

Public Abstract

First Name:Erin

Middle Name:MacNeal

Last Name:Rehrig

Adviser's First Name:Jack

Adviser's Last Name:Schultz

Co-Adviser's First Name:

Co-Adviser's Last Name:

Graduation Term:SP 2010

Department:Plant, Insect and Microbial Sciences

Degree:PhD

Title:The Role of ERF Transcription Factors in Defenses Against Specialist and Generalist Herbivores in *Arabidopsis thaliana*

Plant responses to herbivory are complex, involving differential perception, multiple signaling pathways, and the transcription of defense-responsive genes. Using a whole-genome microarray and bioinformatics tools, we identified transcription factors and cis-elements important in differential responses in *Arabidopsis thaliana* after attack by aphids, the specialist caterpillar, *Pieris rapae*, and the generalist caterpillar, *Spodoptera exigua*. Insect-specific changes in gene expression were observed and involved the fine-tuning of the stress-related hormones, jasmonate, salicylate, and ethylene. Ethylene was produced in response to both insect species, although the amounts and timing of production differed. Additionally, rapid and increased jasmonate and jasmonate-isoleucine elicitation in *Arabidopsis* after attack by both insects confirmed these signals as general herbivore-related responses. Using RT-PCR, we found members of the ERF (Ethylene Response Factor) transcription factor family and AtMYC2 to be differentially regulated in response to the two caterpillars. We assessed the feeding behavior of *S. exigua* and *P. rapae* in wild-type and ERF mutant plants (*erf5*, *erf6*, *erf104*, and *erf105*) using a novel digital phenotyping technique. *S. exigua* maintained similar growth rates despite consuming less mutant tissue. Although induced aliphatic and indolyl glucosinolate (GS) levels were significantly higher in *erf104* plants after *S. exigua* feeding, no consistent relationships between GS and tissue consumption by insects were found. Our results demonstrate clear insect resistance phenotypes in *erf* mutants, suggesting a role for ERFs in the negative regulation of a defense mechanism other than glucosinolate production.