F211 Osmotin gene expression induced by wounding signal follows Jasmonic acid-dependent pathway

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We have identified and characterized the gene encoding osmotin from a cDNA library which is constructed from 3 to 5 day old petunia (*Petunia hybrida*) petal protoplast cultures. RNA gel blot analyses of osmotin mRNAs showed high abundance in the roots. The osmotin gene is induced by environmental signals. Thus, treatments with sucrose, low temperature, and NaCl increased the accumulation in leaves of 4 weeks-old plants. Furthermore, osmotin gene was significantly induced in wounded-leaves whereas its signal was not transferred into the neighbor leaves. To further understand mechanism of wound-induced gene expression, we investigated the effects of JA and JA inhibitors with relation to wounding. JA induced the osmotin gene expression, whereas JA inhibitors down-regulated the effects of JA. Furthermore, osmotin gene expression was decreased in wounded leaves treated with JA inhibitors. Therefore, it is considered that osmotin gene expression induced by wounding signal follows JA-dependent pathway

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The Transcriptional Regulation Role of Gcn4p Recognition Elements within Yeast *ADE3* gene promoter

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The *Saccharomyces cerevisiae ADE3* gene encodes one enzyme called C_1-tetra hydrofolate (THF) synthase which posseses 10-formyl-THF synthetase, methenyl-THF cyclohydrolase and methylene-THF dehydrogenase activities. These activities are required in purine biosynthesis. Through deletional analysis of the *ADE3* promoter, we identified 3 Gcn4p recognition elements (GCREs) as a cis-regulatory element required for *ADE3* gene expression. Gcn4p, a well-known yeast transcription factor, is known to regulate not only many amino acid biosynthetic gene expression but also some genes related with purine biosynthesis. However, the regulation of *ADE3* gene by Gcn4p has not been reported so far. To investigate the expression of *ADE3* is regulated by GCREs and to characterize the roles of each GCREs, TGACT(G/C), was mutated to TGAATTC which is not a binding site for Gcn4p. The most upstream GCRE appears to have a critical role because it was required for the basal expression of *ADE3* as well as inducible expression. When the most downstream GCRE was mutated, the expression level of ADE3-LacZ gene was increased. Therefore, it appears that its role in ADE3 transcription was working in a repressive manner. The GCRE which is located between other GCREs is also necessary to augment the former GCRE although the ability of this for gene expression is weak. These results strongly suggest that transcription factor Gcn4p be involved in a transcriptional regulation of yeast ADE3 gene.