Pectin Activation of WAK Regulated Stress Response in *Arabidopsis thaliana*

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The Wall-associated kinases, WAKs, are integral membrane proteins encoded by five highly similar genes clustered in a 30-kb locus in the Arabidopsis plant. These receptor-like proteins contain a cytoplasmic serine threonine kinase, a trans membrane domain, and a less conserved region that is bound to the cell wall and contains a series of epidermal growth factor repeats. Experimental evidence indicates that WAKs serve as pectin receptors for both short oligo galacturonic acids fragments (OGs) generated during pathogen exposure or wounding, and for longer pectins resident in native cell walls. This ability to bind and respond to several types of pectins correlates with the demonstrated role for WAKs in both the pathogen response via OGs and cell expansion during plant development via native pectin polymers. These two distinct pathways of growth and stress are distinguished by the activation of MPK3 versus MAPK6, respectively, as revealed by biochemical and genetic analysis, and identification of different gene expression patterns. In addition, genetic and biochemical evidence suggests that pectin modification is required for WAK activation.

My project analyzed a pectin methylesterase protein mutant (PME3) that fails to remove a methyl group from pectin, thereby stopping pectin from cross-linking into a more rigid network. By comparing relative gene expressions via qPCR, I found that PME3 suppressed a dominant active allele of WAK (CTAP) that is a hyperactive receptor kinase. I also showed that downstream gene expression changes that are induced by the dominant WAK allele, and are also suppressed by PME3. Thus, de-esterification of pectin is required for WAK activation.

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Funded by the Bowdoin Life Sciences Fellowship

References:
