FUNCTIONAL AND EXPRESSIONAL QUANTITATION OF SELECTED microRNAs IN RESPONSE TO LOW TEMPERATURE AND NaCl STRESS CONDITIONS IN LEAF AND ROOT TISSUES OF Arabidopsis thaliana

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ABSTRACT

The discovery of miRNAs has led to a fundamental change in the understanding of complex biological mechanisms involved in plant responses to stress tolerance. In this study, the expression of seven selected miRNAs was studied in Arabidopsis thaliana plants experiencing low temperature and salt stress separately. The selected miRNAs were in situ hybridized with Locked Nucleic Acid (LNA)-modified oligonucleotide probes. Among the tested miRNAs, expression of miR161, miR168, miR171 and miR397a in leaf tissues and miR171 and miR397a in root tissues was recorded in control plants. Elevated expression of miR171 and miR397a was recorded in both tissue types of low temperature treated plants. A set of four miRNAs viz., miR171, miR395b, miR399e and miR399 showed their up regulation in both tissue types upon NaCl (300 mM) treatment. Expression of miR168 was recorded only in leaf tissues, and on the other hand, down regulation of miR397a was recorded in both tissue types in response to NaCl stress. The miRNA stem-loop RT-PCR assay indicated gradual increase in the expression of miR171 and miR397 with the highest of 4.28 and 3.49 fold changes in leaf tissues of A. thaliana plants experiencing low temperature stress and 6.5 and 6.3 fold up-regulation of miR171, 0.8 and 0.9 fold down-regulation of miR397a and 3.4-3.5 fold up-regulation of miR399 and miR399e in leaf and root tissues, respectively, at 24 hrs of exposure to salt stress. The RT-qPCR assay recorded reduced levels of miRNA target gene transcripts viz., SCL6 III, SCL6 IV, LAC2, and LAC17 in response to low temperature and SCL6 III, SCL6 IV, APS1, APS4 and AGO1 transcripts in response NaCl treatment in both tissue types of Arabidopsis thaliana plants. The study points at the possibility of modulating low temperature and salt tolerance in plants through the down regulation of specific cognate genes.