INFLUENCES OF AUXINS AND AUXIN SYNERGISTS ON ADVENTITIOUS ROOT PRIMORDIUM INITIATON AND DEVELOPMENT

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ABSTRACT

Interpretation of the actions of auxins and auxin synergists on adventitious root primordium initiation and development suggests that cellular dedifferentiation that leads to primordium initiation requires one or more enzymatically synthesized auxin-phenolic conjugates. The "predisposition" of cells in easy-to-root tissues to initiate root primordia apparently resides partly in the availability of active enzymes and substrates necessary for synthesis of the conjugates. On the other hand, difficult-to-root tissues lack necessary active enzymes or substrates, or both.

INTRODUCTION

The present review briefly appraises current knowledge concerning the influences of auxins and their synergists on adventitious root primordium initiation.[1] I have included some information not directly concerned with adventitious root primordium initiation, and speculated freely, in order to demonstrate that existing evidence may explain why auxins alone do not promote primordium initiation in cuttings of all species.

AUXINS

Auxins are the only applied hormones that consistently enhance root primordium development, at least in naturally responsive (i.e., easy-to-root) tissues. However, it remains unclear whether applied natural or synthetic auxins alone can induce primordium initiation in any tissue.

Root primordium initiation begins with dedifferentiation of cells. These dedifferentiated cells attain the meristematic state and become the primordium initials. Primordium

[1] "Primordium initiation" and "primordium development" (Haissig, 1974) and "dedifferentiation" and "initial" (Esau, 1960) have been defined elsewhere. In the present paper, the terms "easy-to-root" and "difficult-to-root" refer to the facility with which propagules initiate induced adventitious root primordia (Haissig, 1974). The terms are not used to distinguish between propagules with and without preformed root primordia.

development occurs through division of the initial cells, and of cells adjacent to the primordium. Adjacent cells may undergo division (most often preceded by dedifferentiation) some time after the initial zone increases in cell number through division of the original dedifferentiated cells and their daughter cells (Haissig, 1974).

Until recently, little consideration had been given to the possible separate influences of auxins on primordium initiation and development. It presently appears that auxins, such as IAA[2], enhance primordium development, but that auxins alone do not induce primordium initiation. For example, Haissig (1970, 1972) found that applied IAA did not induce cellular dedifferentiation that establishes the primordium initials. Rather, the earliest cell divisions during primordium development required endogenous or applied auxin. Auxin deficiency reversed early primordium development because primordium initials differentiated as parenchyma. Shibaoka (1971) concluded that substances other than auxins (such as PCIB) would induce primordium initiation. However, Shibaoka's data may also be interpreted to indicate that, in the presence of endogenous auxin, PCIB did not mimic the effect of applied IAA but the effect of a factor (possibly an auxin synergist) whose coaction may support IAA-induced RNA synthesis (Haissig, 1971) and other processes in primordium initiation and development.

Clearly, IAA alone induces primordium initiation only in predisposed cells, and IAA alone does not cause the necessary predisposition (Haissig, 1970). Therein lies the failure of IAA and other auxins to induce primordium initiation in naturally difficult-to-root cuttings. Voluminous evidence suggests that, prior to primordium initiation, the cells involved in dedifferentiation attain a predisposition to respond to endogenous or applied auxins. Somehow the predisposition manifests itself during the "lag phase" before the maximum root primordium initiating effect of applied auxins can be attained (Fellenberg, 1965, 1966; Haissig, 1970, 1971; Kaminek, 1967; Mitsohashi *et al.,* 1969, Shibaoka, 1971).

Difficult-to-root cuttings apparently lose the predisposition during development, or the predisposition does not manifest itself during the period of propagation. In a following section, I shall explore the possibility that the ill-defined predisposition constitutes a cell's ability to conduct enzymatically catalyzed synthesis of auxin-phenolic conjugates that lead to dedifferentiation and root primordium initiation.

AUXIN SYNERGISTS

The induction of adventitious root primordium initiation, or at least the most abundant initiation, apparently requires the presence of auxin synergists (also called rooting cofactors), in addition to auxin (Ashiru and Carlson, 1968; Bouillenne and Bouillene-Walrand, 1947, 1955; Bouillenne and Went, 1933; Challenger *et al.,* 1965; Cooper, 1935, 1936, 1938; Grace, 1945; Hess, 1961, 1962, 1963, 1964; Hyun and Hong, 1968; Jackson and Harney, 1970; Kawase, 1964, 1970; Libbert, 1956; Ruge, 1957, 1960; Stoltz and Hess, 1966; Went, 1935, 1938; Zimmerman, 1963, and references therein). Immature or embryonic leaves apparently synthesize the synergists, which, like IAA, undergo basipetal transport in cuttings. Somehow the synergists allow or enhance

[2] Abbreviations: IAA—indole-3-acetic acid; IBA—indole-3-butyric acid; NAA—naphthaleneacetic acid; 2,4-D—2,4-dichlorophenoxyacetic acid; 2,4,6-T—2,4,6-trichlorophenoxyacetic acid; PCIB—p-chlorophenoxyisobutyric acid; PHB—p-hydroxybenzoic acid; OHB—o-hydroxybenzoic acid; PPO—polyphenol oxidase (E.C. 1.10.3.1).

auxin-induced root primordium initiation and development. Thus, the type or amount of synergist seems to partially determine whether cuttings initiate root primordia easily, with difficulty, or not at all.

The chemical identities of endogenous synergists remain unclear, but some exhibit the characteristics of phenolics. Various modes of action have been postulated, but none has been unequivocally demonstrated as the prime mode by which the synergists allow or enhance root primordium initiation.

Inhibition of IAA-Oxidase

Plants contain peroxidases (E.C. 1.11.1.7) (IAA-oxidases) that oxidize IAA and supposedly destroy its physiological activity. Monophenols act as cofactors that enhance, whereas polyphenols inhibit, IAA oxidation (Hare, 1964). Like polyphenols, IAA analogs such as indole may also spare IAA from oxidation. A general hypothesis suggests that the regulation of IAA-oxidase activity by phenolics and indole controls endogenous auxin levels and plant growth (Galston, 1967). Thus, phenolics or IAA analogs that inhibit IAA-oxidase should theoretically synergize IAA-induced growth phenomena. Understandably, the possibility has been explored that the modulation of IAA oxidation by IAA-oxidase also determines whether or not, or to what degree, applied or endogenous IAA stimulates root primordium initiation and development (Basu *et al.,* 1969; Gorter, 1958; Van Raalte, 1954).

Present evidence tends to refute the IAA-oxidation growth control theory both generally and in the specific case of root primordium initiation. Chlorogenic acid, a typical polyphenol that should inhibit IAA oxidation, has been shown to reduce the curvature of *Avena* coleoptiles in the presence of high levels of IAA (Tomaszewski, 1964). The actions of the monophenol PHB on the curvature of *Avena* coleoptiles also poses an anomoly. Vieitez and coworkers (Vieitez *et al.,* 1966) observed that PHB at low concentration acted additively with IAA in promoting *Avena* curvature, but PHB should theoretically stimulate IAA oxidation, and inhibit coleoptile curvature. Pilet (1966) also observed that PHB at low concentrations stimulated *Avena* curvature, although PHB only enhanced IAA oxidation at higher concentrations. Tomaszewski and Thimann (1966) found that vanillic acid treatment increased decarboxylation of IAA (increased IAA-oxidase activity) in pea epicotyls but slightly promoted cell elongation. Diethyldithiocarbamate treatment reduced decarboxylation of IAA but did not synergize IAA-induced cell elongation, whereas ethylenediaminetetraacetic acid both reduced decarboxylation of IAA and increased cell elongation. Thus, decarboxylation of IAA, which should theoretically reduce cell elongation, failed to do so in a consistent manner.

As regards root primordium initiation, Basu and coworkers (Basu *et al.,* 1969) have shown synergistic rooting responses between the following: PHB-IAA or NAA, OHB-IAA, NAA, or IBA; gallic acid-NAA, and tannic acid-NAA or IBA. The amount of synergism declined in the series mono-, tri-, poly-hydroxyphenol. However, according to theory, the monohydroxyphenols should have suppressed root primordium initiation and development. Gorter (1962) observed synergism in the rooting of cuttings between the pairs: 1-naphthol-IAA or NAA, 2-naphthol-IAA or NAA, and phenol-IAA. The forementioned are monophenols, and, therefore, supposed stimulators of IAA-oxidase activity. Van Raalte (1954) notes that phenylacetic acid synergistically stimulated rooting in the presence of IAA, although as theory predicts it enhanced IAA--oxidase activity.

Van Raalte (1954) suggested that synergistic rooting responses shown by IAA-indole

combinations arose because indole non-competitively inhibited IAA-oxidase (a strange situation because substrate analogs should function as competitive inhibitors). Gorter (1962) further explored the hypothesis that auxin analogs might spare the analagous auxin from oxidation, but the results nullified the hypothesis. Synergism in inducing rooting occurred between such dissimilar pairs as indole-2,4-D, and phenol-NAA.

The above-cited results lead to one of two general conclusions. First, phenolics and auxin analogs enhance growth and root primordium initiation and development by sparing auxins from destruction by a ubiquitous enzyme system that most probably shows little if any substrate specificity, or specificity for activators and inhibitors. Clearly, such an enzyme could not assume much physiological importance as a precise control mechanism for *in vivo* auxin levels. Second, and most likely, the observed synergism between auxins and phenolics or auxin analogs does not manifest itself through an auxin-sparing mechanism. Several investigators have, in fact, suggested that auxin synergists influence processes other than auxin oxidation (Basu *et al.,* 1969; Gorter, 1962; Pilet, 1966; Vieitez *et al.,* 1966).

Auxin Synthesis

Auxin synergists may enhance root primordium initiation and development because they stimulate auxin synthesis. Gordon and Paleg (1961) demonstrated that enzyme preparations from bean, sunflower, and oats catalyzed the formation of IAA from tryptophane in the presence of catechol or other o-dihydroxyphenols. Phenol partially substituted for catechol.

Thus, phenolics can participate in IAA synthesis, at least *in vitro*. However, the promotive action of auxin synergists in root primordium initiaton and development probably does not arise because of enhanced IAA synthesis. The amount of IAA generated by *in vivo* enzymic reactions could not compare with the massive dosages of auxins used to induce root primordium initiation and development. Therefore, auxin synthesis would not significantly elevate the endogenous auxin level, or not elevate it enough to account for the strong synergistic rooting responses observed after phenolic, followed by auxin, treatment (Basu *et al.,* 1969). Synthesis of IAA on account of added phenolics probably does not occur in physiologically substantial amounts even in non-auxin-treated cuttings because the auxin synergists alone commonly do not enhance root initiation and development (Basu *et al.,* 1969).

Freeing of Auxin

Went (1939) proposed a two stage induction of root primordium initiation and development. Hemi-auxin or auxin could induce the first stage, but only auxin could induce the second. Presumably the hemi-auxin would free auxin for action at the stage II site if the hemi-auxin fully occupied the stage I site under conditions of limiting auxin supply (Gorter, 1958, 1962). Veldstra (1953) entertained similar ideas, for example, that indole, like hemi-auxin, might partially satisfy the requirement for an auxin. Under such circumstances an indole-IAA treatment might evoke a synergistic rooting response (Gorter, 1958, 1962). Veldstra, in fact, stated that hemi-auxins might more properly be termed synergists.

Shibaoka (1971) has shown that PCIB or 2,4,6-T could replace the early influences of IAA in root primordium initiation and development, but not the latter effects. Both the action of these chlorinated phenols, and their biological role, markedly resemble Went's hemi-auxins (Veldstra, 1953). The chlorinated phenols also resemble other auxin

synergists that modify root primordium initiation and development (Basu *et al.,* 1969).

The above evidence suggests that all auxin synergists may enhance root primordium initiation and development by freeing IAA to act at sites that only IAA can satisfy. However, the auxin replacements could only yield a synergistic rooting response under conditions of limiting IAA (auxin), that is, in non-auxin-treated cuttings. On the contrary, evidence has already been cited to the effect that auxin synergists alone do not markedly enhance root primordium initiation or development.

Direct Action

Much attention has been directed to the oxidation of auxins applied to plant organs and tissues. The pathway, products, and mechanisms of IAA oxidation have been partially elucidated (Abramovich and Ahmed, 1961; Fox and Purves, 1968; Hinman *et al.,* 1961; Still, Fukuyama and Moyed, 1965; Still, Olivier and Moyed, 1965). In addition the induction of IAA-oxidase activity by IAA treatment has been investigated (Chandra *et al.,* 1971); Galston *et al.,* 1968).

Scant attention has been directed to the possibility that IAA undergoes oxidation before it acts as a root primordium-inducing substance although one group of investigators has supplied ample evidence to show that not IAA but its oxidation product 3-methyleneoxindole promotes elongation growth (Basu and Tuli, 1972a, 1972b, 1972c; Fukuyama and Moyed, 1964; Moyed and Tuli, 1968; Still, Fukuyama and Moyed, 1965; Still, Olivier, and Moyed, 1965; Tuli and Moyed, 1966, 1969). Furthermore, naturally occurring esters of IAA have frequently been reported (Zenk, 1961, 1964), and some demonstrate physiological activity (Bandursk, *et al.,* 1969; Shantz, 1966). Thus, IAA clearly exists in plants in forms other than the free acid, and forms other than the free acid may have biological activity. Plants also apparently have the ability to synthesize conjugates of synthetic auxins (Goren and Bukovac, 1973).

Little attention has been directed to the chemical modification of applied auxin synergists, yet ample biochemical evidence shows that plant cells contain an enzyme, or enzyme complex (PPO), that oxidizes monophenols to diphenols, and diphenols to quinones (Duckworth and Coleman, 1970; Harel *et al.,* 1964; Kertesz, 1952; Lerner *et al.,* 1950; Sizer, 1953; Tomaszewski, 1959, and references therein). Clearly, phenolics and IAA may not have synergized root primordium initiation or other growth processes in the same chemical form in which they were applied.

Free IAA levels decline in cuttings prior to root primordium initiation and development (Saito and Ogasawara, 1960). The decline may occur because IAA oxidation increases as the result of IAA-induced enhancement of peroxidase activity (Chandra *et al.,* 1971 and Haissig unpublished), or because of increased PPO activity. Briggs and Ray (1956) have demonstrated IAA oxidation coupled to the reduction of quinones that were generated by the PPO-catalyzed oxidation of catechol or pyrogallol. PPO might thus chemically transform both IAA and auxin synergists.

Leopold and Plummer (1961) reported the PPO catalyzed condensation of phenols (catechol, chlorogenic acid, or caffeic acid) and IAA. The authors did not test the conjugates for physiological activity, but Tomaszewski (1959) noted that pretreatment of *Avena* coleoptiles with PPO and chlorogenic acid, followed by IAA treatment, synergized curvature of coleoptiles better than did IAA and chlorogenic acid applied simultaneously to non-PPO-treated coleoptiles.

PPO oxidizes substrates with molecular oxygen, and wounding enhances PPO

activity, as evidenced by the browning of injured tissues. Root primordium initiation and development also require molecular oxygen (Turetskaya and Kof, 1965; Winkler, 1927; Zimmerman, 1930), and wounding of cuttings also enhances root primordium initiation and development (Howard, 1968; Soekarjo, 1965). Wounding, of course, occurs in all cuttings, and roots tend to originate near wounded, oxygen-rich regions, with high PPO activity.

Both wounding and IAA treatment enhance peroxidase activity. Peroxidase catalysed oxidations occur with hydrogen peroxide as a substrate. The influences of endogenous hydrogen peroxide on root primordium initiation and development remain unstudied, but applied hydrogen peroxide enhances rooting (Winkler, 1927).

Fadl and Hartmann (1967) isolated a root-inducing compound from easy- but not from difficult-to-root cuttings of pear. The compound contained phenolic and indole moieties, and appeared in cuttings only after IBA treatment. The phenolic moiety apparently originated in buds. The authors proposed that the root-inducing compound was formed by the condensation of IBA with a phenolic rooting cofactor (synergist). More recently, Girouard (1969) isolated and partially characterized a similar substance from juvenile (easily rooted) *Hedera helix,* and named it p-257. p-257 contained a phenolic moiety, and an unidentified moiety. I found that the U.V. absorption spectrum of the unidentified moiety (published by Girouard) almost perfectly matched published U.V. absorption spectra of 3-methylene-oxindole (Fukuyama and Moyed, 1964; Hinmann *et al.,* 1961), and spectra of 3-methyleneoxindole synthesized by accepted methods (Hinmann and Bauman, 1964a, 1964b) in my laboratory. As noted above, 3-methyleneoxindole is an oxidation product of IAA. Therefore, p-257 may comprise the condensation product of IAA and a phenolic. The phenolic moiety may be derived from caffeic acid, a hydrolysis product of chlorogenic acid present in the juvenile *Hedera* and identified by Girouard.

Some authors have suggested that auxin synergists, particularly phenolics, enhance root primordium initiation or other growth responses apart from effects on auxin levels (Basu *et al.,* 1969; Pilet, 1966; Vieitez *et al.,* 1966). Indeed, the literature suggests that at least some auxin synergists, under appropriate conditions, modify root primordium initiation as directly as does IAA, because with IAA they form the molecule(s) that induce primordium initiation.

It appears that PPO, and possibly peroxidases and other enzymes (Moyed and Tuli, 1968) synthesize the moleculer "keys" that unlock the biochemical apparatus of dedifferentiation concommitant root primordium initiation. The "keys" apparently consist of two parts, an auxin moiety supplied by an indole—or directly from tryptophane (cf. Gorden and Paleg, 1961; Ruge, 1957)—and a phenolic moiety supplied by an "auxin synergist." Full specificity for "locks" (active sites) apparently exists only in the auxin-phenolic conjugate, whereas IAA alone is only partially specific, and the phenolic non-specific unless bonded to the IAA moiety.

The auxin-phenolic conjugates that may induce primordium initiation resemble the rhizocaline complex of Bouillenne and Went (1933), as defined by Bouillenne and Bouillenne-Walrand (1955): 1) "A specific factor, circulating from the leaves and characterized chemically by ortho-diphenol groups." 2) "A non-specific factor, which is also translocated and is auxin itself, biologically circulating at low concentration.", and 3) "An enzyme factor, which is also translocated in particular cells or tissues (pericycle-phloem-cambium). The enzyme is oxydasic (probably of the poly-phenol-oxidase type).

The reaction between the ortho-diphenol group and auxin is only going on where the enzyme is present and gives rise to the complex Rhizocaline . . .". The authors, however, apparently did not conceive of rhizocaline as a molecular entity. They illustrated a scheme of action in which IAA and o-diphenol/o-quinone functioned together but as separate chemical entities.

Libbert (1956) also proposed that rhizocaline was a complex and not a single substance. The complex consisted of two mobile components and an immobile cellular factor. IAA and an undefined Factor-X constituted the mobile components, and physiological age comprised the cellular factor. Libbert suggested that Factor-X (produced in buds and basipetally transported) reacted with IAA by mass action to form dissociable IAA-X. IAA-X elicited a linear root primordium-initiating response; IAA alone elicited a logarithmic response. Thus, Libbert theorized that IAA-X and IAA both stimulated root primordium initiation, but acted at different stages.

Contrary to both the Bouillenne and Libbert hypotheses, I suggest that rhizocaline comprises one or more chemically identifiable molecules composed of covalently bonded indolic and phenolic moieties. However, as those authors suggested, formation of the auxin-phenolic conjugates requires a complex that consists of one or more enzymes, the appropriate substrates, and, possibly, enzyme activators (discussed below). The auxin-phenolic conjugates, unlike auxin or auxin synergists alone, induce adventitious root primordium initiation (*cf.* Fadl and Hartmann, 1967).

Only further investigations will determine what roles, it any, peroxidases (including IAA-oxidase), PPO, and other enzymes (Moyed and Tuli, 1968; Moyed and Williamson, 1967) involved in the metabolism of auxin and phenolics play in production of the conjugates. The available evidence infers too many possible biosynthetic mechanisms and, therefore, too many potential chemical structures for the conjugates to allow other than speculation. The literature indicates, however, that the auxin-phenolic conjugates are unstable at physiological pH (Leopold and Plummer, 1961) and in acid (Girouard, 1969), which would both characterize a possible auxin-phenolic ester. However, other structures seem equally probable (Fig. 1).

Further investigation may also determine how non-indole auxins (such as NAA) and non-phenolic synergists (such as indole) stimulate root primordium initiation. At present, it can only be theorized that non-indole auxins and non-phenolic synergists closely enough resemble indole auxins or endogenous phenolic synergists to form part of the active conjugate, that they influence activity of enzymes that participate in the formation of physiologically active conjugates (Moyed and Williamson, 1967), or that they resemble physiologically active conjugates, and partially mimic their action. Indole, for example, sometimes synergistically increases root primordium initiation and development. It also resembles IAA, and condenses spontaneously with quinones (Bu'lock and Harley-Mason, 1951). Therefore, the indole-phenolic conjugate may mimic action of an IAA-phenolic conjugate. The non-indole auxin, NAA, may regulate IAA metabolism by preventing the conversion of 3-methyleneoxindole to 3-methyloxindole (Tuli and Moyed, 1969). Thus, NAA may increase root primordium initiation by enhancing formation of physiologically active IAA-phenolic conjugates, or NAA may form a phenolic conjugate (Goren and Bukovac, 1973).

The lack of response of difficult-to-root cuttings to auxin treatment may finally be explained, if further research confirms the existence of auxin-phenolic conjugates that

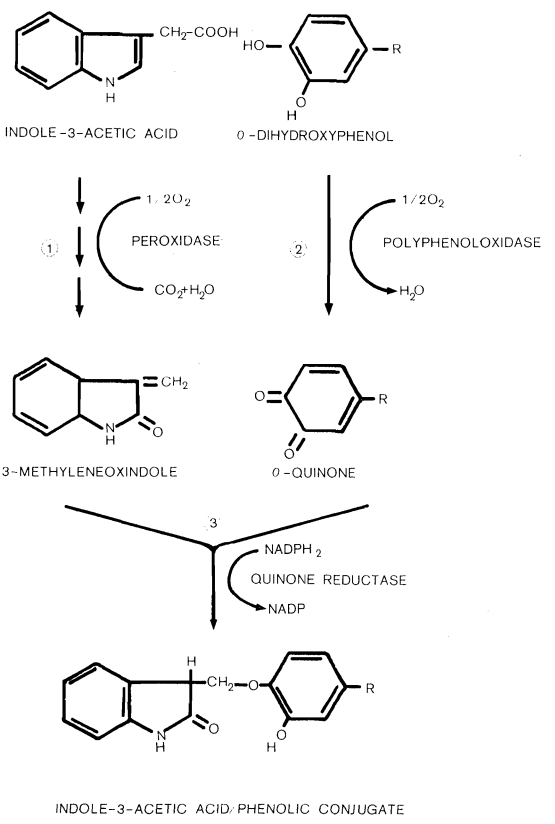

FIG. 1—Hypothetical biosynthesis of an ether conjugate of indole-3-acetic acid and an 0-dihydroxyphenol such as caffeic acid. (1) Peroxidase oxidizes indole-3-acetic acid to 3-methyleneoxindole (Hinman, Bauman, and Lang, 1961) with spontaneous or enzymatic (Basu and Tuli, 1972b) dehydration of intermediate 3-hydroxymethyl-oxindole (not shown). (2) Polyphenoloxidase oxidizes 0-diphenol to the corresponding quinone. (3) Ether synthesis via action of 3-methyleneoxindole reductase (Basu and Tuli, 1972b) or quinone reductase (Moyed and Tuli, 1968). Proposed reaction (3) has not been demonstrated. Monophenols could substitute for 0-dihydroxyphenols in (2) because polyphenoloxidase oxidizes monophenols to 0-dihydroxyphenols. See text for additional references to reactions (1) and (2).

induce root primordium initiation. Past research indicates that endogenous auxin-inhibitor relationships do not satisfactorily or generally account for the susceptibility or predisposition of cells to produce or not to produce root primordia. No universally prevalent relation exists between endogenous auxin levels and root primordium initiating ability of cuttings. Endogenous inhibitors, and their seasonal fluctuations, may lower and, in some instances, prevent, root primordium initiation (Hyun, 1967). However, no convincing evidence demonstrates that endogeonous inhibitors generally control the predisposition of cells to form root primordia.

Rather, the predisposition of cells to initiate root primordia apparently resides in the presence of certain oxidases, certain phenolics, and IAA. The predisposition of cells

to initiate roots becomes physiologically manifest only under circumstances of availability of substrate phenolic and IAA to the necessary activated enzymes.

In these terms, lack of root primordium initiation in response to applied or basipetally transported endogenous auxin may result from: 1) Lack of necessary enzymes to synthesize the root-inducing auxin-phenol conjugates, 2) Lack of enzyme activators, 3) Presence of enzyme inhibitors, 4) Lack of substrate phenolics, or 5) Physical separation of enzymes and reactants on account of cellular compartmentalization.

The most perplexing phenomena regarding woody plants concern the increasing loss of root primordium-initiating ability with age of both the parent plant and of the cutting. Ageing and differentiation of secondary tissues somehow slowly reduce the predisposition of cells within a cutting to initiate root primordia. In terms of the above hypothesis, age affects and the effects of secondary tissue formation bear study from the standpoint of phenol metabolism. Girouard (1969) showed that difficult-to-root mature *Hedera helix* shoots have lower free phenolic levels than do easy-to-root young shoots. Changed phenol metabolism associated with ageing or enhanced rates or amounts of lignification might drastically impair the predisposition of cells to initiate root primordia, if it reduces the synthesis of auxin-phenolic conjugates by limiting the supply of suitable phenolics. Interestingly enough, kinetin treatment of cuttings not only impairs root primordium initiation, but also modifies synthesis of lignin precursors from glucose (Kaminek, 1968).

Variations in phenol metabolism during development might also signal qualitative variations in enzyme systems. Loss of specific species of enzymes (isoenzymes) that synthesize auxin-phenolic conjugates would preclude root primordium initiation or at least greatly reduce the predisposition of cells to initiate root primordia. Lack of specific species of enzymes could easily escape notice because their genera (such as polyphenol oxidases and peroxidases) always have representative species in plant cells, although certain species may exist only during limited periods of development. Thus, the total activity of a certain genus of enzyme may inaccurately reflect root primordium-initiating ability, and confound basically valid physiological experimentation.

The literature adequately demonstrates that diverse chemical and environmental treatments of cuttings inhibit and stimulate root primordium initiation and development. The effects of many treatments may also be interpreted as manifest on enzyme systems that may synthesize the auxin-phenolic conjugates. Precedents exist to support such theories, but further speculation awaits a more complete understanding of auxin-phenolic conjugates and their possible roles in root primordium initiation and other developmental processes.

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