Understanding the resistance response to *Meloidogyne chitwoodi* introgressed from *Solanum bulbocastanum* into cultivated potato

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Meloidogyne chitwoodi (Columbia root-knot nematode; CRKN) is one of the most devastating pest of potato in the Pacific Northwest region of the United States. CRKN is a soil-borne pest that infects potato roots as well as tubers. Tuber damage is evident in the form of internal (blemishes) as well as external (bumps) defects that make the crop unfit for commercial use (Figure 1). Currently, soil fumigation is the preferred method to control this nematode. However, host resistance is viewed as one of the most effective and environmentally friendly approaches to control nematode damage but no commercial potato variety is known to harbor CRKN resistance. CRKN resistance was first identified in the wild diploid species *Solanum bulbocastanum* (clone SB22), and this resistance was introgressed into a tetraploid selection, PA99N82-4 by using protoplast fusion and conventional breeding. A single dominant gene mapped onto Chromosome 11 (Brown et al 1996) controls this resistance but the underlying resistance mechanism is still unknown. In order to unveil the molecular events leading to resistance response, we used RNAseq technology to study the differential gene expression between CRKN resistant PA99N82-4 and a susceptible cultivar Russet Burbank. The resistant and susceptible clones were challenged with 1200 CRKN juveniles and the response to nematode infection was studied at four time points: 48 hours post inoculation (hpi), 7-, 14- and 21 days post inoculation (dpi). RNA sequencing resulted in an average of 33 million reads for each time point, out of which an average of 79% sequence reads mapped to *Solanum tuberosum* reference genome. An average of 3000 genes were differentially expressed between PA99N82-4 and Russet Burbank at each time point, out of which ~50% were up-regulated in the resistant clone. Differentially expressed data showed the genes that are usually triggered in response to the external stimuli were up-regulated in the resistant clone. Most of these genes are known to be involved in transcription factor activity, DNA binding, transporter and kinase-like activity, which in turn are known to trigger various host-pathogen interaction pathways, plant hormone signaling pathways, antioxidant activity, cell wall re-enforcement and polyamine biosynthesis. Based on our gene expression data (fold change ≥1), we hypothesize that CRKN presence in and around the root tissue triggers pathogen associated molecular patterns (PAMP)-triggered immunity (PTI) as an early response. Once the nematodes secrete effectors into the root tissue after their successful penetration, the R-gene mediated effector-triggered immunity (ETI) is initiated in the resistant clone that might be responsible for inhibiting the feeding site formation, an important event for the nematode to reproduce and complete its life cycle. The resistance response is due to the accumulation of reactive oxygen species (ROS) and hypersensitive response (HR) leading to rapid cell death causing localized lesions around the site of nematode infection. The plant hormone salicylic acid (SA) seems to play a significant role in ETI based HR activity, which is indicated by the up-regulation of SA marker, (pathogenesis related) PR-1 gene in PA99N82-4. The ROS scavenging system is also activated in

order to prevent further host tissue damage. In addition, polyamines (spermine and spermidine) are up-regulated in the resistant clone, indicating their role in the resistance mechanism. An active ingredient of the root cell wall re-enforcement, suberin is also playing prominent role in strengthening the host cell wall to protect the tissue from further nematode attacks. The present study is the first ever description of the molecular crosstalk between *M. chitwoodi* and the resistant potato. However, the major genes involved in the defense pathway warrant further validation. The data repository thus generated could be used in breeding for CRKN resistant potato varieties.

Figure 1: *Meloidogyne chitwoodi* infected potato tubers showing external (left) and internal (right) defects.

References

Brown, C. R., Yang, C. P., Mojtahedi, H., Santo, G. S., & Masuelli, R. (1996). RFLP analysis of resistance to Columbia root-knot nematode derived from *Solanum bulbocastanum* in a BC 2 population. *Theoretical and Applied Genetics*, *92*(5), 572-576.