Background:
Plum pox virus (PPV), causing Shanka disease, is one of the main limiting factors for Prunus production worldwide. In apricot (Prunus armeniaca L.) the major PPV resistance locus (PPVres), comprising ~196 kb, has been mapped to the upper part of linkage group 1. Within the PPVres, 68 genomic variants linked in coupling to PPV resistance were identified within 23 predicted transcripts according to peach genome annotation. Taking into account the predicted functions inferred from sequence homology, some members of a cluster of mtrnp and TRAP-C homology domain (MATHd)-containing genes were pointed as PPV resistance candidate genes.

Results:
Here, we have characterized the global apricot transcriptome response to PPV-D infection. None of the PPVres locus genes was differentially expressed between infected/non-infected tissues, suggesting that PPV-D presence does not modulate their expression. However, six PPVres locus genes (ParP-1 to ParP-6) appeared differentially expressed in resistant/susceptible cultivars (Figure 1). Two of them (ParP-3 and ParP-4), that encode MATHD proteins, appear clearly down-regulated in resistant cultivars, as confirmed by qRT-PCR (Figure 2). Concurrently, variant calling was performed using whole-genome sequencing data of 24 apricot cultivars (10 PPV-resistant and 14 PPV-susceptible) and 2 wild relatives (PPV-susceptible) (Figure 3). ParP-3 and ParP-4, named as Prunus armeniaca EPIres MATHd-containing genes (ParPMC1 and ParPMC2 respectively), are the only two genes having allele variants linked in coupling to PPV resistance. ParPMC1 has 1 nsSNP, while ParPMC2 has 15 variants, including a 5-tup deletion within the second exon that produces a frameshift mutation. ParPMC1 and ParPMC2 are highly homologous (87.5% identity) and adjacent in the MATHD genes cluster suggesting they are paralogs originated from a tandem duplication (Figure 4). Cultivars carrying the ParPMC2 resistant (mutated) allele show lack of expression in both ParPMC2 and especially ParPMC1.

Conclusions: Accordingly, we hypothesize that ParPMC2 is a pseudogene that mediates down-regulation of its functional paralog ParPMC1 by silencing. As a whole, results strongly support ParPMC1 and/or ParPMC2 as host susceptibility genes required for PPV infection which silencing may confer PPV resistance trait. This finding may facilitate resistance breeding by marker-assisted selection and pave the way for gene edition approaches in Prunus.