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Determination of AtSCD1 mRNA and protein levels in the temperature sensitive mutant scd1-1

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Plant cells defend themselves against bacterial infection by first detecting bacterial flagellin outside the cell and then initiating defense responses within the cell. Our lab is interested in identifying and characterizing vesicular trafficking components that play a role in defense responses to bacterial flagellin and its active peptide derivative, flg22, in *Arabidopsis thaliana*. Our current research focuses on SCD1*,* a protein implicated in polarized vesicle secretion. Recent results in our lab also indicate that *scd1-1* plants, which contain a point mutation in the SCD1 gene, exhibit reduced flg22-responses, notably in the production of reactive oxygen species (ROS). To gain a better understanding of the role of SCD1 in flg22-responses, we analyzed SCD1 mRNA and protein levels in *scd1-1* plants compared to the wild-type (WT), *Colgl1*, plants using qRT-PCR and protein blot analysis, respectively. SCD1 levels were compared in plants grown continuously at 22°C, the non-permissive temperature, to plants grown initially at 22°C and then shifted for 9 days to the permissive temperature, 17°C. Data indicate that at both 22°C and 17°C, SCD1 mRNA levels were statistically similar in *scd1-1* compared to WT plants. Conversely, SCD1 protein levels were significantly higher in *scd1-1* plants shifted to 17°C compared to those grown at 22°C. An increase in SCD1 protein level also correlated with an increase in flg22-induced ROS production at 17°C. To further confirm a requirement for SCD1 in flg22-repsonses, we showed that in an *scd1-1* mutant plant transformed with the SCD1 gene *(scd1-1/pscd1::SCD1)*, SCD1 protein levels were similar to those in WT grown at 22°C. These results are consistent with the complemented line exhibiting flg22-induced ROS production similar to WT levels. Given these results, both the temperature shift and the complementation experiments support our hypothesis that SCD1 protein is required for full flg22-induced responses.