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Hormonal Control of Gene Expression of Acid Invertase in Mungbean(Vigna radiata I.)

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We have studied a correlation between acid invertasc(Λ I) gene expression and auxin-induced growth in mungbean hypocotyls. Changes in activities and gene expression of AI during growth and along the axis of the hypocotyl were examined. The plant hormone IAA stimulated the growth of hypocotyl segments and induced the accumulation of Λ I transcripts. Under light or dark conditions, the level of AI transcripts were regulated with the same pattern but different kinetics and AI gene expression were reversably regulated. Synthetic auxins, such as 2,4-D, NAA, and protein synthesis inhibitor, cycloheximide, also increased AI transcripts. Treatment of segments with the transcriptional inhibitor α -amanitin resulted in decreased transcript level. These results imply that the increase of AI transcripts is primarily associated with transcriptional activation, but we cannot exclude the possibility of posttranscriptional control.

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Indole-3-acetic acid and N^6 -Benzyladenine Inhibit Ethylene Action in Ethylene-regulated Expressions of Genes Encoding ACC Synthase and Oxidase in Mungbean Hypocotyls

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Gene exepression of ACC oxidase and synthase by ethylene, indole-3-acetic acid (IAA), and N⁶-benzyladenine (BA), was investigated in mungbean hypocotyls by using pVR-ACO1, and pVR-ACS1, respectively as probes. The basal levels of ACC oxidase activity and its transcripts were greatly increased by exogenous application of ethylene, but were reduced by aminooxyacetic acid (AOA) blocking the ethylene biosynthesis. These data suggest that ethylene stimulates ACC oxidase activity primarily at the level of its transcript abundance through a positive feedback mechanism. Expression of ACS1 was inhibited by ethylene, but stimulated by AOA, suggesting a negative feedback loop in ethylene-mediated decrease of ACS1 mRNA. IAA and BA did not show any effect on the level of ACC oxidase mRNA and enzyme activity, but increased the level of ACC synthase mRNA. However, these hormones stimulated ethylene production to the level which was shown to induce ACO1 expression and its enzyme activity, and to suppress ACS1 expression. IAA and BA inhibited ethylene-induced ACO1 expression and its enzyme activity whereas they increased ethylene-suppressed ACS1 expression. Taken together, it is suggested that IAA and BA inhibit ethylene action, resulting in inhibition of a positive feedback of ethylene on ACO1 expression and release from a negative control of ethylene on ACS1 expression.