Effect of Auxin and Ethylene on Elongation of Intact Primary Roots of Maize (*Zea mays* L.)

Kim, Soon Young* and Timothy J. Mulkey[1]

Department of Biology, Andong National University, Andong 760-749, Korea
[1]*Department of Life Sciences, Indiana State University, Terre Haute, IN 47809, USA*

We tested that the hypothesis that root elongation might be controlled by altering the level of ethylene in intact primary roots of maize (*Zea mays* L.). We measured root elongation in a short period using a computerized root auxanometer. Compounds which regulate ethylene production were applied to intact primary roots in different time periods. Root elongation was stimulated by the treatment with ethylene antagonists such as Co^{2+}, aminoethoxy-vinylglycine (AVG) and L-canaline. This result suggested that root elongation was closely related to ethylene level of intact primary roots. Furthermore, IAA- and 1-aminocyclopropane-1-carboxylic acid (ACC)-induced inhibition of root elongation was reversed by treatment with Co^{2+}. The application of ACC to roots which have been exposed to IAA and Co^{2+} have no significant effect on root elongation. However, the inhibition of root elongation by ACC in roots previously treated with IAA and AVG became manifest when the applied IAA concentrations were lower. These results were consistent with the hypothesis that the level of ethylene in intact roots functions to moderate root elongation, and suggested that auxin-induced inhibition of root elongation results from auxin induced promotion of ethylene production.

Keywords: *Zea mays* L., IAA, ethylene antagonists, intact primary root, elongation rate

There have been many attempts to elucidate auxin's effect on root growth since Thimann (1937) observed the dose-response relationships of auxin effects on root and stem elongation.

Indole-3-acetic acid (IAA) is the auxin in most plant species. Auxin is an essential factor in the control of root and stem elongation. Thimann (1937) suggested that the auxin effects on root elongation were similar to the effects observed on stem or coleoptile tissue. Since he was unable to observe significant promotion of root elongation by auxin, he suggested that root cells were more sensitive to auxin than stems or coleoptile cells. Subsequently it was shown that under certain conditions auxin stimulated cell elongation in intact roots at low concentrations (10^{-10} M) (Burstrom, 1969). This auxin effect was minor when compared to inhibition of elongation of roots by high auxin concentration (Evans, 1984).

Burstrom (1969) proposed a two-phase hypothesis to explain auxin effects on wheat root elongation. Naphtylacetic acid (NAA) stimulated the rate of root elongation in high concentrations ranging from 3×10^{-8} M to 3×10^{-7} M (first effect); however, auxin reduced the duration of the promotion of elongation (second effect). He suggested that auxin stimulated cell elongation in both roots and shoots. However, the difference between roots and shoots was the duration of the promotion of elongation by auxin. That is, the duration of promotion of elongation by auxin was shorter in roots than in shoots. In an expansion of this hypothesis, Evans (1984) proposed that roots have suboptimal concentrations of auxin in relation to the rate of cell elongation and supraoptimal concentrations of auxin in relation to the duration of the cell elongation phase of development.

An alternate hypothesis to explain auxin action on elongation of roots was posed by Chadwick and Burg (1967). They suggested that the inhibition of root elongation by auxin resulted from auxin-induced ethylene production. They observed elevated ethylene production within 15 to 30 min after roots were exposed auxin. The rate of elongation of roots decreased and the roots began to swell immediately after exposure to the ethylene gas. They concluded that auxin inhibited the growth of roots by inducing ethy-

lene formation.

Andreae *et al.* (1968) challenged Chadwick and Burg's hypothesis. They suggested that ethylene may mediate the inhibitory action of auxin on root elongation. The inhibition of root elongation by a high concentration of ethylene (100 ppm) is less than inhibition by low concentration of auxin. These data suggested that only a fraction of the inhibition of root growth by auxin could be explained by ethylene. And these data resulted in the opening of new avenues of research into the interaction of auxin and ethylene in the control of growth and development.

Whalen and Feldman (1988) reported inhibition of root elongation in corn within 20 min of ethylene treatment. But they observed a recovery to normal elongation rates within 20 min of removal of ethylene from the system. Thus, short term exposures to ethylene did not result in a permanent alteration of the elongating potential of the plant tissue.

One potential source for the interaction between auxin and ethylene has been discovered. Ethylene is synthesized from methionine through S-adenosyl-L-methionine (AdoMet) and ACC. This pathway has been studied by use of inhibitors, precursors, and labeled precursors (Yang and Hoffman, 1984). The formation of ACC from AdoMet is mediated by the pyridoxal enzyme ACC synthase (Adams and Yang, 1979). ACC synthase is strongly inhibited by AVG, a known inhibitor of pyridoxal phosphate-mediated enzyme reactions (Rando, 1974). And it has been shown that a rapid decline in the rate of ethylene production can result from decreased ACC synthesis or from the conjugation of ACC into n-malonyl ACC (MACC). This reaction is catalyzed by the enzyme ACC malonyltransferase (Yang *et al.*, 1990). The last step in the ethylene biosynthesis pathway is the conversion of ACC to ethylene by ACC oxidase (Yip *et al.*, 1988). It has been known that auxin stimulates ethylene production in plant tissues with an increases the rate of conversion of AdoMet to ACC in the ethylene biosynthesis pathway (Yu and Yang, 1979).

Kim and Mulkey (1997) reported that the inhibition of elongation by IAA could be partially or fully reversed by blocking ethylene biosynthesis or activity, and IAA-induced inhibition of root elongation, at least in part, is a reversible process in intact primary roots of maize.

Based on these facts, the experiments reported in this paper were designed to examine the interaction of auxin and ethylene on root elongation. In this study root elongation was examined in intact primary roots of maize by treatment with various auxin con-

centrations, ethylene precursor, and ethylene biosynthesis inhibitors.

MATERIALS AND METHODS

Plant Material

Maize (*Zea mays* L., Pioneer 3343) seeds were soaked overnight in running tap water and germinated between wet paper towels on opaque plastic trays placed in a vertical position. The trays were kept in a growth chamber at 27°C. Little light reached the seeds and the seedlings were used after 1.5 d when the primary roots were about 15 to 20 mm long.

Measurement of Root Elongation

Root elongation was measured using a computerized root auxanometer similar to one previously described (Kim and Mulkey, 1997).

Chemicals

Ethephon was purchased form Carolina Biological Supply Company (Burlington, NC, USA). All other chemicals were purchased from Sigma (St. Louis, MO, USA). To observe the effect of silver ions (Ag^{2+}), silver thiosulfate was prepared with silver nitrate and sodium thiosulfate as mentioned previously (Kim and Mulkey, 1997).

RESULTS

Fig. 1 illustrates the effects of application of several compounds involved in ethylene biosynthesis on the rate of elongation of intact primary roots of maize. AVG (10^{-6} M), an inhibitor of ACC synthase, promoted root elongation up to 130% of the control rate within minutes of exogenous application of the compound (solid line). Co^{2+} is an inhibitor of ethylene biosynthesis through the inhibition of the conversion of ACC to ethylene. Co^{2+} (10^{-4} M) stimulated an increase in the elongation rate of roots. The maximum rate of promotion was approximately 80% of the control rate (dotted line). L-Canaline,(10^{-9} M) is an inhibitor of ethylene biosynthesis which stimulates root elongation after a lag period of approximately 1 h. The elongation rate of L-canaline-treated roots increased to a maximum of 60% of the control rate (long dashed line).

Application of 10^{-6} M ACC, a precursor of ethylene, was performed to increase the endogenous ethy-
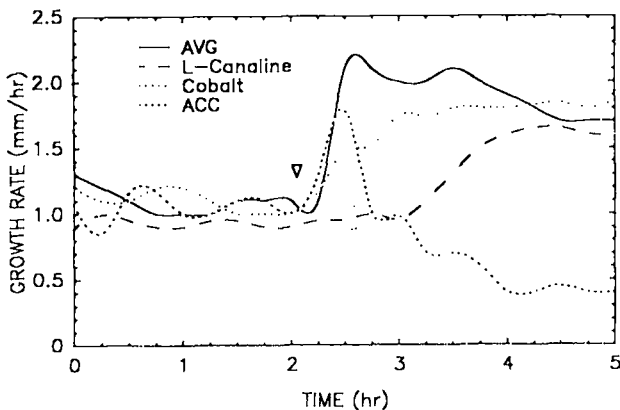
Fig. 1. Effect of several compounds relating to ethylene biosynthesis on the rate of elongation of intact primary roots of maize. Roots were treated with inhibitors or a promoter of ethylene biosynthesis 2 h after steady growth was observed. AVG (10^{-6} M) (solid line), 10^{-9} M L-canaline (long dashed line), and 10^{-4} M Co^{2+} (dotted line) are inhibitors of ethylene biosynthesis. ACC (10^{-6} M) is a precursor of ethylene (short dashed line).
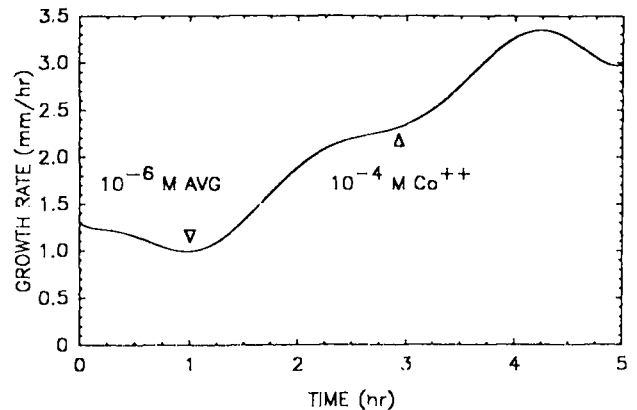

Fig. 2. Effect of ethylene biosynthesis inhibitors on the rate of elongation of intact primary roots of maize. AVG (10^{-6} M) was added to roots after steady growth was observed (first arrow). Roots were treated with 10^{-4} M Co^{2+} after AVG treatment (second arrow).

lene concentration within the tissue. Since the conversion of ACC to ethylene is an enzymatic process and ACC has been shown to be permeable within plant cell membranes, exogenous application of ACC provides an acceptable method of providing an endogenous source of ethylene. One difficulty associated with this method is that quantitation of the elevated ethylene levels is not possible. Thus, only qualitative conclusions could be drawn from these data. However, ACC induced a transient promotion of elongation followed by a decrease in the rate of elongation approximately 1 h after treatment. The final elongation rate was approximately 40% of the control rate within 2 h (short dashed line). The initial stimulation of root elongation may be the result of acidification of the cell wall as a result of the uptake by the root cells of ACC. The subsequent inhibition of root elongation was interpreted as the result of the endogenous conversion of ACC to ethylene and the expression of ethylene effects on root elongation. These data suggested that root elongation was closely related to the ethylene level of primary roots of maize.

Two inhibitors of ethylene biosynthesis were applied to roots in different time periods (Fig. 2). AVG inhibits the ethylene production by inhibiting ACC synthase activity. IAA stimulates ACC synthase activity. Co^{2+} block the conversion of ACC to ethylene. The stimulation of root elongation was observed after treatment of the roots with 10^{-6} M AVG. This treatment promoted root elongation to 180% of control

rate. Subsequent application of 10^{-4} M Co^{2+} to AVG treated roots resulted in an additional increase in the rate of root elongation to 230% of control rate. The magnitude of this combined treatments was approximately the same as the sum of the individual treatments such as AVG treatment (130% increase) and Co^{2+} treatment (80% increase) (Fig. 1). These data suggested that AVG only suppressed ethylene production. The addition of Co^{2+} to AVG-treated roots blocked the conversion of the low levels of ACC to ethylene.

Table 1 illustrates the effect of Co^{2+} and ACC on the various concentrations of IAA-induced elongation rate of primary roots of maize. Application of 10^{-6} M

Table 1. Effect of ACC and Co^{2+} on the various concentrations of IAA-induced elongation rate in intact primary roots of maize. Roots were treated with several concentrations of IAA (10^{-6} M-10^{-11} M), then 10^{-6} M ACC was applied 1 h after IAA treatment. Co^{2+} (10^{-4} M) was added 2 h after ACC application. Elongation rates were measured in 30 min after IAA application, 2 h after ACC, and 2 h after Co^{2+} treatments.

IAA concentrations (M)	Elongation Rate (%)[a]		
	IAA[b]	ACC[c]	Co^{2+d}
10^{-6}	22	30	52
10^{-7}	31	35	77
10^{-8}	58	70	125
10^{-9}	73	90	150
10^{-10}	87	110	185
10^{-11}	95	135	282

a: % of control elongation rate
b: measurement in 30 min after IAA treatment
c: measurement in 2 h after 10^{-6} M ACC treatment
d: measurement in 2 h after 10^{-4} M Co^{2+} treatment

IAA caused severe inhibition of elongation within 30 min. Posttreatment with 10^{-6} M ACC showed no affect root elongation. Co^{2+} (10^{-4} M) was added 2 h after ACC application. Root elongation recovered to approximately 60% of the control rate within 2 h. Roots were treated with 10^{-7} M IAA which inhibited the roots to 30% of the control rate within 1 h. Treatment with 10^{-6} M ACC promoted the rate of elongation temporarily within 1 h, then the rate of elongation decreased. Application of 10^{-4} M Co^{2+} allowed for recovery in the rate of elongation to 77% of the control rate within 1 h. Depending on the IAA concentrations, root elongation recovered to 125%, 150%, 185%, and 280% of the control rate within 2 h after the addition of 10^{-4} M Co^{2+}. In experiment, 10^{-6} M ACC seemed to promote root elongation within 30 min. However, this effect was transient as shown in Fig. 1. This growth data (Table 1) indicated that Co^{2+} could partially reverse the IAA- and ACC-induced inhibition of root elongation. The recovery effect by Co^{2+} appeared to be closely related to the IAA concentrations.

Table 2 demonstrates the effect of reversing the order of the treatments illustrated in Table 1. Treatment with 10^{-6} M IAA inhibited root elongation to 16% of the control rate. Co^{2+} (10^{-4} M) and 10^{-6} M ACC were applied to roots at 2 h and 4 h, respectively. These applications neither inhibited nor stimulated the 10^{-6} M IAA-induced inhibition of root elongation.

In roots exposed to 10^{-7} M IAA, addition of 10^{-4} M Co^{2+} 1 h after IAA treatment allowed reversal of

10^{-7} M IAA-induced inhibition of elongation. The elongation rate of these roots returned to 40% of the control rate within 2 h of Co^{2+} application. Application of ACC 2 h after the addition of 10^{-4} M Co^{2+} resulted in neither promotion nor inhibition of root elongation.

Addition of 10^{-4} M Co^{2+} increased the rate of root elongation in roots previously exposed to 10^{-8} M IAA. ACC appeared to increase further the rate of root elongation. However, the combined increase in the rate of elongation resulting from the application of Co^{2+} and ACC returned the elongation rate to a level which was not significantly high when compared to the original elongation rate prior to addition of IAA.

When ACC was applied to roots which have been previously treated with an auxin range of 10^{-9} M to 10^{-11} M IAA and with 10^{-4} M Co^{2+}, the rate of elongation was only slightly affected by the IAA treatment. The application of Co^{2+} allowed for reversal of any IAA inhibition in the rate of elongation (Table 2). The inhibition of root elongation by 10^{-6} M ACC returned the elongation rate to approximately the rate of elongation prior to the application of the test compounds. Treatment with Co^{2+} resulted in reversal of part or all of the IAA-induced inhibition of root elongation in these experiments (Table 2). The application of 10^{-6} M ACC to roots after the Co^{2+} treatment had no significant effect on root elongation.

The effects of 10^{-6} M AVG and 10^{-6} M ACC on IAA-induced root elongation were illustrated in Table 3. Treatment of roots with 10^{-6} M AVG allowed for reversal of the inhibition off elongation which resulted

Table 2. Effect of Co^{2+} and ACC on the various concentrations of IAA-induced elongation rate in intact primary roots of maize. Roots were treated with several concentrations of IAA (10^{-6} M-10^{-11} M), then 10^{-4} M Co^{2+} was applied 1 h after IAA treatment. ACC (10^{-6} M) was added 2 h after Co^{2+} application. Elongation rates were measured in 30 min after IAA application, 2 h after Co^{2+}, and 2 h after ACC treatments.

IAA concentrations (M)	Elongation Rate (%)[a]		
	IAA[b]	Co^{2+}[c]	ACC[d]
10^{-6}	16	20	12
10^{-7}	23	40	40
10^{-8}	65	100	115
10^{-9}	77	130	100
10^{-10}	85	132	100
10^{-11}	96	163	125

a: % of control elongation rate
b: measurement in 30 min after IAA treatment
c: measurement in 2 h after 10^{-4} M Co^{2+} treatment
d: measurement in 2 h after 10^{-6} M ACC treatment

Table 3. Effect of AVG and ACC on the various concentrations of IAA-induced elongation rate in intact primary roots of maize. Roots were treated with several concentrations of IAA (10^{-6} M-10^{-11} M), then 10^{-6} M AVG was applied 1 h after IAA treatment. ACC (10^{-6} M) was added 2 h after AVG application. Elongation rates were measured in 30 min after IAA application, 2 h after AVG, and 2 h after ACC treatments.

IAA concentrations (M)	Elongation Rate (%)[a]		
	IAA[b]	AVG[c]	ACC[d]
10^{-6}	12	30	30
10^{-7}	40	85	85
10^{-8}	65	125	100
10^{-9}	80	138	92
10^{-10}	92	133	88
10^{-11}	98	180	100

a: % of control elongation rate
b: measurement in 30 min after IAA treatment
c: measurement in 2 h after 10^{-6} M AVG treatment
d: measurement in 2 h after 10^{-6} M ACC treatment

from the pretreatment of roots with 10^{-6} M IAA. However, subsequent application of 10^{-6} M ACC neither inhibited nor stimulated the elongation of these roots.

Roots treated with 10^{-7} M IAA exhibited inhibition of root elongation. subsequent application of 10^{-6} M AVG allowed recovery in the rate of elongation to approximately 85% of the control rate. The addition of 10^{-6} M ACC to roots which were treated with 10^{-7} M IAA and 10^{-6} M AVG have no significant effect on the rate of elongation.

Application of 10^{-6} M ACC induced a mild inhibition of root elongation in roots exposed to 10^{-8} M IAA and 10^{-6} M AVG. This inhibition by ACC of root elongation increased in roots treated with 10^{-9} M IAA and AVG. Treatment of roots with 10^{-6} M AVG induced full recovery and a subsequent promotion of the elongation rate in roots exposed to 10^{-10} M IAA. Application of ACC inhibited the rate of elongation in these roots.

Addition of ACC induced a strong inhibition in the rate of elongation in roots which were pretreated with 10^{-11} M IAA and AVG. The inhibition of root elongation by ACC in roots previously treated with IAA and AVG became manifest when the applied IAA concentrations were low.

DISCUSSION

Auxin has been demonstrated to be involved in the control of physiological processes such as cell elongation and differentiation in plant tissues. Some auxin actions are known to be mediated by the ethylene produced in response to auxin (Abeles et al., 1992).

Ethylene production is stimulated by several factors. These factors include other plant hormones, and physical and chemical injury (Abeles et al., 1992). It is known that auxin stimulated ethylene production in plant tissues, including roots (Kang et al., 1971; Mulkey et al., 1982). The auxin-induced promotion of ethylene production is associated with an increase in ACC synthase levels. There is evidence that auxin increases the rate of conversion of AdoMet to ACC in the ethylene biosynthesis pathway (Yu and Yang, 1979). There is a linear relationship between the rate of ethylene production and ACC content in plants (Yoshii and Imaseki, 1981).

Both AVG and L-canaline are known inhibitors of ethylene biosynthesis (Abeles et al., 1992). These compounds inhibited the conversion of AdoMet to ACC. These inhibitors promoted the rate of root elongation to 160%-180% of the control rate (Fig. 1). Treatment of roots with AVG promoted root elongation

with little or no lag period. However, application of L-canaline resulted in a lag period of approximately 1 h prior to significant stimulation of root elongation. The effect of AVG and L-canaline on ethylene production is dependent on the plant tissue. Both AVG and L-canaline were equally effective in intact lettuce seedling roots (Abeles and Wydoski, 1987). In melon seedlings, L-canaline was less effective than AVG (Toppan et al., 1982). The data presented in here suggested that AVG may be more effective than L-canaline in intact primary roots of maize (Fig. 1).

Co^{2+} are inhibitors of ethylene production at the step in which ACC is converted to ethylene by ACC oxidase (Yu and Yang, 1979). In this experiment, Co^{2+} promoted root elongation gradually to a maximum of 180% of the control rate.

Treatment of intact roots with inhibitors of ethylene production resulted in an increase in the rate of root elongation without externally applied auxin. This suggested that maize roots have supraoptimal auxin concentrations. These supraoptimal auxin levels induce ethylene biosynthesis under normal growth conditions. These low levels of ethylene may regulate root elongation by acting to slow the growth of roots. The presence of supraoptimal auxin concentrations in roots would explain the inhibition of root elongation by the exogenous application of almost any concentration of IAA. Although Eliasson et al. (1989) observed that ethylene production in pea roots treated with IAA for 24 h was less than that in roots treated with ACC for 24 h, they still observed inhibition of pea root elongation in both treated roots in relatively long period. The results presented in this experiment suggest that the elongation of intact primary roots of maize can be regulated by the internal level of ethylene within 6 h. Whalen and Feldman (1989) reported that inhibition of root elongation by treatment of ethylene was reversible, and there is evidence that post- or pretreatment of ethylene antagonists reverse the auxin-induced inhibition of root elongation within 4 h to 6 h (Kim and Mulkey, 1988).

On the other hand, ACC is a precursor of ethylene and is rapidly converted to ethylene (Apelbaum and Burg, 1972; Adams and Yang, 1979). Root elongation was inhibited by applied ACC (Fig. 1). However, ACC induced a transient (40 min) increase in root elongation. This effect may be due to the uptake of the acidic tricyclic compound (ACC). This uptake stimulates the efflux of hydrogen ions to maintain a constant cytoplasmic pH. Application of ACC and the endogenous conversion of ACC to ethylene inhibited root elongation to 40% of the control rate

within 3 h. Treatment of intact roots with AVG and Co^{2+} stimulated root elongation due to their effects on endogenous pools of ACC.

Reversal of IAA- and ACC-induced inhibition of root elongation could be observed upon application of 10^{-4} M Co^{2+} (Table 1). Both IAA and ACC are compounds which promote ethylene production in plant tissues. The effect of Co^{2+} can be explained by the fact that Co^{2+} blocks the conversion of ACC, either induced by IAA or supplied from an exogenous pool, to ethylene.

Application of ACC to roots previously exposed to IAA did not further inhibit root elongation. ACC induced mild stimulation of root elongation in the presence of lower concentrations of IAA (10^{-11} M to 10^{-9} M). This phenomenon may result from increasing the rate of H^+ efflux from the root cell over the rate of H^+ influx associated with inhibition of root growth. Under mild inhibition of root growth, the low level of H^+ influx could be compensated for the acidification resulting from the uptake of ACC.

The converse treatments with ACC and Co^{2+} in the presence of several concentrations of IAA were shown in Table 2. In this experiment, Co^{2+} enhanced recovery from IAA-induced inhibition of root elongation. Cobalt ions blocked the inhibitory effect on root elongation of subsequent treatment with ACC. Therefore, ethylene formation from ACC was inhibited by Co^{2+}. Additionally endogenous pools of ACC should be elevated due to the effects of exogenously applied IAA. Thus, these roots should have high endogenous pools of ACC which should decrease the uptake of externally-applied ACC.

AVG inhibits the action of ACC synthase resulting in inhibition of ethylene production (Yu and Yang, 1979). Roots treated with AVG exhibited recovery from IAA-induced inhibition of root elongation (Table 3). AVG counteracts the effect of IAA in the stimulation of ACC synthase. Therefore, the internal ACC level in roots should change depending on the concentration of applied IAA in relation to the concentration of AVG. Addition of ACC to roots exposed to higher concentrations of IAA (10^{-6} M and 10^{-7} M) and AVG did not show inhibition of root elongation. However, ACC caused inhibition of root elongation when roots were exposed to lower concentrations of IAA (10^{-11} to 10^{-8} M) and AVG. These results indicated that 10^{-6} M AVG may not be the optimal level to counteract the effects of the higher concentrations of IAA; but application of higher concentrations of AVG to roots resulted in numerous nonspecific effects (Abeles et al., 1992).

The experimental results presented in this study were consistent with the hypothesis that the level of ethylene in primary roots of maize functions to moderate root elongation. Additionally, these data demonstrated that root elongation can be controlled by the compounds which regulate ethylene production. Furthermore, these results supported the hypothesis that auxin-induced inhibition of root elongation results from auxin-induced promotion of ethylene production.

LITERATURE CITED

Abeles, F.B. and S.G. Wydoski. 1987. Inhibitors of ethylene synthesis and action: a comparison of their activities in a lettuce root growth model system. J. Amer. Soc. Hort. Sci. 112: 122-125.

Abeles, F.B., P.W. Morgan and M.E. Saltveit, Jr. 1992. Ethylene in Plant Biology. Academic Press, Inc., San Diego, CA.

Adams, D.O. and S.F. Yang. 1979. Ethylene biosynthesis: identification of 1-aminocyclopropane-1-carboxylic acid as an intermediate in the conversion of methionine to ethylene. Proc. Natl. Acad. of Sci., USA 76: 170-174.

Andreae, W.A., M.A. Venis, F. Jurasic and T. Dumas. 1968. Does ethylene mediate root growth inhibition by indole-3-acetic acid? Plant Physiol. 43: 1375-1379.

Apelbaum, A. and S.P. Burg. 1972. Effects of ethylene and 2,4-dichlorophenoxyacetic acid on cellular expansion in Pisum sativum. Plant Physiol. 50: 125-131.

Burstrom, H. 1969. Influence of the tonic effect of gravitation and auxin on cell elongation and polarity in roots. Amer. J. of Botany. 56: 679-684.

Chadwick, A.V. and S.P. Burg. 1967. An explanation of the inhibition of root growth caused by indole-3-acetic acid. Plant Physiol. 42: 415-420.

Eliasson, L., G. Bertell and E. Bolander. 1989. Inhibitory action of auxin on root elongation not mediated by ethylene. Plant Physiol. 91: 310-314.

Evans, M.L. 1984. Functions of hormones at the cellular level of organization. In Encyclopedia of Plant Physiology, New series Vol. 10. T.K. Scott (ed.). Springer-Verlag, Berlin, pp. 23-79.

Kang, B.G., W. Newcomb and S.P. Burg. 1971. Mechanism of auxin induced ethylene production. Plant Physiol. 47: 504-509.

Kim, S.Y. and T.J. Mulkey. 1988. Effects of ethylene antagonists on recovery from auxin-induced inhibition of root elongation. Curr. Topics in Plant Biochem. and Physiol. 7: 228.

Kim, S.Y. and T.J. Mulkey. 1997. Effect of ethylene antagonists on auxin-induced inhibition of intact primary root elongation in maize (Zea mays L.) J. Plant Biol. (in press).

Mulkey, T.J., K.M. Kuzmanoff and M.L. Evans. 1982. Promotion of growth and shift in the auxin dose/response relationship in maize roots treated with the e-

thylene biosynthesis inhibitors, aminoethoxyvinylglycine and cobalt. *Plant Science Letters.* **25**: 43-48.

Rando, R.R. 1974. Chemistry and enzymology of kcat inhibitors. *Science* **185**: 320-324.

Thimann, K.V. 1937. On the nature of inhibition caused by auxin. *Amer. J. of Botany.* **24**: 407-412.

Toppan, A., D. Roby and M.T. Esquerre-Tugaye. 1982. Cell surface in plant-microorganisms interaction. III. *In vivo* effect of ethylene on hydroxyl proline-rice glycoprotein accumulation in the cell wall of diseased plants. *Plant Physiol.* **70**: 82-86.

Whalen, M.C. and L.J. Feldman. 1988. The effect of ethylene on root growth of Zea mays seedlings. *Canadian J. of Botany.* **66**: 719-723.

Yang, S.F. and N.E. Hoffman. 1984. Ethylene biosynthesis and its regulation in higher plants. *Ann. Rew. of Plant Physiol.* **35**: 155-189.

Yang, S.F., W.K. Yip, S. Satoh, J.H. Miyazaki, X. Jiao, Y. Liu, L.Y. Su and G.D. Peiser. 1990. Metabolic aspects of ethylene biosynthesis. *In* Plant Growth Substances 1988, R.P. Pharis, and S.B. Road (eds.). Springer-Verlag, Berlin, pp. 291-299.

Yip, W.K., X.Z. Jiao and S.F. Yang. 1988. Dependence of *in vivo* ethylene production rate on 1-aminocyclopropane-1-carboxylic acid content and oxygen concentrations. *Plant Physiol.* **88**: 553-558.

Yoshii, H. and H. Imaseki. 1981. Biosynthesis of auxin-induced ethylene. Effects of indole-3-acetic acid, benzyladenine and abscisic acid on endogenous levels of 1-aminocyclopropane-1-carboxylic acid (ACC) and ACC synthase. *Plant Cell and Physiol.* **22**: 369-379.

Yu, Y.B. and S.F. Yang. 1979. Auxin-induced ethylene production and its inhibition by aminoethoxyvinylglycine and cobalt ion. *Plant Physiol.* **64**: 1074-1077.