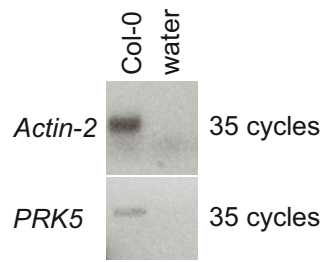


**Fig. S1. A 20 aa peptide derived from GRI is sufficient to induce cell death in *Arabidopsis* leaves.**

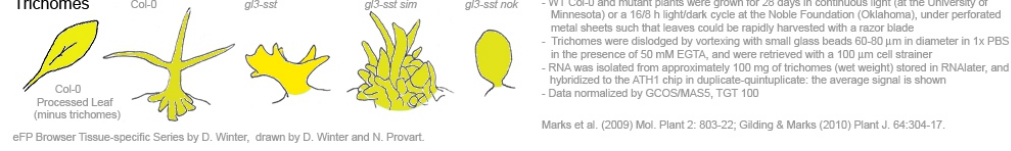
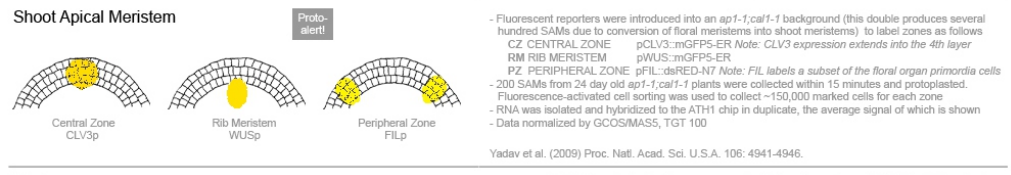
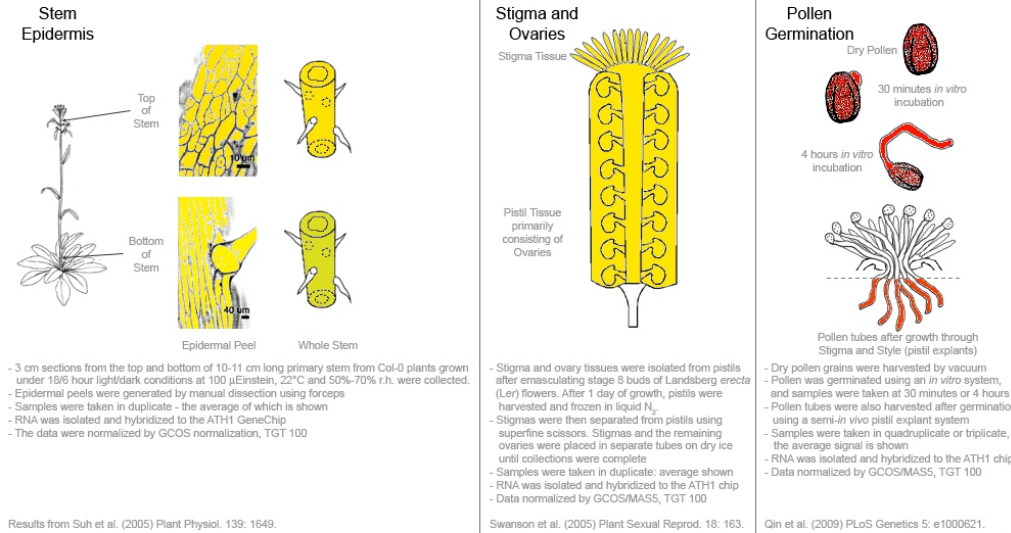
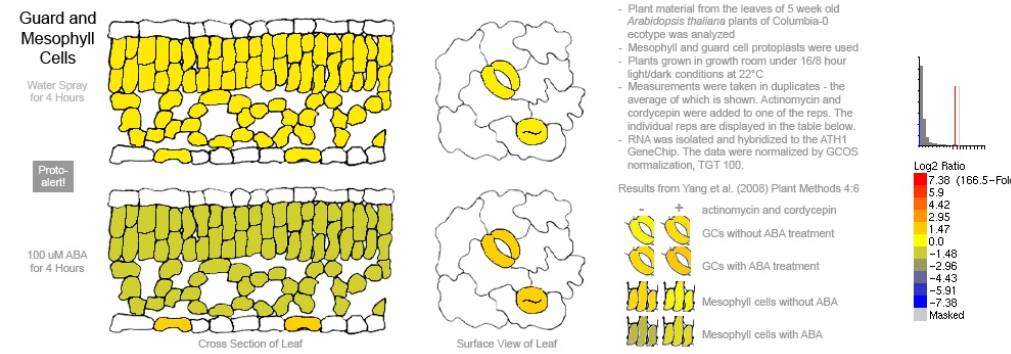
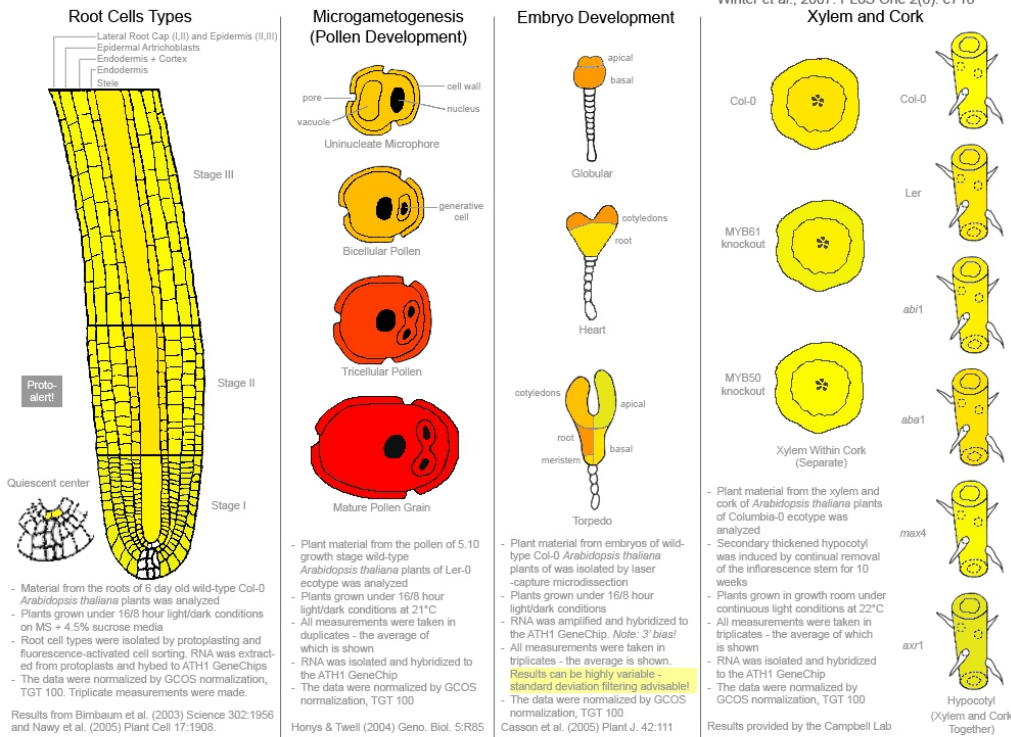
**A** Schematic representation of GRI protein and GRI-peptides. Sequences of GRip<sup>31-96</sup>, GRip<sup>31-51</sup>, GRip<sup>47-68</sup>, GRip<sup>65-84</sup> and GRip<sup>80-96</sup> are shown underneath.

**B** Col-0 leaves were infiltrated with 37 nM GST, GRip<sup>31-96</sup> or GRip<sup>65-84</sup> and subsequently stained with Trypan blue. Pictures were taken under a light microscope at 4x and 10x magnification, respectively.

**Data information:** The experiment has been repeated six times with similar results.



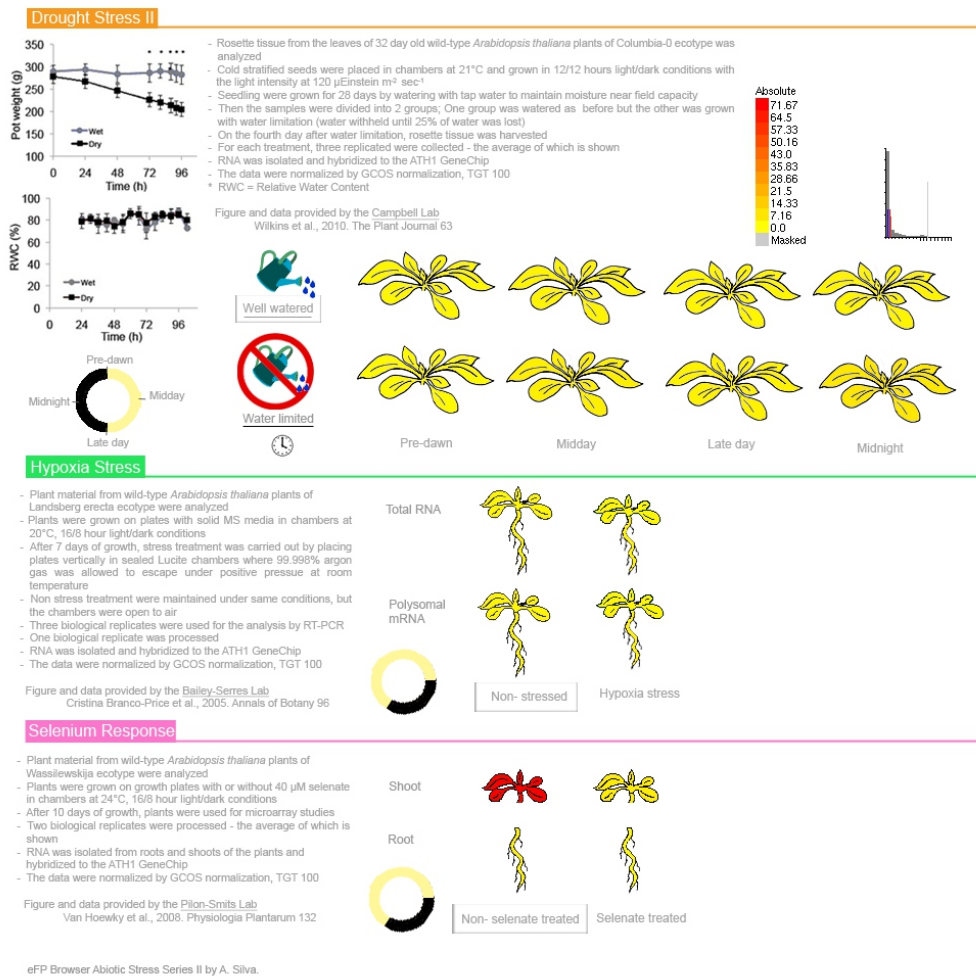
**Fig. S2. *PRK5* transcript is present in low levels in the leaves of wild type *Arabidopsis* Col-0 plants.**



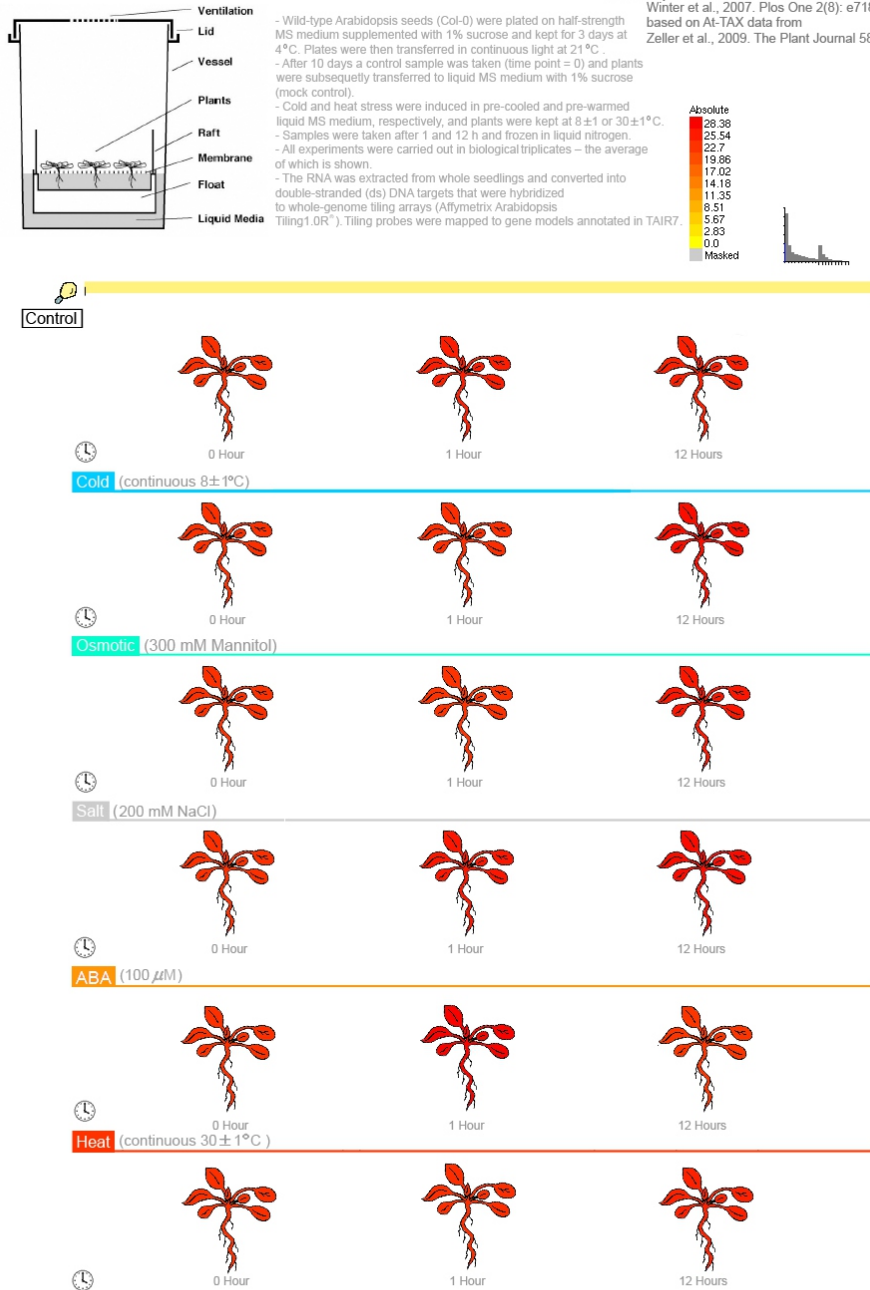
**Fig. S3. Expression of PRK5 transcript Arabidopsis tissues (eFP browser, developmental map; <http://bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi>).**



**Fig. S4. *PRK5* transcript levels are altered in response to a variety of abiotic stresses in *Arabidopsis* (eFP browser, Abiotic stress I; <http://bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi>).**

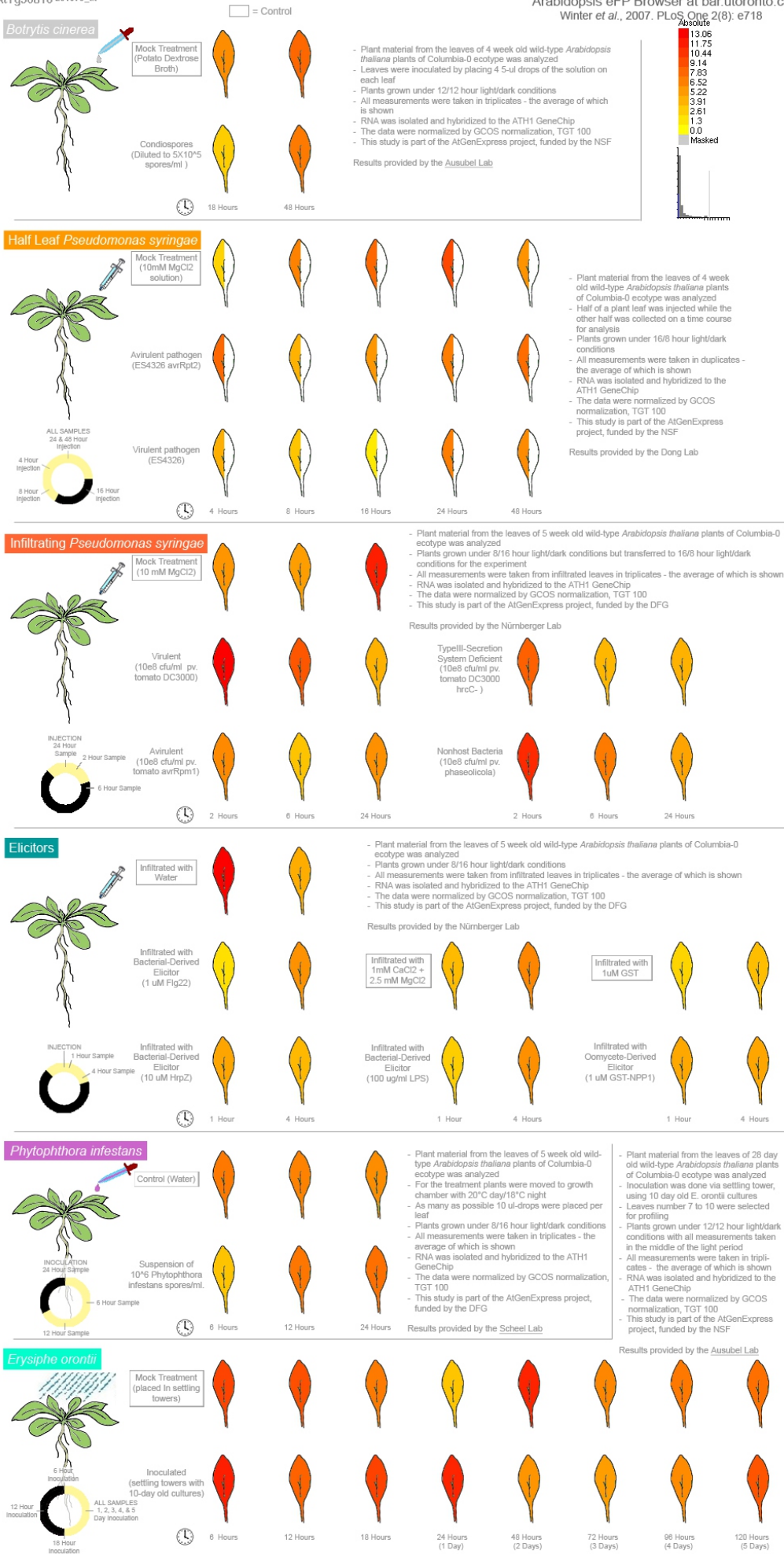


**Fig. S5. *PRK5* transcript levels are altered in response to a variety of abiotic stresses in *Arabidopsis* (eFP browser, Abiotic stress II; <http://bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi>).**



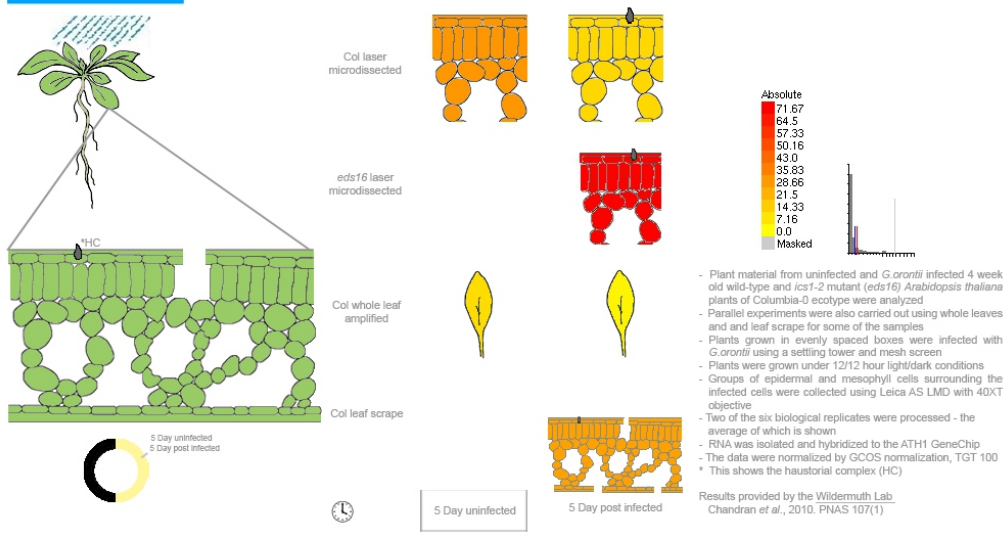
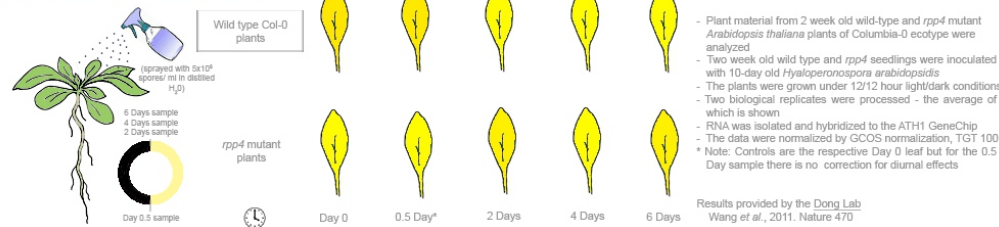
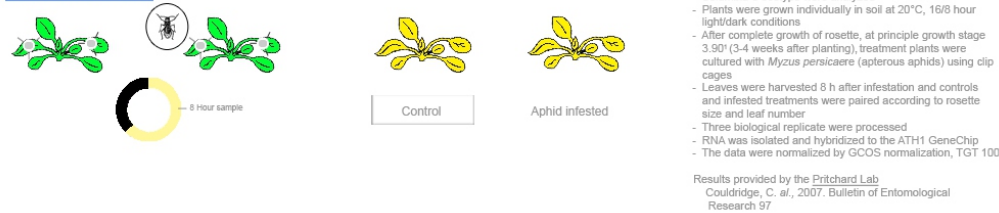
eFP Browser Stress Series by B. Vingar, D. Winter, and Y. Ji, drawn by D. Winter and Y. Ji. Data from At-TAX Abiotic Stress Series from Zeller et al., (2009, Plant J. 58: 1068-82)

**Fig. S6. *PRK5* transcript levels are altered in response to a variety of abiotic stresses in *Arabidopsis* (eFP browser, Abiotic stress At-TAX; <http://bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi>).**



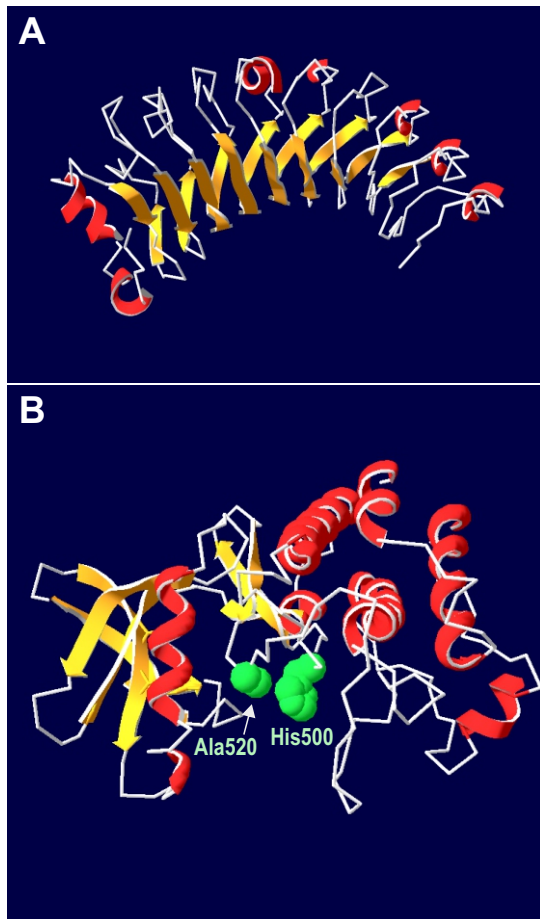
eFP Browser Stress Series by B. Vinegar and D. Winter. Data from AIGenExpress Pathogen Series.

**Fig. S7. PRK5 transcript levels are altered in response to pathogen infection in *Arabidopsis* (eFP browser, Biotic stress I; <http://bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi>).**

**Golovinomyces orontii****Hyaloperonospora arabidopsidis****Myzus persicaere**

**Fig. S8. *PRK5* transcript levels are altered in response to pathogen infection in *Arabidopsis* (eFP browser, Biotic stress II; <http://bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi>).**



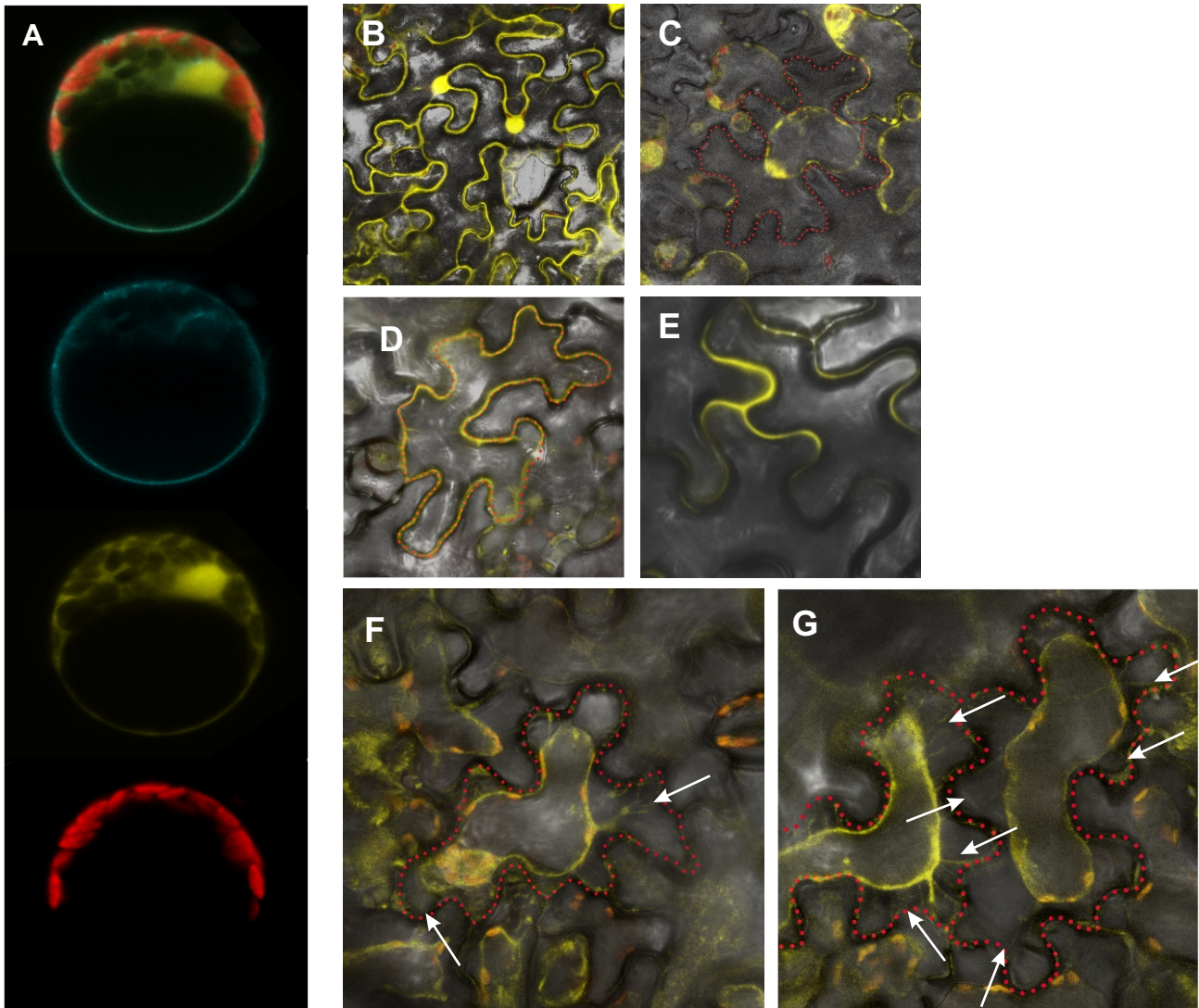


**Fig. S9. Structural prediction of PRK5 extracellular and intracellular domains.**

**A** I-Tasser prediction of the extracellular domain of PRK5 (residues 40-281).

**B** Phyre<sup>2</sup> prediction of the intracellular kinase domain of PRK5 (residues 375-645).

Histidine residue 500 and alanine residue 520 are shown in green.



**Fig. S10. PRK5-YFP localises to the plasma membrane in *Arabidopsis* protoplasts and in *Nicotiana benthamiana*.**

**A** Localization of PRK5-CFP and cytosolic YFP in Col-0 mesophyll protoplasts. Panels from top to bottom: overlay, PRK5-CFP, YFP, chloroplast.

**B** YFP (expressed under the control of the 35S promoter) localises to the cytosol and nucleus in epidermal cells of *Nicotiana benthamiana*.

**C** After plasmolysis with 0.8 M NaCl for 15 minutes YFP is visible in the cytosol and nucleus in *Nicotiana benthamiana* epidermal cells.

**D, E** PRK5-YFP (expressed under the control of the 35S promoter) localises to the plasma membrane in *Nicotiana benthamiana* epidermal cells.

**F, G** After plasmolysis with 0.8 M NaCl for 15 minutes PRK5-YFP is visible in the plasma membrane in *Nicotiana benthamiana* epidermal cells as indicated by the appearance of Hechtian strands (see arrow heads).

**Data information:** Experiments in this figure have been repeated three times with similar results.

**A**

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PRK5  -ASAENVLGGTFFGASYYKAAISSG-QTLVVK--RYKHMNNVGRDEFHEHMRRLGRNLHPNI 56
PRK4  -ASAENVLGGSGFGSSYKGTGINSQ-QMLVVK--RYKHMNNVGRDEFHEHMRRLGRNLKHPNL 56
BIR2  -----IVSTRGTGTYKALLPDG-SALAVK--HLSTCK-LGEREFYEMNQWLWELRHSNL 50
BRI1  FHNDSLIGSGGFGDVYKAILKDG-SAVAIAK--KLIHVSGQGDREFFMAEMETIGIKHRNL 57
FLS2  FNSANLIGSSSLSTVYKQLEDG-TVIAVVLNKLKEFSAESDKWFYTEAKTSLQKLRNL 59
EFR   FSSNTLIGSGNFGNVFKGLLGPENKLVAVKVLNLLKHGATKS--FMAECETFFKGRHRNL 58
CRK7  FSENNKIGRGGFGDVYKGTFSNG-TEVAVKRLSKTS--EQGDTEFKNEVVVVANLRHKNL 57
SUB   FSEENII GEGSIGNVYRAELRHG-KFLAVKLSNTINRTQSDGEFLNLSVNLKLRGHI 59
      :      .      :      :      :      :      :      :      :      :      :
      :      :      :      :      :      :      :      :      :      :

PRK5  LPLVA-----YYYRREEKLLVTEFMPNSSLASHLHANNSA-----GLDWITRLKIIKG 104
PRK4  LPIVA-----YYYRREEKLLIAEFMPNRS LASHLHANHSVDQP-----GLDWPTRLKIIQG 107
BIR2  APLL-----PCVVEEEKFLVYKYMSNCTLHSLLDNSRGE-----LDWSTRFRIGLG 97
BRI1  VPLL-----YCKVGDRLVVEFMKYGSLEDVLDHPKKAG-V-----KLNWSTRRRIATG 107
FLS2  VKIL-----FAWESGKTALVLPFMEGNLEDTHGSAAPIG-----SLEKIDLCVH 108
EFR   KGLITVCSLDSGNDFRALVVEFMKPGSLDMWLQLEDLERNVDHSRSLTPAEKLNIAD 118
CRK7  VRILG-----FSIEREERILVVEYVENKSLDN-FLFDPAKKGQ-----LYWTQRYHIIGG 106
SUB   LLLG-----YCNEFQORLIVVEYCPNGSLQDALHLDRKLHKK-----LTWNVRIIALG 109
      :      :      :      :      :      :      :      :      :      :
      :      :      :      :      :      :      :      :      :      :

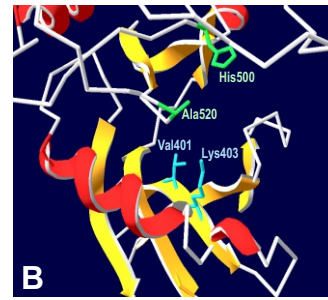
PRK5  VAKGLSYLFDLPLTLTI PHGHMKSSNIVLDDSFEPLLT DVALRPMMS---SEHAHN--FM 159
PRK4  VAKGLSYLFDLPLTLTI PHGHMKSSNIVLDDSFEPLLT DVALRPMMS---SEQSHN--LM 162
BIR2  AARGLAWLHHGCR-PPI LHQNICSSVILIDEDFDARI IDSGLARLMV---PSDNNESSFM 153
BRI1  IASGDLAFLLHNCSS-PHI IHRDMKSSNIVLLENLEARVSDDFC MARLMS---AMDTHLSVST 163
FLS2  IASGIDVLLHSGYG-FPIVHCDLKPANILLSDRVAHVSDDFC TARILG--FREDGSTTAST 165
EFR   VASALEYLHVHCH-DPVAHCDIKPSNILLDDTLAHVSDDFC LAQLLYKYDRESFLNQFSS 177
CRK7  IARGILYLLHQDSR-LTI IHRDLKASNILLDADMPNPKVADDFC MARIFG---MDQQTQNT 160
SUB   ASKALQFLHEVCQ-PPVVHQNFKSSKVLDDGKLSVNVADSGLAYMLP-----PRPTSQMA 163
      :      :      :      :      :      :      :      :      :      :
      :      :      :      :      :      :      :      :      :      :

PRK5  T-----AYKSPPEYRPSKQIITKKTDVWCQFVLLILEVLTGRFPENYLTQGYDSNMSLVT 213
PRK4  I-----SYKSPPEYS-LRG-HLTKKTDVWCQFVLLILELTLGRFPENYLSQGYDANMSLVT 214
BIR2  TGDGLGFEFGYVAPPEYS--TTMLASLKGVDYGLGVVLELATG--LKAVGGEGFKG--SLVD 207
BRI1  LAG--TPGVVPEYY--QSFRCTKGDVYSYGVVLELTLGKRPTDSDPFDDNN---LVG 216
FLS2  SAFEGTIGYLAPEFAMRK--VTTKADVFSFGIIMMELMTKQRPDSDNDESDQ-MTLRQ 222
EFR   AGRGTIGYLAPEYGMGQ--PSIQGDVYSFGILLLEMFSGKKPT--DESPAGD-YNLHS 232
CRK7  SRIVGTGYMSPPEYAMRGQ--FSMSKDVYSFGVLEIISGRKNNS-FIETDDA-QDLVT 216
SUB   G-----YAAPEVEY-GS--YTCQSDVFSLGVVMELELTGRRPFD-RTRPRGH-QTLAQ 211
      *      *      :      :      :      :      :      :      :      :      :

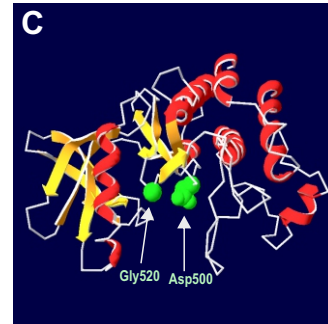
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PRK4  WVSNMVKE---KKTGDVFD-KEMTGGKKNCAEMINLLKIGLSCCEDEEERRMEMRDAVE 269
BIR2  WVKGLESS---GRIAEFFD-ENIRGK-GHDEEISKFVEIALNCVSSRPKERWSMFQAYQ 261
BRI1  WVKQHAK---LRI SDVFDPELMKEDPALEIQLHLVAVACLDRAWRRPTMVFQVMA 271
FLS2  LVEKSI GNRKGMVRLDMELGDSIVSLKQEAIEDFLKLCFCTSSRPEDRPDMNEILT 282
EFR   YTKSILSG-----CTSSGGSNAIDGLRLVLQVGIKCSSEYPRDRMRMTDEAVR 280
CRK7  HAWRLWRN-----GTALD-LVDVPIADSCRKSEVVRCTHIGLLCVQEDPVKRPAMTISV 270
SUB   WAI PRLHD-----IDALTRMVDPSLHGAYPMKSLSRFADIIISRSLQMEPGFRPPISEIVQ 266
      .      :      :      :      :      :      :      :      :      :

PRK5  MV----- 270
PRK4  KI----- 271
BIR2  SL----- 263
BRI1  MF----- 273
FLS2  HL----- 284
EFR   EL----- 282
CRK7  MLTSNTMALPAPQPGF 287
SUB   DLQHMI----- 272

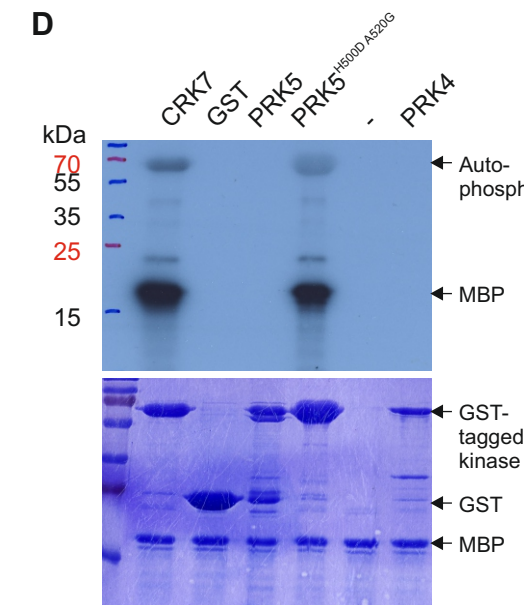
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**B** PRK5 ATP binding site (Phyre<sup>2</sup> model)



**C** PRK5<sup>H500D A520G</sup> kinase domain (Phyre<sup>2</sup> model)



**Fig. S11. Conservation of the kinase domain and *in vitro* kinase activity of PRK5 and PRK5<sup>H500D A520G</sup> kinase domains.**

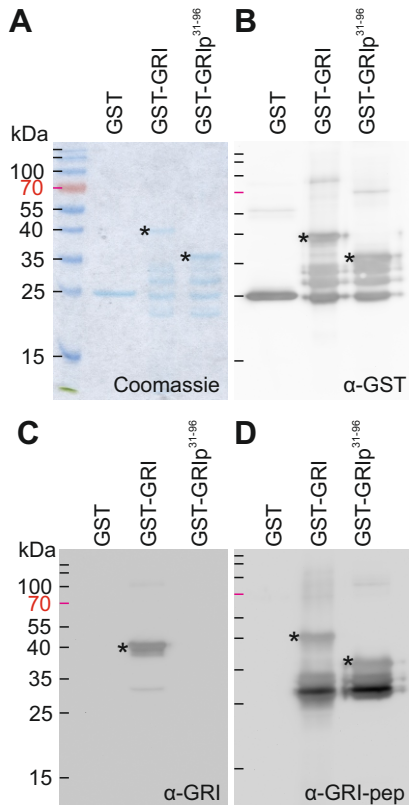
**A** Alignment of kinase domains of the inactive kinases PRK4, PRK5, BIR2 as well as SUB with the active kinase domains of BRI1, FLS2, EFR and CRK7.

**B** Phyre2 prediction of the catalytic core of the intracellular kinase domain of PRK5. Histidine (H) at position 500 and alanine (A) at position 520 are shown in green.

**C** Phyre2 prediction of the intracellular kinase domain of PRK5; introduced mutations H500D and A520G. D and G in the catalytic core are highlighted in green.

**D** *In vitro* phosphorylation assays of GST-PRK5 kinase domain in the presence of 10 mM MgCl<sub>2</sub>. GST-PRK5 and GST-PRK4 did not show kinase activity while mutation of H500D and A520G restored GST-PRK5<sup>H500D A520G</sup> kinase activity. GST-CRK7 was used as a positive control. Upper panel shows autoradiograph, lower panel shows the Coomassie-stained 15% SDS-polyacrylamide gel. Arrows indicate the GST-tagged RLK kinase domains, the size of free GST and myelin basic protein (MBP; used as artificial substrate).

**Data information:** Experiments have been repeated three times with similar results.



**Fig. S12. Western blot of GST, GST-GRI and GST-GRip<sup>31-96</sup>.**

**A** Coomassie brilliant blue stained polyacrylamide gel of bacterially produced GST, GST-GRI (asterisk) and GST-GRip<sup>31-96</sup> (asterisk).

**B** Western blot of bacterially produced GST, GST-GRI (asterisk) and GST-GRip<sup>31-96</sup> (asterisk) with α-GST antibody.

**C** Western blot of bacterially produced GST, GST-GRI (asterisk) and GST-GRip<sup>31-96</sup> with α-GRI antibody.

**D** Western blot of bacterially produced GST, GST-GRI (asterisk) and GST-GRip<sup>31-96</sup> (asterisk) with α-GRI-pep antibody.

**E** Sequence alignment of PRK5 and PRK4 protein sequences.

**F** Interaction of GST-GRI and GST-GRip<sup>31-96</sup> with the ectodomain of FLS2. GST-GRip<sup>31-96</sup> showed strongest interaction with the ectodomain of PRK5 and weak interaction with PRK4. No interaction was detected with the ectodomain of FLS2.

**E**

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PRK5 MRNWEDPFTLACNTALKKNLPSCIFIIIFISVLCVPVAMSQ-----VVVPDSADADCLLRFK 55
PRK4 MLTWETPVMLASNTASTKKLAFITFTFLIIIVLCPTVMVMSQPQADVLPASADADCLLRFK 60
* . * * * . * * * * * . * * * * . : * * * * * : * * * * * * * * * * *

PRK5 DTLANGSEFRSVDPLSSPCQGNANTANWFVGLCSN-YVWGLQLEGMGLTGKLNLDPLVPMKN 114
PRK4 DTLVNASFISWDPISISCKRNSENWFVGLCVTGNVWGLQLEGMGLTGKLDLEPLAAIKN 120
* * * . * * : * * * * * * * * * * * . * * * * * * * * * * * * * * * * *

PRK5 LRTISFNMNNFNGPMPQVKRFTSLKSLYLSNNRFSGEIPADAFGLMPLLKKILLANNAFR 174
PRK4 LRTLSEFMNKNFNGMSPVKNFGALKSLYLSNNRFTGEIPADAFDMHHLKLLANNAFR 180
* * * : * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

PRK5 GTIPSSLASLPLMLELRNLGNQFQGQIPSFQKDLKLSFENNDLGGPIPESLRNMDDPGS 234
PRK4 GSIPSSLAYLPLMLELRNLGNQFHGEIPIYFKQDLKLSFENNDLEGPPELSNMDPVS 240
* . * * * : * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

PRK5 FAGNKGLCDAPLSPCSSSPGVPVVPVSPVDPKSTSPPTGKKAGSFYTLAIILVIGIIL 294
PRK4 FSGNKNLCGPPLSPCSSDSGSSPDLSPSPEKN-----KNQSFFTIAIVLIVIGIIL 292
* : * * * . * . * * * * * . * : * * * : : * * * : * * * * * * * * * *

PRK5 VI IALVFCFVQRRRNFLSAYPSSAGKERIESYNYHQSTNKNNKPAESVN--HTRRGSM 352
PRK4 MI ISLVVCILHTRRRKLSAYP-SAGQDRTEKYNDQSTDKD-KAADSVTSYTSRRGAVP 350
: * * * . * . : : * * * : * * * * * * * * * * * * * * * * * * * * *

PRK5 DPGGRLLFVRDDIQRFDLQDLLRASA EVLGSGTFGASYKAAISSGQTLVVKRYKHMNVG 412
PRK4 DQN-KLLFLQDDIQRFDLQDLLRASA EVLGSGSFGSSYKTGINSQMLVVKRYKHMNVG 409
* . : * * * : * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

PRK5 RDEFHEHMRRRLGRLNHPNLLPLVAYYYRREEKLLVTEFMPNSSLASHLHANNSA---GLD 469
PRK4 RDEFHEHMRRRLGRLKHPNLLPIVAYYYRREEKLLIAEFMPNRSASHLHANHSVDQPGLD 469
* * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

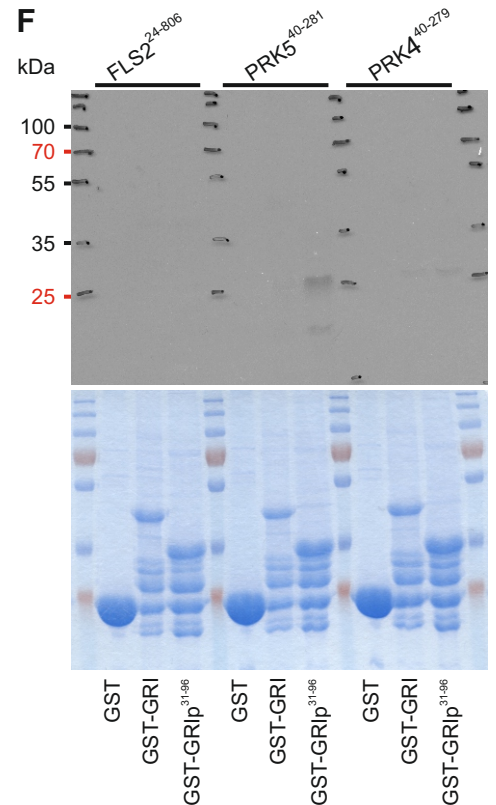
PRK5 WITRLKIIKGVAKGLSYLFDLPTLTIIPHGHMKSSNIVLDDSFEPDLLTDYALRPMMSSEH 529
PRK4 WPTRLKIIQGVAKGLGYLFNELLTTLTIIPHGLKSSNVVLDSEFEPDLLTDYALRPMVNSEQ 529
* * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

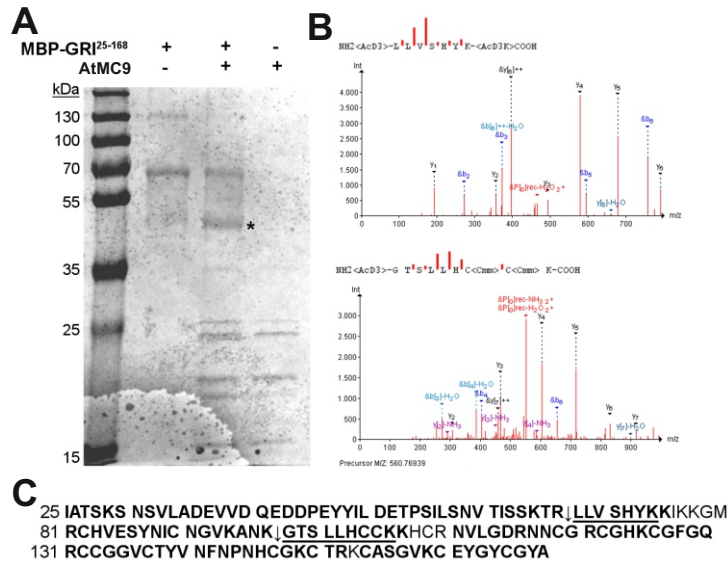
PRK5 AHNFMAYKSPPEYRPSKQIITKKTDVVCFVGLILEVLVTGRFPENYLTQGYDSNMSLVTW 589
PRK4 SHNLMISYKSPPEYS-LKG-HLTKKTDVWCLGVLELLTGRFPENYLSQGYDANMSLVTW 587
: * * * : * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

PRK5 VNDMVKEKKTGDVFDKEMKGNCKAEMINLLKIGLRCEEEEEERRMDREVVEMVEMLR 649
PRK4 VSNMVKEKKTGDVFDKEMTGNCKAEMLNLLKIGLSCCEDEERRMEMRDAVEKIERLK 647
* . : * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

PRK5 EGESEDDFGSMDRHGTNNVYSMLLDDDDDFGFSMNR 686
PRK4 EGEFDNDFASTTH-----NVFASRLIDDDDFGFAMNR 679
* * * : * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

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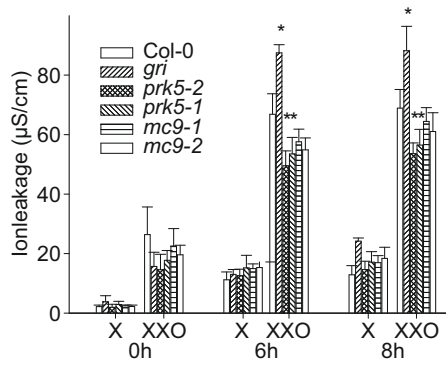
**Fig. S13. GRI is cleaved by AtMC9 after K65, R67, K78 and K97, as revealed by *in vitro* cleavage assays and mass spectrometry analysis.**

**A-C** Cleavage of recombinant MBP-GRI<sup>25-168</sup> fusion protein by recombinant AtMC9, followed by trypsin treatment and LC-MS/MS analyses using a Thermo LTQ Orbitrap XL mass spectrometer.

**A** Coomassie stain of representative samples on a 12% SDS-PAGE. (\*) After 90 min incubation at 30°C, a clear lower molecular weight band of MBP-GRI<sup>25-168</sup> was visible after proteolysis by AtMC9.

**B** After AtMC9 treatment, cleavage products of MBP-GRI<sup>25-168</sup> were N-terminally labeled in solution with trideutero- acetyl (<AcD3>). The annotated spectra covering the neo N-terminal peptides NH<sub>2</sub><AcD3>-68LLVSHYK74 (Mascot score 60; Identity threshold score 33) and NH<sub>2</sub><AcD3>-98GTSLLHCCKK107 (Mascot score 47; Identity threshold score 32) are shown.

**C** Position of the AtMC9 cleavage sites (↓) in the GRI<sup>25-168</sup> amino acid sequence. The identified <AcD3>-labelled peptides are underlined. Peptide coverage of the protein (93,7%) is represented in bold. Spectra of the <AcD3>-labeled peptides were visualized by Peptizer (Helsens *et al.*, 2008).

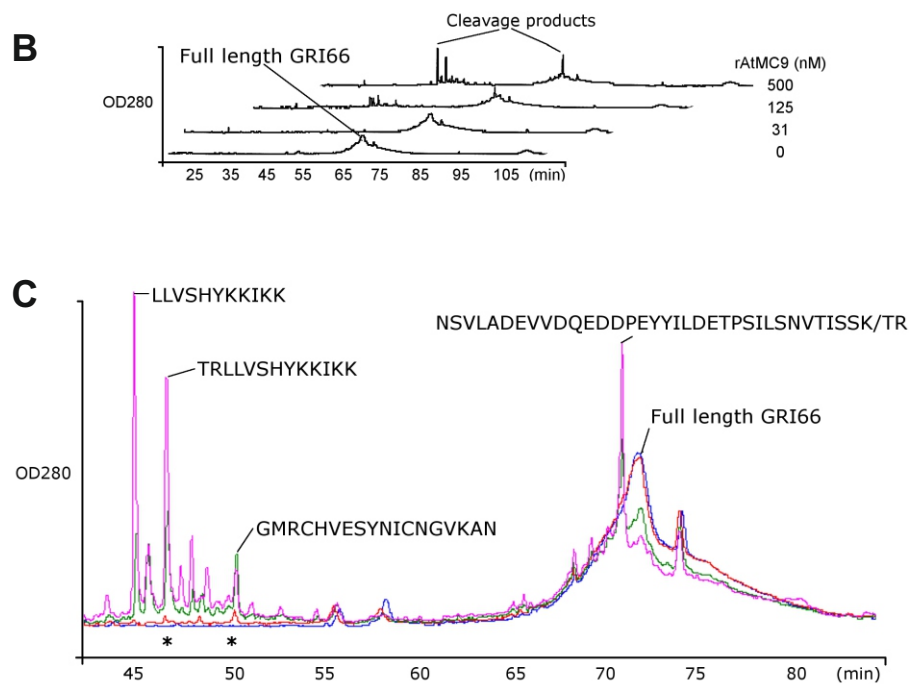


**Fig. S14. Xanthine/xanthine oxidase infiltration of Col-0, *gri*, *prk5* and *atmc9* plants.**

Enzymatic superoxide production from xanthine/xanthine oxidase (XXO) infiltration into Col-0, *prk5-1*, *prk5-2*, *atmc9-1*, *atmc9-2* and *gri* leaves ( $\pm$ SD of 4 replicates consisting of 4 leaf disks each). X is buffer control containing xanthine without xanthine oxidase.

**Data information:** Asterisks mark statistically significant differences from infiltration with GST according to Sidak's test ( $P < 0.05$ ). The experiment was repeated three times with similar results.

**A** NSVLADEVVDQEDDPEYYILDETSPILSNVTISSK↓TR↓LLVSHYKKIKK↓GMRCHVESYNICNGVKAN



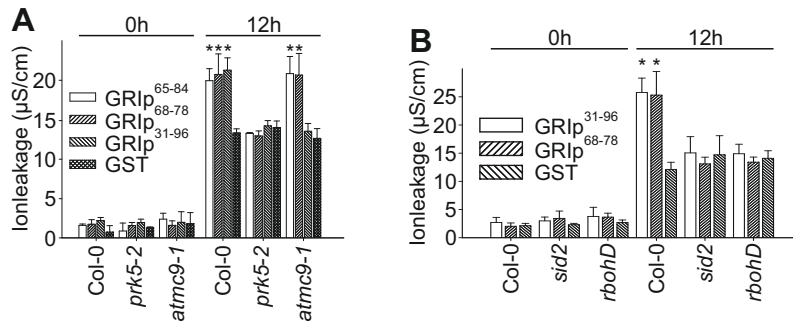
**Fig. S15. GRI is cleaved by AtMC9 after K65, R67, K78 and K97, as revealed by *in vitro* cleavage assays and mass spectrometry analysis.**

**A-C** Cleavage of biochemically pure GRIp<sup>31-96</sup> peptide (66 AA) by recombinant AtMC9, followed by immediate separation of cleavage products by reverse-phase chromatography (RP-HPLC) and size analysis by MALDI-TOF MS (Ultraflex, Bruker).

**A** GRIp<sup>31-96</sup> peptide was incubated with increasing concentrations of recombinant AtMC9 (rAtMC9) and cleavage products were separated on a C18 reverse-phase column. Cleavage sites in the GRIp<sup>31-96</sup> sequence are indicated with (↓). The experiment was performed twice with similar results.

**B** 3D view of the RP-HPLC UV absorbance chromatograms corresponding to the four concentrations of rAtMC9 used (as indicated in the figure).

**C** Overlapping view of the four RP-HPLC UV absorbance chromatograms indicating the peptide sequence of the main cleavage products (peptide mass was deduced by MALDI-TOF MS). The blue trace corresponds to 0 nM rAtMC9 sample, red trace to 31 nM rAtMC9 sample, green trace to 125 nM rAtMC9 sample and pink trace to 500 nM rAtMC9 sample. The first cleavage products to appear (highest in red trace and green trace) are indicated with asterisks. The mass of the full length GRIp<sup>31-96</sup> peptide was deduced by LC-MS/MS using a Thermo LTQ Orbitrap XL mass spectrometer, because it was not possible to deduce the mass by MALDI-TOF MS. The slash (/) in NSVLADEVVDQEDDPEYYILDETSPILSNVTISSK/TR indicates that both the longer (cleavage after R) and shorter (cleavage after K) cleavage products were present in the peak.



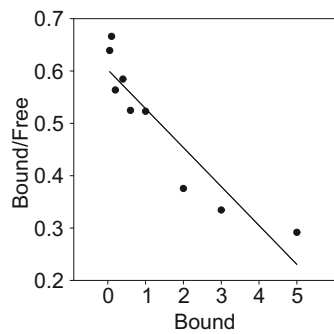
**Fig. S16. Cell death by GRI-peptides is dependent on salicylic acid production and extracellular superoxide production.**

**A** Infiltration of wild type, *prk5-2* and *atmc9-1* leaves with GRIp<sup>31-96</sup>, GRIp<sup>65-84</sup>, GRIp<sup>68-78</sup> or GST. GRIp<sup>65-84</sup> and GRIp<sup>68-78</sup> but not GRIp<sup>31-96</sup> were able to induce elevated ion leakage in the *atmc9-1* mutant ( $\pm$ SD of 4 replicates of 4 leaf disks each).

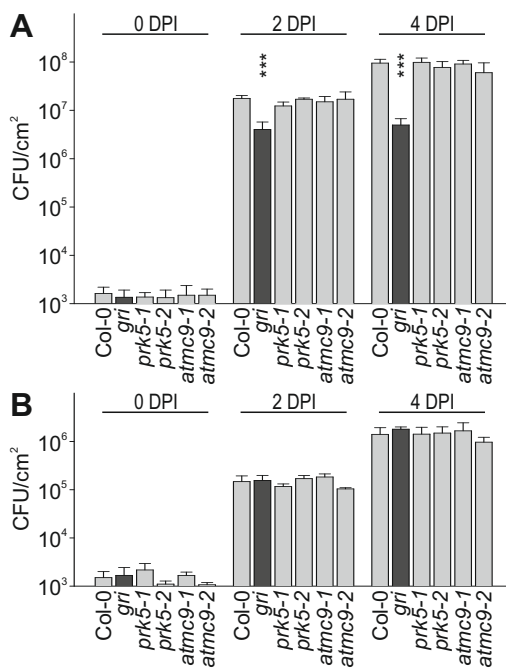
**B** Infiltration of wild type, *sid2* and *rbohD* leaves with 66 aa-long GRIp<sup>31-96</sup>, 11 aa-long GRIp<sup>68-78</sup> or GST. GRIp<sup>31-96</sup> and GRIp<sup>68-78</sup> were able to induce elevated ion leakage in Col-0 but not in the mutants *sid2* and *rbohD* ( $\pm$ SD of 4 replicates of 4 leaf disks each).

**Data information:** Asterisks mark statistically significant differences from infiltration with GST according to Sidak's test ( $P < 0.05$ ). The experiments were repeated three times with similar results.





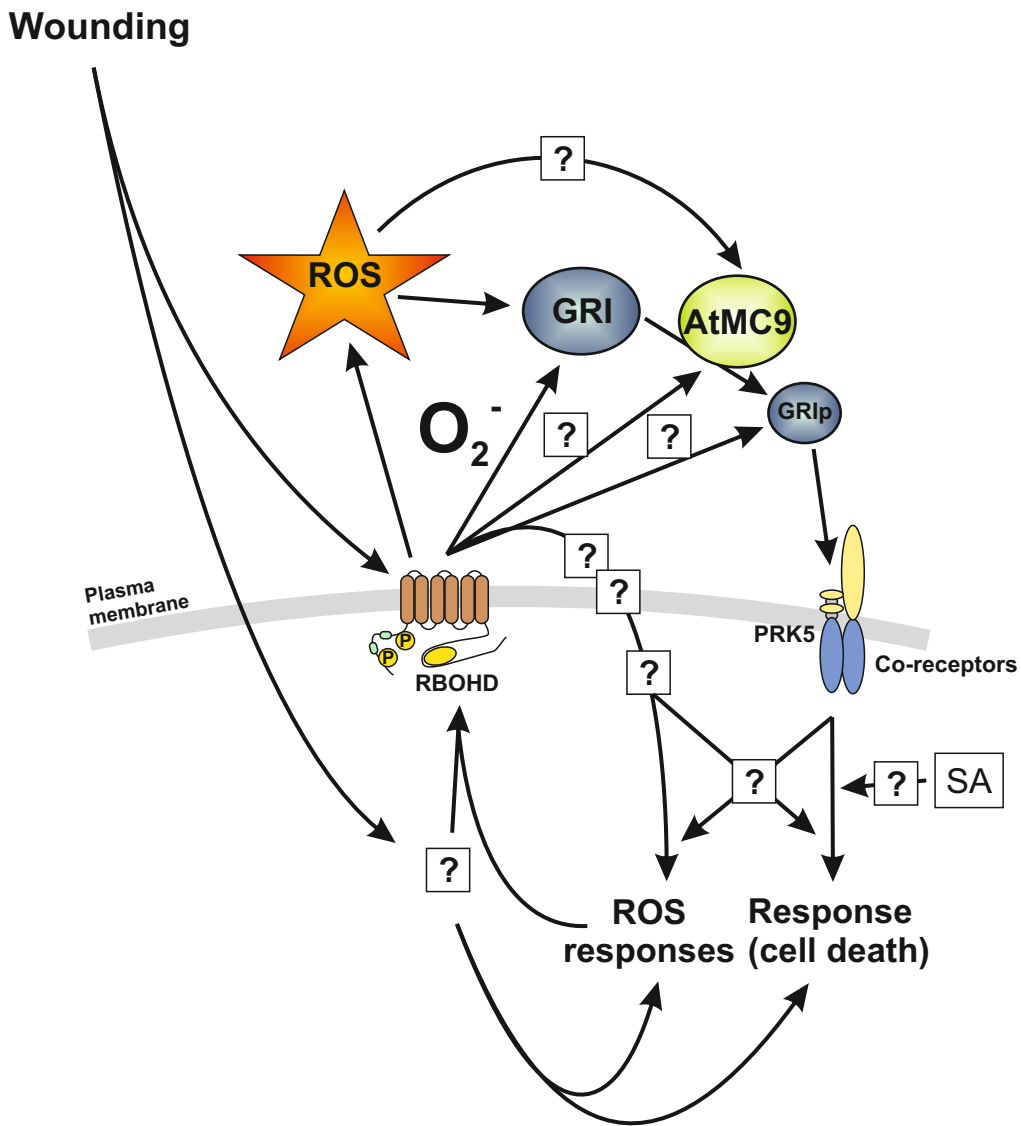
**Fig. S17. Scatchard plot from data in Fig. 4G.**



**Fig. S18. Pathogen susceptibility in *gri*, *prk5* and *atmc9* plants.**

4-week old plants were hand-infiltrated with 10<sup>5</sup> CFU/ml of *Pseudomonas syringae* pv. *tomato* (*Pto*) DC3000 (**A**) or DC3000 avrB (**B**), respectively. Subsequently, 4 leaf disks (total area 1 cm<sup>2</sup>) were homogenized in 10 mM MgCl<sub>2</sub> and the bacterial count was determined by dilution plating one hour, two days and four days after infiltration.

**Data information:** Bars represent mean and error bars represent standard deviation of 3 replicates of 4 leaf disks each. Asterisks mark statistically significant differences from bacterial growth in Col-0 according to two-way anova with Tukey's HSD post-hoc test ( $P < 0.001$ ). For **A** bacterial growth in all lines is significantly different after two days from zero days, and for Col-0, *prk5-1*, *prk5-2*, *atmc9-1* and *atmc9-2* different at four days from two days post-infection. For **B** bacterial growth in all lines is significantly different after two days from zero days, and after four days from two days post-infection. All experiments were repeated three times with similar results.



**Fig. S19. Model of GRI function in ROS-induced cell death regulation.**

Stress (for example wounding through peptide infiltration with a blunt syringe) leads to reactive oxygen species (ROS) production in the extracellular space by the NADPH oxidase RBOHD (producing superoxide,  $O_2^-$ ). The secreted GRI protein is cleaved by AtMC9 and the resulting peptide (GRIP) is subsequently perceived by the enzymatically inactive RLK PRK5. It is currently unclear at what level ROS signalling interacts with GRI processing and perception. Alternatively signalling through ROS and GRI could represent parallel pathways which are only integrated at the intracellular response level. Salicylic acid (SA) is required for cell death induced by GRIP infiltration but the details of this interaction are currently not known. Co-receptors that are required for PRK5 action are hypothetical.