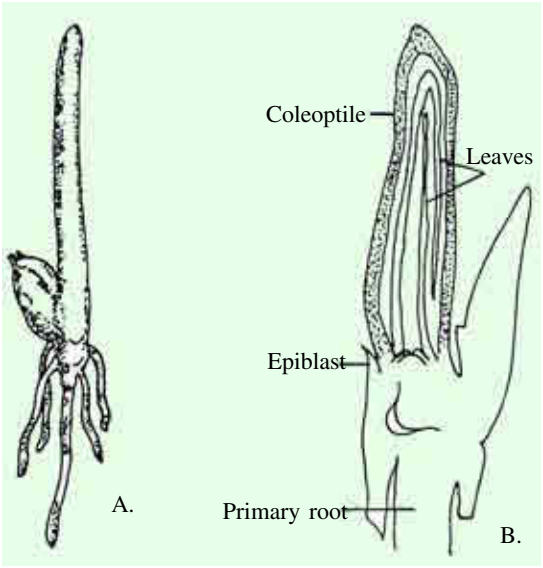


Fig. 32–1. Structure of oat coleoptile

Oat Coleoptile and the Auxins

Since long the action of auxin has been clearly demonstrated in the leaf sheath or coleoptile of oat plant (*Avena sativa*). The coleoptile (Fig. 32–1) is a tubular structure with a conical top and encloses the first-formed leaf in it. A transection of the coleoptile reveals that it consists of an epidermal layer and a few parenchyma cells with two vascular bundles running longitudinally.

The experimental utility of oat coleoptile lies in the fact that the process of cell elongation can be easily studied over here. After four days, the primary leaf breaks through the coleoptile at its tip and the organ stops growing. Thus, cell divisions cease relatively early in the development of oat coleoptile and further growth in length takes place as a result of cell elongation only. Elongation is confined to a region about 1 cm below the tip of the coleoptile. The rate of elongation is most rapid (about 1 mm per hour) when the seedling is about 3 days old. At this time, the coleoptile may measure about 3 cm in length. It is mostly at this stage that it is experimentally used for conducting growth experiments.

Hormone Concept

The presence of growth-regulating hormones in plants was first suggested by **Julius von Sachs** in 1980. He proposed that there were certain '*organ-forming substances*' in plants which were produced in the leaves and translocated downward in plant body.

Also in 1880, **Charles Darwin**, an evolutionist, studied the effect of unilateral light on plant movements. While conducting his experiments on canary grass (*Phanaris canariensis*), he found that if the coleoptile tip is provided light from one side only (*i.e.*, unilateral illumination), the tip would bend towards light. In the absence of illumination, however, no curvature could be induced (refer Fig. 32–2 for this and other subsequent experiments).

The material nature of the hormones was first conclusively demonstrated by a Dane, **Peter Boysen-Jensen** (1910). He first cut off (or decapitated) the coleoptile tip a few millimeters from the apex, then put a block of gelatin on the decapitated stump and ultimately replaced the cut tip on the gelatin block. Upon unilateral illumination, the coleoptile showed curvature towards light.

This he attributed to the fact that the 'stimulatory effect' can diffuse through gelatin and is a type of soluble material.

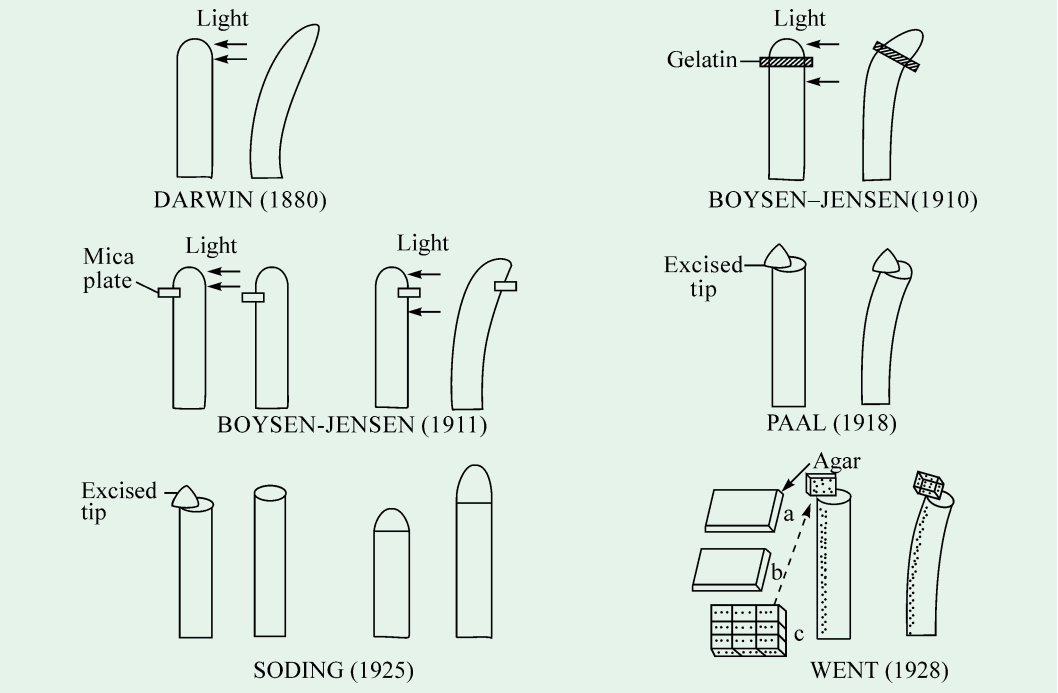


Fig. 32–2. Major experiments in the discovery of auxins

A year later (*i.e.*, in 1911) in another experiment, he made a transverse slit halfway through the coleoptile below the tip on one side and inserted a piece of mica in the slit. Upon unilateral illumination from the side opposite the slit, no phototropic curvature was observed. If, however, the operated coleoptile is illuminated on the side of the slit, the phototropic bending towards the source of light is seen. From this was inferred that the stimulus for bending passes down the dark side of the coleoptile.

In 1918, the Hungarian plant physiologist, **Arpad Paál** of the University of Budapest in an experiment decapitated coleoptile and replaced the tip eccentrically. He found that the coleoptile showed a negative curvature, *i.e.*, bent away from the side with the tip even in the dark. Paal, therefore, concluded that the material substance (called as '*correlation carrier*' by him) diffuses from the tip downwards and stimulates growth of the cells below the tip.

Soding (1925) found that decapitation of a coleoptile markedly retards the rate of cell division. But reheading of the tip on the cut stump resulted in resumption of vertical growth due to cell elongation.

The task of isolation, extraction and bioassay of these growth-promoting substances was admirably done by a Dutch botanist, **Frits W. Went**, in 1928. He worked in his father's laboratory at the University of Utrecht in the Netherlands. He placed numerous freshly-cut coleoptile tips on an agar block for some time. Later, this block was divided into small rectangular blocks (*a*, *b* and *c*). These agar blocks were, then, placed eccentrically on decapitated coleoptiles for 2 hours in dark. In all cases, the coleoptiles showed curvature towards the side opposite to the agar blocks (*i.e.*, negative curvature). Went also devised a method for the bioassay (to be described later) of these substances.

Cholodny in Russia also worked on similar lines independently and arrived at the same conclusions.

The availability of a technique of extracting the hormone opened up new vistas in this field and within a short span of about 8 years (from 1928 to 1936) extensive work was done. With the result, three different hormones (auxin *a*, auxin *b* and heteroauxin) were identified and their characteristics noted mainly on account of the efforts of Kögl and his colleagues in Holland.

Extraction of Auxins

The two forms of auxins (free and bound) appear to be in a dynamic state as there are many examples where the bound auxin is released in free state during extraction. With the result, the strictly separate measurement of free and bound auxins is often difficult. There are, however, two methods commonly employed for auxin extraction.

A. Diffusion method. It was devised by Went (1928) at Utrecht. In this method the growing tip (or other organ to be tested), under conditions of low transpiration, is severed (or cut) and is then placed on an agar block (usually of 1.5 concentration) for about an hour or so. During this period, the auxin diffuses from the cut tip into the agar block.

This method, though simple, has some major *drawbacks* and is, henceforth, not widely used.

(*a*) Excessive transpiration may prevent the accumulation of the auxin in the agar block.

(*b*) Severing the tip results in lowering the amount of auxin from the cut surface.

(*c*) The method cannot be widely adopted on account of the presence of growth inhibitors in many green plants.

B. Solvent extraction method. Here, the tissues are grinded in some organic solvents like chloroform, ether, ethyl alcohol or even water and the liquid is then filtered. The auxin is separated from the filtrate by chromatographic technique.

This method is widely employed for the extraction of auxins, *esp*., the bound auxins. But this one also suffers from certain *drawbacks*.

(*a*) Use of chloroform as a solvent causes slow accumulation of chlorine which is a toxic substance and probably an auxin inactivator too.

(*b*) Diethyl ether, if used as a solvent, brings about oxidation of the auxin in the presence of a spontaneously formed peroxide. This can, however, be avoided if the solvent is distilled with ferrous sulfate and calcium oxide before use.

(c) During auxin extraction, a new auxin may be produced which may thus contaminate the auxin to be extracted. The difficulty can be overcome by employing Gustafson's technique which involves the boiling of plant material for about a minute prior to extraction.

Bioassay of Auxins

The term *bioassay* refers to determining the amount of active substance present in the plant tissues. In the various methods employed for bioassay, the activity of the auxin is in general determined by making it available in certain concentration to a seedling (or an ovary or a root) and the degree of either acceleration or inhibition of growth is recorded.

One of the most commonly employed methods of bioassay of auxins, as devised by Went (1928), is known as **Avena curvature test**. The test involves the following steps (Fig. 32–3).

Went found that the degree of angular curvature of coleoptile tip is proportional, within limits, to the concentration of auxin present in the agar block. This fact he made the basis of his *Avena test*.

Kögl and Haagen-Smit (1931) used *Avena test* as the unit of measurement. It is termed *Avena Einheit* and is abbreviated as A.E. One A.E. is defined as the amount of auxin present in an agar block ($2 \times 2 \times 1$ cm) which produces a curvature of 10° to a decapitated *Avena* coleoptile when placed eccentrically on it for 90 minutes.

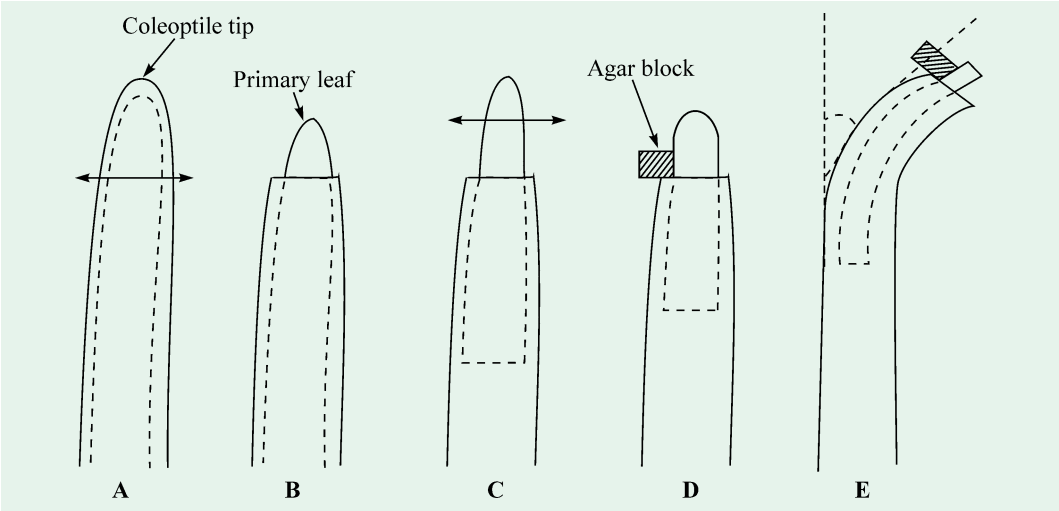


Fig. 32–3. Steps involved in Avena curature test

A. Decapitation of the coleoptile tip containing the primary leaf.
B. Pulling out of the exposed primary leaf so that its elongation may not dislocate the agar block.
C. Cutting the tip of primary leaf.
D. Affixing the agar block containing the auxin unilaterally to the cut tip.
E. Reading the angle resulting from movement of auxin into the side of coleoptile on which agar block is placed.

Biochemistry of Auxins

Application of *Avena* test to a wide variety of substances led to the discovery of auxins in human urine. **Kögl and Haagen-Smit** (1931) isolated 40 mg of auxin (which they named as *auxin a*) from as much as 33 gallons of human urine. Three years later, Kogl and his associates isolated two more compounds with auxin activity. These were named as *auxin b* and *heteroauxin* and were obtained from corn germ oil and human urine respectively. Their structure is shown in Fig. 32–4.

1 gallon = 3.78533 litres (U.S. units)
= 4.54596 litres (British units)

1. **Auxin a, auxentriolic acid.** It occurs at the meristematic apices (buds and growing leaves) both in the free state and bound to plasma proteins. It is a weak acid and is soluble in water, alcohol, ether and chloroform. It is stable in acid solutions but decomposes in alkaline soultions (*i.e.*, acid-stable and alkali-labile).

2. **Auxin b, auxenolonic acid.** It is present in corn germ oil, other vegetable oils, malt and a fungus called *Rhizopus*. It is also a weak acid and is soluble in water, alcohol, ether and chloroform. It is both acid-labile and alkali-labile. This as well as auxin *a* both are derivatives of cyclopentene.

3. **Heteroauxin, indole-3-acetic acid.** It is of universal occurrence in plants and is also synthesized by microorganisms including certain bacteria, yeasts and fungi like *Rhizopus*. It is resistant to alkalies whereas destroyed by acids and undergoes rapid decomposition on heating. Unlike the first two, it can be easily synthesized in the laboratory. Chemically, it is a monobasic acid of a relatively simple structure.

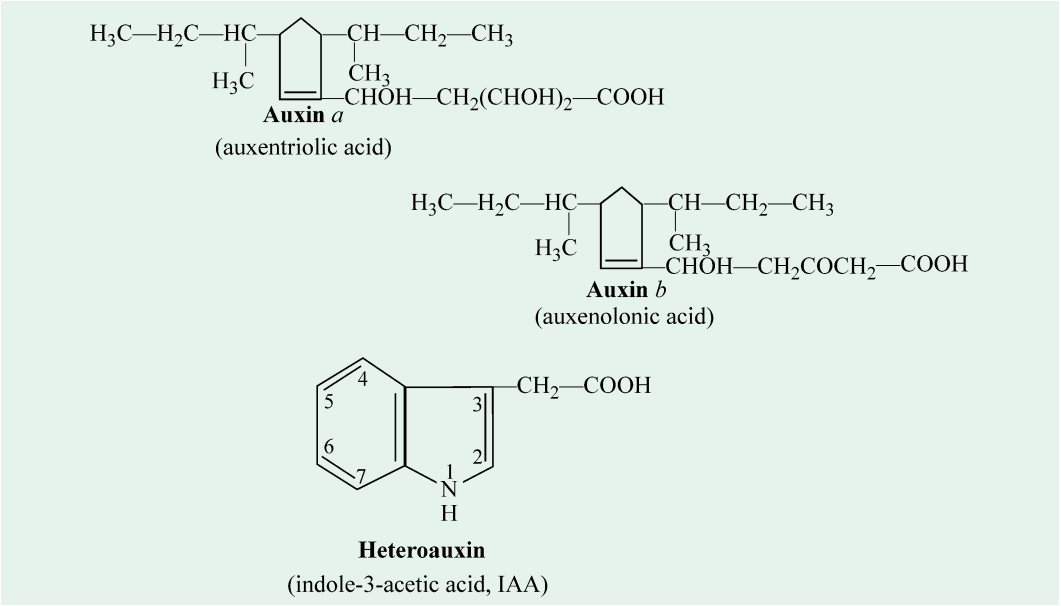


Fig. 32–4. Natural hormones

Natural Auxins

Besides the above-mentioned auxins, certain other compounds have recently been shown to occur in plants. These show similar behaviour in terms of their effect on growth, although usually less intense. The principal naturally-occurring auxins in plants that have been definitely identified, isolated, purified and their chemical structure determined are all indole derivatives, (refer Fig. 32-5), as mentioned below :

1. Indole-3-acetic acid, IAA
2. Indole-3-acetonitrile, IAN
3. Indole-3-acetaldehyde, IAc
4. Ethylindoleacetate
5. Indole-3- pyruvic acid, IPyA
6. Indole-3-ethanol, IEtOH

Controversy exists regarding whether ethylindoleacetate occurs naturally or is produced during extraction as an artifact. The presence of indole-3-ethanol is also not conclusively established.

Non-indole compounds, *e.g.*, some fatty acids may also possess auxin-like properties. But they have not yet been properly characterized.

The position of the side chain in the ring structure in indole-3-acetic acid (IAA) appears to be highly specific for activity, since 1-, 2- and 4-indole acetic acids are only very slightly active in bioassay. Substitution with halogens, like fluorine and chlorine, can result, however, in very active derivatives, *e.g.*, the 4-chloro and 6-chloro compounds. The replacement of an aromatic CH of IAA by N can give rise either to 7-aza (indole-3) acetic acid, a synthetic auxin (see Fig. 32-6 for the structure of this and other synthetic auxins) which is less active in the *Avena* test or to another synthetic auxin, indazole-3-acetic acid which has activity equal to IAA. Indole-3-acetonitrile (IAN) may be converted into either IAA or IAc, depending on whether C ≡ N is replaced by COOH or CHO, respectively. Replacement of the imino group (NH) in IAA by a S atom produces another synthetic auxin called thianaphthen-3-indoleacetic acid which is quite active in various biossay tests (Allsopp, 1965). Indole-3-propionic acid (IPA) is less active than IAA, but indole-3-butyric acid (IBA) is less active than IAA, but indole-3-butyric acid (IBA) is more active than

IPA; however, in some tests as in the case of rooting of cuttings, IBA

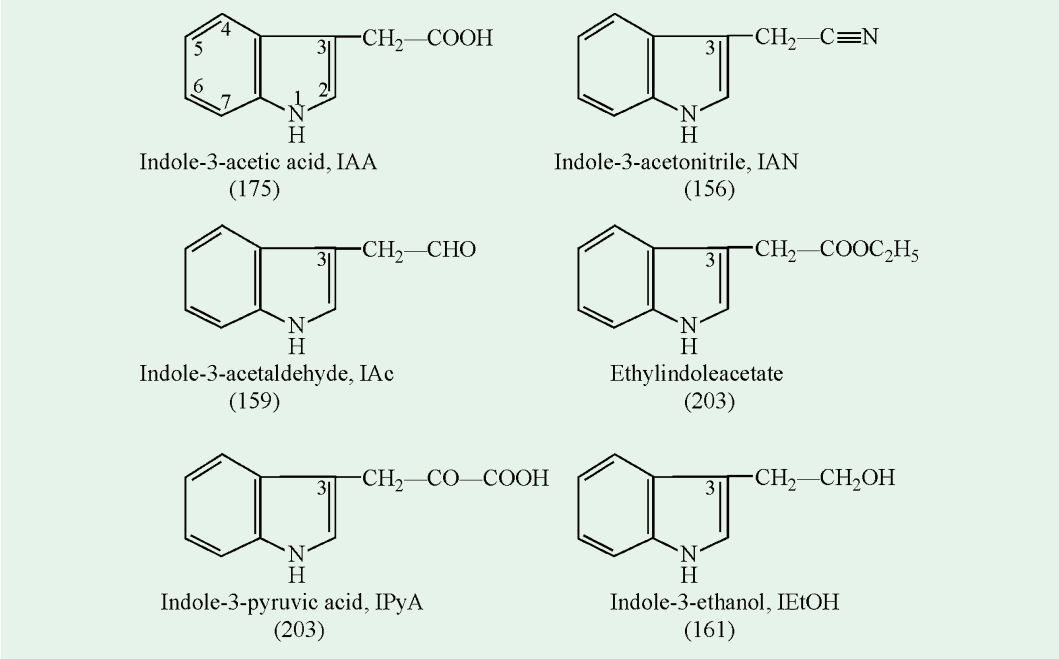


Fig. 32–5. Some natural auxins (IAA and its derivatives)
Bracketed numbers underneath each compound represent its molecule weight.

tests as in the case of rooting of cuttings, IBA is even as active as or more active than IAA. It is noteworthy that the side chains having even number of C atoms are more active than those having an odd number. It is hypothesized that the side chain is oxidized to IAA before the compound shows a biological activity.

Indoleacetonitrile (IAN), a nitrile derivative of IAA, is a neutral substance and has been obtained in crystalline form from many plant materials. It is believed that the nitrile derivative (*i.e.*, IAN) as such is inactive and has to be converted to IAA in order to be active. IAN, upon alkali hydrolysis, yields IAA. IAN promotes growth of only those plant organs (like *Avena* coleoptile, bean seeds, tomato ovary etc) which possess the enzyme *indoleacetonitrilase*, while tissues (like pea roots)which cannot convert IAN to IAA because of the absence of indoleacetonitrilase, are unreactive to IAN. However, in higher concentrations, IAN is inhibitory like IAA. In addition to acting as an auxin precursor, IAN is also known to be an activator or booster of IAA responses.

Ethylindoleacetate is the ethyl ester derivative of IAA and has been isolated from many plant tissues. Both IAN and ethylindoleacetate seem to be more active than IAA, in those tissues which are capable of converting them into IAA. IAN, ethylindoleacetate and **indoleacetaldehyde**, **IAC** (the aldehyde derivative of IAA), all three exist in nature as auxin precursors and all of which can be converted into IAA.

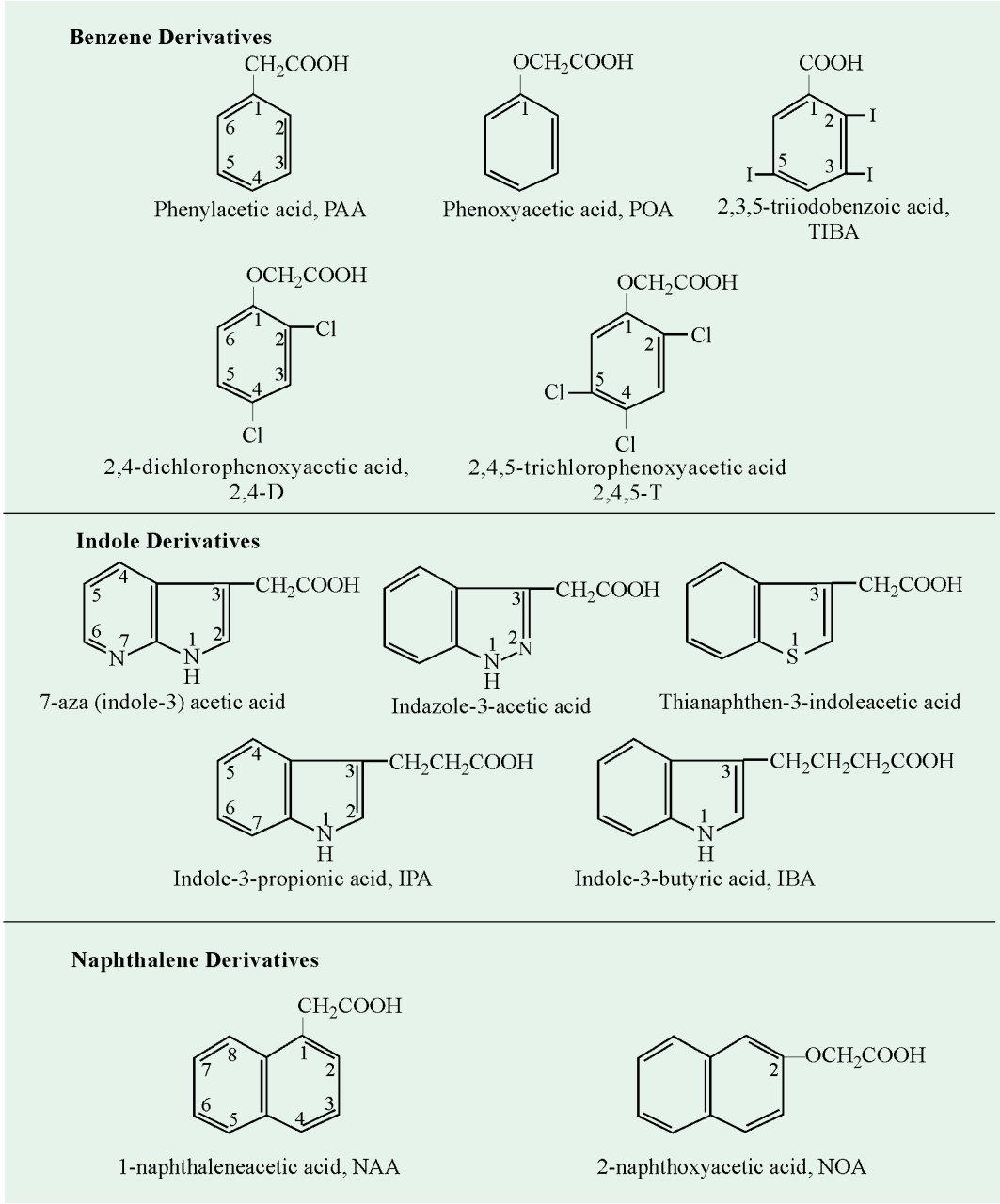


Fig. 32-6. Some synthetic auxins

Synthetic Auxins

Several small organic molecules have been synthesized (Fig. 32-6) which show biologic properties characteristic of indole-3-acetic acid (IAA), though not in all respects. They are usually derivatives of benzoic acid, indole-3-acetic acid or naphthalene acetic acid. *An acidic side chain, unsaturation in the ring and an unfilled ortho position usually characterize many active molecules*; although no general rule can be framed as some thiocarbamates, which do not satisfy most of the requirements, are quite active. *The D-isomer is more active than the L-isomer. Cis*-cinnamic acid

is an auxin whereas *trans*- cinnamic acid behaves as an antiauxin. The 3-'D' structure of the molecule and the spatial relationship between the side chain and the aromatic ring, if present, determine biologic activity. Some of the potent synthetic auxins are TIBA, 2-4-D, 2,3,5-T, NAA and NOA. Phenylacetic acid (PAA) is, however, a weak auxin.

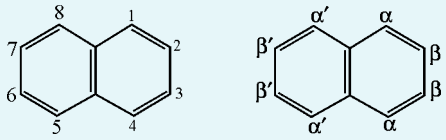
Biogenesis (= Synthesis) of Auxins

Folke Skoog (1937) of the University of Wisconsin, for the first time, experimentally proved that tryptophan is an auxin precursor in higher plants. Since then a wide variety of plant tissues (like leaf, stem, buds, coleoptile, ovary, pollen, embryo, endosperm and callus tissue) have been shown to convert tryptophan to indoleacetic acid (Larsen, 1951). In fact, it is quite probable that all living plant tissues may have the capability of bringing about this conversion. Although tryptophan is the primary precursor of IAA, about half a dozen indole compounds have been found to serve as potential precursors of IAA.

For the enzymic production of IAA, a plausible hypothesis has been suggested by Wildman, Ferri and Bonner in 1946. According to this hypothesis (Fig. 32–7), tryptophan, liberated by hydrolysis of proteins, undergoes either oxidative amination first and then decarboxylation or vice versa to yield indoleacetaldehyde (IAc). IAc is then oxidized to yield the free auxin. Indoleacetaldehyde, thus, acts as an intermediate metabolite as well as an immediate precursor of IAA.

System of Carbon Numbering in Naphthalene

A molecule of naphthalene consists of two benzene rings joined together. The various carbon atoms in naphthalene (as also in other organic compounds) are numbered using either the Arabic numerals (*Arabic system*) or Greek alphabets (*Greek system*), as shown below :



Two systems of carbon numbering in naphthalene

Positions of carbon atoms numbered 1, 4, 5, and 8 in Arabic system are similar; hence, they all are called as α in Greek system. However, to differentiate between these two pairs of C atoms (1–4 pair of one ring and 5–8 pair of another ring) in the Greek system, carbon atoms 5 and 8 of the second ring are numbered by putting a prime (′) sign on the right of α as superscript, thus numbering them as α′. Similarly, position of carbon atoms numbered 2, 3, 6 and 7 in Arabic system are similar; hence they all are called as β in Greek system. By the same reasoning, carbon atoms numbered 6 and 7 are designated by β′.

The various positions of carbon atoms, denoted by Arabic numerals, have been given specific names in Greek system. These are as follows :

Position 1, 2 is known as *ortho*.

Position 1, 3 is known as *meta*.

Position 1, 4 is known as *para*.

Position 1, 5 is known as *ana*.

Position 1, 6 is known as *epi*.

Position 1, 7 is known as *kata*.

Position 1, 8 or 4, 5 is known as *peri*.

Position 2, 6 or 3, 7 is known as *amphi*.

Position 2, 7 or 3, 6 is known as *pros*.

The capability of living plant tissues to form auxin from tryptophan has been demonstrated for spinach (Wildman *et al*, 1947), pineapple (Gordon and Nieva, 1949) and many others. The three essential conditions for IAA synthesis are the presence of light, zinc and an enzyme system.

Distribution of Auxins

The greatest concentration of auxins is usually found in the growing apices of the plant, *i.e.*, in the coleoptile tip, in buds and in the growing tips of leaves and roots. However, auxin is found widely distributed throughout the plant body. In general, it may be stated that where there is active growth, there is auxin production. The formation of auxin by a mature organ like leaf, however, suggests that growth may not be the pre-requisite to auxin production.

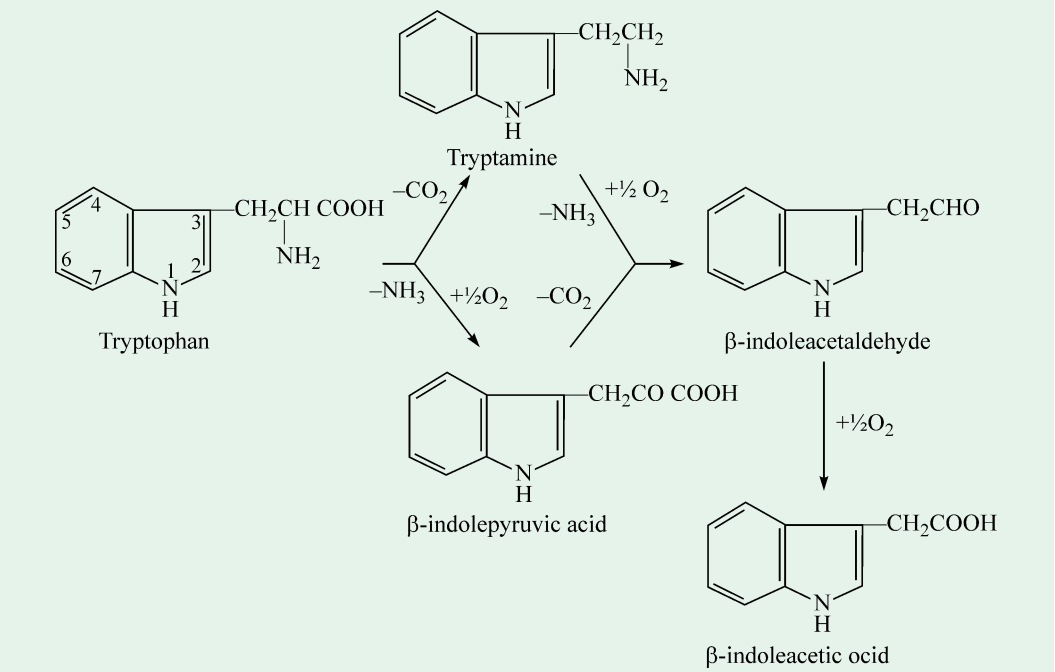


Fig. 32–7. Possible possible pathways of auxin synthesis from tryptophan

Thimann (1934) studied the distribution of auxin, in detail, in etiolated *Avena* coleoptile (Fig. 32–8). He found that the concentration of auxin drops as one progresses from the coleoptile tip to its base; the highest concentration being at the tip and the lowest at the base. If one progresses further from the base of the coleoptile along the root, there is a steady increase in auxin content till a maximum is reached in the root tip. Of the two maximal values, that for the stem tip is much higher than that for the root tip.

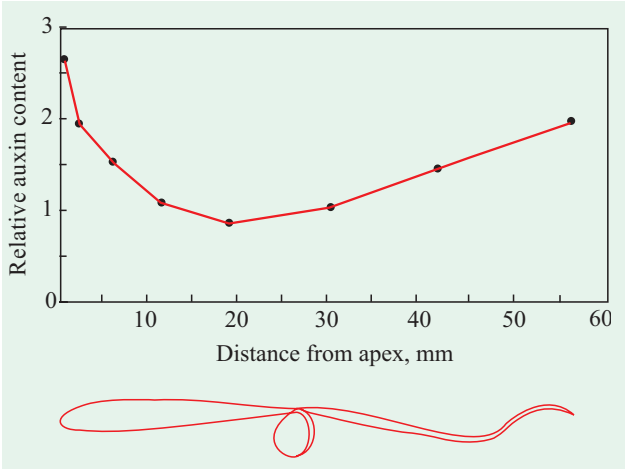


Fig. 32–8. Distribution of auxin in an etiolated Avena seedling

(*After Thimann KV, 1934*)

Thimann and Skoog (1934), while working on *Vicia faba* seedlings grown

in light, found the concentration of auxins in various organs in the following descending order :

Apical buds > Young leaves > Mature leaves

The amount of diffusible auxin per hour for these organs was found to be approximately in the ratio of 12 : 2 : 1.

Van Overbeek (1947) studied the distribution of both free and bound auxins in pineapple. He found that large quantities of free auxin occurred in apical buds and lowest amount in mature leaves. For bound auxin, however, the condition was found to be reverse.

A few other examples of auxin concentration studies are given in Table 31–1.

Table 32–1. Amount of auxin present in different organs of some plants

S.N.	*Plant*	*Organ*	*Maximum Concentration Found (in µg IAA equivalent)*
1.	Corn	Endosperm	105, 000
2.	Lily	Stem tip	83,900
3.	Oat	Grain	1,000
4.	Rice	Endosperm	250
5.	Turimp	Seed	250

Concentration of Auxins

The concentration of auxins, which has a profound influence on growth changes, varies from organ to organ (Fig. 32–9). The effect of auxin concentration on the shoots is quite different from

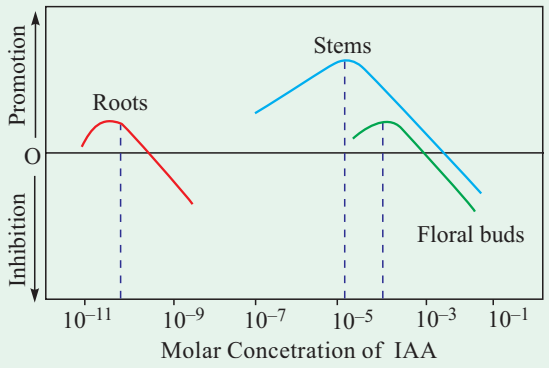


Fig. 32–9. Graph showing the interrelationship between growth promotion (or growth inhibition) and various plant organs such as roots, stems and floral buds

The optimum concentrations for growth promotion were found to be between 10–11 and 10–9 for roots, 10–8 and 10–3 for stems and 10–5 and 10–3 for floral buds.

(*After Leopold AC and Thimann KV, 1949*)

that on the roots. Higher concentration of auxin, which has growth-stimulatory effect on the shoots, is growth-inhibitory for the roots ; the latter growing better at much lower concentration. Thus, *in general, the optimum range of concentration for elongation in stems is much higher than for the roots*. The stems grow best at an auxin concentration of 1.0 mg/litre, whereas the optimum concentration for the roots is of 0.001 mg/litre.

Translocation (= Movement) of Auxins

The auxins are transported in plants from one organ to the other. The usual direction of auxin transport is downward but when added to the soil these are absorbed by the roots and carried

upward along with transpiration stream to various plant organs. The prevailing downward movement takes place through the living phloem cells whereas the upward movement occurs through the dead xylem elements.

The most striking characteristic of auxin movement is its almost strict *basipetal* (from apex to base) *polarity*. This has been demonstrated in various plant organs such as oat coleoptiles (Went and White, 1939), petioles of leaves and herbaceous and woody strems (Oserkovsky, 1942). The actual proof of polar transport was furnished by Went (Fig. 32–10). He showed that if an agar block containing auxin be affixed to the morphologically upper end of a coleoptile segment and a block of pure agar to the lower end, auxin would move and collect in the agar block at the lower end. But if the coleoptile segment is inverted, no translocation of auxin would occur. The translocation of auxins in oat takes place through the parenchyma tissue.

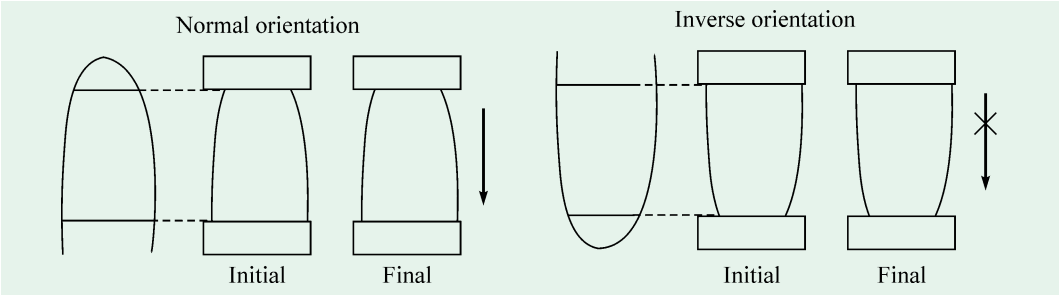


Fig. 32–10. Experiment demonstrating basipetal movement of auxin in oat coleoptile

(*After Went FW, 1935*)

In *Coleus*, however, Leopold and Guernsey (1953) have shown that the basipetal polarity becomes progressively weaker as the distance from the shoot apex increases. Further, in the flowering stems there is some acropetal (from base to apex) movement also even at the stem tip. Jacobs (1961) has also shown that in *Coleus* stem sections, the ratio of basipetal to acropetal transport of auxin is 3 : 1. In fact, there exists a polarity gradient in *Coleus* from a complete basipetal polarity in vegetative apex to a complete acropetal polarity in root apex with a gradual transition in between.

The velocity of auxin transport varies from 26 mm per hour to 6.4 mm per hour (Rajgopal, 1967). These rates are higher than the rates of diffusion. The velocity of auxin transport is unaffected by temperature, although the amount of auxin transported is proportional to the temperature. Also, the distance over which the transport occurs does not influence the velocity.

Mechanism of Auxin Action

The action of auxins, like growth itself, seems to be a complex of many functions. Although the mechanism of auxin action may, in part, be attributed to each of these functions, none of these can account for *in toto* the multifarious effects of these growth substances. The various views put forward to explain auxin behaviour may be, for convenience, grouped under following five headings :

1. Molecular reaction theories. Skoog (1942) expressed the view that the auxin may act like a coenzyme and serves as a point of attachment for some substrate onto an enzyme regulating growth. The molecular configuration of the auxin affects the activity by altering the fit and functioning of this molecular union. The higher auxin concentrations would inhibit growth owing to separate molecules combining with the enzyme and the substrate.

Muir *et al* (1949) advanced the hypothesis that the auxins (*esp*., phenoxy acids) may combine with some material (*e.g*., protein) in the cell at two points, the ortho position of the ring and the acid group of the side chain

Foster and his associates (1952) put forward a theory of auxin action by *2-point attachment*.

It is presumed that the enzyme is attached to a substrate to form an enzyme-substrate complex and that the complex may, then, dissociate to produce the end product of the reaction (*i.e.*, growth) and regenerate the enzyme. Considering the enzyme as the material with which auxin reacts, the theory may thus be expressed as :

$$E + S \rightleftharpoons ES \longrightarrow \text{Growth} + E$$

[where E = auxin receptor ; S = auxin ; ES = complex]

The inhibitory effect of high auxin concentration on growth may well be explained by this hypothesis. This would result from two auxin molecules becoming attached to the receptor substrate, one at each of the points of attachment and each preventing the functioning of the other (Fig. 32–11).

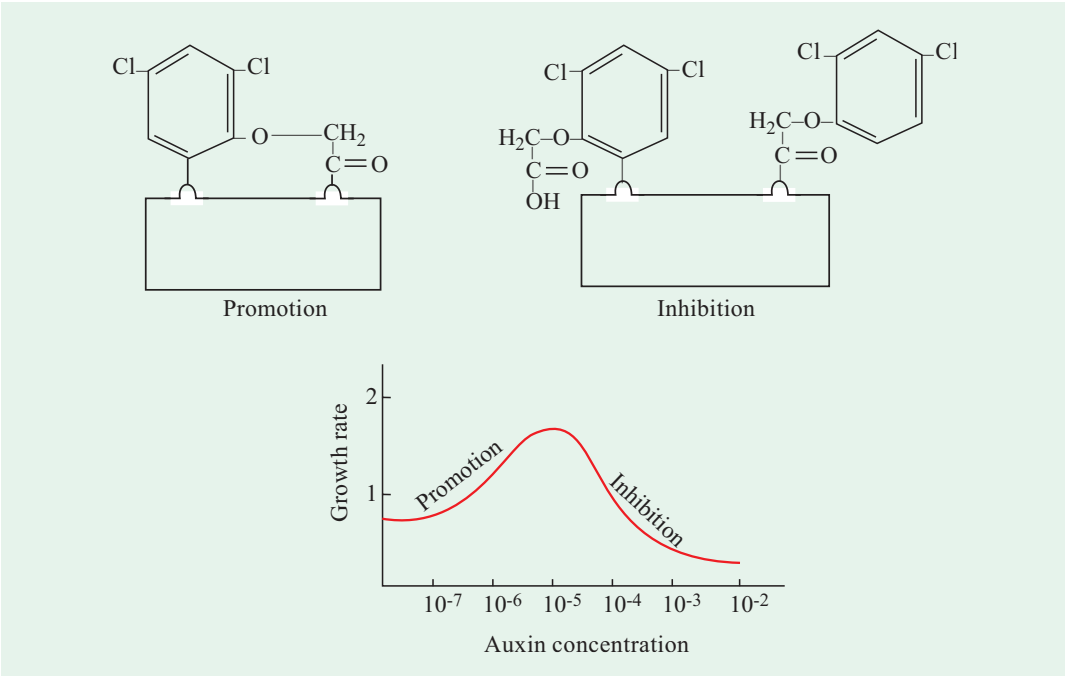


Fig. 32–11. Effect of auxin concentration on growth

2. Theories of enzymatic effects. The fact that growing tissues, upon treatment with auxin, show an increased activity of a number of enzymes has proved to be the basis of these theories. Northen (1942) pointed out that the auxin causes decrease in cytoplasmic viscosity and also brings about dissociation of the cytoplasmic proteins. The latter effect would result in an increase in water permeability and also in the osmotic value of the cytoplasm. These effects ultimately lead to enhanced enzymic activity.

Burger and Avery (1942) demonstrated that some *dehydrogenases*, under certain conditions, could be stimulated by auxin. Thimann (1951), however, found that the auxins act as agents protecting certain growth enzymes from destruction rather than as substances activating enzymes.

3. Theories of osmotic effects. During the process of growth, the cell increases in volume due to water uptake. The uptake of water occurs due to changes in the cytoplasm itself (*esp.*, changes in the osmotic value) or due to changes in permeability of the cell wall and the cell membranes.

Czaja (1935), for the first time, stated that the auxin may increase the volume of the cell which could result directly in water uptake and growth. Van Overbeek (1944), however, pointed out that growth is not necessarily associated with an increase in osmotic value.

Commonor and his associates (1942–43) suggested that since water uptake in growth may be linked with respiration, the process of growth may be explained as due to the osmotic uptake of water which is, in its turn, activated by respiratory uptake of salts.

Two important *objections* put against this theory are as follows :

(*a*) In some instances (*e.g.*, potato slices), growth cannot be a function of salt accumulation causing an increase in osmotic value (Van Overbeek, 1944).

(*b*) Reinders (1942) points out that the auxin-induced growth can occur in pure water in the absence of salt uptake. She describes water uptake as dependent upon oxidative metabolism.

Thus, water uptake is not the cause but the consequence of growth (Burström, 1953).

4. Theories of cell wall effects. Hyen (1940) attributed growth to the dynamic function of the cell wall instead of the cytoplasm. He observed that auxin application increases flexibility and extensibility of the cell wall. This results in lowering the wall pressure around the cell wall, thus permitting water uptake due to this simple drop in turgor pressure.

The elasticity of cells always increases at the start of cell stretching but decreases again before the cells have reached maturity. Thus, increasing elasticity cannot cause elongation but is connected with the elongation process. Ruge (1942) has suggested that cell elongation proceeds in two different phases :

(*a*) an increasing extensibility of the wall without synthesis of new wall material.

(*b*) a hardening of wall with a deposition of new wall material through either intussusception or apposition.

The theory envisages that the effect of auxin upon growth is attained through activating cell wall growth. The wall is believed to be made plastic enough for cell extension and the deposition of new cell wall material, then, causes cell enlargement. To keep up with the growing wall, the cytoplasm must be capable of taking up sufficient water through osmosis.

5. Theories of toxic metabolism. Besides promoting growth, the auxins can also inhibit elongation. In short this inhibition is brought about by high auxin concentration whereas in roots the inhibition is induced even by relatively low auxin concentrations. This inhibition is thought to be due to excess auxin molecules inactivating the sites of auxin action and, thus, checking maximum growth response.

Van Overbeek (1951) proposed that growth regulator toxicity may be a result of an alternation of metabolism in such a manner that unsaturated lactones are accumulated in plant tissues. These are toxic to plants when applied in higher concentrations. Such toxic compounds may accumulate in plant tissues following application of such hormones as 2,4-D.

Thus, it may be concluded that the mechanism of auxin action remains unsolved, although the various theories put forward provide some clues regarding various functions which the auxins perform.

Physiological Roles of Auxins

It was previously thought that the sole function of auxins was to promote cell enlargement. But the work done in later years has proved them to be deeply associated with a variety of functions. In some cases they act as a stimulating agent, in others as an inhibitory agent and in still others as a necessary participant in the growth activity of other phytohormones such as gibberellins and cytokinins. The various growth processes in which the auxins (both natural and synthetic) play their role are discussed below :

1. Cell elongation. It is usually considered that cell elongation occurs only in the presence of auxins and also that the rate of elongation is directly proportional to the amount of auxin applied provided no other factors are limiting. But relatively high concentrations usually exert inhibitory effect on this phase of growth.

AUXIN AS HERBICIDE

Some commercial weedkillers are chemically similar to auxin and have similar effects. They cause affected plant cells to elongate and the plant grows. The exact mechanism in not yet understood, but the auxin seems to interfere with DNA transcription and RNA translation. The amount of auxin applied in a weedkiller is far greater than the amount produced within the plant and so the rate of growth produced is much greater than normal. The plant cannot sustain this rate of growth : it becomes weakened, unable to reproduce, and then it dies. The auxin like herbicides have a much greater effect on dicots than on monocots, such as grasses. This is partly because dicot leaves have a larger surface area than monocot leaves and so absorb more herbicide.

Auxin-like herbicides as efficient means of getting rid of weeds like dock

As already discussed, the various plant organs like roots, buds and stems all react in a comparable way to auxins : their growth being promoted by relatively low and inhibited by relatively high auxin concentrations. Elongation of roots is promoted only at very low concentrations ; at higher concentrations their growth is retarded. Stems and coleoptiles respond similarly except that optimum range of concentrations for elongation is much higher than for roots. Flowers require still higher concentration for growth.

Auxins also play a significant role in the elongation of petiole, mid rib and major lateral veins of the leaves. Thus, adenine favours enlargement in detached leaves of radish and pea. Similarly, coumarin has been shown to promote expansion of leaves in some plants.

An osmotic equilibrium exists in a cell where the turgor pressure developed is counterbalanced by the wall pressure acting in opposite direction. Regarding the mechanism of cell elongation, it is thought that auxins stimulate cell elongation by modifying certain conditions responsible for this equilibrium (Devlin, 1969). These modifications include:

(*a*) an increase in osmotic contents of the cell

(*b*) an increase in permeability of the cell to water

(*c*) a decrease in wall pressure

(*d*) an increase in wall synthesis and

(*e*) an inducement of specific RNA and protein synthesis.

2. Cambial activity. In the spring season, the trees exhibit growth by developing buds which later on open. This is then followed by elongation of the young stems. This resumption of growth by cambial cells is activated by the auxins which move basipetally in the stems from developing buds. Snow (1935) has shown that a steady supply of auxin a at 1/1,000,000 mg per hour (or of IAA at 1/500,000 mg per hour) from a gelatin block, upon affixing it to the cut end of a decapitated shoot of sunflower (*Helianthus annuus*) seedling, stimulated meristematic activity of the cambium.

The suggestion by Jost (1940) seems to imply that the major function of hormones that migrate from the developing apex of the epicotyl is to activate the differentiation of procambial strands.

3. Callus formation and galls. Besides acting as stimulants of cell elongation, the auxins may also activate cell division. This may be illustrated by applying 1% IAA in lanolin paste to a debladed petiole of a bean plant. This causes prolific division of parenchyma cells resulting in the

formation of a swelling or callus tissue at a point where the auxin in applied. The amount of callus tissue formed is directly proportional to the concentration of IAA applied (Ropp, 1950).

Bezerinck, in as early as 1885, investigated the production of cecidomid galls in *Poa nemoralis.* He compared gall production to callus formation and thought of a substance coming from the larval body as a causative agent. He regarded this action of animal substance as analogous to that of the inner causes which lead to the formation of roots in normal plants.

4. Rooting of stem cuttings (= Formation of adventitious roots). It is a common observation that the presence of buds on a cutting favours development of roots when the lower end is dipped in a suitable rooting medium. Developing buds are effective in accelerating root formation. Young leaves also favour the initiation of roots on the cuttings. These observations led to the suggestion that the root formation is favoured by the auxins which are synthesized in the buds and young leaves and are later translocated to the basal part of the cutting.

Two techniques are usually employed to introduce auxins into the cuttings. In one, "*dry form*" *method*, the auxin is first mixed with an inert powder such as talc in the proportion of 500 to 2,000 parts of the auxin to 1,000,000 parts of the talc. Later, the basal end of the cutting is first dipped in water and then in powder before immersion into the rooting medium.

In the other, "*quick dip*" *method*, the basal end of the cutting is dipped for about 5 seconds into a relatively concentrated solution of auxin (5,000 to 10,000 ppm in water or 5% ethyl alcohol) before immersing in the rooting medium.

The auxins most commonly employed for this purpose are IAA, NAA, 2,4-D, naphthalene acetamide (NAd) etc.

Auxin-induced rooting is not only of academic interest but also of enormous horticultural value as it helps propagation of certain plants by cuttings. Although some success has been achieved in bean and alike plants, yet certain other plants like apples do not respond to it (Thimann and Behnke, 1947).

5. Apical dominance. It has been generally observed that so long as the apical bud is intact on the plant, the growth of the lateral buds remains suppressed. Upon removal of the apical bud, the lateral bud nearest the apical bud establishes its dominance over the remaining buds, causing them to become inactive again. *This inhibitory effect of a terminal bud upon the development of the lateral buds is called apical dominance and produces a cone-shaped plant.* This is why a gardener keeps on trimming the hedge occasionally in order to obtain a denser growth. Plants that are tall and unbranched exhibit strong influence of apical dominance than those which are short and branched.

The characteristic conical shape of most **Christmas trees** results from apical dominance. Almost all Christmas-tree-shaped trees are conifers. Ever since U.S. President Franklin Pierce (a politician, otherwise known for putting stickum on the backs of postage stamps) first had a Christmas tree in the White House in 1856; people have been choosy about these trees, preferring ones that are 2 metres high and 1.3 metres wide at the base. Scotch pine (*Pinus sylvestris*) is the most popular Christmas tree in India, the United States and elsewhere. It is a fast-grower and takes only 8 years to grow to a height of 2 metres. Although the tree has long needles, but they stay on the tree much longer than those of balsam fir (*Abies balsamia*) or spruce (*Picea* sp.), the previous best sellers.

The relation of apical dominance with the auxin supply was first reported by Skoog and Thimann (1934). They demonstrated that when agar block containing auxin *b* or IAA was kept on the decapitated shoot of broad bean (*Vicia faba*), the lateral buds, as might be expected, resulted in the usual suppression of growth as if the terminal buds were present. But when the same decapitated shoot was reheaded with an agar block containing no auxin, these lateral buds resumed growth. Similar results were also obtained with field-grown tobacco plants using NAA as the auxin.

Although the exact mechanism behind this growth correlation is not yet known, the best explanation for this has been furnished by Snow (1939, 40). According to him, under the influence of auxin, a growth inhibitor is formed that is responsible for the inhibition of growth of the lateral buds. The growth inhibitor is synthesized in some unknown manner when the auxin moves in its usual downward direction. The theory lends support from the fact that certain growth inhibitors have chemical structure not much different from that of the auxins. It may, thus, be visualized that such a synthesis of growth inhibitor from the auxin may take place in the tissues.

The knowledge of apical dominance has been utilized practically in solving the problem of storage of the potatoes. Potatoes, stored for some time, sprout and become sweet in taste, thus causing financial loss to the grower as the sweet taste is disliked by its consumers. But spraying the potatoes with auxins like indole butyric acid (IBA) and NAA would prevent sprouting (or in other words, would prolong dormancy) by inhibiting the development of buds or 'eyes' ; the effect persisting for as long a period as 3 years. Although such a treatment is in the interest of the breeder but certainly not in that of the consumer.

6. Delay (or inhibition) of abscission of leaves. The abscission of leaves can be delayed or inhibited by the application of auxins on the surface of the lamina or on the cut surface of a debladed petiole. The controlling behaviour of the auxins on the abscission was first noted by Laibach (1933) who showed that the extract of orchid pollinia is capable of preventing the leaf fall. Since then, enough work has been carried out in this direction. Addicott and Lynch (1955) have proved conclusively the delaying effect of IAA on the abscission of various plant organs.

As to the mechanism of abscission, it has been suggested that the leaf fall is retarded by the basipetal migration of a hormone from the blade to the base of the petiole. Removal of the leaf blade eliminates the supply of hormone to the abscission zone and thus induces leaf fall.

The correlation between the amount of diffusible auxin and the age of the organ concerned (Fig. 32–12) was demonstrated by Shoji et al in 1951. They found high auxin contents in the young leaf blades of bean plant as compared to their petioles. But on ageing, the auxin contents in the leaf blade and the petiole fall almost to the same level.

7. Flowering. A flowering hormone, *florigen*, is produced in the leaves under correct light and dark period. It moves first down the petiole and then up the stem to the growing apex where it causes the development of floral buds in place of vegetative buds (Cajlachjan, 1936). Auxins are useful in modifying flowering in one of the following ways :

A. *Altering earliness*— Leopold and Guernsey (1953) conducted experiments on oat, corn, barley, peas etc., and found that when their seeds were treated with auxins followed by low temperature treatment at 4°C for 5—15 days, they produced quantitative increase in earliness. Chakravarti (1955)

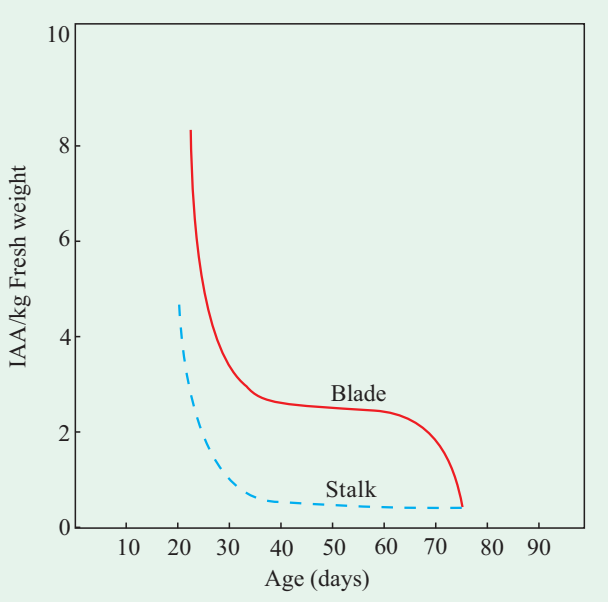


Fig. 32–12. Efect of ageing of the petiole and the leaf blades of bean plant on the auxin contents of these organs (*After Shoji et al, 1951*)

also produced earliness and greater yield in mustard when auxin was applied before chilling. No gains were, however, obtained when the auxin was applied after chilling.

B. *Inducing flowering*— The flowering in pineapple has been successfully promoted in Hawaii islands by treating the fields with sodium naphthalene acetate at some time during growing period. In Caribbean areas, however, 2, 4-D is more commonly used.

Leopold and Thimann (1949) have, likewise, found low concentrations of auxins such as a-NAA or IAA as effective in inducing flowering in barley and Teosinte. Furthermore, 2, 4-D has been successfully used in control of flowering in sweet potato.

C. *Preventing or delaying flowering*— Although natural flowering of many species of plants is inhibited by high endogenous auxin concentrations, attempts to attain this inhibitory effect by applying auxins exogenously (*i.e.*, from outside) usually fail as the plants are damaged before effective control is obtained. But experimentally in certain plants like cabbage and celery, the bolting is prevented by applying *p*-chlorophenoxypropionic acid during cold periods which would otherwise normally induce flowering.

8. Fruiting. Auxins play significant role in fruiting by modifying it in one of the following ways :

A. *Fruit setting—Fruit set* refers to the changes in the ovary leading to the development of the fruit. These changes are usually induced after pollination and fertilization— the two processes which are in some way concerned with the release of some stimulus of hormone nature. But the development of fruit without fertilization, *i.e.*, parthenocarpy (*parthenos*^{G} = virgin; *carpos*^{G} = fruit), however, is also a common feature in the plant world and henceforth occurs frequently in nature.

Such a parthenocarpic development of fruits nowadays has also been induced artificially. For example, Yasuda (1934) demonstrated it by application of pollen extracts to cucumber flowers. An analysis of the extract showed that auxins were present in it. Later, Gustafson (1936, 39) also observed that ovaries of many plants (orange, lemon, grape, banana, tomato etc.) could be induced to develop into seedless fruits by application of IAA in lanolin paste to their stigmas. The various other auxins employed for this purpose are IPA, IBA, α-NAA, phenoxyacetic acid (POA), α-naphthoxyacetic acid (NOA) etc.

B. *Fruit thinning*— In many instances, the trees bear extensively large number of fruits. This causes the trees to fail to produce average number of new flower buds. Such trees, therefore, have to produce fruits either at alternate years (*alternate bearing*) or if yearly, the number of fruits is greatly reduced (*infrequent bearing*). These trees, obviously, require thinning.

Fruit thinning was, for the first time, done in apple when naphthalene acetic acid applied to flowers failed to set the fruits and, in fact, caused a decrease in fruit set.

It is surprising to note that *naphthalene acetic acid appears to be the only successful auxin which brings about thinning of fruits*. However, other examples of auxins employed for fruit thinning are a-2,4,5-trichlorophenoxyacetic acid for thinning of pears (Griggs, 1951) and *p*-chlorophenoxyacetic acid for thinning of grapes (Weaver and Winkle, 1952).

C. *Control of premature fruit dropping*— In many fruit trees, the unripe fruits fall off on account of the formation of an abscission layer, thus causing serious losses in yield to the gardeners. This problem has now been successfully overcome in many cases like apples by the application of auxins which prevent the formation of abscission layer and thus check preharvest drop of the fruits. Besides apples, such as control has also been induced in citrus fruits (like oranges and lemons) using 2,4-D and 2,4,5-trichlorophenoxyacetic acid as auxins.

D. *Improving fruit quality*— The various processes like colouration, softening, sweetening and ripening are all involved in improving the quality of the fruit.

Auxin effects on fruit colouration are most pronounced in apples where use of 2,4, 5-trichlorophenoxyacetic acid has greatly increased red pigments. 2,4-D when applied to bananas hastened the process of ripening as the auxin facilitates the conversion of starch to sugars. By injecting 2,4-D, IBA or maleic hydrazide, the accumulation of sugars was reported in sugarcane.

9. Increase in respiration. James Bonner (1953), for the first time, recognized that auxins stimulate the process of respiration. And as such a direct relation between growth due to auxin treatment and the rate of respiration has been found. *Greater the growth, higher is the rate of respiration.*

Such a knowledge about the behaviour of auxins has been applied in controlling the development of weeds which grow obnoxiously in the crop fields. This has been successfully achieved by spraying 2,4-D which acts as a *weed killer*. In fact, this hormone, which operates only on broad-leaved herbaceous plants, increases the rate of respiration so much so that the plants die of over-oxidation and exhaustion. And fortunately most of the weeds are broad-leaved dicots and the crop plants are usually narrow-leaved monocots which, henceforth, escape destruction on spraying 2,4-D.

Another hormone 2,4-dichloropropionic acid (or dalapon), however, destroys graminaceous weeds.

The defoliation is Vietnam forests was protested during the war by many botanists, but the problems have proved to be more serious than the destruction of the forests. Synthesis of 2,4,5-T, a component of Agent Orange, also produces **dioxin** (2,3,7,8-tetrachloro-dibenzo-para-dioxin). *Dioxin is one of the most toxic synthetic chemicals known*. It is also known to be carcinogenic as well as endocrine disruptor in nature and is highly persistent in the environment, besides being extremely resistant to chemical or physical breakdown. Thousands of U.S. pilots and Vietnamese citizens exposed to Agent Orange (which is often contaminated with dioxin) have had higher frequencies of miscarriages, birth defects, leukemia and other types of cancer. Although the **Environmental Protection Agency** (EPA), in 1979, banned the use of 2,4,5-T in the United States, its effects continue to plague many Vietnamese citizens, especially the veterans.

10. Increased resistance to frost damage. In parsnip, the tops resist damage by frost on treatment with 2,4,5-T. Similarly, the application of 2,4,5-T in apricot fruits before the onset of frost resulted in less damage than the untreated fruits.

11. Great weapon of war. Auxins when applied in greater concentrations on enemy crop fields by air may cause devastation of land and thus form the basis of what is called *biological warfare*.

The synthetic auxins (such as 2,4-D, 2,4,5-T and NAA) have been widely used since 1940s. Their use has decreased production costs by reducing the amount of labour and mechanical weeding, needed to grow and effectively harvest a crop. Unfortunately, the effects of synthetic auxins have not all been positive. Most of the negative effects can be traced to a defoliant, once used in Vietnam War, by the code name Agent Orange. **Agent Orange** was a 1 : 1 mixture of 2,4-D and 2,4,5-T that was sprayed throughout the jungles of vietnam and followed by napalm bombs. This resulted in the destruction of hundreds of square kilometres of Vietnam forests.

GIBBERELLINS

The gibberellins are another class of compounds whose minute quantities profoundly stimulate the growth of many plants.

Discovery

The gibberellins were discovered in an interesting and incidental way. In early part of the twentieth century, Japanese farmers noted that some plants in rice fields were taller, thinner and paler than the normal plants; had longer and narrower leaves markedly overgrowing their unaffected neighbours; and were sometimes devoid of fruits too. They named this disease as "*bakanae*",

meaning foolish asseedlings. Sawada (1912) suggested that the disease is due to a 'substance' secreted by a parasitic as comycetous fungus, *Gibberella fujikuroi* (the perfect form, occurring only occasionally; the imperfect form is *Fusarium moniliforme*), in infecting the diseased plants. This suggestion was experimentally supported by Ewiti Kurosawa (1926) who demonstrated that sterile filtrates of the fungus could initiate symptoms of bakanae disease in healthy rice seedlings. Later in 1939, Yabuta and Hayashi isolated this growth promoting substance in crystalline form and named it as gibberellin A, which has now been shown as a mixture of many growth promoters, collectively known as **gibberellins**.

Since that time, gibberellins and allied substances have been found in higher plants also by Mitchell et al (1951), West and Phinney (1957) and Sumiki and Kawarada (1961).

Definition

A *gibberellin* (abbreviated as GA, for gibberellic acid) may be defined as a compound which is active in gibberellin bioassays and possesses a gibbane ring skeleton (refer page 753). There are, however, other compounds (like kaurene) which are active in some of the assays but do not possess a gibbane ring. Such compounds have been called *gibberellin-like* rather than gibberellins.

Isolation, Distribution and Biosynthesis

About 29 gibberellins were previously isolated and their chemical structures known. These

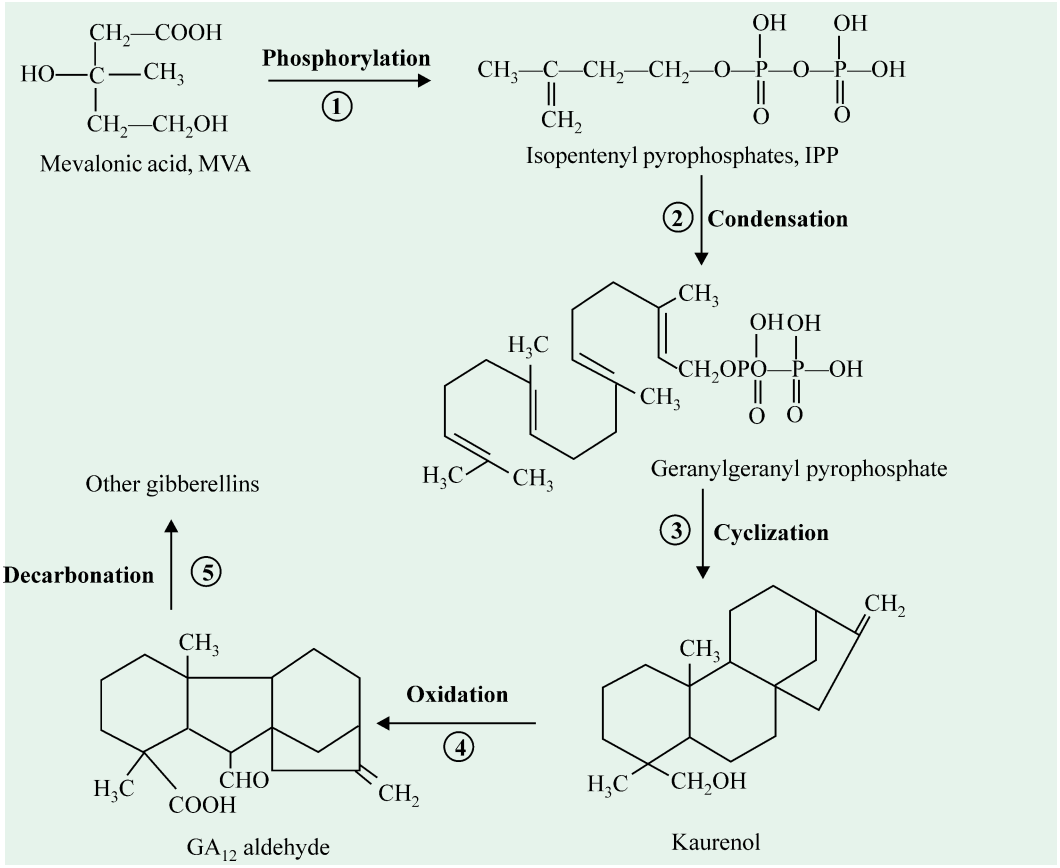


Fig. 32–13. The mevalonic acid (MVA) pathway for the synthesis of gibberellins

Although the biosynthesis of gibberellins from mevalonic acid occurs through some 18 or more steps, 5 key steps culminating in GA production have been outlined here. Mevalonate, in its turn, is produced from acetate.

(*Adapted from Moore R, Clark WD and Stern KR, 1995*)

have been named as gibberellin A_1 (GA_1), gibberellin A_2 (GA_2) and so on up to gibberellin A_{29} (GA_{29}). Of these, Cross *et al* (1961) have isolated 6 gibberellins from the fungus, *Fusarium moniliforme* and designated them as GA_1, GA_2, GA_3, GA_4, GA_7 and GA_9. The same year, MacMillan *et al* isolated 3 gibberellins from bean seeds and named them as GA_5, GA_6 and GA_8. The GA_{10} and GA_{13} have been discovered by Mulholland (1963). All these compounds are sometimes referred to as constituting the **gibberellin A series**. Till date, more than 80 gibberellins have been isolated from various plant sources.

Although the gibberellins were originally isolated from a fungus, but now they have been shown to be present in almost all the groups of plant kingdom including angiosperms, gymnosperms, ferns, mosses and algae but are unknown in bacteria. For example, GA_1, and GA_5 have been isolated from immature seeds of *Phaseolus vulgaris* by West and Phinney (1959). Although all the organs of the flowering plants contain gibberellins, but the highest level has been detected in seeds. Young leaves and roots are also rich in them. It may, thus, be generalized that r*apidly growing and developing regions of the plant possess higher concentrations of gibberellins.*

The gibberellins can exist in more than one form within the plant. Hashimoto and Rappaport (1966) suggested that the esterified forms of gibberellins (*i.e.*, neutral gibberellins) act as reservoir of active gibberellins. The active acidic form may be drawn from the neutral form as and when needed. In addition, bound forms of gibberellins also exist (Mc Comb, 1961).

Many angiospermous plants have now been used as bioassay for gibberellins and gibberellin-like substances. A few of them are *Avena sativa* (leaf section), *Pisum sativum* (intact seedling), *Triticum vulgare* (excised coleoptile) and *Rudbeckia bicola* (resetted plants).

Gibberellins are synthesized via the mevalonic acid (MVA) pathway. In fact, the biosynthesis of GA_3 from MVA proceeds via 18 or more steps or intermediates and about 15 related compounds. The biosynthetic studies were conducted using cell-free systems of *Gibberella fujikuroi* and systems from immature seeds of *Echinocystis macrocorpa* and *Cucurbita maxima*. In essence, the MVA pathway (Fig. 32–13) comprises of 4 major steps for the formation of C_{20} gibberellins, from which C_{19} gibberellins (such as GA_3) are produced by decarbonation, the 5th major step.

Many commercial compounds inhibit the synthesis of gibberellins. These inhibitors (Fig. 32–14), which are called **growth retardants**, include B-Nine, Cycocel (CCC), Phosphon D and Amo-1618. Growth retardants inhibit stem elongation whereby producing stunted plants. Growth retardants are frequently sprayed on growing chrysanthemums to produce flowers with thicker, sturdier stalks.

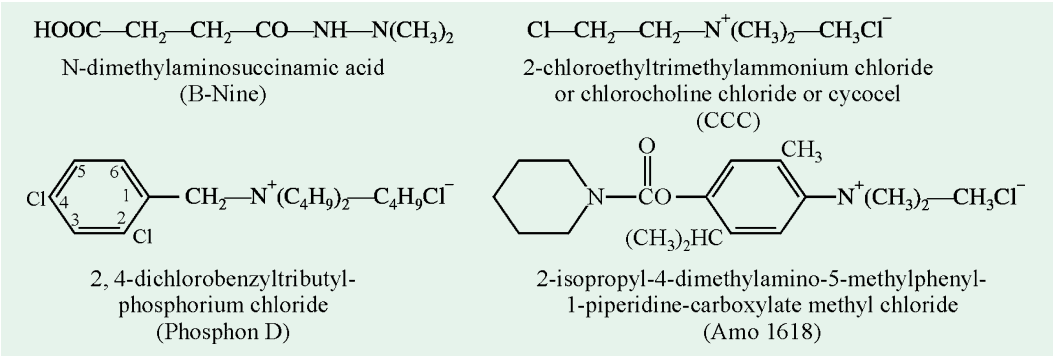


Fig. 32–14. Some growth retardants for the synthesis of gibberellins

Unlike the transport of IAA and other auxins, the transport of gibberellins is not polar: it moves in all directions (*i.e.*, nonpolarly) in the xylem and phloem.

Chemistry

The chemical structure of the gibberellins was established by Cross *et al* in 1961. They showed that the gibberellins (Fig. 32–15) are a group of closely related compounds with one or

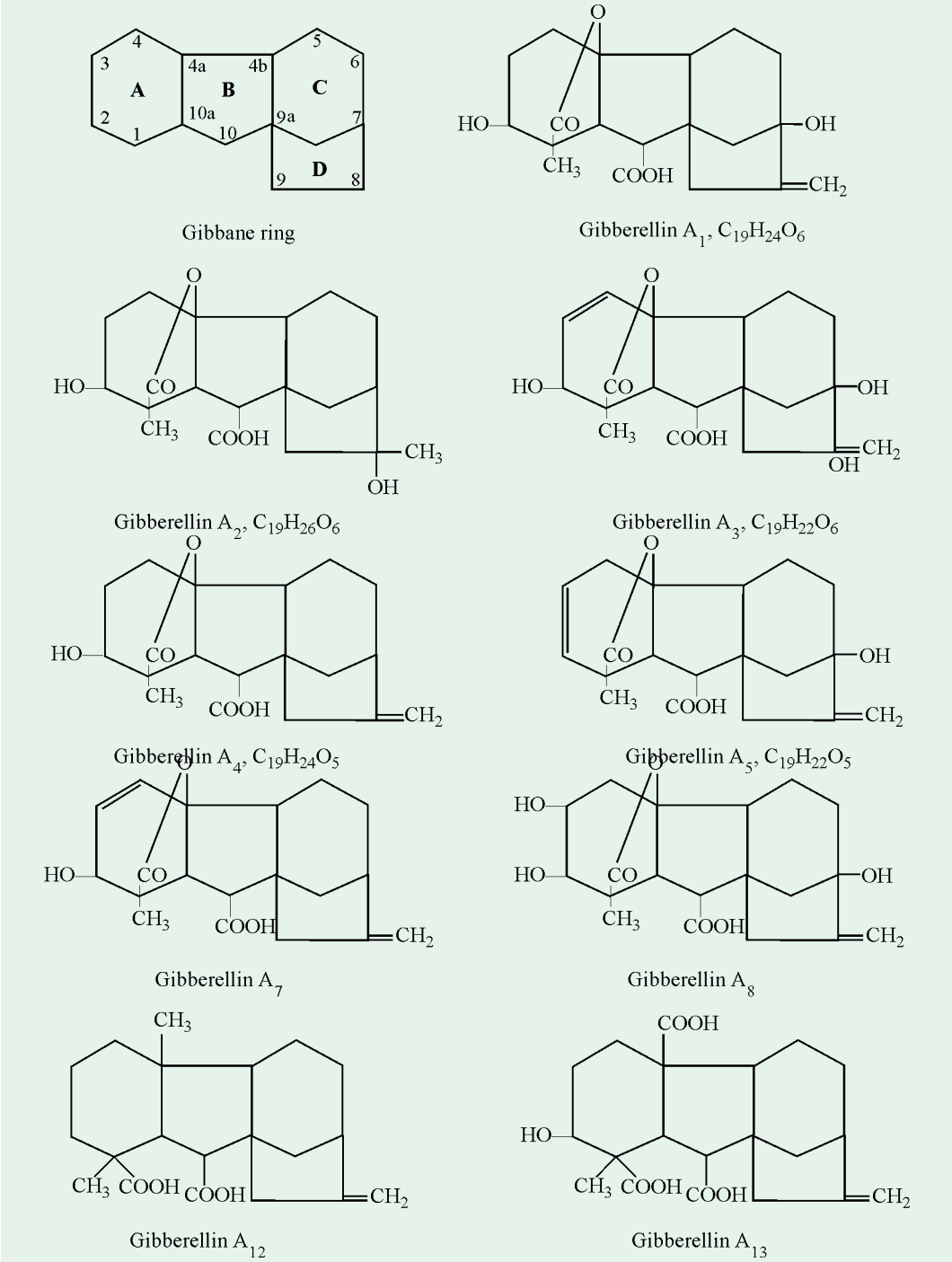


Fig. 32–15. Chemical structure of some gibberellins

Note the minor but significant differences between any two gibberellins. Differences in biologic potency are sometimes associated with presence or absence of double bonds, particular position of the OH groups(s), presence of the lactone ring and the number of carboxyl groups. Also note that while most of the gibberellins have only one carboxyl group, GA_{12} and GA_{13} have two and three carboxyl groups, respectively.

more carboxyl groups that impart acidic properties to the molecule and possess a common feature, the *gibbane ring skeleton*. The gibbane ring consists of a carbon skeleton with 4 interlocking rings, designated as A, B, C and D. They are, hence, described as tetracarbocyclic compounds. Of the 29 gibberellins, previously isolated, 19 are C_{19} compounds and the other 10 have 20 carbon atoms each. Eighteen gibberellins are monocarboxylic, 7 are dicarboxylic and 4 are tricarboxylic acids. The various gibberellins differ from each other in the number and position of the functional groups present in the molecule. In *gibberellin* A_1, the functional groups are a carboxyl, one ethylenic double bond, two alcoholic hydroxyl groups (one secondary and the other tertiary), a saturated lactone and one methyl group. *Gibberellin* A_3 differs from GA_1 in the presence of one more ethylenic double bond in the ring A. It is, thus, more unsaturated than GA_1.

The *gibberellin A_2 and gibberellin A_4* both have structures similar to that of GA_1 except for the difference in position of the tertiary hydroxyl group and the absence of a double bond in GA_2 and in the absence of tertiary hydroxyl group in GA_4. The gibberellin A_5 is, in fact, a dehydrogibberellin A_1 where the secondary hydroxyl group is eliminated from ring A, making the compound more unsaturated.

Gibberellin A_3 has been usually shown to be biologically most active followed by GA_1, GA_4 and GA_2 in descending order of their activity.

$$GA_3 > GA_1 > GA_4 > GA_2$$

Certain gibberellins have been found in plant tissues to occur in bound or conjugated form with other compounds; *free gibberellins* can be released from '*bound ones*' by enzymatic treatment with *emulsin*. In pea seedlings during the first few days of growth, the necessary gibberellin needed for this phase of growth, may partly be released from bound ones. Among the bound forms of gibberellins, those deserving mention are:acetyl-GA_3, D-glycopyranosyl-GA_3, D-glucopyranosyl-GA_8, D-glucopyranosyl-GA_{27} and D-glucopyranosyl-GA_{28}.

Physiological Roles

Gibberellins may be regarded as natural phytohormones on account of their wide range of distribution in plants and specificity of response of individual flowering plants to the exogenously applied gibberellins. The gibberellins, however, play important roles in the following processes:

1. Genetic dwarfism. In certain plants, dwarfism is caused by the mutation of a single gene. Such individuals are called '*single gene dwarfs*'. In these plants, dwarfism is due to shortening of internodes rather than a decrease in the number of internodes. Application of gibberellins on such dwarfs causes them to elongate so much as to become indistinguishable from the tall normal plants. Elongation of the stem, in fact, takes place due to an elongation in the internodes rather than an increase in the number of internodes. Thus, genetic dwarfism has been successfully overcome by gibberellin A_3 treatment in many single gene dwarf mutants like *Pisum sativum, Vicia faba and Phaseolus multiflorus* (Brian and Hemming, 1955).

The gibberellins, thus make most plants grow taller by causing the internodes to elongate considerably (Fig. 32–16).

Two views have been put forward regarding the mechanism of control of dwarfism by gibberellins.

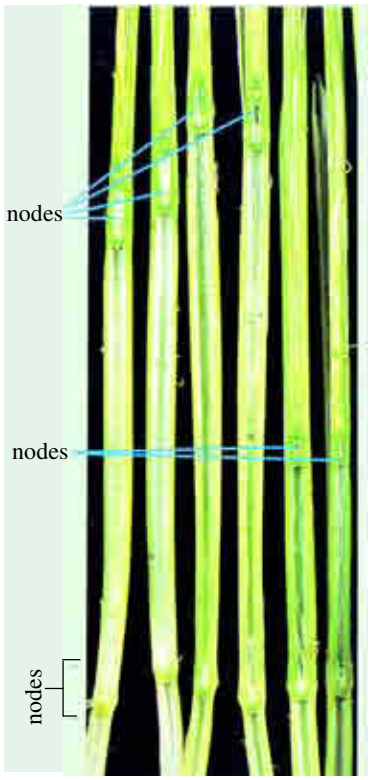


Fig. 32-16. Wheat stems, cut lengthwise, to show the modes
The 4 plants on the left, treated with gibberellic acid, have internode lengths much greater than the intermodes of the two untreated on the right.

(*a*) It is due to the lack of endogenous gibberellins in dwarf plants or if at all present, they are in traces as to have no effect.

(*b*) A natural inhibitor is present in those plants which retard growth. And the gibberellin, when applied, nullifies the effect of this inhibitor.

2. Bolting and flowering. '*Rosette plants*' are characterized by their profuse leaf development and retarded internodal growth. But prior to the reproductive phase, there occurs striking elongation in the internode so that the plant attains 5 to 6 times the original height. Treatment of these `rosette' plants with gibberellins, under conditions that would normally maintain the rosette form, induces them to bolting (or shoot elongation) and flowering (Lang, 1957). By regulating the amount of gibberellin applied, it is also possible to separate shoot elongation from flowering ; *with low dosages of gibberellins, the plant will bolt but not flower* (Phinney and West, 1961).

It is, therefore, not amazing to find a direct correlation between the amount of gibberellin present and the habit of the plant, whether rosetted or bolted. Native gibberellin-like substances are found in higher concentrations in the bolted forms than in the nonbolted ones. This has been experimentally demonstrated in quite a few plants including the biennial *Hyoscyamus niger* by Lang (1957) and the cold-requiring plant *Chrysanthemum morifolium* and a long-day plant *Rudbeckia speciosa* by Harada and Nitsch (1959).

As far as the use of gibberellins in agriculture is concerned, it may be possible to grow cold-requiring plants in warm countries and long-day plants in short-day conditions at lower altitudes.

Gibberellic acid (GA) hastened flowering and improved the flower yield in *Coriandrum sativum* (coriander). This was accomplished by a decline in starch content and an increase in reducing sugars, as well as enhanced amylase activity. It was inferred that GA_3 hastened flowering probably through its influence on carbohydrate metabolism (Amrutavalli, 1979).

3. Light-induced inhibition of stem growth. Light-grown plants reveal suppressed stem growth than the dark-grown (or etiolated) plants, indicating that light has an inhibitory effect on stem elongation. But this inhibitory effect of light on stem elongation can be reversed at least in some plants (*like Pisum sativum*) by the application of gibberellins on these plants. This clearly suggests that endogenous gibberellin is the limiting factor in stem elongation.

Lockhart (1961) has given a possible explanation for it. According to him, exposure to light lowers the level of available gibberellins present in the plant. The lowered available gibberellin contents then, in turn, decrease the plasticity of cell walls, thus inhibiting stem growth. The theory has, however, not won the universal support on account of the following *drawbacks* :

(*a*) Stem elongation is also induced in mustard seedlings, grown in dark, upon application of gibberellin.

(*b*) In some plants, gibberellin-stimulated stem growth has been found to be partially due to enhanced cell division and has nothing to do with cell wall plasticity.

(*c*) Germination of the seeds of *Lactuca sativa* is not only promoted by gibberellins but by red light too.

4. Parthenocarpy. Like auxins, the gibberellins are also capable of inducing parthenocarpic fruit-set. *Gibberellins are, in fact, more efficient than the auxins in inducing parthenocarpy*. For example, Wittwer and Bukovac (1957) have found gibberellin to be about 500 times more effective than IAA in inducing parthenocarpy in tomatoes. Moreover, there are cases where auxins have failed to induce parthenocarpy while gibberellins are effective, as shown experimentally for apples (Davison, 1960) and stone fruits (Crane *et al*, 1960).

Gibberellin-induced parthenocarpy has been reported in many plants such as *Cucumis sativus* (cucumber), *Solanum melongena* (brinjal) and *Zephyranthes* sp. Whether the production of parthenocarpic fruits is a direct action of gibberellins or an interaction with the natural auxins of the plant has not been conclusively proved.

5. Breaking dormancy of seeds. The light-sensitive seeds (lettuce, tobacco) show poor

germination in dark and on exposure to light their germination starts vigorously. But when these seeds are treated with GA_3, the light requirement is alleviated and they germinate in dark.

6. Breaking dormancy of buds. In temperate areas, the buds produced in winter remain dormant until the next spring due to very low temperature. The dormancy in such cases is overcome by gibberellin treatment. Thus, GA_3 treatment to birch buds has replaced the light requirement for breaking dormancy (Eagles and Wareing, 1964). Gibberellins are also capable of breaking dormancy in potato tubers.

7. Role in abscission. GA_3 treatments have shown accelerated rate of abscission in explants of bean (Chatterjee and Leopold, 1964) and of *Coleus* (Gupta and Kaushik, 1969).

8. Stimulation of enzyme activity in cereal endosperm. Yomo (1960) and Paleg (1960) working independently showed that the gibberellins applied exogenously could stimulate *amylase* activity in isolated barley endosperm. It was then shown that it is the aleurone layer of the endosperm which is sensitive to the gibberellin. Subsequent researches by Paleg (1964) and Varner (1964) revealed that GA treatment of isolated aleurone can cause release of the enzymes, *amylase* and *proteinase*. Finally, Jacobson and Varner (1967) showed that the two enzymes (amylase and proteinase) induced by GA treatment arise through *de novo* synthesis. These enzymes participate in the breakdown of the stored starch to simple sugars. These sugars are then translocated to the growing embryo where they provide energy for growth.

de novo is a Latin phrase, meaning anew.

9. Sex expression. Gibberellins are also capable of altering the sex of the flowers. Galun (1959) could induce maleness by foliar application of GA_3 to the female flowers of *Cucumis*. Also, the antheridia have been induced to develop in many fern gametophytes by GA_3 treatment.

10. Juvenility. Many plants in their life cycle exhibit two different stages of growth: a juvenile stage and an adult stage. For example, in a species of eucalyptus (*Eucalyptus globulus*), the juvenile leaves are oppositely-placed and are shorter, softer and with emarginate apex; while the leaves of the adult stages are spirally-arranged and are larger, harder and acicular with pointed apex (Fig. 32–17). Gibberellins may help determine whether a particular part of a plant is juvenile or adult. For instance, the buds of adult branches usually develop only into adult branches, but

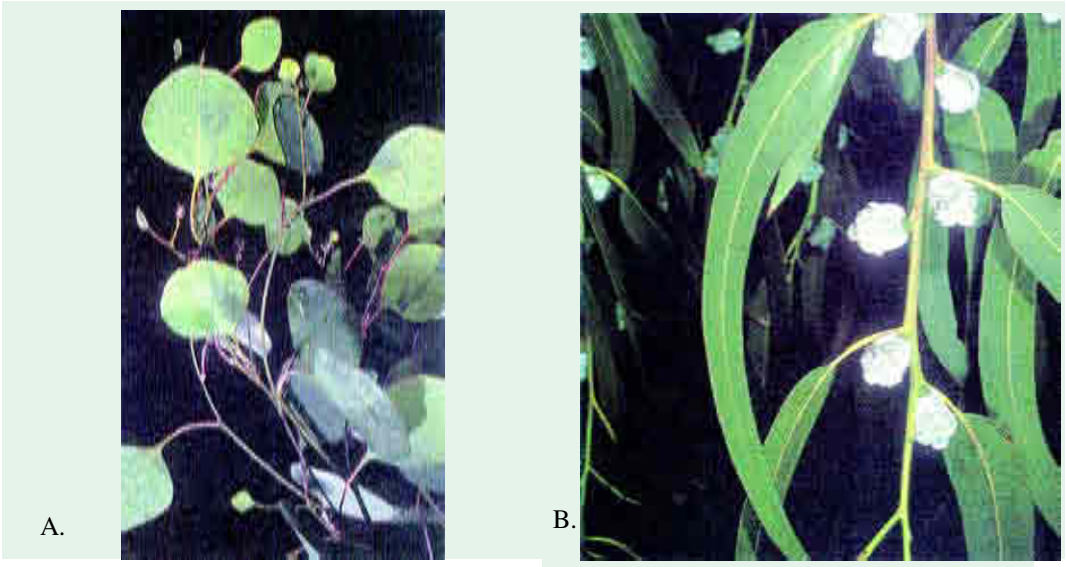


Fig. 32–17. Photographs of two growth stages of *Eucalyptus globulus* leaves
A. Juvenile stage B. Adult stage

The difference in the leaves of 2 stages is also exhibited in their anatomy. The juvenile leaves have palisade cells only on the upper surfaces, whereas the adult leaves contain palisade parenchyma on both sides.

treating them with gibberellin causes them to grow into juvenile branches.

Relationship between Auxins and Gibberellins

Auxins and gibberellins are similar to each other in that both promote cell elongation, flowering and parthenocarpy. These, however, differ form each other in many of the physiological activities. These differences are listed in Table 32–2.

Table 32–2. Differences between auxins and gibberellins

S. .N	*Physiological activity*	*Auxins*	*Gibberellins*
1.	Transport polar	Yes	No
2.	Promote root initiation	"	"
3.	Inhibit root elongation	"	"
4.	Delay leaf abscission	"	"
5.	Inhibit lateral buds	"	"
6.	Induce callus formation	"	"
7.	Promote epinastic responses	"	"
8.	Control dwarfism	No	Yes
9.	Promote seed germination and the breaking of dormancy	"	"
10.	Promote bolting and flowering in non-vernalized biennials and in long-day plants	"	"

(*Adapted from Galston and Purves, 1960*)

Accumulated evidences indicate that auxins and gibberellins act both independently and together, depending upon the type of plant and the conditions under which the plant grows. The fact whether the auxins and the gibberellins interact or not is not conclusively proved.

CYTOKININS (= KININS)

Discovery and Nomenclature

Auxins and gibberellins, besides inducing cell elongation, also do promote cell division under certain conditions. But this behaviour of them is an exception rather than a rule. However, there exist in plants many substances inducing cell division. For example, Van Overbeek *et al* (1941) found coconut milk as an active stimulant of cell division. Later, in 1955 Carlos Miller *et al* isolated a *"cell-division-stimulating factor*" from yeast DNA. It was named as kinetin because of its amazing power to stimulate cell division (cytokinesis) in the presence of an auxin. In subsequent years, many other compounds promoting cell division have been synthesized. Miller and his associates (1956) have grouped all such compounds including kinetin under a generic name kinin. D.S. Leetham (1963) of New Zealand proposed the term cytokinins for such substances. This term is the most acceptable one.

Fairley and Kilgour (1966), however, prefer to use the term **'phytokinins'** for such substances in order to distinguish them from the peptide hormones of animal gastrointestinal tract.

Definition

Skoog, Strong and Miller (1965) have defined cytokinins as chemicals which, regardless of their activities, promote cytokinesis (cell division) in cells of various plant organs.

Fox (1969) has defined cytokinins as chemicals composed of one hydrophilic adenine group of high specificity and one lipophilic group without specificity.

Isolation, Distribution and Biosynthesis

Although kinetin does not occur in nature but other kinins are found occurring widely, if not universally, in plants. The naturally-occurring kinins do not occur free in nature but are normally bound to a pentose sugar, ribose and sometimes to an inorganic phosphate, the ribonucleotide.

Fruits and endosperm are the richest sources of kinins. Coconut milk and corn endosperm possess the active substance. Substances with cytokinin activity have also been reported in tomato juice, in floral extracts of apples and pears and also in cambial tissues of certain plants. A *kinetin-like substance* is also present in peach embryo (Powell and Pratt, 1964) and sunflower root exudate (Kende, 1964).

Diphenylurea and many of its derivatives have cytokinin activity. Several synthetic cytokinins are available. These include benzimidazole and 6-benzyladenine. Adenine also has some cytokinin activity.

In angiosperms, the cytokinins are synthesized mostly in roots and probably originate at the root tips. Whether the shoots also synthesize cytokinins or else receive their cytokinin requirement from the roots is not certain. Contrary to what was first believed, the cytokinins are not breakdown

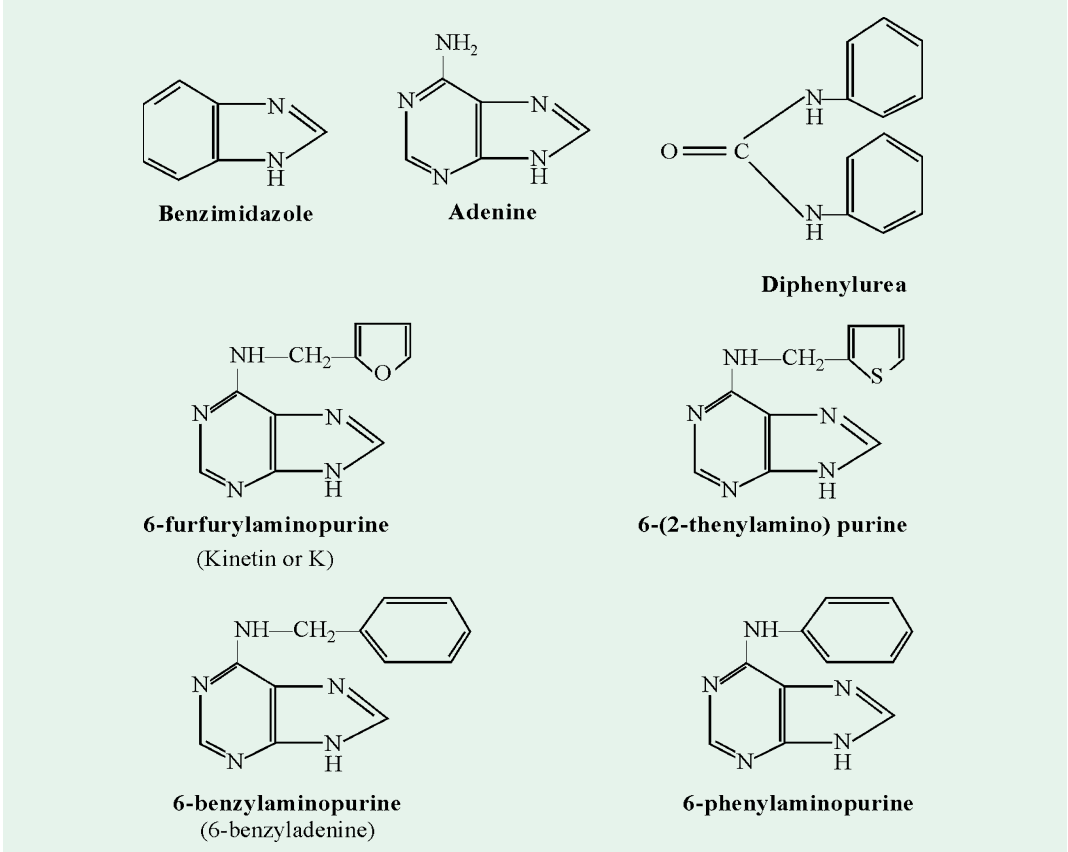


Fig. 32–18. Structural formulae of some kinins

Note that all cytokinins are structurally related to the purine derivative, adenine, *i.e.*, they have a side chains rich in carbon and hydrogen and attached to nitrogen protruding from the top of the adenine ring.

products of DNA. Rather, they are made *via* the mevalonate pathway, the same pathway used to make gibberellins. Like gibberellins, the cytokinins move nonpolarly in xylem, phloem, and parenchyma cells.

Chemistry

Chemically, kinetin ($C_{10}H_9ON_5$) is 6-furfurylaminopurine. It is formed from deoxyadenosine which is a degradation product of DNA (Hall and de Ropp, 1955). The structural formulae of kinetin and its 3 structural analogues are given in Fig. 32–18. All these substances promote cell division.

Apart from the above-mentioned kinins, Letham (1963) successfully isolated a cytokinin in pure crystalline form from immature maize seeds. It was named as zeatin (Fig. 32–19) and identified as 6-(4-hydroxy 3-methylbut-trans-2-enyl) aminopurine. *Zeatin is more powerful than any other known cytokinin probably because of the presence of a highly reactive allylic OH group in its side chain.* Zeatin riboside (Letham, 1966) and zeatin ribotide (Letham, 1966; Miller, 1967) also occur naturally in plants. A *cis*-ribosyl zeatin and a *ms*-ribosyl zeatin have also been extracted from plant tissue.

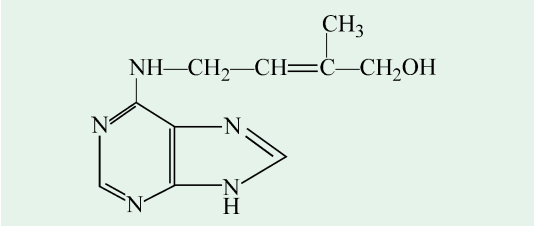


Fig. 32–19. Zeatin
[6-(4-hydroxy 3-methylbut-*trans*-2-enyl) aminopurine]

Fleissner and Borek (1962) have described compounds such as N^6-methylaminopurine and N^6, N^6-dimethylaminopurine. These are widespread in plants and have cell division stimulating property. Later, a cytokinin called N^6-purine was isolated from serine-transfer RNA of yeast cells by Hall and others in 1966.

Physiological Roles

Certain physiological processes which are influenced by the cytokinins *esp*., kinetin are given below :

1. Cell division. *Kinins are notable for their stimulatory effect on cell division*. Using tobacco pith cultures (Fig. 32–20), Skoog and Miller (1957) found that, in addition to IAA, kinetin is also needed for growth. The growth response is much more pronounced when both IAA and kinetin are used together in right ratio of concentrations. When either of them is used alone, a little response is produced which is due to the presence of small amounts of endogenous kinetin-like substances and IAA, already present in the tissues.

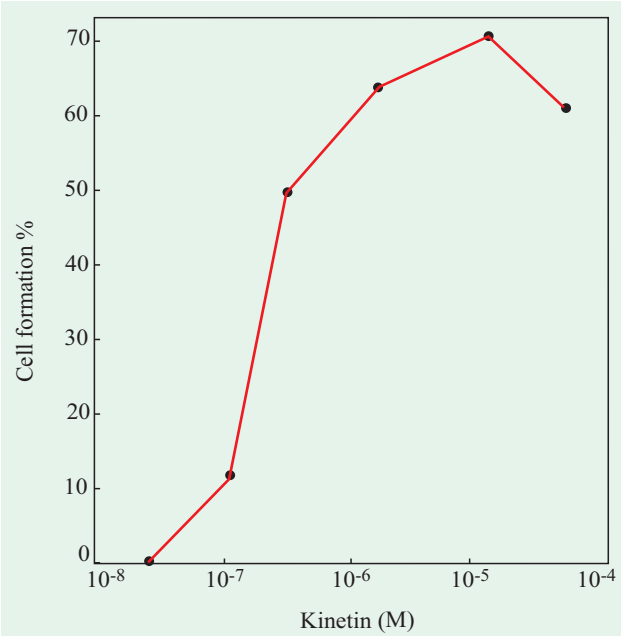


Fig. 32–20. Effect of kinetin in stimulation of cell division in tobacco pith cultures
[2mg/litre of IAA was also present in the medium]
(*After Skoog and Miller, 1957*

If a mixture of cytokinin and auxin is added to unspecialized cells (Fig. 32–21), they will begin to differentiate. A high cytokinin to auxin ratio will lead to the formation of shoots, buds and leaves while a low cytokinin to auxin ratio will

lead to root formation. The one treatment followed by the other provides a means of forming small plantlets. Such *in vitro* culture methods have become widely adopted for the rapid propagation of new plant varieties. The technique allows growers to produce very large numbers of plants, quite rapidly and in a small space. The alternative conventional propagation methods can take several years and cover a large area of land.

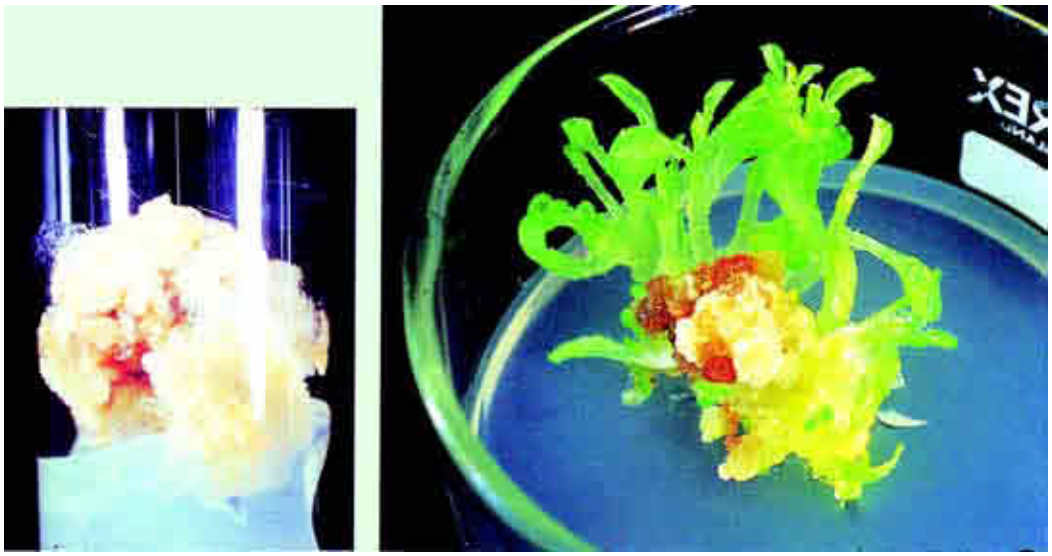


Fig. 32–21. Callus formation and production of plants from callus cells

(a) If a small piece of pith from a shoot is placed on agar in aseptic conditions, it will grow into a mass of unspecialized cells called callus.

(b) Plants can be grown from these cells.

The process of cell division completes in 3 steps, *viz*., DNA synthesis, mitosis and cytokinesis. Studying the specific influence of IAA and kinetin alone on any of these 3 steps, Patau, Das and Skoog (1957) found that IAA is involved in the first two steps of cell division (i.e., in DNA snythesis and mitosis) and that the last step (*i.e*., cytokinesis) is controlled by kinetin. It has been suggested that the adenine moiety of the kinetin molecule is essential for cell division.

2. Cell elongation. Besides auxins and gibberellins, kinetin also promotes cell elongation. Such promotion after kinetin treatment has been observed in tobacco pith cultures (Glasziou, 1957), tobacco roots (Arora et al, 1959) and bean leaf tissues (Powell and Griffith, 1960). Since cell elongation induced by kinetin has been well established, the kinetin should not be regarded as exclusively a cell division factor.

3. Root growth. Kinetin is capable of stimulating as well as inhibiting root development. Skoog and Miller (1957) found stimulatory effect of kinetin, when applied along with IAA, on root initiation and development in stem callus cultures. Similarly, kinetins also induced increase in dry weight and elongation of the roots of lupin seedlings (Fries, 1960).

4. Shoot growth. The callus tissue of tobacco can be kept in an undifferentiated state so long as the proper balance of IAA and kinetin is maintained. If, however, the amount of kinetin is increased, leafy shoots are initiated to develop. Bean seedlings, soaked in kinetin solution, also showed an increase in dry weight and a marked elongation of stem and petioles (Miller, 1956).

5. Organogenesis. Cytokinins can cause organogenesis (*i.e.*, the formation of organs) in a variety of tissue cultures. For instance, Skoog and Miller (1957) observed that tobacco pith callus can be made to develop either buds or roots by changing the relative concentrations of kinetins and auxins. High kinetin and low auxin contents result in the production of buds. In reverse

condition (high auxin and low kinetin), however, the roots appear on the pith.

The kinins also stimulate the production of buds in leaf segments of various plants such as *Saintpaulia ionantha, Bryophyllum sp and Begonia* sp.

In addition to the root and shoot differentiation, the cytokinins also bring about other morphogenetic responses. These are :

(a) maturation of proplastids into plastids

(*b*) differentiation of tracheids

(*c*) induction of parthenocarpy

(*d*) induction of flowering

6. Counteraction of apical dominance. As discussed earlier (refer page 763), the auxins emanating from the apical bud inhibit the growth of lateral buds (apical dominance). Wickson and Thimann (1958) studied the antagonistic effect of auxin and kinetin in apical dominance using pea stem sections in culture solutions. They found, as might be normally expected, that the growth of lateral buds is inhibited when the culture medium contained IAA and is uninhibited when the culture medium does not contain IAA. They further noted that addition of kinetin, along with IAA, stimulates the growth of lateral buds.

The above workers also conducted experiments with entire shoots, *i.e.*, with the apical bud intact. As long as the apical bud is present, the lateral buds do not develop but removal of the apical bud leads to the stimulation of growth of the lateral buds. If, however, the intact shoot is soaked in kinetin solution, the inhibition of lateral buds is checked to a great extent or, in other words, the lateral buds tend to develop, although less vigorously, as if the apex of the shoot has been cut off. The above findings point out towards the possibility of controlling apical dominance by maintaining a proper balance of concentrations between IAA and the endogenous kinetin-like substances.

Studies conducted in subsequent years by Sachs and Thimann (1964, 67) and Panigrahi and Audus (1966) also indicated that the cytokinins are strong promoters of lateral bud growth.

7. Breaking dormancy of seeds. Cytokinins are also effective in breaking seed dormancy in lettuce, tobacco, white clover and carpet grass. Thimann (1963) suggested that the site of cytokinin action in such cases is the cotyledon. Furthermore, the inhibitory effect of infrared light on germination of lettuce seeds is also alleviated by kinetin treatment.

The seeds of parasites such as Striga asiatica require the presence of host plant for germination. But when treated with kinetin, the seeds germinate even in the absence of their host.

8. Delay of senescence (= Richmond-Lang effect). The term *senescence* refers to the ageing of the leaves which is associated with the loss of chlorophyll and the breakdown of proteins. Richmond and Lang (1957) showed that the senescence in the detached leaves of Xanthium could be postponed for many days by kinetin treatment. This effect of kinetin in retarding senescence (or ageing) is known as *Richmond-Lang effect.* According to Mothes and Engelbracht (1961), the cytokinins have the ability to attract certain substances including auxins and to prevent the movement of leaf components out of the treated area. However, the mobilizing effect of cytokinin may actually induce senescence in others parts of the plant. Osborne (1962) suggested that the high protein content in kinetin-treated areas is probably due to enhanced protein synthesis than their breakdown. The protein synthesis, in its turn, is dependent on RNA synthesis, a process governed by kinetins. It may, however, be emphasized that the cytokinin-induced delay in leaf senescence occurs only in detached leaves; cytokinins have little or no effect on senescence in attached organs. Leaf senescence is also delayed by the formation of adventitious roots. As the roots are rich in cytokinins, the transport of these cytokinins from roots to leaves could account for the delayed senescence.

A correlation between the age of the leaf and the kinetins has been established. Mature leaves of tobacco respond more vigorously to kinetin treatment in delaying senescence than the young leaves.

Cytokinins are sometimes used commercially to maintain the greenness of excised plant parts, such as cut flowers. However, their use on edible crops such as broccoli is banned in some countries. This is possible because any compound like cytokinin that resembles a nucleic acid component is automatically a suspected carcinogen.

9. Role in abscission. Cytokinins can accelerate as well as retard the process of abscission in leaf petioles depending on the site of their application (Osborne and Moss, 1963). In explant petioles of *Coleus blumei*, Gupta and Kaushik (1969) reported accelerated abscission on kinetin application."

10. Effects on cotyledons. Cytokinins promote cellular division and expansion in cotyledons. Cellular expansion results from cytokinin-induced increases in wall plasticity that do not involve wall acidification. Cytokinins also increase the amount of sugars (especially glucose and fructose) in cells, which may account for the osmotic influx of water and the resulting expansion of cytokinin-treated cells in cotyledons.

OTHER NATURAL GROWTH HORMONES IN PLANTS

In addition to the 3 well-established categories of plant hormones described above, some other compounds with hormonal actions have been identified in plants which fall under 4 categories: ethylene, traumatic acid, calines and vitamins. Their account follows.

1. ETHYLENE

Discovery

During the 1800s, the city streets of Germany were illuminated by lamps that burned "illuminating gas". Soon after these lamps were installed, city residents made a curious observation: plants growing near the lamps had short thick stems and leaves falling from most of them. The mystery was solved in 1901 by a Soviet plant physiologist, Dimitry Neljubow who identified ethylene as the combustion product of "illuminating gas" that was responsible for defoliation and stunted growth of plants growing near the lamps. He also showed that only micro quantities are needed to bring about these effects, *i.e.*, only 0.06 ppm of ethylene.

ppm is part(s) per million;
1 ppm = 1 millilitre in 1,000 litres

Later in 1910, an annual report submitted to the Japanese Department of Agriculture recommended that *oranges be not stored with bananas*, because oranges released something that caused premature ripening of the bananas; this "something" was in 1934 identified by R. Gane as ethylene which is made by plants. Subsequent researches showed that ethylene has all the characteristics which warrant its inclusion under plant hormones, *i.e.*, it is made in one part of a plant and transported to another, where it induces a physiological response. Thus was discovered the *gaseous plant hormomes: ethylene*. Later, the presence of ethylene was shown in certain fungi (*Penicillium digitatum, Alternaria citri*) and in the leaves, flowers and fruits of many higher plants; its recognition as a natural plant hormone was confirmed by Pratt and Goeschl only in 1969.

Distribution and Biosynthesis

All parts of angiospermous plants produced ethylene but especially large amounts are released into the air by roots, the shoot apical meristem, nodes, senescing flowers and ripening fruits (for example, the dark flecks on a ripening banana peel are concentrated pockets of ethylene). Because most ethylene-induced effects result from ethylene in the air, *the effects of ethylene can be contagious:*

ethylene made by one "bad" (*i.e.*, overripe) apple can "spoil" (*i.e.*, induce rapid ripening of) an entire bushel of apples. Ethylene also occurs in minute quantities in city gas and in tail gases of blast furnaces. It is a volatile gas of peculiar odour and is sparingly soluble in water but a little more in ethanol and ether. It is inflammable and hence the ignition of a mixture of ethylene with air leads to explosion.

$$H_2C = CH_2$$

Ethylene, ETH

Ethylene is made from methionine, a S-containing amino acid (Fig. 32-22). Its synthesis, which requires O_2, is inhibited by CO_2 When plants are placed in pure CO_2 (or O_2-free air), ethylene synthesis decreases dramatically.

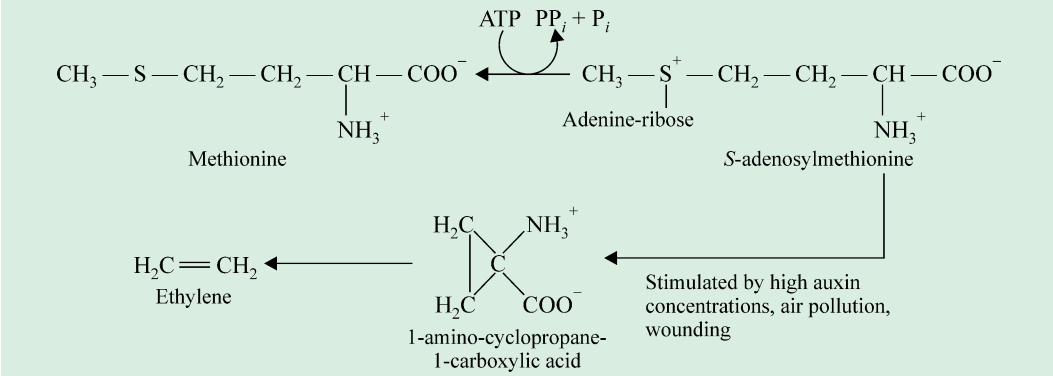


Fig. 32–22. Biosynthesis of ethylene from methionine

Note that a cyclic compound, 1-amino-cyclopropane-1-carboxylic acid is the immediate precursor of ethylene.

Physiological Roles

1. Stimulates fruit ripening. The ancient Chinese knew that fruits would ripen faster if placed in a room containing burning incense. The factor responsible for this hastened ripening was not heat, but ethylene released as the incense burned. The stimulation of fruit ripening by ethylene is a consequence of many ongoing processes such as:

(*a*) the breakdown of chlorophyll and synthesis of other pigments; for example, apples changing from green to red during ripening,

(*b*) fruit softening due to breakdown of cell walls by cellulase and pectinase, and

(*c*) conversion of starches and acids to sugars.

And ethylene stimulates each of these processes, leading ultimately to fruit ripening.

We often say that 'one rotten apple spoils the rest of the barrel' because pieces of fruit near to a rotten one start to go bad quickly. This is because a damaged on fungus-infected fruit starts to produce ethene. And in a closed barrel the concentration of ethene is high enough to quickly trigger the ripening process in neighbouring fruits making them more vulnerable to infection (Fig. 32–23).

Fig. 32–23. The oranges infected with the fungus *Penicillium digitatum*

The fungus itself produces large amounts of ethene, which accelerates the post-harvest maturation of oranges, making them more vulnerable to infection.

Some fruits (such as tomatoes and apples) show a conspicuous increase in respiration just before fruit ripening. This increase in respiration is called a **climacteric**, and fruits that display it are referred to as **climacteric**

fruits. The climacteric begins just after a huge increase (up to a 100-fold) in ethylene production. Thus, *the climacteric and fruit ripening are both triggered by ethylene.*

Fruit growers often take advantage of ethylene's capability of stimulating fruit ripening for making fruits available for sale out-of-season. For instance, many apples are plucked in September and October when they are green and immature. These are then stored in rooms containing air that has small amounts (1–3%) of O_2 large amounts (5–10%) of $CO_2$and no ethylene. As these conditions inhibit protein synthesis, the fruits can be stored without the fear of their being ripened. When these unripe fruits are needed for sale, producers expose them to normal air containing 1 ppm of ethylene, which is enough to induce climacteric and ripening of fruits. Thus the "fresh" apples one buys in March are the ones harvested in September/October of the previous year. This "ripening on demand" is also used in the case of tomatoes, lemons and oranges. It is for these reasons that, in common parlance, ethylene is known as '*ripening hormone*'.

However, some other fruits (such as grapes and cherries) cannot be ripened by ethylene. Such fruits are called nonclimacteric and are insensitive to ethylene. Table 31–3 lists the climacteric and nonclimacteric type of fruits.

2. Promotes flowering. Although ethylene inhibits flowering in most species, but induces it in a few plants including mangoes, pineapples and some ornamentals. The Filipino mango growers and the Peurto Rican pineapple growers, who knew this effect long ago, set bonfires near their crops. The fires produced ethylene which initiated flowering of their plants. Nowadays, the pineapple growers in Hawaii produce pineapple fruits round the year by spraying plants with ethepon. Ethepon splits under neutral and alkaline conditions to release ethylene.

Table 32-3. Climacteric and nonclimacteric fruits

*Climacteric**		*Nonclimacteric**
Apple	Persimon	Bell pepper
Avocado	Plum	Cherry
Banana	Tomato	Citrus
Contaloupe		Grape
Cherimoya		
Fig		Pineapple
Mango		Snap beam
Olive		Strawberry
Peach		Watermelon
Pear		

* Note that the term **'climacteric'** can be used either as a noun, as in "most fruits exhibit a climacteric during ripening" or as an adjective, as in "a climacteric rise in temperature". The term **'nonclimacteric'** however, is used only as an adjective.

3. Hastens leaf abscission. Abscission zone in leaves causes the increased production of ethylene which triggers the breakdown of middle lamella, thus leading to the initiation of abscission (Fig. 32–24). This effect is also utilized by horticulturists to minimize the harvesting period of such fruits as cherries, grapes and blueberries. These fruits are sprayed with ethepon to coordinate abscission, thereby allowing growers to harvest their crops in shorter periods of time.

4. Induces leaf epinasty. When a plant's roots are kept submerged in water for long periods, water fills the intercellular spaces. Since these spaces are the primary routes of gas exchange with the atmosphere, the submerged roots become waterlogged and anaerobic. The symptoms of waterlogging (*e.g.*, leaf chlorosis, shorter and thicker shoots and wilting) can also be induced by

Fig. 32–24. Photograph of a plant showing leaf fall

Leaf abscission is strongly influenced by hormones, esp., ethylene.

placing roots in O_2-free air. Because O_2 is required to produce ethylene, its synthesis is greatly checked in roots of waterlogged plants. The small amount of ethylene that is made in these roots is trapped, where it accumulates and eventually stimulates the activity of enzymes like cellulase and pectinase. These enzymes break down the cell walls. This leads to the formation of many intercellular spaces, characteristic of hydrophytes. Concomittantly, the ethylene precursors in the shoot are also converted to ethylene, which causes parenchyma cells on the upper side of the petiole to expand and point the leaf down, a physiologic response called epinasty.

5. Controls stem elongation. Mechanical disturbances such as shaking decrease stem elongation. This effect, which is called **thigmomorphogenesis**, is mediated by ethylene. Mechanical disturbances enhance ethylene production several times. Ethylene so produced causes cells to arrange their cellulose microfibrils *longitudinally.* This lengthwise reinforcement inhibits cellular elongation, causing cells to elongate radially; this leads to the formation of short and thick stems. This effect is opposite to that of auxin, which causes cells to orient their microfibrils *transversely*, thereby accounting for cellular elongation.

6. Determines sex expression. Both ethylene and gibberellins determine the sex of flowers on monoecious plants, i.e., plants having male and female flowers on the same individual. As an instance, cucumber (*Cucumis sativus*) buds, on treatment with ethylene, become carpellate (♀) flowers, whereas those treated with gibberellins become staminate (♂) flowers. Correspondingly, buds that ultimately become flowers produce more ethylene than do buds that become male flowers.

Ethylene versus Auxin

IAA stimulates ethylene production, thereby linking the responses of these two hormones. But

ethylene does not account for all the effects induced by applying IAA. For example, IAA's stimulation of cellular elongation and the formation of lateral roots occur independently of ethylene. Similarly, leaf epinasty, decreased elongation of roots and shoots, and determination of sex are responses to ethylene application rather than IAA.

2. TRAUMATIC ACID

Most plants form callus when they are injured. This observation of Gottlieb Haberlandt (1913) led to postulate that the injured cells secrete a '**wound hormone**' which induces the neighbouring uninjured cells to become meristematic and divide till the injured part is healed up. Later, the active substance was isolated and identified as traumatic acid by Bonner and English in 1938. Traumatic acid is an open-chain dicarboxylic acid with a single double bond.

$$COOH—CH = CH—(CH_2)_8—COOH$$

Traumatic acid or **Traumatin** or **Nekrohormone**

Traumatic acid is specifically effective in inducing cell division in bean pods. It, however, seems to be of confined activity as it has failed to develop meristematic activity in a number of plant tissues including tobacco pith. Davies (1949), henceforth, regards this as a specific wound hormone for bean pods.

3. CALINES

Some indirect evidences accumulated during the past few decades have established beyond doubt the presence of some hormones which are needed for initiating the action of auxins on roots, stems and leaves. For example, the presence of leaves and buds is necessary for a better rooting caused by auxin application. These hormones have been collectively called as **calines** or **formative hormones**. The various postulated calines are :

A. *Rhizocaline*— It is a root forming hormone. It is produced by the leaves and transported down the stem.

B. *Caulocaline*—It is a stem forming hormone. It is synthesized mostly in the roots and translocated up in the stem.

C. *Phyllocaline*— It stimulates the development of mesophyll in the leaves. It is synthesized probably in the cotyledons from where it moves to its site of action.

None of these calines has been isolated from plants as yet.

4. VITAMINS

Their account follows in the succeeding two chapters.

GROWTH INHIBITORS

Introduction. While all the hormones discussed so far possess the power of growth promotion, the plants also do possess a few substances which inhibit growth. Although auxins when present in higher concentrations also inhibit growth, the growth inhibitors do so *irrespective of their concentration.* They retard such processes as seed germination, root and stem elongation and bud opening. As a matter of fact, the growth inhibitors act as chemical check upon plants preventing the seeds from germinating and the buds from opening under unfavourable conditions. For example, the sprouting of lateral buds during cold weather is inhibited by the presence of growh inhibitors produced within the plant body during the previous growing season.

Characteristics. The growth inhibitors are, in general, characterized by some common features (Addicott and Lyon, 1969 ; Milborrow, 1969). These are :

1. The amount of growth inhibitors decreases during active growth period of plants and

increases during the period of growth suppression.

2. They counteract the activities of growth promoters.
3. They inhibit the growth of various isolated organs and tissues.
4. They do not evoke the strong stimulatory effects specific for auxins, gibberellins and kinins.
5. They are actively synthesized in green tissues and are found associated with auxins, gibberellins and kinins. However, they alone are accumulated in the absence of these growth substances in senescent and resting organs.
6. They are accumulated in woody plants during the period of dormancy.

Types. There are two types of growth inhibitors :

A. *Phenolic inhibitors*— These are better known and are widespread in plant organs. These are derivatives of either benzoic acid (*e.g.*, *p*-hydroxybenzoic, salicylic and gallic acids) or cinnamic acid (e.g., p-coumaric acid) and as well as coumarin.

B. *Abscisic acid and other substances*— Besides abscisic acid, these also include pyridine derivatives (*e.g.*, fusarinic and picolinic acids), quinones and flavonoids (*e.g.*, naringenin, phloridzin).

The structure of some common growth inhibitors is given in Fig. 32–25.

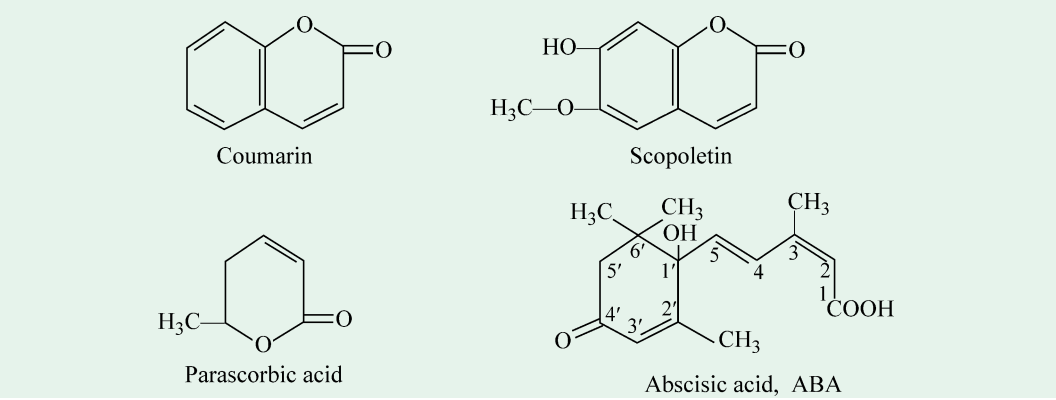


Fig. 32–25. Some common growth inhibitors

ABSCISIC ACID or ABSCISIN II or DORMIN

Discovery

Most of the effects first discovered for plant hormones were stimulatory. For example, IAA stimulates cellular elongation and cytokinins stimulate cell division. But near the end of the decade 1940s, Torsten Hemberg of Sweden reported that dormant buds of ash and potato contained inhibitors (rather than stimulators) that blocked the effects of IAA. When the buds germinated, the amount of these inhibitors decreased. Later, Eagles and Wareing (1963) isolated an inhibitor from the birch (Betula pubescens) leaves held under short day conditions. When this substance was reapplied to the leaves of birch seedlings, apical growth was completely arrested. As this substance induced dormancy, they named it as **dormin**. Later in 1965, Ohkuma et al isolated an inhibitor from cotton fruits and named it **abscisin II**. The same year, Cornforth and his associates isolated a growth inhibitor from sycamore and pointed out that both dormin and abscisin II are identical. Abscisin II is peculiar in that it is effective in much lower concentration than phenolic inhibitors and is accumulated under short day conditions. This compound was later named as abscisic acid (abbreviated as ABA)— an

unfortunate name, because subsequent research has shown that *ethylene rather than abscisic acid controls abscission*.

Distribution and Biosynthesis

Abscisic acid occurs in angiosperms and gymnosperms but apparently not in liverworts. ABA in plants is made from carotenoids. Once synthesized, ABA moves throughout a plant in xylem, phloem and parenchyma. *Like gibberellins and cytokinins, abscisic acid moves nonpolarly. There are no synthetic abscisic acids.*

Chemistry

Abscisic acid (Fig. 32–25) is a sesquiterpene with molecular formula $C_{15}H_{20}O_4$. Its molecule contains a corboxyl groups, 4 double bonds and an identical number of methyl groups.

Physiological Roles

1. Closure of stomata. It is a known fact that during drought, leaves synthesize large amounts of ABA which causes stomata to close. Thus, ABA acts as a messenger and enables plants to conserve water during drought. Because ABA-induced closure of stomata occurs within 1 to 2 minutes, this effect probably occurs independently of protein synthesis. As to its mechanism, ABA probably produces its effect by binding to proteins on the outer surface of the plasmalemma of guard cells. This renders the plasmalemma more positively-charged. thereby stimulating transport of ions (especially K^+) from guard cells to epidermal cells. The loss of these ions causes water to leave guard cells (via osmosis) which then collapse, thus closing the stomatal aperture.

2. Delays seed dormancy. In many species, applying ABA delays seed germination. Similarly, in many other plants, the amount of ABA in their seeds decreases, when seeds germinate. Thus, it may be inferred that ABA controls seed dormancy in some cases. However, this conclusion may not be generalized, since germination of many seeds occurs without any changes in the amount of abscisic acid.

3. Controls bud dormancy. Bud dormancy was previously thought to be controlled solely by ABA. But when leaves are treated with radioactive ABA, no radioactivity has been detected in buds. This suggests that, besides ABA, bud dormancy is probably also influenced by cytokinins and IAA-induced synthesis of ethylene.

4. Counteracts the effects of other hormones. ABA counteracts the stimulatory / inhibitory effects of other hormones. For example,

(*a*) ABA inhibits cell growth promoted by IAA.

(*b*) ABA inhibits amylase produced by seed treated with gibberellin.

(*c*) ABA promotes chlorosis that is inhibited by cytokinins.

This may be due to the fact that *ABA is a* Ca^{2+} *antagonist* and its inhibition of the stimulatory effects of IAA and cytokinin may be due to its interference with Ca^{2+} metabolism. Although ABA usually inhibits growth, it is not toxic to plants as are inhibitors of RNA / protein synthesis. ABA often decreases gene acctivity, but there are instances of ABA stimulating genes. For example, ABA stimulates the synthesis of mRNAs for storage proteins in developing wheat grains.

MORPHACTINS

Recently, a group of growth substances called morphactins (meaning morphologically active substances) has diverted the attention of physiologists on account of its regulatory effect on the growth and development of plants (Merck, 1970). *The role of morphactins is usually of inhibitory nature.*

Chemically, the morphactins are derivatives of fluorene-9-carboxylic (Fig. 32-26) acid and are absorbed through the seeds, roots, and leaves. Their action is systematic but slow and can be transported both acropetally and basipetally. Considering from phylogenetic viewpoint, the morphogenic action of morphactins starts with the complex brown algae, the mosses and ferns and is exhibited well in higher plant groups. *Unicellular plants and simple filaments are, however, not affected by morphactins.*

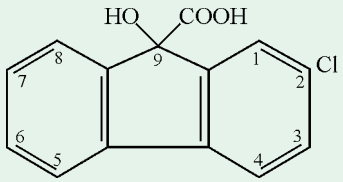


Fig. 32–26. Chlorofluorenol or morphactin

Although morphactins are much alike to abscisic acid in action, yet some of their effects are highly specific to them. Some such effects are :

1. inhibit sprouting of rosette plants
2. complete abolition of polarity in plants.
3. inhibition of mitosis in apical meristems.
4. stimulation of cell division in cambium of intact plants and cuttings.
5. reinforcement of the apical dominance in tap roots so that the formation of lateral roots is strongly inhibited.
6. abolition of geotropic and phototropic responses.
7. reduction of apical dominance in the main shoot so that the formation of branches is promoted, resulting in a broomlike growth of the plant.

OLIGOSACCHARINS AND OTHER PLANT HORMONES

Plant hormones are not only restricted to those mentioned above. For example, fragments of the cell wall called oligosaccharins control plant growth, differentiation, reproduction and defense against disease and hence function as plant hormones. However, oligosaccharins typically differ from other plant hormones because they elicit specific effects:

1. Different oligosaccharins can induce cultured cells to form undifferentiated callus roots, shoots, or flowers in a variety of plants.
2. Oligosaccharins that inhibit flowering and promote vegetative growth in one species have the same effect on other species.
3. Oligosaccharins induce effects by impressively small amounts: about 100 to 1,000 times less oligosaccharin than IAA or cytokinin to induce a response.

Recent researches have revealed the presence of a variety of other compounds that act as hormones in different groups of plants. For example:

(*a*) Yams contain **batasins** which induce dormancy of bulbils (vegetative reproductive structures) that form from lateral bulbs.

(*b*) **Brassosteroid**s are plant hormones present in tea, bean and rice plants that stimulate the growth of stems. Their chemical structure resembles that of ecdysone, an insect moulting hormone.

HORMONAL INTERACTIONS

It is very rarely, if ever, that the plant hormones work alone; rather *plant growth and development usually result from interactions of plant hormones* (Fig. 32–27).

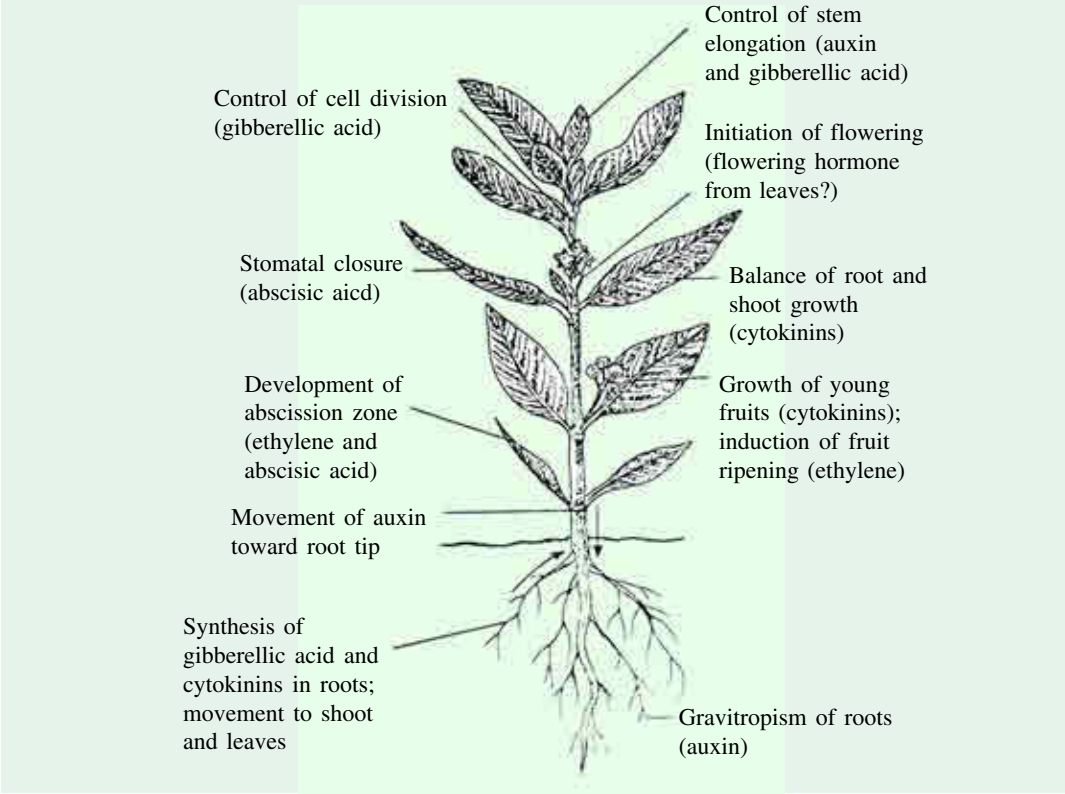


Fig. 32-27. Hormonal interactions in a plant

Note that numerous hormonal interactions influence plant growth and development.

An intriguing question is what controls the amounts of hormones and thereby their ratios and the resulting interactions? The amount of hormones is controlled in two ways:

(*a*) **Regulation of the rate of synthesis.** Many factors influence the rate of hormone production. For example, daylength can stimulate the synthesis of IAA, and the cold temperatures trigger the synthesis of gibberellins.

(*b*) **Regulation of the rate of breakdown or inactivation.** Inactivation of a hormone usually takes place either by its oxidation or by its conjugation with other compound.

PLANT HORMONES VERSUS ANIMAL HORMONES

Since plant hormoes were discovered later than animal hormones, early research was based on the assumption that plant and animal hormones had basically similar physiological effects. However, there are important differences in plant and animal hormones (Table 32–4)

1. There is no evidence to indicate that the fundamental actions of plant and animal hormones are the same.
2. Plant hormones are not made in tissues specialized for hormone production, whereas animal hormones are produced in specialized organs, mostly in endocrine glands.
3. Plant hormones do not have definite target areas, whereas animal hormones do act at target .areas.
4. Animal hormones are usually very specific in their actions, while plant hormones seldom have such specific effects.

Table 32–4. Comparison of animal and plant hormones

Characteristic	*Animal hormones*	*Plant hormones*
Site of production	Specific endocrine glands specialized for homone production.	Produced by actively metabolizing tissues that have other functions.
Target tissues	Each hormone acts on a specific target tissue or organ.	Each hormone acts on a variety of tissues.
Number of hormones	Many, each with a specific function.	Relatively few, each with a variety of functions.
Primary function	Affect homeostasis and are regulatory in action; effects are reversible.	Affect growth and development; effects are permanent.

Despite these differences, botanists have traditionally suggested that plant hormones function like animal hormones; that is, the response elicited by a plant hormone is determined by the concentration of the hormone. Certain responses *can* be obtained by applying hormones to various parts of the plants, but this does not necessarily mean that such responses are naturally controlled by the hormone. As a result, some botanists now question the propriety of plant hormones being called as hormones, in a sense as applied to animal hormones. These botanists, therefore, refer the plant hormones as **plant growth regulators** and suggest that plant hormones are intergrating agents that are necessory for, but do not *control*, the response. For example, cytokinins regulate the conversion of buds from a dormant to nondormant state, but do not control the subsequent growth of the bud. Thus, cytokinins are necessary for bud growth , but do not control it. According to this perspective, "the response elicited by a hormone is determined not by the amount of hormone, but rather by the sensitivity of the tissue to the hormone" (Moore, Clark and Stern, 1995).

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PROBLEMS

1. Describe two general chemical features that distinguish plant homones from animal hormones.
2. Name two plant hormones that are antagonistic in their effects.
3. The primary effect of gibberelllins is on :
   (*a*) mitosis (*b*) meiosis (*c*) cell enlargement
   (*d*) flowering (*e*) root growth