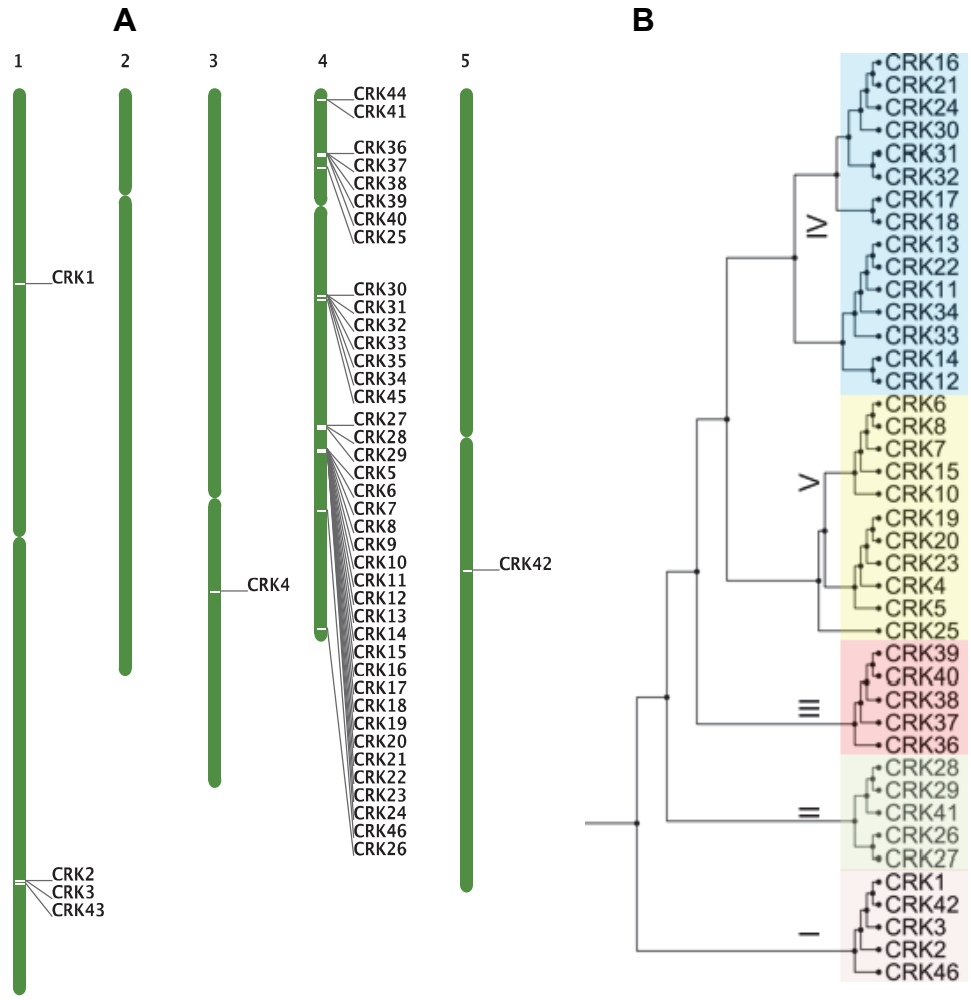

Fig S1. The chromosomal location and phylogenetic tree of CRKs in *Arabidopsis* genome. A) The majority of the *Arabidopsis* CRKs are tandemly located on chromosome 4. B) A phylogenetic tree of all canonical CRKs. Numbers and color shade indicate the different clades of CRKs

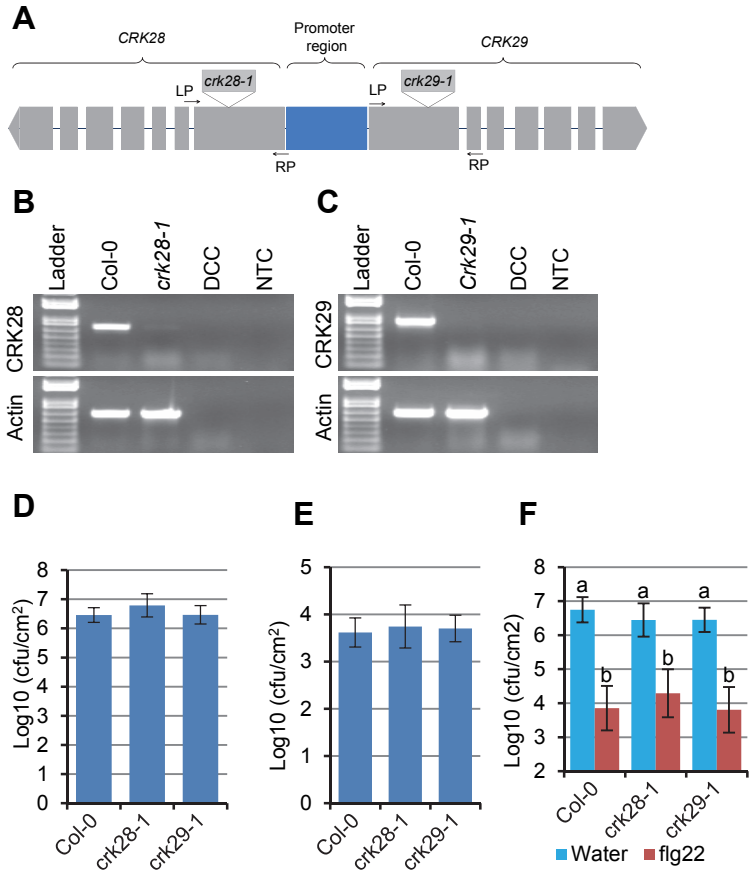

Fig S2. Genotyping and disease phenotyping of *CRK28* and *CRK29* T-DNA knockout lines. A) Schematic representation showing the position of the T-DNA insertion sites in *CRK28* and *CRK29*. B) RT-PCR showing the expression of *CRK28* and *CRK29* in their respective T-DNA lines compared to Col-0. Primer pairs flanking the insertion site were used for RT-PCR. The *Arabidopsis Actin 2 (ACT2)* gene was used as an endogenous control. DCC = DNA contamination control, NTC = no template control. D) and E) Quantification of the *Pst DC3000* (D) and *Pst DC3000* Δ*hrcC* (E) in the wild type Col-0, *crk28-1*, and *crk29-1* plants four days post-infiltration and spray inoculation, respectively. Error bars indicate standard deviation, n = 6. No statistically significant differences were observed between the T-DNA knockouts and Col-0. F) Quantification of *Pst DC3000* in Col-0, *crk28-1* and *crk29-1* 2dpi. For the flg22 protection assay, leaves were infiltrated with 1μM flg22 and 24h later, the same leaves were infiltrated with *Pst DC3000* ($1x10^5$ CFU/mL). Error bars indicate standard deviation, n = 8. Blue and red bars indicate water and flg22 treated plants. These assays were repeated at least three times with similar results. No statistically significant differences in flg22 protection were observed between the T-DNA knockouts and Col-0.

ATGGAACATGTCAGAGTTATCTTTTTCTTCTTTGCTTGTGTCCTAAAGATTGTACCATTTATCTGCTTAGCACAGAA
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TCTTCCACAGAACCGTTATTAATGTCCTTGAATGATGTCACTGTTTCTGAGTTATCTCCTCGTTAG

Fig S3. CRK28's cDNA sequence from Col-0. CRK28 possesses seven exons and six introns. Nucleotides with blue and red font colors show the fourth and fifth exons, respectively.

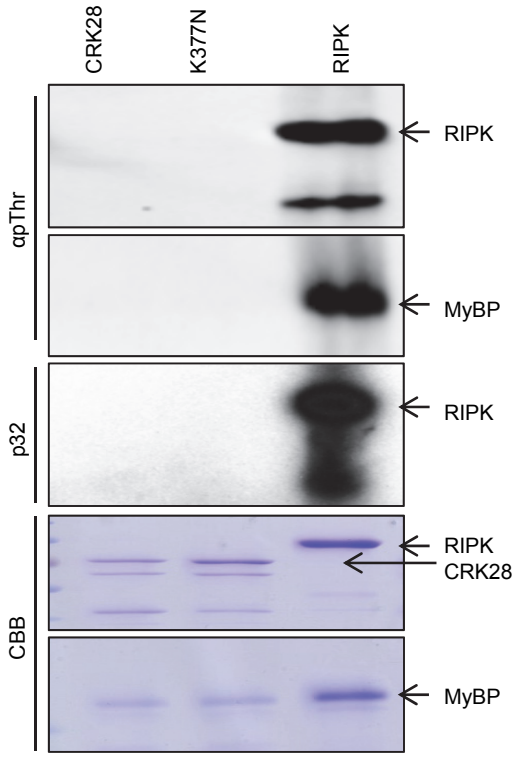
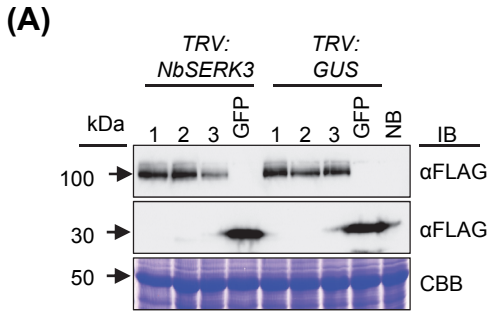
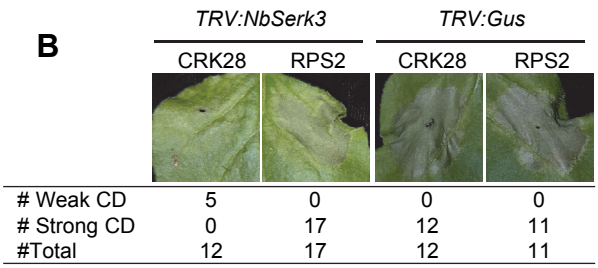


Fig S4. *In vitro* CRK28 kinase activity assay. CRK28's kinase domain purified from *E. coli* shows neither autophosphorylation nor transphosphorylation of the artificial kinase substrate myelin basic protein (MyBP). Three µg of CRK28's purified recombinant kinase domain and the kinase dead variant ($CRK28^{K377N}$) were mixed with the kinase substrate myelin basic protein (MyBP) and subjected to a kinase activity assay. The RIPK (AT2G05940) kinase was included as a positive control. The reaction was separated by SDS-PAGE and immunoblotted with anti-phospho-threonine antibody. A radioactive (^{32}P) kinase activity assay was also performed as described in (Lin et al., 2015). The bottom panel shows coomassie brilliant blue (CBB) stain showing total loaded protein.





# Weak CD	5	0	0	0
# Strong CD	0	17	12	11
#Total	12	17	12	11

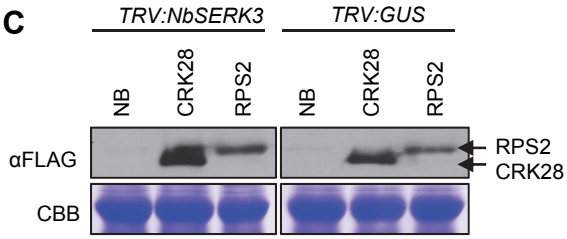


Fig S5. Silencing *NbSerk3* does not inhibit cell death elicited by the resistance protein RPS2. A) Anti-FLAG immunoblot detecting CRK28-FLAG and 35S:CBL-GFP-FLAG expression in silenced plants. The bottom panel is a gel stained with coomassie brilliant blue (CBB) to show protein loading. B) The cell death phenotype caused by infiltration with *CRK28*-FLAG and *RPS2-FLAG* in *NbSerk3* silenced and *TRV:GUS* infiltrated *N. benthamiana* plants. The numbers below the picture represent the number of leaves that showed weak or strong cell death per total number of leaves infiltrated. These experiments were repeated at three times with similar results. C) Anti-FLAG immunoblot verifying the expression of CRK28-FLAG and RPS2-FLAG in silenced and control plants at 20hpi. CBB = coomassie brilliant blue.