Understanding molecular interactions between plants and pathogens is a necessary step in the development of disease prevention and management strategies. Plants utilize a multifaceted innate immune response to prevent infection by pathogens. The first layer of this response is Pattern Triggered Immunity (PTI). In the model plant Arabidopsis thaliana, MAP Kinase Phosphatase 1 (MKP1) has been identified as a negative regulator of PTI. As a result, mkp1 mutants are more resistant than wild type plants to bacterial infection. Bacteria are a diverse and damaging class of plant pathogens. Many bacteria, such as Pseudomonas syringae, utilize a Type III Secretion System (T3SS) to inject host plants with effector proteins that disable the host immune system. The components of this molecular syringe are expressed upon detection of chemical secretions from host plants. Interestingly, exudate samples collected from mkp1 mutant plants contain lower amounts of T3SS-inducing chemical signals than wild type plants. We hypothesize that the decreased abundance of these chemicals in mkp1 exudate is due to the activity of the defense-related E3 Ubiquitin ligase E3L2. Consistent with this, our preliminary data suggest that e3l2 knockout mutants cannot properly manifest PTI. To determine whether E3L2 is genetically linked to MKP1, double knockout mutants have been generated from a cross of the two single mutants. Assays performed with these plants and P. syringae will shed light on the potential genetic interactions between E3L2 and MKP1 during PTI, leading to a more complete understanding of plant innate immunity.