A common bean truncated CRINKLY4 kinase controls gene-for-gene resistance to the fungus *Colletotrichum lindemuthianum*

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Running title: **A truncated CRINKLY4 kinase controls resistance to a fungus**

30   **Highlight**

31   CRINKLY4 receptor-like kinases have been mainly reported to be involved vascular

32   plant development. Here we report an unusual truncated CRINKLY4 kinase, with an

33   unexpected role in resistance against a pathogen.

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35

36   **Abstract**

37   Identifying the molecular basis of resistance is critical to promote chemical free

38   cropping system. In plants, NLR constitute the largest family of disease resistance (*R*) genes

39   but they can be rapidly overcome, prompting research of alternative source of resistance.

40   Anthracnose, caused by the fungus *Colletotrichum lindemuthianum*, is one of the most

41   important diseases of common bean. This study aimed to identify the molecular basis of *Co-x*,

42   an anthracnose *R*-gene conferring total resistance to the extremely virulent *C. lindemuthianum*

43   strain 100. To that end, we sequenced the *Co-x* 58kb target region in the resistant JaloEEP558

44   (*Co-x*) and identified KTR2/3, an additional gene encoding a truncated and chimeric

45   CRINKLY4 kinase, located within a CRINKLY4 kinase cluster. KTR2/3 presence is strictly

46   correlated with resistance to strain 100 in a diversity panel of common beans. Furthermore,

47   KTR2/3 expression is upregulated 24 hours post-inoculation and its transient expression using

48   *Agrobacterium*-transformation in susceptible genotype increases resistance to strain 100.

49   Altogether, our results provide molecular evidence that *Co-x* encodes a truncated and

50   chimeric CRINKLY4 kinase probably resulting from an unequal recombination event that

51   occurred recently in the Andean domesticated gene pool. This atypical *R*-gene might act as a

52   decoy involved in indirect recognition of a fungal effector.

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54   **Keywords:** Common bean, *Phaseolus vulgaris*, NLR, disease resistance gene,

55   CRINKLY4 kinase, anthracnose, unequal crossing-over

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Introduction

Plants are under constant pathogen challenge. However, successful infection is not an easy accomplishement for the potential pathogens that have to overcome several plant immunity layers. A first layer of immunity resides in the recognition of Microbe/Pathogen-Associated Molecular Pattern (MAMP or PAMP) by plant Pattern Recognition Receptor (PRR), the so-called PAMP-Triggered Immunity (PTI) (Bigeard *et al.*, 2015). PRR are surface-localized ligand-binding transmembrane proteins from the Receptor-Like Protein (RLP) or Receptor-Like Kinase (RLK) families (Macho and Zipfel, 2014). Although PTI can keep a number of pathogens at bay, some adapted pathogens can counter this first defense layer by releasing effectors in the extracellular matrix or into the plant cell (Toruño *et al.*, 2016). A second layer of plant immunity, called Effector-Triggered Immunity (ETI), is based on the recognition of these effector molecules, that were originally referred to as avirulence (Avr) proteins, by the product of plant Resistance genes (*R* genes) (Jones *et al.*, 2016). Strikingly, regardless of the plant, the type of pathogen or the diversity of pathogen Avr proteins, the majority of *R* genes cloned to date encode intracellular Nucleotide-binding Leucine-rich repeat Receptors (NLR) (Baggs *et al.*, 2017; Jones *et al.*, 2016). NLRs can be divided into two major sub-groups by their N-terminal domain: the CNLs, with a Coiled Coil (CC) domain and the TNLs, with a Toll/Interleukin-1 Receptor (TIR) domain (Monteiro and Nishimura, 2018). NLR-based immunity often triggers a localized cell death known as the hypersensitive response (HR) (Balint-Kurti, 2019).

It has initially been proposed that NLRs recognize pathogen Avr proteins *via* direct interaction (Keen, 1990). However, cases of direct interaction have been demonstrated for only few R-Avr combinations, such as for the TNL N from tobacco that directly recognizes the *Tobacco mosaic virus* (TMV) protein p50 (Ueda *et al.*, 2006) or for the TNLs L5, L6 and M from flax which recognize the Avr proteins AvrL567 and AvrM, respectively, from *Melampsora lini*, the agent of flax rust (Catanzariti *et al.*, 2010; Dodds *et al.*, 2006). Since no physical interaction has been observed for many R-Avr pairs, and because effectors/Avr have a role in promoting infection by manipulating virulence target, another model of indirect recognition of the Avr by the R protein emerged. In this model, called the guard model, the R protein recognizes modified-self (the modification of a plant virulence target by the effector/Avr protein), rather than non-self (the effector/Avr itself). The R protein is therefore monitoring or guarding the virulence target specific modification by the effector, resulting in immunity activation (van der Hoorn and Kamoun, 2008). One well-studied example

illustrating the guard model is the RIN4 protein of *Arabidopsis thaliana*, which is the virulence target of at least three different *Pseudomonas syringae* effectors, AvrB, AvrRpm1, and AvrRpt2 (Belkhadir *et al.*, 2004; Kim *et al.*, 2005). AvrB and AvrRpm1 directly interact with the RIN4 protein and induce its phosphorylation, perceived by the CNL RPM1 (Mackey *et al.*, 2002). AvrRpt2 triggers RIN4 cleavage, which is in turn perceived by the CNL RPS2 (Axtell *et al.*, 2003; Day *et al.*, 2005; Mackey *et al.*, 2003). Interestingly, an alternative model to the guard model has been proposed: the decoy model. In this model, effector manipulation of plant targets does not always increase pathogen fitness because plant possesses decoys, which mimic virulence targets. The manipulation of these decoys by the effectors does not directly affect the pathogen fitness but can be recognized by an NLR (van der Hoorn and Kamoun, 2008). For example, the non-functional kinase ZED1 from Arabidopsis is suspected to be a decoy. ZED1 is guarded by the NLR ZAR1 which recognizes ZED1-threonine acetylation by the *P. syringae* effector HopZ1a (Lewis *et al.*, 2013).

Common bean (*Phaseolus vulgaris* L.) is a major pulse crop that is extensively cultivated around the world as a dry grain or fresh vegetable. It is the most important grain legume for human consumption worldwide especially in developing countries in Central and South America and Southeastern Africa (Broughton *et al.*, 2003). In these countries, common bean is a staple food and represents an important source of protein and micronutrients and is consequently a critical component to combat malnutrition for hundreds of millions of smallholder farmers (Messina, 2014) (http://faostat.fao.org/). In addition to its agronomic importance, common bean is an ideal model for crop evolutionary studies thanks to its complex evolution which led to wild forms grouping in three gene pools widely distributed from Mexico to South America. Cultivated germplasm arose from two of these ecogeographical gene pools by independent domestication events (Bitocchi *et al.*, 2017). Indeed, recent data suggest that wild *P. vulgaris* originated in Mesoamerica and subsequently colonized the Southern hemisphere, giving rise to the Peruvian–Ecuadorian wild populations and the wild Andean gene pool (Bitocchi *et al.*, 2012 ; Bitocchi *et al.*, 2017 ; Rendon-Anaya *et al.*, 2017)*.* The divergence between the Andean and Mesoamerican wild gene pools was estimated to have occurred ~110,000 to 165,000 years ago (Mamidi *et al.*, 2013; Schmutz *et al.*, 2014). The lower genetic diversity in Andean compared to Mesoamerican wild germplasm is in agreement with the occurrence of a bottleneck prior to domestication in the Andes, that narrowed the subsequent domestication bottleneck (Bitocchi *et al.*, 2017 ; Rendon-Anaya *et al.*, 2017). Subsequently, common bean was independently domesticated

125 from the Mesoamerican and Andean gene pools ~8000 years ago, while no domestication

126 event occurred in the Peruvian-Ecuadorian wild population (Bitocchi *et al.*, 2013).

127   Sequencing of plant genomes has become increasingly routine since the advent of the

128 next-generation sequencing (NGS) technology (Bilsborough, 2013). Common bean is an

129 autogamous diploid (2n = 2x = 22) species with a relatively small genome ~630 Mb

130 (Arumuganathan *et al.*, 1991). In that context, three genome assemblies of cultivated common

131 bean of contrasting origin are available, one for genotype G19833 of Andean origin (Schmutz

132 *et al.*, 2014), and two for genotypes of Mesoamerican origin, BAT93 (Vlasova *et al.*, 2016)

133 and UI111 (Dash *et al.,* 2016).

134   Common bean yield stability is affected by a number of pests and diseases.

135 Anthracnose, caused by the hemibiotrophic fungus *Colletotrichum lindemuthianum*, is one of

136 the major disease of common bean worldwide, especially in temperate regions with cool and

137 humid environmental conditions (Pastor-Corrales and Tu, 1989). As use of resistant

138 genotypes is an economic and environmentally friendly way for controlling plant diseases,

139 efforts have been made to genetically characterize anthracnose *R* genes in common bean. The

140 interaction between common bean and *C. lindemuthianum* fits the gene-for-gene model and

141 nearly 20 specific *R* genes have been localized in the common bean genome (Meziadi *et al.*,

142 2016). As expected, most of these *R* genes are organized in clusters of genes co-localized with

143 NLR rich regions (Meziadi *et al.*, 2016). One notable exception is the *Co-x R* gene, present in

144 the Andean genotype JaloEEP558. Indeed, *Co-x* is located at one end of chromosome 1 in a

145 region devoid of any NLR sequences (Geffroy *et al.*, 2008; Richard *et al.*, 2014) strongly

146 suggesting that *Co-x* corresponds to a non-canonical *R* gene. In addition to an academic

147 interest, *Co-x* is also an important *R* gene at the agronomic level because it confers resistance

148 to *C. lindemuthianum* strain 100, a highly virulent strain corresponding to race 3993, that

149 overcomes nearly all the known *R* genes of Mesoamerican origin (Richard *et al.*, 2014).

150 Because of the academic interest and agronomic importance for *Co-x*, as a long term goal we

151 have sought to clone *Co-x*, and toward this effort we previously delimited the *Co-x* locus into

152 a 58 kb genomic DNA region that contains eight candidate genes in the sequence of the

153 reference Andean genotype G19833 (Richard *et al.*, 2014).

154   The objective of the present study was to identify the molecular basis of *Co-x*. To that

155 end, we PCR-amplified long-range fragments in the resistant genotype JaloEEP558 (*Co-x*) to

156 carry out a detailed sequence analysis of the *Co-x* 58kb target region. Analysis of the *Co-x*

157 locus sequence allowed us to identify an additional gene (KTR2/3) present in JaloEEP558

158 within a small cluster of CRR3 CRINKLY4 kinases. The presence of KTR2/3 is strictly

correlated with the resistance to strain 100 of *C. lindemuthianum* in a diversity panel of common bean, and KTR2/3 is upregulated in leaves at 24 hours post-inoculation (hpi). In addition, transient expression experiments confirmed a role for KTR2/3 in the resistance to strain 100. Altogether, our results provide molecular evidence that *Co-x* corresponds to a non-canonical *R* gene encoding a truncated and chimeric CRR3 CRINKLY4 kinase which is the result of an unequal recombination event that occurred recently in the Andean domesticated gene pool.

Materials and methods

Common bean material

To infer whether *Co-x* is a dominant or recessive resistance gene, 149 F2 individuals derived from a cross between the Mesoamerican breeding line BAT93 (*co-x*) and the Andean landrace JaloEEP558 (*Co-x*) were inoculated with strain 100. In order to study the origin of *Co-x*, a total of 192 cultivated and wild common bean lines of various geographical origins were studied. The evaluated materials include 123 cultivated common bean lines from the Bean Coordinated Agriculture Project (BeanCAP) diversity panel. The BeanCAP panel consists of cultivars, germplasm releases, and important breeding lines primarily from North America, and was later augmented with Andean materials from Africa and South America, from the International Center for Tropical Agriculture (CIAT) breeding program, and included some landraces (Cichy *et al.*, 2015). These cultivated beans can be separated into subpopulations based on genepool Andean vs Middle American and market type dry bean *vs* snap bean. The BeanCAP diversity panel was complemented by 27 cultivated lines from our Orsay collection and 41 wild accessions (21 Andean, 17 Mesoamerican and 3 Peruvian-Ecuadorian) provided by the CIAT, the United States Department of Agriculture Western Regional Plant Introduction Station (USDA-WRPIS), the National Botanic Garden of Belgium, and Roberto Papa (Università Politecnica delle Marche; one wild accession collected from Mexico). The AND277 genotype was a gift from Celeste Gonçalves-Vidigal (Universidade Estadual de Maringa; Brazil).

Inoculation assay with *Colletotrichum lindemuthianum* strain 100

Infections of common bean with *C. lindemuthianum* were carried out as previously described except for the plants used for the RT-qPCR analysis which were grown in soil instead of vermiculite (Richard *et al.*, 2014). Briefly, 7 days post-sowing in soil, seedlings of

193   *P. vulgaris* were inoculated with the *C. lindemuthianum* strain 100 by spraying an aqueous

194   spore suspension (2×10^6 spores/mL) on both sides of the two cotyledonary leaves. For the F2

195   individuals and diversity panel lines, symptoms were scored 7 days after infection. For the

196   diversity panel, symptoms were scored on two independent replicates (4 plants in each

197   replicate). The kinetics experiments were carried out on JaloEEP558, and water was used as a

198   mock (negative control). A time course gene expression analysis was conducted at 6, 24, 48,

199   72, and 96 hpi in JaloEEP558 seedlings infected with the strain 100. For each time, one of the

200   two cotyledonary leaves from three different inoculated plants and control plants were

201   sampled, flash frozen in liquid nitrogen for RNA isolation and RT-qPCR analysis.

202

203   **Amplification of *Co-x* target region by Long-range PCR, purification and**

204   **sequencing**

205   Long-range PCR was used to amplify *Co-x* target region in seven overlapping

206   fragments of ∼10 kb using the primers listed in Supplementary Table S1. PCR reactions used

207   100 ng of JaloEEP558 DNA added to PCR mix containing milliQ H_2O, LA Taq buffer

208   (TaKaRa), 400 μM of each dNTPs, 0.5 μM of forward and reverse primers, 1.25 units of LA

209   Taq (TaKaRa) to a total volume of 25 μL. PCR cycles were performed in a Thermal Cycler

210   (Applied Biosystem). An initial 2-minutes incubation at 94°C was followed by 30 PCR cycles

211   of denaturation (15 seconds at 94°C), annealing (45 seconds) and extension (1 minute per kb

212   at 68°C). After the tenth cycle, 10 seconds of extension were added at each cycle. The

213   reaction was completed by a final extension of 15 minutes at 68°C. Amplification products

214   were separated through a 1% agarose gel containing Ethidium Bromide in cold TBE 0.5X

215   buffer (previously cooled down at 4°C). Amplification products were excised from the gel

216   and the DNA was extracted using the NucleoSpin® Gel and PCR Clean-up kit (Macherey-

217   Nagel) following supplier's protocole and eluted in 30 μL of milliQ H_2O pre-warmed at 50°C.

218   Purified DNA from LR2.2, LR5, LR6 and LR7 was sequenced at the CNRGV (Toulouse,

219   France) using 454 GS-Junior technology (Roche). LR1, LR3 and LR4 were sequenced using

220   Pacific Biosciences PacBio RS II at GATC Biotech (Konstanz, Germany). Annotation was

221   carried out as described in David *et al.* (2009). Genbank accession numbers are MW328721

222   and MW325717.

223

224   **PCR amplification of *KTR2/3***

225   The presence of *KTR2/3* was tested using the primers RepetJalo1F and RepetJalo1R

226   (Supplementary Table S1) using Go Taq DNA Polymerase kit (Promega, Charbonnières-les-

Bains, France) in a standard PCR program (start: 94°C for 5 min; amplification: 35 cycles of 94°C for 30 s, 64°C for 30 s, 72°C for 3 min; termination: 72°C for 5 min). PCR products were resolved on 1% agarose gels containing ethidium bromide, run in 0.5X TBE buffer and visualized under UV light. In genotypes carrying *KTR2/3* this PCR reaction generates a 3179 bp fragment (referred to as "3.1 kb band") while in genotypes that do not carry *KTR2/3* it generates a 1639 bp fragment (referred to as "1.7 kb band").

Cloning and sequencing of *KTR2/3*

Primers KTR2/3exp-F2 and KTR2/3exp-R were used to amplify a 1563 bp fragment containing *KTR2/3* ORF using standard PCR program and an Advantage HF-2 PCR Taq Polymerase (ClonTech) (Supplementary Table S1). For the 14 genotypes analyzed (Supplementary Table S2), the resulting fragment was cloned into pGEM®-T vector (Promega, Charbonnières-les-Bains, France) for sequencing analysis.

RNA extraction and RT-qPCR analysis

Total RNA was extracted by using the kit for plants NucleoSpin RNA plus (Macherey-Nagel, Hœrdt, France). The RNA quantity and purity were determined on a NanoDrop 8000 spectrophotometer (Thermo Fisher Scientific, Waltham, USA) and integrity was checked by electrophoresis on a 1% agarose gel. cDNA was synthesized from 1 μg of RNA using the ImProm-II™ Reverse Transcription System according to the manufacturer protocol. Quantitative RT-PCR (RT-qPCR) was performed with a LightCycler® 96 Instrument in 15 μL volume reaction containing 5 μL of 6 times diluted cDNA, each specific primer with a final concentration of 0.1 μM, 7.5 μL SYBR green premix (LightCycler® 480 SYBR Green I Master, Roche) and distilled water. Results were analyzed by using the software LightCycler® 96 version 1.1.

The expression analyses of the genes KTR2/3, KTR2, KTR3 and KFL genes were performed using the gene specific primers listed in Supplementary Table S1. Gene expression was normalized with four reference genes (*PvUkn1*, *PvUkn2*, *PvIDE* and *PvAct11*) (Borges *et al.*, 2012) (Supplementary Table S1). Gene expression in mock condition was used to calibrate gene expression in infected plants for each gene and time point. Relative gene expression in inoculated leaves compared to mock leaves was calculated using the method $2^{-\Delta\Delta Ct}$ on two biological replicates and three technical replicates (Livak and Schmittgen, 2001).

Transient transformation of common bean hypocotyles and disease assay with *C.*
lindemuthianum

KTR2/3 open reading frame (ORF) and its putative promoter sequence (corresponding to the 345 bp located between the transcription start codon of KTR2/3 and the stop codon of KTR3; Fig. 1) was PCR-amplified using High Fidelity polymerase (Advantage HF2, ClonTech), following the supplier's protocol, with KTR2/3exp-F2 and KTR2/3exp-R primers (Supplementary Table S1) on JaloEPP558 gDNA and was cloned into the expression vector pCambia0390 in *Bam*HI restriction site. The resulting vector (pCambia0390+KTR2/3) or the empty vector (pCambia0390) were transformed into *Agrobacterium tumefaciens* strain EHA105. Bacteria were grown in YEB liquid culture (5 g/L beef extract, 1 g/L yeast extract, 5 g/L peptone, 5 g/L sucrose and 2 mM $MgSO_4$) supplemented with 200 μM of acetosyringone, overnight at 28°C. After reaching an OD_{600nm} between 0.4 and 1.2, the bacterial suspension was pelleted and resuspended in MMAi medium containing 10 mM MES pH 5.8, 10 mM of $MgCl_2$ and 150 μM of acetosyringone at an OD_{600nm} of 1 and incubated 1 hour at room temperature. Meanwhile, the hypocotyls of 8 days-old BAT93 plants, susceptible to strain 100, were cut to have pieces of hypocotyl of 5 to 6 cm. Hypocotyls were agroinfiltrated under vacuum for 15 minutes in a beaker containing one of the *Agrobacterium* suspension, pCambia0390+KTR2/3 or empty pCambia0390. After infiltration, the hypocotyls were dried on towel paper and their extremities were coated with paraffin wax. The hypocotyls were placed on little racks in humid transparent boxes and kept in the dark for one day prior to *C. lindemuthianum* infection. *C. lindemuthianum* was prepared as described in Richard *et al.* (2014). The hypocotyls were inoculated with a spore suspension of *C. lindemuthianum* strain 100 at 5×10^6 spores/mL 24 h after agroinfiltration. On each hypocotyl, 3 drops of 5 μL each were carefully pipetted. The hypocotyls were then incubated for 7 days in the humid transparent box in a growth chamber at 19°C with 16 h light before disease scoring. For each condition (pCambia0390+KTR2/3 or empty pCambia0390), ~60 hypocotyls (~180 droplets) were tested. After 7 days, for each droplet, the symptoms were scored as "resistant" (no symptoms) or susceptible (lesions developing). We performed a chi square test (one degree of freedom) to test if the agroinfiltration with pCambia0390+KTR2/3 has a significant effect on the resistance, compared to controlled conditions with the empty vector.

Sequence analysis

The targeted *Co-x* region in BAT93 was annotated using an automatic annotation pipeline described in David *et al.* (2009). The pipeline uses a combination of gene-finding

programs and sequence homology with known genes and proteins: the two *ab initio* gene prediction programs FGENESH (Burset and Guigo, 1996) and GeneMarkhmm (Lukashin and Borodovsky, 1998), BLAST (Altschul *et al.*, 1997) analyses against the GenBank nonredundant database and all the *Phaseolus* ESTs available at GenBank (Ramirez *et al.*, 2005). All this information was imported into the annotation platform Artemis (Rutherford *et al.*, 2000) for further manual analysis.

Multiple amino acid sequence alignment of the conserved kinase domain of *CRINKLY 4* protein sequences was generated using MUSCLE (Edgar, 2004a, b) with default parameters and edited in Seaview for manual adjustments (Gouy *et al.*, 2010). Alignments were subjected to maximum likelihood (ML) analysis using the JTT+I+G model as implemented in PhyML (Guindon and Gascuel, 2003). Relative support for clades was assessed with 100 bootstrap replicates. The resulting phylogenetic tree was displayed using MEGA5 (Tamura *et al.*, 2011).

Trypan and Aniline blue staining

For the Trypan blue staining, at 8 days post-infection (dpi), leaf discs of JaloEEP558 infected with *C. lindemuthianum* strain 100 were boiled 1 minute in a 3:1 mixture of 96% ethanol and staining solution (100 mL lactic acid, 100 mL phenol, 100 mL glycerol, 100 ml H_2O, and 100 mg Trypan blue (Sigma-Aldrich™, 93590)), rinsed with water and destained overnight in 2.5 g/mL chloral hydrate in water before observation on an optical microscope.

For the aniline blue staining, at 8 dpi, leaves of JaloEEP558 infected with *C. lindemuthianum* strain 100 were destained and fixated in 1:3 acetic acid/ethanol until complete discoloration. The saturated destaining/fixation solution was replaced, if necessary. Discs of fixated leaf were incubated in K_2HPO_4 100 mM pH=9.0 solution for 2 h before being transferred in aniline blue staining solution (0.1% aniline blue in K_2HPO_4 100 mM pH=9.0) and observation on an optical microscope.

Results

Co-x **is a dominant resistance gene**

In order to infer the dominant/recessive status of *Co-x*, 149 F2 individuals derived from a cross between BAT93 (susceptible) and JaloEEP558 (resistant) were inoculated with strain 100. The observed segregation data fits the expected 3:1 ratio for one dominant resistance gene (122 resistant and 27 susceptible; $\chi^2_{1df}=3.76$, *P*=0.0525).

Sequencing of the *Co-x* region in JaloEEP558 using Long-Range PCR

We previously fine-mapped *Co-x* in JaloEEP558 between the marker P05 and K06 (Richard *et al.*, 2014), defining a 58 kb interval in the genome of the reference genotype G19833 (Schmutz *et al.*, 2014) (Fig. 1). Annotation of the corresponding sequence in BAT93, for which a genome sequence is available (Vlasova *et al.*, 2016), reveals the presence of the same eight genes in G19833 and BAT93: three phosphoinositide-specific phospholipases C (PLC3, PLC2, PLC1), one zinc finger transcription factor (ZnF) and a small cluster of four CRINKLY4 kinases (KTR1, KTR2, KTR3, KFL) (Fig. 1). In addition, the intergenic regions are also highly similar between G19833 and BAT93 (>90% nucleic identity) (Fig. 1).

In order to recover the target region in JaloEEP558 (*Co-x*), we PCR-amplified this region in 7 Long-Range (LR) overlapping segments of ~ 9 kb and sequenced these LR fragments using a combination of Illumina and PacBio sequencing. The resulting sequences were annotated and compared to the sequence of the susceptible genotype BAT93 (*co-x*) (Fig. 1). The sequence of JaloEPP558 contains the 8 genes previously annotated in both G19833 and BAT93 (PLC3, PLC2, PLC1, ZnF, KTR1, KTR2, KTR3, KFL) and highly similar intergenic regions (Richard *et al.*, 2014). However, an additional gene referred to as KTR2/3 (1185 bp) was identified only in JaloEEP558 (*Co-x*).

The truncated and chimeric CRR3 CRINKLY4 kinase (KTR2/3) is a strong candidate for the *Co-x* resistance gene

Unlike the kinases KTR1, KTR2, KTR3 (for <u>K</u>inase <u>TR</u>uncated) and KFL (for <u>K</u>inase <u>F</u>ull <u>L</u>ength) present both in the susceptible and resistant genotypes, KTR2/3 is only present in JaloEEP558 (*Co-x*) (Fig. 1 and Fig. 2A). KTR2/3, like KTR1, KTR2, KTR3 and KFL, belongs to the CRINKLY4 kinase family. In *A. thaliana,* the CRINKLY4 kinase family is composed of five genes, *ACR4*, and four CRINKLY4-RELATED (CRR): *AtCRR1*, *AtCRR2*, *AtCRR3* and *AtCRK1*. Phylogenetic analysis revealed that the five kinases present at the *Co-x* locus *(*KTR1, KTR2, KTR3, KTR2/3 and KFL) belong to the CRINKLY4-RELATED 3 (CRR3) subfamily (Supplementary Fig. S1). However, while KFL corresponds to a classical full length CRINKLY4 kinase, the four other kinases present at *Co-x* locus, KTR1, KTR2, KTR2/3, and KTR3 are truncated proteins lacking both the extracellular (Crinkly repeats and TNFR domain) and transmembrane domains classically found in CRINKLY4 kinases (Fig. 2B). Instead, these truncated kinases are composed of an unknown N-terminal domain of ~100 amino-acid followed by a predicted cytoplasmic serine/threonine

kinase catalytic domain classically found in CRINKLY4 kinase (Fig. 2B; Supplementary Fig. S2). BLASTP analysis of KTR2/3 protein sequence revealed that this kind of truncated CRINKLY4 kinases containing this ~100 aa domain, are only found in legumes belonging to the *Phaseoleae* tribe. Indeed, truncated CRINKLY4 kinase were identified in common bean, at the *Co-x* locus (this study), in soybean, in the syntenic regions of *Co-x* locus located on chromosomes Gm11 and Gm18, as well as in *Vigna angularis*, *V. unguiculata*, *V. radiata* and *Cajanus cajan*. Sequence comparisons of the different truncated kinases present in *P. vulgaris* at the *Co-x* locus revealed that KTR2/3 is a chimera between the beginning of the KTR2 (first 232 bp) and the end of KTR3 (953 bp). Interestingly, the junction region of KTR2/3 contains internal related repeats of either 69 or 78 bp supporting the dynamic nature of the region in terms of recombination (Supplementary Fig. S3 and S4). More precisely, in the ~100 aa unknown domain, KTR2/3 contains two 78 bp tandem repeats derived from KTR2 followed by one 69 bp repeat derived from KTR3 (Supplementary Fig. S2, S3 and S4). The exact size of the N terminal unknown domain is variable depending on the number of repeats in each gene: 182 aa in KTR1, 72 aa in KTR2, 110 aa in KTR2/3, 127 aa in KTR3 (Supplementary Fig. S2, S3 and S4). Truncated forms of CRR3 were also found in *Ricinus communis* but with a different and also unknown, N-terminal domain (Supplementary Fig. S2). Conversely, the unknown ~100 aa is also present in the genome of *Spatholobus suberectus*, a traditional chinese medicine also belonging to the *Phaseoleae* tribe (Qin et al 2019). However, in this latter species, the ~100 aa domain is not associated with truncated CCR3 kinase, but present in the N-term of a nuclear inhibitor of protein phosphatase, and in a Mitogen-activated protein kinase kinase kinase 1 (MAP3K1). In conclusion, even if truncated CCR3 or the ~100 aa unknown domain are found separately in very few species, truncated CCR3 containing this ~100 aa domain, are only found in legume species from the *Phaseoleae* tribe.

Diversity panel of wild and cultivated common beans

On the basis of these genomic results, KTR2/3 is a strong candidate gene for *Co-x R* gene. In order to confirm that *KTR2/3* is *Co-x*, we used a diversity panel of 192 cultivated and wild common beans from various geographical origins. To infer the presence of KTR2/3, we developed a couple of PCR primers amplifying specifically a 3.1 kb fragment on genotypes containing KTR2/3, while a 1.7 kb fragment is observed for genotypes that do not have KTR2/3 (Fig. 2A, Supplementary Table S2). We identified 37 genotypes presenting the 3.1 kb fragment, thus potentially containing KTR2/3. A 1.7 kb fragment was amplified from the other 154 genotypes, implying of the absence of KTR2/3 in those genotypes. The same 192

genotypes were also scored for disease resistance after infection with strain 100 of *C. lindemuthianum*. Strikingly, all the genotypes presenting the 3.1 kb band (KTR2/3) were resistant to strain 100 (Supplementary Table S2). Reciprocally, we identified 115 genotypes susceptible to strain 100 and none of them contained KTR2/3. Altogether, these results strongly suggest that KTR2/3 is *Co-x*. Furthermore, 25 genotypes were resistant to strain 100 but did not contain KTR2/3, suggesting that these genotypes possess resistance to strain 100 conferred by another *R* gene. Most of the genotypes containing KTR2/3 are cultivated genotypes of Andean origin (Supplementary Table S2). However, four Meso-American genotypes (Raven, Newport, Phantom, Jaguar) were also identified as carrying KTR2/3. Notably, all 41 wild genotypes tested, including 21 Andean wild genotypes, present the 1.7 kb fragment, suggesting that KTR2/3 is not present in wild germplasm. We sequenced KTR2/3 gene in 14 common bean genotypes of Andean (10 genotypes) and Mesoamerican (4 above-mentioned genotypes) origin belonging to various market classes. The KTR2/3 coding sequence (1185 bp) was 100% identical for all 14 genotypes.

Expression pattern of KTR2/3

In order to ascertain the involvement of KTR2/3 in anthracnose resistance, we performed an expression analysis of KTR2, KTR2/3, KTR3 and KFL in the leaves of JaloEEP558 after infection with *C. lindemuthianum* strain 100 (incompatible interaction), using RT-qPCR and gene-specific primers. In particular, to discriminate between KTR2, KTR3 and KTR2/3 we developed primers bordering the junction area of KTR2/3 (Fig. 2; Supplementary Table S1). Temporal gene expression analysis revealed that KTR2/3 is 3 fold up-regulated after infection compared to mock control at 24 hpi (Fig. 3). Conversely, the expression of *KTR2*, *KTR3* and *KFL* was not modified upon *C. lindemuthianum* infection (Supplementary Fig. S5; Supplementary Table S3).

The function of *Co-x* confirmed by transient agrobacterium infiltration

In order to confirm the function of KTR2/3 as resistance protein against *C. lindemuthianum* strain 100, we developed a transient expression system by vacuum-infiltrating *Agrobacterium* in bean hypocotyls. BAT93 hypocotyls were agroinfiltrated with an expression vector containing or not *KTR2/3* ORF (and its own promotor). Transformed hypocotyls were then inoculated with droplets of strain 100 spore suspension and symptoms were scored at 7 dpi. We scored as "resistant" when no symptoms were observed and as "susceptible" when symptoms were observed at the site of deposition of the droplets

430   (Supplementary Fig. S6). In total 165 and 177 *C. lindemuthianum* droplet-inoculations were

431   scored on hypocotyls agroinfiltrated with the empty pCambia0390 vector and the

432   pCambia0390+*KTR2/3*, respectively. These experiments showed that agroinfiltration with

433   pCambia0390+KTR2/3 (68 droplets scored as "resistant" and 109 as "susceptible") has a

434   significant effect on the resistance compared to controlled conditions with the empty vector

435   (40 droplets scored as "resistant" and 125 as "susceptible") (χ^2_{3df}=19.37; *P* = 1.08E-05*)*,

436   supporting that KTR2/3 is *Co-x*.

437

Cytological characterization of the *Co-x* mediated resistance

439   The interaction between *P. vulgaris* and *C. lindemuthianum* has been extensively

440   studied at the cytological level in seminal papers (Oconnell and Bailey, 1988; Oconnell *et al.*,

441   1985). Because the resistance gene KTR2/3 does not encode a classical *R* protein from the

442   NLR family, a characterization of KTR2/3-mediated immunity at the cytological level was

443   conducted. To that end, leaves of JaloEEP558 were inoculated with spores of the strain 100,

444   stained with Trypan or aniline blue and observed under the optical microscope at 8 dpi.

445   Fungal structures, such as conidia and appressoria, were observed on the surface of the

446   epidermal cells (Figure 4). However, no fungal structure could be observed intracellularly,

447   suggesting that KTR2/3-mediated resistance blocked the fungus at an early step of the

448   infection. In fact, cell death was observed at the site of penetration of the fungus, in the

449   epidermal cells, suggesting the triggering of hypersensitive response (HR), which is a typical

450   early output of NLR-triggered immunity (Figure 4B). Furthermore, callose papillae

451   depositions were observed in some epidermal cells, coinciding with the presence of fungal

452   appressoria (Figure 4A, C and D). Consequently, these observations suggest that, even if

453   KTR2/3 encodes a non-canonical resistance gene, KTR2/3-mediated immunity resembles

454   canonical NLR-mediated immune responses at the cytological level.

455

Discussion

457

458   In common bean, as in other crops, identifying the molecular basis of resistance is

459   critical to promote chemical free cropping systems (Adachi *et al.*, 2020 ; van Esse *et al.*,

460   2020). In that context, the *Co-x R* gene is interesting for both applied and academic reasons.

461   At the agronomic level, *Co-x* confers resistance to an extremely virulent strain of *C.*

462   *lindemuthianum*. From a fundamental point of view, we present here molecular evidence that

463   *Co-x* is a non-canonical resistance gene encoding a truncated and chimeric CRINKLY4

464   kinase belonging to the CRR3 subfamily.

465   Thanks to the recent improvement in sequencing technologies, reference genome

466   sequences are now available for most crop species (Hickey *et al.*, 2019; Michael and

467   VanBuren, 2020). In that context, a major bottleneck for disease *R* gene cloning is the

468   generation of high-quality sequence information from the genotype of interest carrying the

469   target *R* gene (Thind *et al.*, 2017). In the present study, the development of a 11X non-gridded

470   BAC library of JaloEEP558 was unsuccessful since after screening with PCR-based markers

471   developed from the *Co-x* locus (Richard *et al.*, 2014), no positive BAC clone was identified

472   (data not shown). In contrast, long-range PCR amplification in combination with long read

473   sequencing enabled sequencing of the *Co-x* locus in JaloEEP558 (~60 kb). This allowed us to

474   identify an additional gene (*KTR2/3*) present only in the resistant JaloEEP558 (*Co-x*). KTR2/3

475   is a chimeric gene consisting of fragments of the two adjacent genes present at the *Co-x* locus.

476   Consequently, it is worth noting that a re-sequencing strategy based on mapping of short reads

477   would have been inefficient to identify KTR2/3. The democratization of long read sequencing

478   offers new perspectives by enabling not only *de novo* genome but also pan-genome assembly

479   (Michael and VanBuren, 2020). A seminal study in wheat has already exploited pan-genome

480   variation, instead of performing GWAS (Genome-Wide Association Studies) on a reference

481   genome to rapidly clone *R* genes (Arora *et al.*, 2019).

482   The agronomical importance of the *Co-x* cluster is illustrated by the numerous recent

483   papers dealing with this cluster (Chen *et al.*, 2017 ; Goncalves-Vidigal *et al.*, 2020; Mahiya *et*

484   *al.*, 2019; Murube *et al.*, 2019; Padder *et al.*, 2016; Wu *et al.*, 2020). However, these studies

485   are based on the reference genome of G19833 (Schmutz *et al.*, 2014), where *KTR2/3* is not

486   present, blurring the conclusions not only with regard to the gene content but also for the

487   expression analysis by RT-qPCR. Indeed, without knowing the existence of KTR2/3, primer

488   pair supposed to be specific to KTR2 or to KTR3 can also amplify KTR2/3, because these

489   genes present highly similar regions (Supplementary Fig S3 and S4). In the present study, by

490   using primer pair specific to KTR2/3 we showed that KTR2/3 is up-regulated in leaves after

491   infection, while KTR2 and KTR3 are not, supporting the involvement of KTR2/3 in resistance

492   (Fig. 2; Fig.3; Supplementary Table S1, Supplementary Fig. S5). Consequently, the *Co-x*

493   resistance cluster in common bean clearly exemplified the importance of working with the

494   genome carrying the target *R* gene (*ie* JaloEEP558) and not only a reference genome.

495   Using a combination of approaches including comparative genomics, diversity panel studies,

496   RT-qPCR experiments and transient expression experiments, we identified *KTR2/3* as the

molecular basis of *Co-x*. KTR2/3 and the other kinases from the *Co-x* locus (KTR1, KTR2, KTR3 and KFL), belong to the CRINKLY4 (CR4) family of receptor-like kinases and more precisely to the CRR3 subfamily (Supplementary Fig. S1). CR4 kinases were initially described in maize, and have been mainly reported to be involved in plant growth and development in several vascular plants (Becraft *et al.*, 1996; Czyzewicz *et al.*, 2016). More recently, an unexpected role in plant defense was also described for an *A. thaliana* CR4 homolog (ACR4), against the pathogenic fungus *Botrytis cinerea* since *acr4* mutant displayed an enhanced resistance to the fungus (Czyzewicz *et al.*, 2016; e-Zereen and Ingram, 2012). Furthermore, ACR4 has been shown to localize at the plasma membrane, and more specifically at the plasmodesmata in *Arabidopsis* or when transiently expressed in *Nicotiana benthamiana* (Stahl and Simon, 2013). Plasmodesmata are plasma membrane-lined tubes that directly connect the cytoplasm of adjacent cells and ACR4 is suspected to have a role in the regulation of the trafficking *via* these plasmodesmata (Stahl and Faulkner, 2016). So how can KTR2/3, a chimeric and truncated CR4 kinase from common bean, act as an *R* gene against *C. lindemuthianum*? We propose that KTR2/3 could act as a decoy in an indirect recognition system (van der Hoorn and Kamoun, 2008). Indeed, some plant pathogens such as *Colletotrichum* or *Magnaporthe oryzae* use plasmodesmata as an effective pathway for intercellular hyphal passage and for effector spread in plant tissues (Kankanala *et al.*, 2007; Liao *et al.*, 2012; Ohtsu *et al.*, 2019). It is also suspected that fungal effectors could manipulate plasmodesmata to promote their opening (Cao *et al.*, 2018 ; Ohtsu *et al.*, 2019). Consequently, it is tempting to speculate that an effector from *C. lindemuthianum* strain 100 could manipulate bean plasmodesmata by targeting members of the CR4 family and that bean has evolved a decoy, the truncated CR4 KTR2/3 kinase, guarded by a NLR protein. The resistance against strain 100 segregates as a single dominant gene in the RIL population BAT93 x JaloEEP558. Thus, the putative NLR guarding KTR2/3 could be either monomorphic between BAT93 and JaloEEPP558 or different NLRs in the same genomic region (same NLR cluster) in BAT93 and JaloEEPP558 genomes may guard KTR2/3 leading to an apparent non segregation among NLRs. Notably, we showed that KTR2/3 lacks the extracellular and transmembrane domains classically observed for CR4 proteins such as KFL which supports the hypothesis that KTR2/3 could be a decoy, that mimics a virulence target (Fig. 2, Supplementary Fig. S2). Indeed, decoys are thought to usually evolve after duplication of an ancestral guardee by diversifying selection and are supposed to have no clear biological, cellular, or physiological functions compared to guardees (van der Hoorn and Kamoun, 2008). In agreement with the role of *KTR2/3* as a decoy/guardee in indirect recognition, we have shown that *KTR2/3*-mediated

531 resistance implies HR and callose deposition, which are a hallmark of NLR-mediated
532 resistance. Many kinases and pseudokinases are essential for NLR-mediated immunity and are
533 common targets for pathogen effectors, acting as guardees or decoys (Sun *et al.*, 2020). This is
534 the case, for example, of Pto from tomato (Mucyn *et al.*, 2006), and PBS1, ZED1 and CRCK3
535 from Arabidopsis (Ade *et al.*, 2007; Lewis *et al.*, 2013; Zhang *et al.*, 2017). Conversely, the
536 wheat *Stb6 R* gene encoding a wall-associated receptor kinase (WAK)-like protein has been
537 shown to encode the *R* gene *per se* but in that case, it confers pathogen resistance without HR
538 (Saintenac *et al.*, 2018). The dominant nature of *Co-x* fits this model of indirect recognition.
539 Most of the above cited examples of guardees/decoys are involved in indirect recognition of
540 bacteria effectors, thus *KTR2/3* constitutes an original example of indirect recognition of an
541 effector from a fungal pathogen. Interestingly, KTR2/3 belongs to a small class of kinases
542 termed non- RD kinase that has been proposed to function in plant immunity (Dardick and
543 Ronald; 2006). Furthermore, KTR2/3 presents the residues required for catalytic activity in the
544 catalytic loop but not in the ATP-binding pocket and in the P-loop (Dardick and Ronald; 2006)
545 (Supplementary Fig. S2). This suggests that KTR2/3 is not functional which is in agreement
546 with our hypothesis that KTR2/3 is a decoy. In order to test whether KTR2/3 is a decoy, the
547 susceptibility to *C. lindemuthianum* strain 100 should be measured in plants lacking the NLR
548 guarding the decoy (KTR2/3), in presence and absence of KTR2/3. If KTR2/3 is a decoy, rather
549 than a virulence target, the pathogen should not benefit from manipulating KTR2/3 and
550 therefore no differences in susceptibility should be observed between plants with or without
551 KTR2/3. Finally, our results raise the question of what can be considered as an *R* gene. Indeed,
552 a distinction between PTI and ETI, cannot strictly be maintained (Thomma et al. 2011), and in
553 fact PRRs, NLRs, and any signaling protein could be considered an *R* gene if the genetics
554 follows.

555   Unequal crossing-over (UCO) has been shown to be one of the main driving forces for
556 genome differences (Thind *et al.*, 2018). Concerning *R* gene clusters, it has been identified
557 that UCO can increase or decrease the number of paralogs and can in some cases lead to new
558 *R* specificities (Cai and Xu, 2007; Thind *et al.*, 2018). We found that *KTR2/3* is a chimeric
559 and additional gene in JaloEEP558 (*Co-x*) presenting sequence similarities with the
560 surrounding genes *KTR2* and *KTR3*. This strongly indicates that *KTR2/3* is the result of an
561 intragenic UCO event. The internal related tandem repeats of either 69 bp or 78 bp present in
562 both KTR2 and KTR3 are also located at the junction between KTR2 and KTR3
563 corresponding sequences in KTR2/3 (Supplementary Fig. S2, S3, S4). This suggests that these
564 tandem repeats have served as template for this intragenic UCO event. The importance of

565 UCO in the gain or loss of *R* genes has been well described for various NLR clusters such as

566 the maize rust Rp1 cluster or the soybean *Phytophthora Rps* cluster (Ashfield *et al.*, 2012;

567 Richter *et al.*, 1995; Sandhu *et al.*, 2004; Sudupak *et al.*, 1993). Strikingly, using a diversity

568 panel of common bean, we showed that *KTR2/3* was present only in cultivated Andean

569 genotypes strongly suggesting that the UCO that gave rise to *KTR2/3* occurred recently, after

570 domestication, in the Andean cultivated gene pool. Since domestication is dated 8,000 years

571 ago, the emergence of *KTR2/3* is younger than this date. In agreement with the recent origin

572 of *KTR2/3*, sequencing of *KTR2/3* in 14 genotypes belonging to various market classes

573 showed that *KTR2/3* sequence is 100% identical in all these genotypes. Four genotypes of

574 Mesoamerican origin were also shown to present *KTR2/3*. However, this does not disprove

575 our hypothesis since these genotypes are known to result from recent breeding programs

576 where Andean resistance genes located at the *Co-x* cluster were introgressed by back-cross in

577 a Mesoamerican elite background.

578 The unknown domain located in the N terminal part of the truncated CRR3

579 CRINKLY4 kinase remains intriguing. Our research reveals that the sequence coding this

580 domain is only present in the *Phaseolea* tribe suggesting a recent origin. In addition, this

581 domain exhibits variable number of repeats in the different KTR genes from *P. vulgaris*, and

582 is located at the junction between corresponding KTR2 and KTR3 parts of KTR2/3

583 suggesting a dynamic role in recombination. Most puzzling, this unknown domain was found

584 associated in other genes such as MAP kinase in *Spatholobus suberectus* which supports its

585 dynamic role in recombination and suggests a functional role in association with kinases.

586 Additional analysis will be required to understand the origin, the mechanism of integration in

587 genes and the potential function of this unknown domain.

588 Notably, several additional anthracnose *R* specifies have been located in the same

589 genomic region as *Co-x*, and described as an allelic serie: *Co-1, Co-1², Co-1³* (Melotto and

590 Kelly, 2000)*, Co-1⁴* (Goncalves-Vidigal *et al.*, 2011), *Co-1⁵* (Goncalves-Vidigal and Kelly,

591 2006). Except for genotype Widusa carrying *Co-1⁵*, all the genotypes carrying a *Co-1* allele

592 (*Co-1, Co-1², Co-1³*, and *Co-1⁴*) contain KTR2/3 (Supplementary table S2) which could

593 suggest the involvement of KTR2/3 or an allele of KTR2/3 in these resistance. Further work

594 will be needed to clarify the molecular basis of these *R* genes.

595 Wild populations undoubtedly possess a large reservoir of *R* genes, but our results

596 show that *R* gene diversity can be created after domestication. Maintaining evolutionary

597 processes in crops is the core concept behind a dynamic management of genetic resources,

598 and mutation and recombination have been highlighted as efficient mechanisms in

599 experimental populations (Enjalbert *et al.*, 2011; Raquin *et al.*, 2008). Our results reinforce

600 dynamic resistance management in germplasm collections, where the evolution of R genes in

601 cultivated crops constitutes a critical leverage for their adaptation to emerging diseases.

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Figure legends:

614   **Figure 1:** The comparison of *Co-x* target region between JaloEEP558, G19833 and
615 BAT93 reveals the presence of an additional gene (*KTR2/3*) in JaloEEP558. A schematic
616 representation of the PCR-amplified and sequenced regions of JaloEEP558 genomic DNA are
617 depicted on the top part. The identified genes and their orientation are depicted by black
618 arrows.

619

620   **Figure 2:** Representation of the kinase-containing region at *Co-x* locus and details of
621 their protein structure. **A.** Location and orientation of the kinase-encoding genes present at
622 *Co-x* locus in JaloEEP558 sequence and in the corresponding regions in G19833 and BAT93.
623 The primers used are depicted by arrows. **B.** The kinases present at *Co-x* locus belong to the
624 CRINKLY 4-RELATED 3 kinase type (CCR3). The full length kinase (KFL) present the
625 typical protein structure of CRINKLY 4 kinase with a predicted extracellular part containing
626 the Crinkly repeats and the Tumor Necrosis Factor Receptor (TNFR) domain, a
627 transmembrane domain, and an intracellular kinase domain and C-terminal part. The truncated
628 kinases present in *Co-x* locus (KTR2, KTR2/3 and KTR3) lack the extracellular and
629 transmembrane part, which is replaced by an ~100 aa unknown domain boxed in yellow.

630

631   **Figure 3:** Time-course expression analysis of the *P. vulgaris* KTR2/3 gene in
632 JaloEEP558 after inoculation with *C. lindemuthianum* strain 100. The relative abundance of
633 *KTR2/3* transcripts was calculated by comparing KTR2/3 cDNA level in inoculated leaves
634 with mock controls at each respective timepoint at 6, 24, 48, 72 and 96 hours post inoculation
635 (hpi). Data was normalized using the *PvUkn1*, *PvUkn2*, *PvIDE* and *PvAct11* reference genes.
636 Bars represent the mean ± SD, n=2 independent experiments.

637

638   **Figure 4:** Micrographs of Trypan blue (A and B) and aniline blue (C and D) stained
639 leaves of JaloEEP558 inoculated with the *C. lindemuthianum* strain 100 under white (A, B
640 and C) and UV (D) light at 8 dpi. Fungal conidia and appressoria are indicated by black
641 arrowheads and black arrows, respectively. Plant epidermal cell responses, such as, cell
642 browning and callose papillae are indicated by white asterisk and white arrows, respectively.
643 Bars = 10 μM

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645

Supplementary data

Figure S1: Maximum likelihood (ML) tree based on the protein multiple sequence alignment of CRINKLY 4 kinase domain from various species.

Figure S2: Multiple alignment of CRINKLY 4 related 3 (CRR3) protein sequences from *Arabidopsis thaliana, Zea mais, Oryza sativa, Glycine max, Medicago truncatula, Ricinus communis* and *Phaseolus vulgaris.*

Figure S3: Multiple alignment of nucleic sequences of KTR2 (G19833, JaloEEP558 and BAT93) and KTR2/3 (JaloEEP558) showing that the first 232 bp of KTR2/3 are highly similar to the sequence of KTR2.

Figure S4: Multiple alignment of nucleic sequences of KTR3 (G19833, JaloEEP558 and BAT93) and KTR2/3 (JaloEEP558) showing that the last 953 bp of KTR2/3 are highly similar to the sequence of KTR3.

Figure S5: Time-course expression analysis of the *P. vulgaris* genes KTR2, KTR3 and KFL of JaloEEP558 in response to *C. lindemuthianum* strain 100.

Figure S6: Transient expression of KTR2/3 in common bean hypocotyls and *C. lindemuthianum* disease assay scoring at 7 dpi.

Table S1: List of primer sequences used in this study (RT-qPCR, cloning).

Table S2: Common bean genotypes of various geographical origins tested for resistance to strain 100 of *C. lindemuthianum* and for the presence of KTR2/3 encoding gene.

Table S3: Raw data of the RT-qPCR experiments.

686　**Conflict of interest**

687　The authors declare no conflict of interest.

688

689　**Author contribution**

690　VG, and MMSR designed the project; MMSR, AG, JCAD, VT, SP, CM, SB, PNM,

691　WM, EB and VG, performed the experiments; MMSR, AG, JCAD, VT, SP, PNM, EB, RP

692　and VG analyzed and interpreted the data; MMSR, PNM and VG wrote the paper with

693　significant input from all authors.

694

695　**Data Availability Statement**

696　All data supporting the findings of this study are available within the paper and within

697　its supplementary materials published online.

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Figure 1
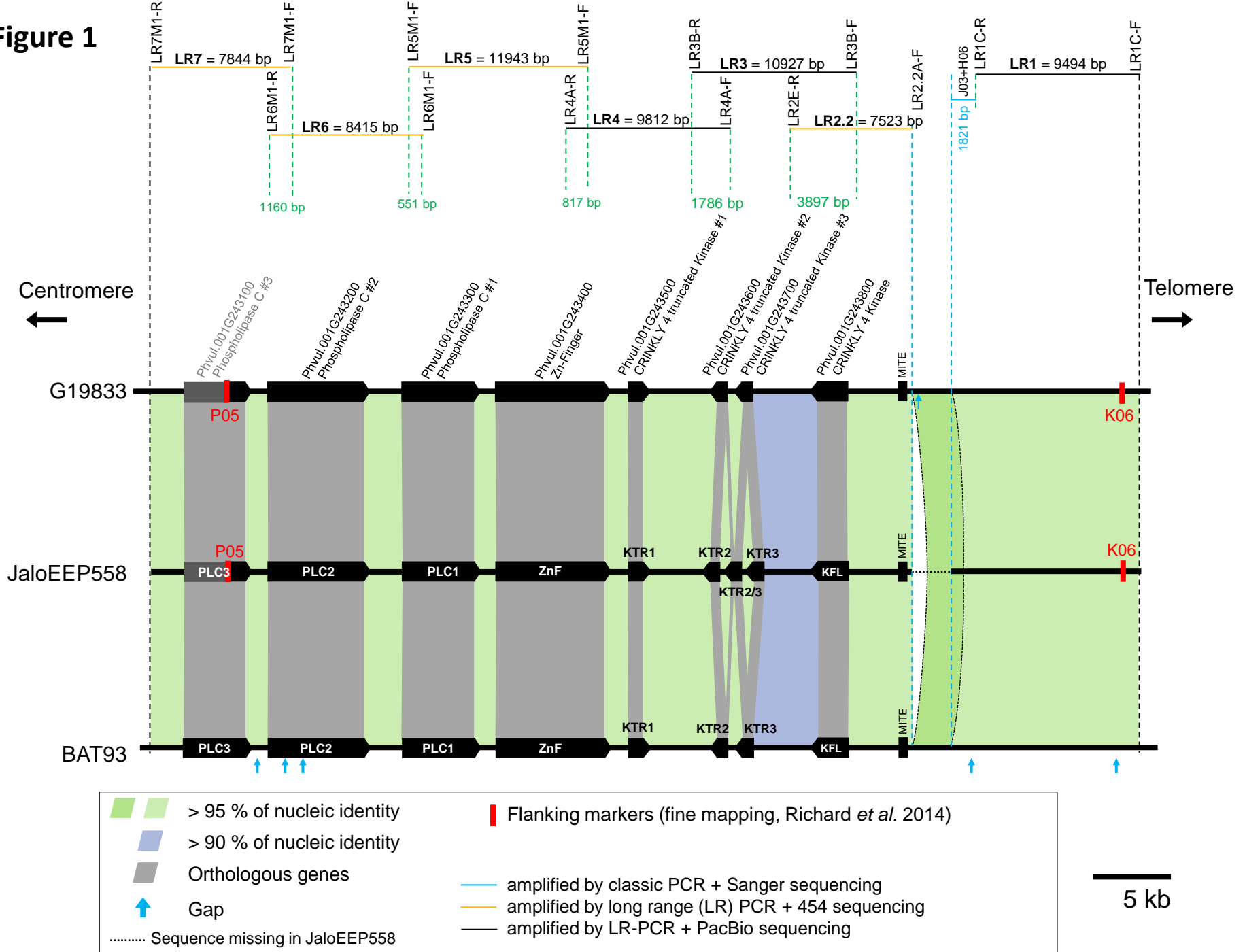
Figure 1: The comparison of *Co-x* target region between JaloEEP558, G19833 and BAT93 reveals the presence of an additional gene (*KTR2/3*) in JaloEEP558. A schematic representation of the PCR-amplified and sequenced regions of JaloEEP558 genomic DNA are depicted on the top part. The identified genes and their orientation are depicted by black arrows.

Figure 2
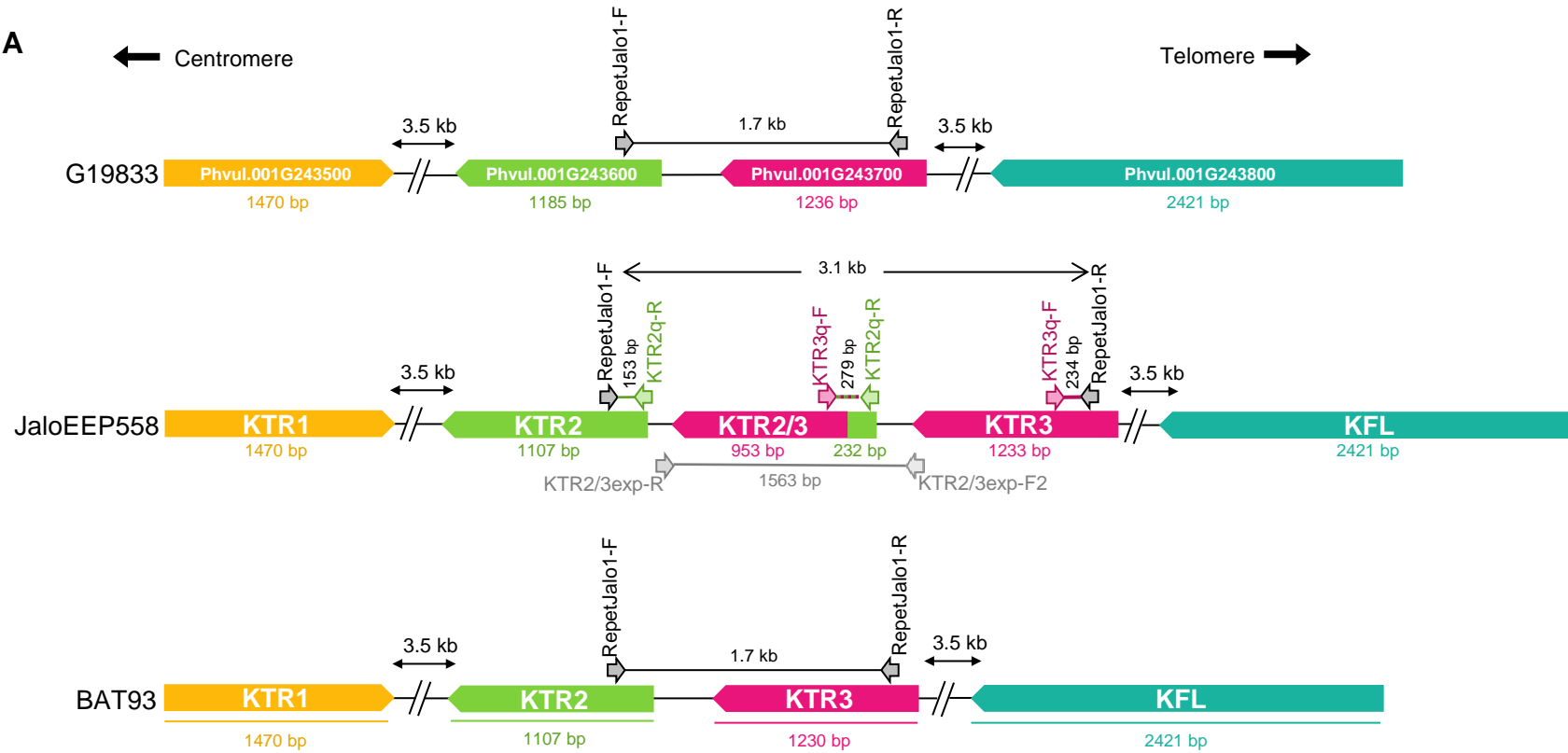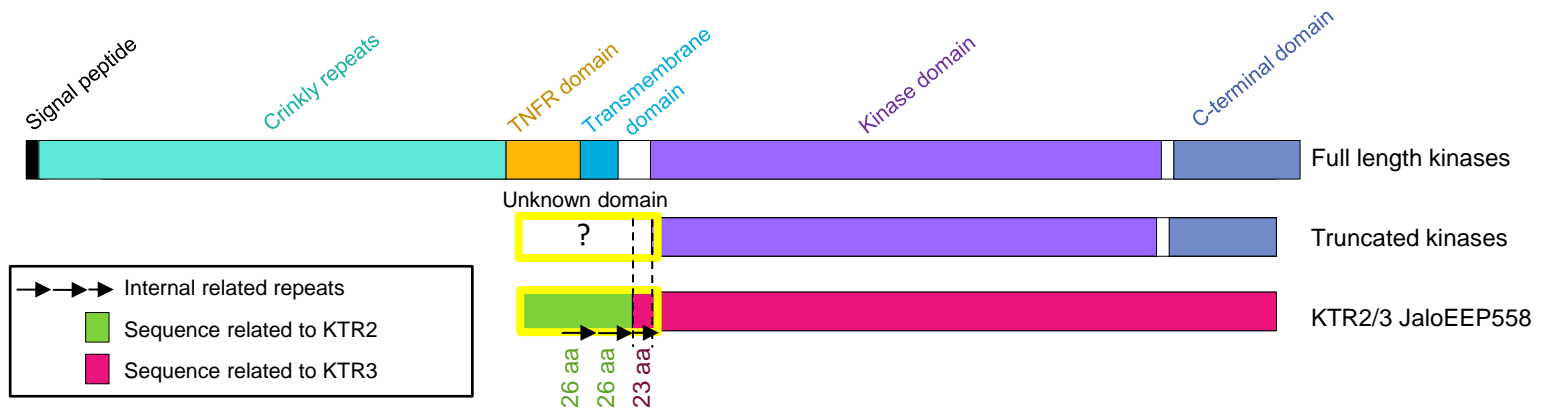
Figure 2: Representation of the kinase-containing region at *Co-x* locus and details of their protein structure. **A.** Location and orientation of the kinase-encoding genes present at *Co-x* locus in JaloEEP558 sequence and in the corresponding regions in G19833 and BAT93. The primers used are depicted by arrows. **B.** The kinases present at *Co-x* locus belong to the CRINKLY 4-RELATED 3 kinase type (CCR3). The full length kinase (KFL) present the typical protein structure of CRINKLY 4 kinase with a predicted extracellular part containing the Crinkly repeats and the Tumor Necrosis Factor Receptor (TNFR) domain, a transmembrane domain, and an intracellular kinase domain and C-terminal part. The truncated kinases present in *Co-x* locus (KTR2, KTR2/3 and KTR3) lack the extracellular and transmembrane part, which is replaced by an ~100 aa unknown domain boxed in yellow.

Figure 3

Time-course expression analysis of the *P. vulgaris* KTR2/3 gene in JaloEEP558 after inoculation with *C. lindemuthianum* strain 100. The relative abundance of *KTR2/3* transcripts was calculated by comparing KTR2/3 cDNA level in inoculated leaves with mock controls at each respective timepoint at 6, 24, 48, 72 and 96 hours post inoculation (hpi). Data was normalized using the *PvUkn1*, *PvUkn2*, *PvIDE* and *PvAct11* reference genes. Bars represent the mean ± SD, n=2 independent experiments.
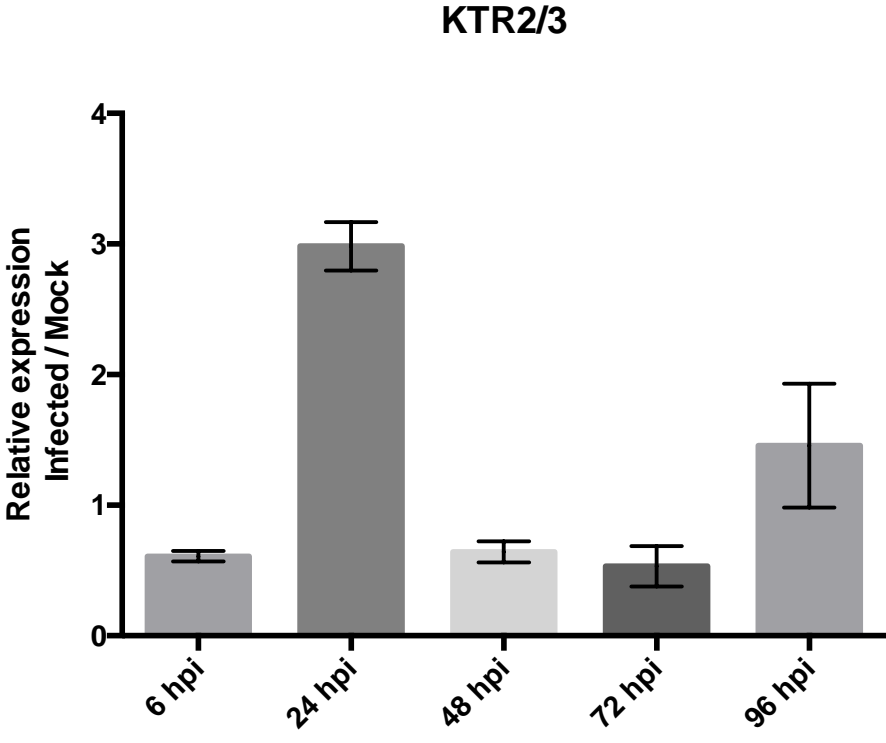
Figure 4

Micrographs of Trypan blue (A and B) and aniline blue (C and D) stained leaves of JaloEEP558 inoculated with the *C. lindemuthianum* strain 100 under white (A, B and C) and UV (D) light at 8 dpi. Fungal conidia and appressoria are indicated by black arrowheads and black arrows, respectively. Plant epidermal cell responses, such as, cell browning and callose papillae are indicated by white asterisk and white arrows, respectively. Bars = 10 µM
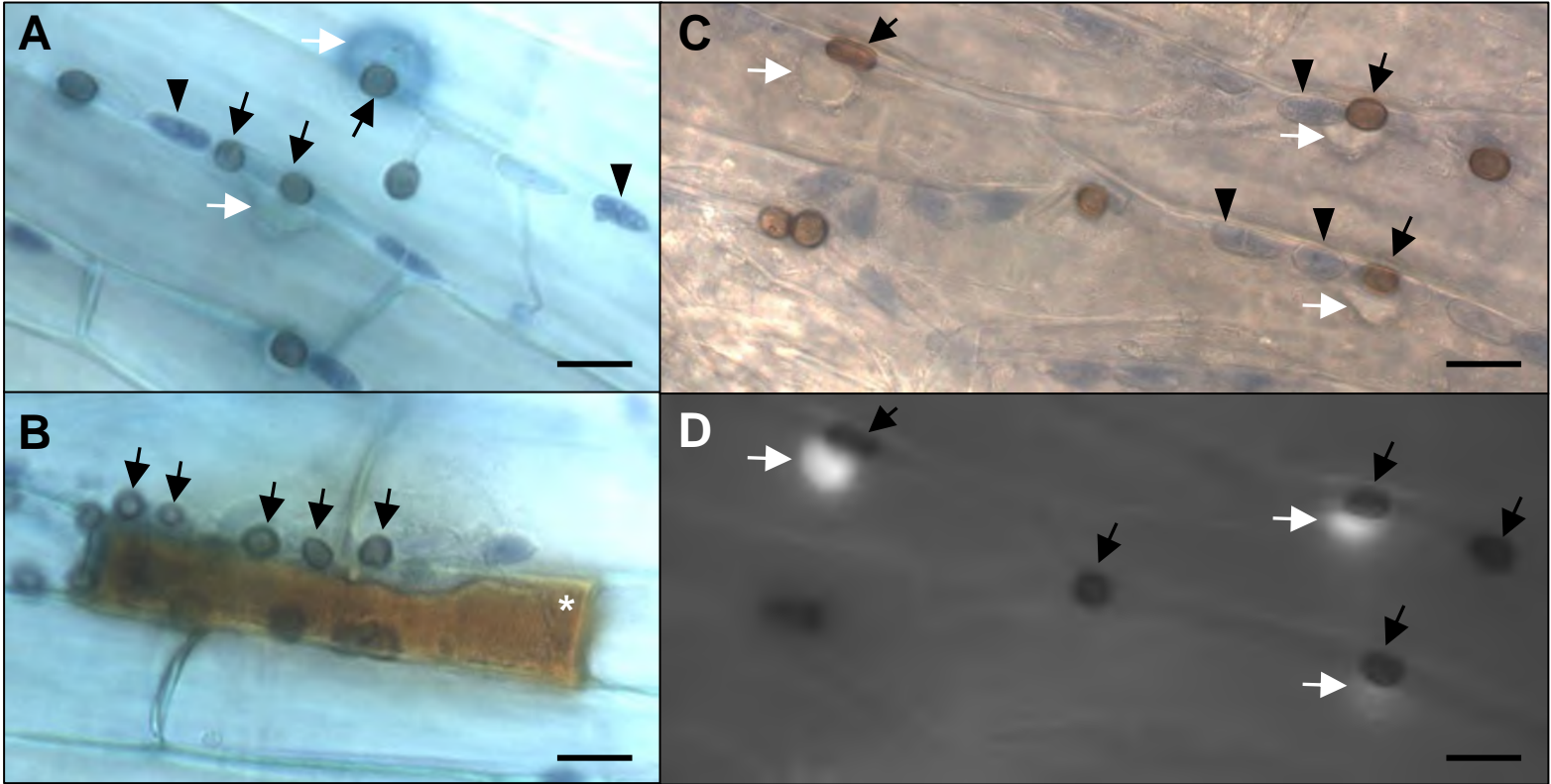
Fig. S1: KTR2/3 belongs to the CRINKLY 4-RELATED 3 Kinase clade. Maximum likelihood (ML) tree based on the proteic multiple sequence alignment of CRINKLY 4 kinase domain from various species.
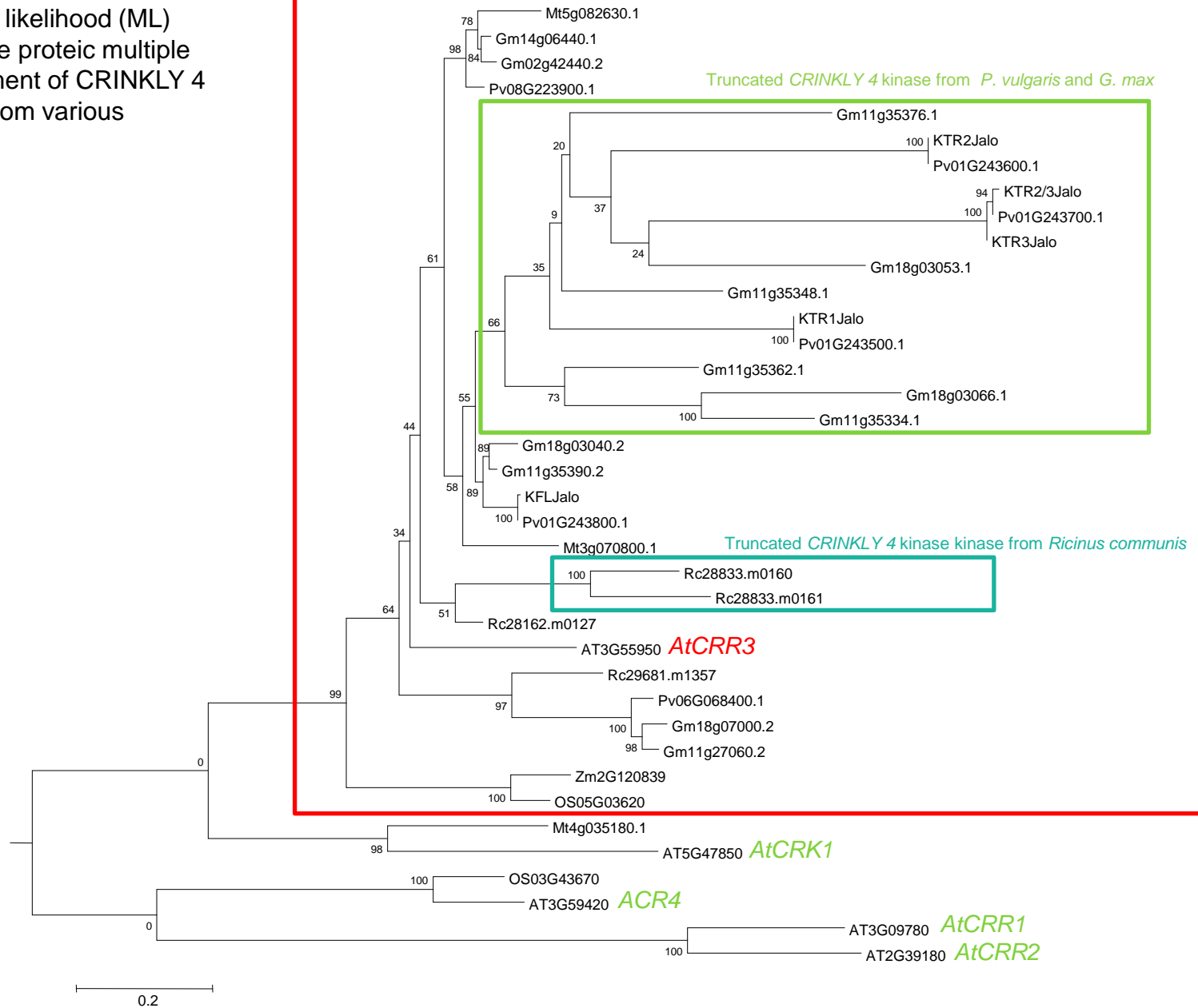
Fig. S2: Multiple alignment of CRINKLY 4 related 3 (CRR3) protein sequences from *Arabidopsis thaliana, Zea mais, Oryza sativa, Glycine max, Medicago truncatula, Ricinus communis* and *Phaseolus vulgaris*. Predicted domains are depicted on the alignment according to Cao *et al.* 2005, as described on the legend below. Truncated kinases (which names are boxed in red) present in *Co-x* region in common bean, *R. communis* and *Glycine max* present only the kinase domains compared to the classical full length *CRINKLY 4* kinases. Note that the truncated CRR3 kinases from *R. communis* lack the 100 aa unknown domain, but possess a different domain instead. Note that the truncated CRR3 kinases from *R. communis* don't present the 100 aa unknown domain, but a different one. The two related repeats of 78 or 69pb are boxed in green or pink arrows, respectively, on the KTR2/3 sequence.

- ─────────── 7 CRINKLY repeats
- ●───────────● 3 TNFR (Tumor Necrosis Factor Receptor) repeats
- ·················· Transmembrane domain
- ◆───────────◆ 12 conserved kinase domains

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                                                                    I                                          II
            10        20        30        40        50        60        70        80        90       100       110       120       130       140
          ....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|
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KFLJalo_p     MKTLTSSSVTLCL---VVAALLILSLPRSSHGLGSGATLTISDASSTVCAVVARESTRRIECYRQGQ-----IASITPNASFS-----------TISGGRN-YFCGLRSSNSDLLCWNTSSS---FERRRLYND--SSV
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Rc28833.m0160 ------------------------------------------------------------------------------------------------------------------------------------------
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Pv01G243600.1 ------------------------------------------------------------------------------------------------------------------------------------------
KTR2Jalo_p    ------------------------------------------------------------------------------------------------------------------------------------------
Pv01G243700.1 ------------------------------------------------------------------------------------------------------------------------------------------
KTR3Jalo_p    ------------------------------------------------------------------------------------------------------------------------------------------
KTR2/3Jalo_p  ------------------------------------------------------------------------------------------------------------------------------------------
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```

Top block — secondary structure labels: III IV V

Position ruler: 150 160 170 180 190 200 210 220 230 240 250 260 270 280

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Pv01G243700.1
KTR3Jalo_p
KTR2/3Jalo_p
Gm18g03053.1
Pv01G243500.1
KTR1Jalo_p
Gm11g35376.1
Gm11g35334.1
Gm18g03066.1
Gm11g35362.1
AT5G47850
Mt4g035180.1
AT2G39180
AT3G09780

Bottom block — secondary structure labels: VI VII I II

Position ruler: 290 300 310 320 330 340 350 360 370 380 390 400 410 420

(The figure is a multiple sequence protein alignment; each named row above contains an aligned amino-acid sequence. A subset of sequences, boxed in red at the left, shows gap-only rows across the upper region and short C-terminal sequence segments in the lower region, with a portion highlighted by a yellow box near positions 360–415.)

III   TM

```
         430       440       450       460       470       480       490       500       510       520       530       540       550       560
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AT3G59420
OS03G43670
OS05G03620
Zm2G120839
Pv01G243800.1
KFLJalo_p
Gm11g35390.2
Gm18g03040.2
Mt3g070800.1
Gm02g42440.2
Gm14g06440.1
Pv08G223900.1
Mt5g082630.1
Rc28162.m0127
AT3G55950
Gm11g27060.2
Gm18g07000.2
Pv06G068400.1
Rc29681.m1357
Rc28833.m0161
Rc28833.m0160
Gm11g35348.1
Pv01G243600.1
KTR2Jalo_p
Pv01G243700.1
KTR3Jalo_p
KTR2/3Jalo_p
Gm18g03053.1
Pv01G243500.1
KTR1Jalo_p
Gm11g35376.1
Gm11g35334.1
Gm18g03066.1
Gm11g35362.1
AT5G47850
Mt4g035180.1
AT2G39180
AT3G09780

I   II   III   IV   V

```
         570       580       590       600       610       620       630       640       650       660       670       680       690       700
```

AT3G59420
OS03G43670
OS05G03620
Zm2G120839
Pv01G243800.1
KFLJalo_p
Gm11g35390.2
Gm18g03040.2
Mt3g070800.1
Gm02g42440.2
Gm14g06440.1
Pv08G223900.1
Mt5g082630.1
Rc28162.m0127
AT3G55950
Gm11g27060.2
Gm18g07000.2
Pv06G068400.1
Rc29681.m1357
Rc28833.m0161
Rc28833.m0160
Gm11g35348.1
Pv01G243600.1
KTR2Jalo_p
Pv01G243700.1
KTR3Jalo_p
KTR2/3Jalo_p
Gm18g03053.1
Pv01G243500.1
KTR1Jalo_p
Gm11g35376.1
Gm11g35334.1
Gm18g03066.1
Gm11g35362.1
AT5G47850
Mt4g035180.1
AT2G39180
AT3G09780

Alignment — domains VI-A, VI-B, VII, VIII, IX (positions 710–840)

```
                        VI-A              VI-B      VII          VIII              IX
                 710       720       730       740       750       760       770       780       790       800       810       820       830       840
                 ....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|

AT3G59420        -NKALKEQLDWVKRVTIAVQAARGIEYLHGYACPP-VIHRDIKSSNILIDEEHNARVADFGLSLLGPVDSGSPLAELP--------AGTLGYLDPEYYRLHYLTTKSDVYSFGVLLEILSGRKAIDMHYEEG-------
OS03G43670       -DPNLKKRLNWARRVTIAVQAARGIEYLHGYACPP-VIHRDIKSSNILIDEDHNARVADFGLSILGPADSGTPLSELP--------AGTLGYLDPEYYRLHYLTTKSDVYSFGVLLEILSGRKAIDMQFEEG------
OS05G03620       SPSPSPVATSWKLRIKILLDASRGIDYLHSYAVPP-IIHRDIKSSNILLDGSWVARVSDFGLSLMG-PETEEVKH------LSMKAAGTVGYMDPEYYGLHHLTVKSDVYGFGVVMLESLTGRRAIFKEA-EGG-----S
Zm2G120839       SAAPSPVASSWKLRIKILLDASRGIDYLHSYAVPP-IIHRDIKSSNILLDGGWTARVSDFGLSLTGSPEPSSEESRSQQLAVTTKAAGTVGYMDPEYYGLHHLTVKSDVYGFGVVMLESLTGRRAIFKEA-EGG-----S
Pv01G243800.1    VEKGSSVLNYWKMRIKIALDAARGIEYLHNYAVPS-IIHRDIKSSNILIDVTWTARVSDFGLSLMS-PEPDRDY-------RPMKAVGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKYGEDGG-----T
KFLJalo_p        VEKGSSVLNYWKMRIKIALDAARGIEYLHNYAVPS-IIHRDIKSSNILIDVTWTARVSDFGLSLMS-PEPDRDY-------RPMKAVGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKYGEDGG-----T
Gm11g35390.2     VEKESSVLNNWKMRIKIALDASRGIEYLHNYAVPS-IIHRDIKSSNILLDATWTARVSDFGLSLMS-PEPDRDH-------RPMKAAGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKYGEDGG-----T
Gm18g03040.2     VEKESSVLNNWKMRIKIALDASRGIEYLHNYAVPS-IIHRDIKSSNILLDATWTARVSDFGLSLMS-PEPDRDH-------RPMKAAGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKYGEDGG-----T
Mt3g070800.1     VEKGSNLLNSWKMRIKIALDASRGIEYLHNYAVPS-IIHRDIKSSNILLDSTWTARVSDFGLSLMS-PESDIDY-------KPTKTAGTVGYIDPEYYGLNLLTAKSDVYGLGVVLLELLTGKRAIFRNGEDGG-----N
Gm02g42440.2     VDKSSSSVLNSWRMRIKVALDASRGIEYLHNYAVPS-IIHRDIKSSNILIDATWTARVSDFGLSLMS-PESNHDF------RPMKAAGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKNDENGG-----G
Gm14g06440.1     VDKSSSSVLNSWRMRIKVALDAARGIEYLHNYAVPS-IIHRDIKSSNILLDATWTARVSDFGLSLMS-PESDHDY------QPMKAAGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAVFKPEDGS------G
Pv08G223900.1    VDKSSSVLNSWRMRIKIALDAARGIEYLHNYAVPS-IIHRDIKSSNILIDATWTARVSDFGLSLMS-PESDLDY------RPTKAAGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKNDENGG-----T
Mt5g082630.1     VDKNSSLLNSWKMRIKVALDAARGIEYLHNYAVPS-IIHRDIKSSNILIDADWTARVSDFGLSLMS-PDSDHDY------RPTKAAGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKNDENGG-----T
Rc28162.m0127    IKKNSSSVINSWKMRIKIALDAARGIEYLHNYAVPS-IIHRDIKSSNILIDANWIARVSDFGLSLMG-PESDRDY------RPMKAAGTVGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAIFKGDDNGG-----T
AT3G55950        VEKHSSSLINSWKMRIKIALDAARGIEYLHNYAVPP-IIHRDIKSSNILLDSNWVARVSDFGLSLMG-PVLGKDHNP---YQRPTKAAGTVGYIDPEYYSLNVLTDKSDVYGLGVVLLELLTGKRAIFRNNGDVEEEEGCV
Gm11g27060.2     VDKSSSILNSWRMRIKIALDAARGIEYLHNYAVPP-IIHRDIKSSNILLDSNWNARVSDFGLSKIW-HETEQEL------MSSTKAVGTVGYIDPEYYVLNVLTTKSDVYGLGVVMLELLTGKRAVFKPEDGS------G
Gm18g07000.2     VDRSSNILNSWRMRIKIALDAARGIEYLHNYAVPP-IIHRDIKSSNILLDSNWNARVSDFGLSKIW-PETEQEL------MSS-KAVGTVGYIDPEYYVLNVLTTKSDVYGLGVVMLELLTGKRAVFKPEDGS------G
Pv06G068400.1    TEKSSSILNSWRMRIKIALDAARGIEYLHNYAVPP-IIHRDIKSSNILLDASWTARVFDFGLSLMG-PETEQEL------MST-KAVGTVGYIDPEYYVLNVLTTKSDVYGLGVVMLELLTGKRAVFKTEDGS------S
Rc29681.m1357    VEKDSSSILNSWKMRIKIALDAARGIEYLHNYAVPP-IIHRDIKSSNILLDSNWNARVSDFGLSLMG-PESDQEL------MST-KAVGTVGYIDPEYYVLNVLTTKSDVYGLGVVLLELLTGKRAIFKTEEAED----TG
Rc28833.m0161    NEKNSSVINSWKTRIKIALDVARGIEYLHNHAVPL-VIHRDIKSSNILLDSAWTARIAEFALSLMV-PESEHGY------RLMKAVGTAGYINPEYYALNILTEKSDVYSFGVVLLELLTGKKVIFYDDNSEG-----T
Rc28833.m0160    NEKNNSVINSWKMRIKIALDAARGIEYLHNYAVPP-VIHRQIKSSNILIDANWTARVAEFAFSLKA-PESEHGY------RTLSAVGTVGYIDPENYSRNVLTAKSDVYSFGVVLLELLTGKRAIFQDDDSGG------
Gm11g35348.1     ---------------------------------------------------------------------------------------MKKTAGTVGYIDPEYLAGHVVTAKSDVYGFGVVLLELLTGKTAIFKSGDDGG------
Pv01G243600.1    VDRESSALNSWRMRIKVALDASRGIEYLHKHVVPS-IIHRDINSSNILLDATWTARVSGFESSCFMSPE----------------------AEHVYSDTRVLTAKSDVYGLGVVLLELLTGKKTTLKFGINRE------
KTR2Jalo_p       VDRESSALNSWRMRIKVALDASRGIEYLHKHVVPS-IIHRDINSSNILLDATWTARVSGFESSCFMSPE----------------------AEHVYSDTRVLTAKSDVYGLGVVLLELLTGKKTTLKFGINRE------
Pv01G243700.1    ---GSSVLNSWKMRIKIALDASRGIEYLHKFGVPS-PVHGDINPSNILLDATWTAKVSNIGKA--------------------AGTFGYIDPEYIDLNVLTTKSDVYGFGVVLLELLTGK---------NGG--TILH
KTR3Jalo_p       ---GSSVLNSWKMRIKIALDASRGIEYLHKFGVPS-PVHGDINPSNILLDATWTAKVSNIGKA--------------------AGTFGYIDPEYIDLNVLTTKSDVYGFGVVLLELLTGK---------NGG--TILH
KTR2/3Jalo_p     ---GSSVLNSWEMRIKIALDASRGIYYLHKFGVPS-PVHGDINPSNILLDATWTAKVSNIGKA--------------------AGTFGYIDPEYIDLNVLTTKSDVYGFGVVLLELLTGK---------NGG--TILH
Gm18g03053.1     VEKGSTMLNSWKMRIKISLDASRGIEYIHNH--LY-SIHGNIKSSNILLDATWTAKVSDFGKVS---------------------AGT----------RHVLTEKSDVCGFGVVLLELLTGKKPILKNEEDGS-----T
Pv01G243500.1    ---GSVELNSWKMRIKIALDASRGIKYLHYYAGTN-IYR-DIKSSNILLDDTWTARVSDFGLSSLMKA-----------------VGTI--IDPEYYGQYVMTAKSEVYAFGVVLLELLTGKR-HILCGEDGG-----T
KTR1Jalo_p       ---GSVELNSWKMRIKIALDASRGIKYLHYYAGTN-IYR-DIKSSNILLDDTWTARVSDFGLSSLMKA-----------------VGTI--IDPEYYGQYVMTAKSEVYAFGVVLLELLTGKR-HILCGEDGG-----T
Gm11g35376.1     ---GSSVLNSWEMRIKIALDASRGIYYLHYYAGTN-IYR-DIKSSNILLDDTWGWK-FMRNRR-----------------RIIENTDPEYVIRGALSAESDVHGLGVVTLELLTGKRPTFVYGEDGG--TLLS
Gm11g35334.1     VEKASSVLNSWIMRIKIALDASRGIQYLHSYAVPSIIIHIDIKPSNILLDATWTARVFNFDLSFMNRESDCDDVHT-------EPVARTVGYICPEYL-GVVTTKCDVYGLGVVLLELLTGKTAFFKSEEDGG-----P
Gm18g03066.1     VDKDSSVLNSWRMRIKIAWDASLGIQYLHNYANPS-IIHRDIKSSNILLDASWTARVFDFELSFICPEP--DDEYD------- EIIMGTMGYMCPEYMTRGVLTAKCDVYGLGVVLLELLTGKRAILLESEEDG-----P
Gm11g35362.1     --KGSSVLNSWKMRIKIAVAVSEGIQYLHNYAS---IIHKNIMSSNILFDATWTARVSGFDLWLVNPEPD-RDYRT-------MWTVGTFGYIDPEYYGLNVLTAKSDVYGLGVVLLELLTGKRAKFKYEGKDFVCTPIK
AT5G47850        ---QFDP-LSWQTRLMIALDAARGIQYLHEFFVPP-VIHRDIKSSNILLDSEWTARVSDFGLSQMGPTEEDDVSHLS------LHAAGTLGYIDPEYYKFQQLTTKSDVYSFGVVLLLLSGHKAIHNNE-------DEN
Mt4g035180.1     ---QTSTIMSWSGRIKVALDAARGIEYLHKYAQPP-IIHRDIKTSNILLDSKWVAKVSDFGLSLMGP--EDEESHLS------LLAAGTVGYMDPEYYRLQYLTSKSDVYSFGVVLLLSGYKAIHKNE-------NGV
AT2G39180        -----LSQLDWSMRLKIMLQAARGLDYLHNEVDPP-IIHRDVKTSNILLDGEMCARIADFGLVSSNE-------------------------RDSSNSDREGDVYDFGIVLLEILSGRKAIDRES---------D
AT3G09780        -----FSPLSWSLRIKIAMQTAKGLEYLHNEAEPR-IIHGDVKSSNVLLDSEWVARVADFGLVTSS-------------------------NEKNLDIKRDVYDFGVVLLEILTGRKRYDRDC---------D
```

Alignment — domains X, XI (positions 850–980)

```
                        X                      XI
                 850       860       870       880       890       900       910       920       930       940       950       960       970       980
                 ....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|

AT3G59420        --NIVEWAVPLIKAGDINA-----LLDPVLKHP----SEIEALKRIVSVACKCVRMRGKDRPSMDKVTTALERALAQLMGNPSSEQPILPTEVVLGSSRMHKKSWRIGSKRSGSEN----------TEFRGGSWITFPSVT
OS03G43670       --NIVEWAVPLIKAGDISA-----LLDPVLSPP----SDLEALKIAAVACKCVRMRAKDRPSMDKVTTALERALALLMGSPCIEQPILPTEVVLGSSRMHKKVSQRSSNHSCSENDLVDGDDQRIEYRAPSWITFPSVT
OS05G03620       PVSVVVDYAVPSIVAGELSK-----VLDARAPEP--SAHEAEAVELVAYTAVHCVRLEGKDRPAMADIVANLETAVALCEDSATGGGAAGHGNSSSSS---------
Zm2G120839       PVSVVVDYAVPSIMGGELAG-----VLDPRAPEP--AAHEAEAVELVAYTAVHCVQLEGKDRPAMADIVANLETAFALCEGSDRDRGTGAFGNSSSSS---------
Pv01G243800.1    PLSLVVDFAVPRILAGEMVK-----ILDPRVGTP--DEKEAEAVELVAYTAIYCVNLEGKDRPTMADIVVNLERALGICESS--------HDSISSG
KFLJalo_p        PLSLVVDFAVPRILAGEMVK-----ILDPRVGTP--DEKEAEAVELVAYTAIYCVNLEGKDRPTMADIVVNLERALGICESS--------HDSISSG
Gm11g35390.2     PLSVVVDFAVPAILAGELVK-----ILDPRVGPP--DVNEAEAVELVAYTAIHCVNLEGKDRPTMADIVSNLERALAICESS--------HDSTSSG
Gm18g03040.2     PLSVVVDFAVPSILAGELVK-----LLDPRVEPP--NVTESEAVELVAYTAIHCVNLEGKDRPTMADIVSNLERALAICESS--------HDSISSG
Mt3g070800.1     PLSIVVDFAVPAILAGELMK-----ILDSRVGTPPTESNESEAVELMAYTAVHCVNLEGKDRPTMADIVANLERALLICEGGSNHE----IESISSV
Gm02g42440.2     PVSVVVDFAVPLIMAGELAK-----ILDPRVEPP--EMNETEAVELVGYTAMHCVNLEGKDRPTMADIVANLERALALCDSS--------HGSISSG
Gm14g06440.1     PVSVVVDFAVPVIMTGELAK-----ILDPRVKPP--EMNETEAVELVGYTAMHCVNLEGKDRPTMADIVANLERALALCDSS--------HGSISSG
Pv08G223900.1    PVSLVVDFAVPVIMAEELAK-----ILDPRVEPP--ELNETEAVELVAYTAMHCVNLEGKDRPTMADIVANLERALALCDTS--------HGSISSG
Mt5g082630.1     PISIVVDFAVPIIMKGELMK-----ILDQRVGPP--EMNETEAVELVSYTAMHCVHLEGKDRPTISDIVANLERAFTLCDSSS-------HGSISSKNNFSLKNKTGAGSYGVVFKQMYDHLHDKNNVDKNSSLLNSWKIR
Rc28162.m0127    PTSIVVDFAVPRIMSGELAK-----VLDHRVGPP--ELNEAEAVELVAYTALHCVNLEGKDRPTMADIVANLERALSLCDGS--------HGSISSG
AT3G55950        PVHLVVDYSVPAITADELST-----ILGEGDAVELVAYTALHCVNAEGRNRPTMTDIVGNLERALDLCGDS----------HGSISSG
Gm11g27060.2     PMGVVEYTGPKIASGELWS-----VLDYRVGHP--EVNEVESIQIMAYTAMHCVNLEGKERPEMTDIVANLERALAFIEGT--------PTSLSFA
Gm18g07000.2     PMGVVEYTGPKIASGELWS-----VLDYRVGQP--EVNEVESLEIMAYTAMHCVNLEGKERPEMTDIVANLERALAFIEGT--------PTSLSIA
Pv06G068400.1    PIGVVEHTGPKIASGELWS-----VLDYRVGQP--EVNEVESLEIMAYTAMHCVNMEGKERPEMTDIVANLERALAFIEGS--------PTSISLA
Rc29681.m1357    PMGVAEYAAPLISAGKMQR-----ILDTRVGPP--EMHEVEAVEVMAYTALHCVNLEGKERPDIVDIVSNLERALACEEF--------PARVCTN
Rc28833.m0161    PTSLVVDFAMLRILSGDFVK-----ILDPRVNPP--EFNEEEAVELVAYTAIHCVNLEGRNRPTMTDVVANLERSLALCIDS--------SNEDEM
Rc28833.m0160    --SVVVDFAVPKIMSGEFVR-----ALDPRVSQP--ELSEAEAVEMVAYTALHCVNLEGKERPKMIDVVANLERSLSICDAS--------RDNDQ
Gm11g35348.1     --SVVVDFAVPHILSGEWVK-----ILDPRIGPP--DENEADAVELVYTALHCVNLEGKDRPTIDEVVVNLESALASCCHS------GTIPVVSVVLPTKKKKKNFCCFTRKYC----------
Pv01G243600.1    --TSMVKIAGRVILGWKMVK-----ILDPRVGAP--HVNEAEAEALEIVAHTAVSCVNSKRKDRPTMTQVVANLETALALCDSADYAVVVGHTFGASKE----------
KTR2Jalo_p       --TSMVKIAGRVILGWKMVK-----ILDPRVGAP--HVNEAEAEALEIVAHTAVSCVNSKRKDRPTMTQVVANLETALALCDSRPS----------
Pv01G243700.1    VPS-AEVSIL---GGDFVKNLDKRVGEPRL-------NEAKALKLVARTAINCVNVEGKVRPTSAQVVFNLERAFAYFRRRY----------
KTR3Jalo_p       VPS-AEVSIL---GGDFVKNLDKRVGEPRL-------NEAKALKLVARTAINCVNVEGKVRPTSAQVVFNLERAFAYFRRRY----------
KTR2/3Jalo_p     VPS-AEVSIL---GGDFVKNLDKRVGEPRL-------NEAKALKLVARTAINCVNVEGKVRPTSAQVVFNLERAFAYFRR----------
Gm18g03053.1     RLHVIDFAEPAILAGELVK-----IKDPRVGPP--GVNEAKALELVADTAIHCVKREGKDRPTMADIVLNLERALATCGSDHRSPEKRMAL----------
Pv01G243500.1    PLSVVEFAVPPILDGDLAK-----ILDPRVGAP--HVNEAKAVELMAFTAIHCVNLEGKDRPSLAEIVVNLKRALAIICDSST------HDSISNH
KTR1Jalo_p       PLSVVEFAVPPILDGDLAK-----ILDPRVGAP--HVNEAKAVELMAFTAIHCVNLEGKDRPSLAEIVVNLKRALAIICDSST------HDSISNH
Gm11g35376.1     RKHLVDFAVTAISNGFLEK-----ILDQRAGQP--DVNEAEAVKLVADTAIRCVNLKLKDRPTMADIVVRLERALAIC----E------YDSIFRG
Gm11g35334.1     ALYVVEFAVRAILAGDLVE-----ILDSRVGPP--HEKEAEALEILAYTAICCVKRKKDRPTMNDIVASLELALALCGSSLY------YENFSS--ALQSHESFHMSMYPLHLEEFYTRRSSISLSCWGINIPVNTEDV
Gm18g03066.1     PFYLVDFAVPPILAGDLVE-----ILDSRVGPP--DLKEAEAKILAHTAIRCVEEEPRQRPTMNDIVVDLDRAFAICGSSLY------DENFSYSIALPSHESNHMTPDPLDTDS--------------CDIPIYEENT
Gm11g35362.1     S--VVDFAVPVILTGELVK-----ILDPRVGPP--DVNEAEAVKLVAYTAIHCVNLKGKDRPTMADIVANLGRALAIC------------ESSHI
AT5G47850        PRNLVEVVVPYILLDEAHR-----ILDQRIPPP--TPYEIEAVAHVGYLAAECLMPCSRKRPSMVEVVSKLESALAACLTAP------KTETVSRSNTY----------
Mt4g035180.1     PRNVVVDFVVPYIVQDEIHR-----ILDTKLPPP--TPFEIEAVTFVGYLACDCVRLEGRDRPNMSHVVNSLEKALEACLAQPIFCE--STRTTSTNVSYE----------
AT2G39180        PAGIAEWAVPLIRKGKAAA-----IIDRNICLP----RNVEPLLKLAELAELAVRENSNERPNIRNILCFLDLIVKSGLTF----------
AT3G09780        PPEIVEWTVPVIREGKAAA-----IVDTYIALP----RNVEPLLKLADVAELCVREDPNQQPTMSELANWLEHVARDALIF----------
```

```
                              990      1000      1010      1020      1030      1040      1050      1060      1070
                   ....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|....|
AT3G59420          SSQRRKSSASEGDVAE-EEDEGRKQ----QEALRSLEEEIGPASPGQSLFLHHNF-----------------------------------
OS03G43670         SSQRRKSSASEADMDGRTTTDGRNVGSSIGDGLRSLEEEISPASPQENLYLQHNF-----------------------------------
OS05G03620         ------------------------------------------------------------------------ASLSITSMELSRMD-
Zm2G120839         ------------------------------------------------------------------------ASLSVTSMDRSGALA
Pv01G243800.1      ------------------------------------------------------------------------SISVVSE-------
KFLJalo_p          ------------------------------------------------------------------------SISVVSE-------
Gm11g35390.2       ------------------------------------------------------------------------TISVVSE-------
Gm18g03040.2       ------------------------------------------------------------------------TISVVSE-------
Mt3g070800.1       ------------------------------------------------------------------------TIS-VSD-------
Gm02g42440.2       ------------------------------------------------------------------------TISIVSD-------
Gm14g06440.1       ------------------------------------------------------------------------TISIVSD-------
Pv08G223900.1      ------------------------------------------------------------------------TVSIVSD-------
Mt5g082630.1       IKIALDAPRGIEHLHNHTVPSIIHRDIKSSTILIDMNWMARVSHIGWLVSYTAMHCVHLEGKDRPTISDIVCISCVTISIGSD-------
Rc28162.m0127      ------------------------------------------------------------------------TISVVSE-------
AT3G55950          ------------------------------------------------------------------------ICSIVSD-------
Gm11g27060.2       ------------------------------------------------------------------------SFSAPLE-------
Gm18g07000.2       ------------------------------------------------------------------------SFSAPLE-------
Pv06G068400.1      ------------------------------------------------------------------------SFSAPLE-------
Rc29681.m1357      ------------------------------------------------------------------------RFSIPPKLRFPC--
Rc28833.m0161      ------------------------------------------------------------------------NIKHQSE-------
Rc28833.m0160      -------------------------------------------------------------------------------------
Gm11g35348.1       -------------------------------------------------------------------------------------
Pv01G243600.1      -------------------------------------------------------------------------------------
KTR2Jalo_p         -------------------------------------------------------------------------------------
Pv01G243700.1      -------------------------------------------------------------------------------------
KTR3Jalo_p         -------------------------------------------------------------------------------------
KTR2/3Jalo_p       -------------------------------------------------------------------------------------
Gm18g03053.1       -------------------------------------------------------------------------------------
Pv01G243500.1      ------------------------------------------------------------------------TIFDVSE-------
KTR1Jalo_p         ------------------------------------------------------------------------TIFDVSE-------
Gm11g35376.1       ------------------------------------------------------------------------TIYDVSE-------
Gm11g35334.1       LMYIPVYA-----------------------------------------------------------------------------
Gm18g03066.1       NS-----------------------------------------------------------------------------------
Gm11g35362.1       -------------------------------------------------------------------------------------
AT5G47850          -------------------------------------------------------------------------------------
Mt4g035180.1       -------------------------------------------------------------------------------------
AT2G39180          -------------------------------------------------------------------------------------
AT3G09780          -------------------------------------------------------------------------------------
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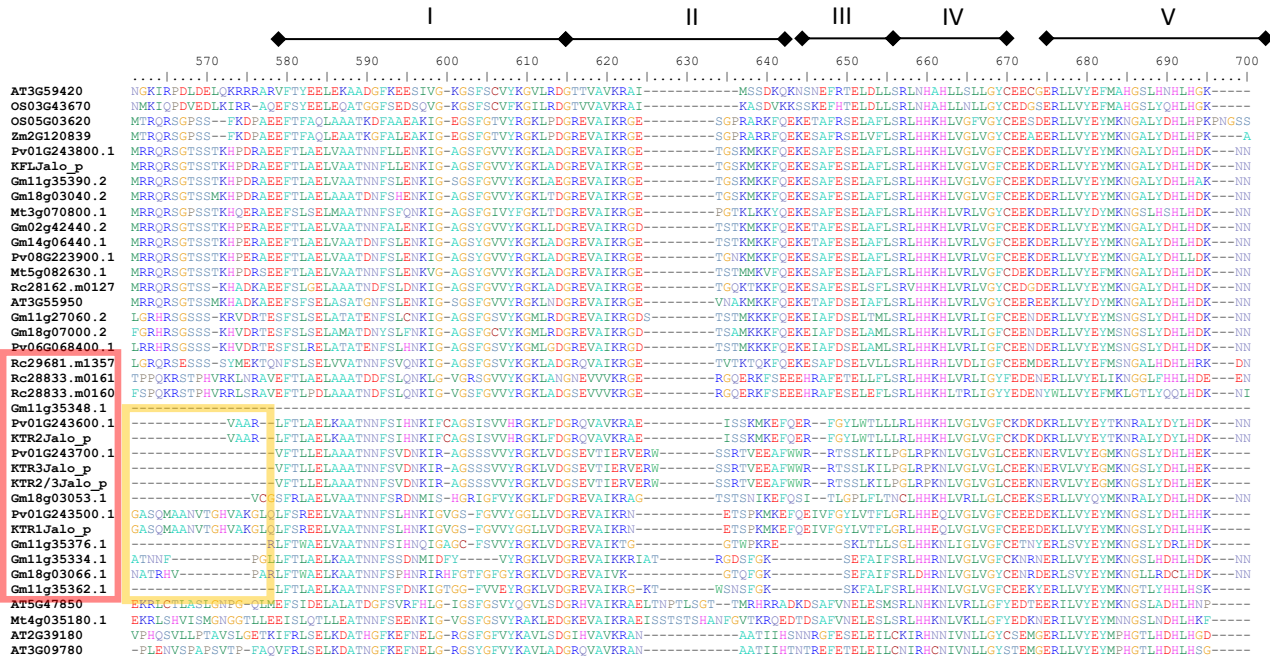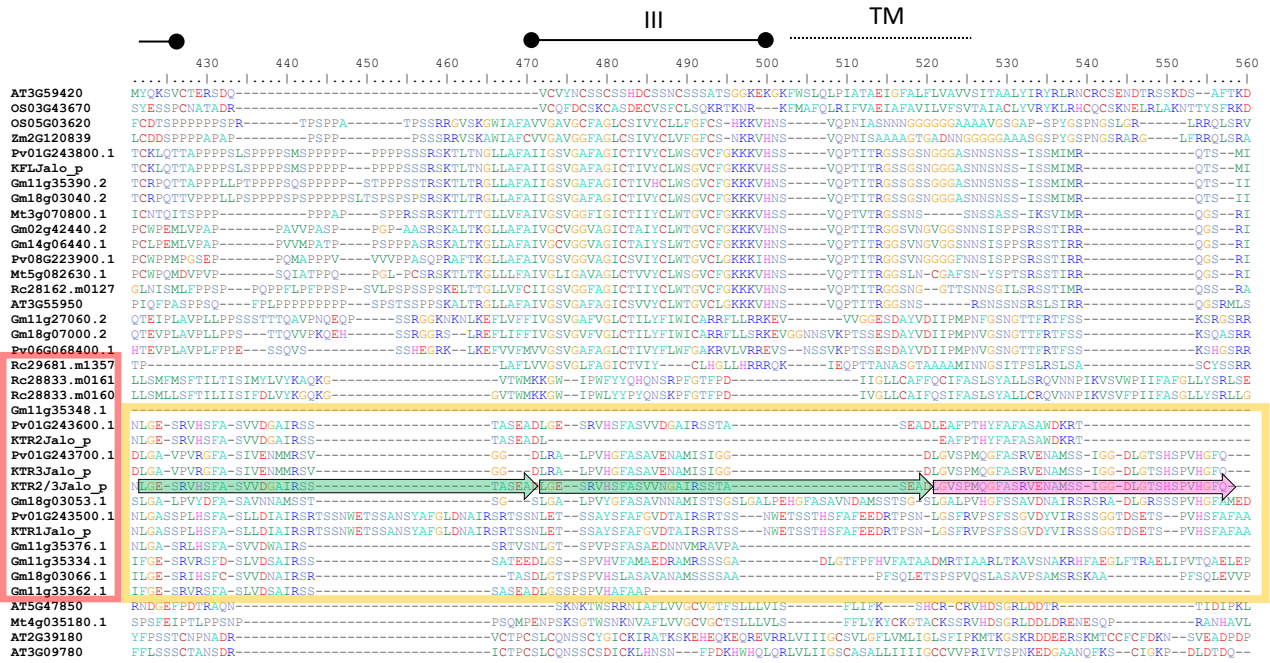
Figure S3: Multiple alignment of nucleic sequences of KTR2 (G19833, JaloEEP558 and BAT93) and KTR2/3 (JaloEEP558) showing that the first 232bp of KTR2/3 are highly identical to the sequence of KTR2. The unknown domain of ~ 100 amino-acid present in the truncated CRR3 kinases is boxed in yellow. The three related repeats of 78 or 69pb are shown by arrows.
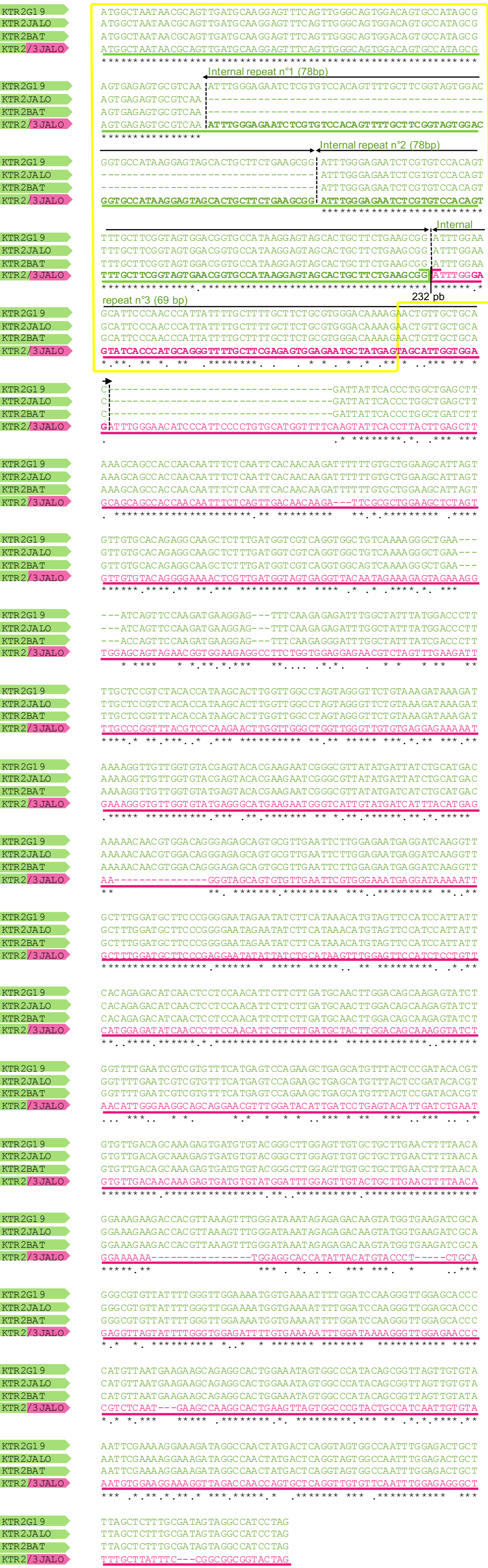
Figure S4: Multiple alignment of nucleic sequences of KTR3 (G19833, JaloEEP558 and BAT93) and KTR2/3 (JaloEEP558) showing that the last 953bp of KTR2/3 are highly identical to the sequence of KTR3. The unknown domain of ~ 100 amino-acid present in the truncated CRR3 kinases is boxed in yellow. The three related repeats of 78 or 69pb are shown by arrows.

Fig. S5: Time-course expression analysis of the *P. vulgaris* genes KTR2, KTR3 and KFL of JaloEEP558 in response to *C. lindemuthianum* strain 100. The relative abundance of *KTR2/3* transcripts was calculated by comparing KTR2/3 cDNA level in inoculated leaves with mock controls at each respective timepoint at 6, 24, 48, 72 and 96 hpi. Data was normalized using the *PvUnk1*, *PvUnk2*, *PvIDE* and *PvAct11* reference genes. Bars represent the mean ± SD, n=2 independent experiments.
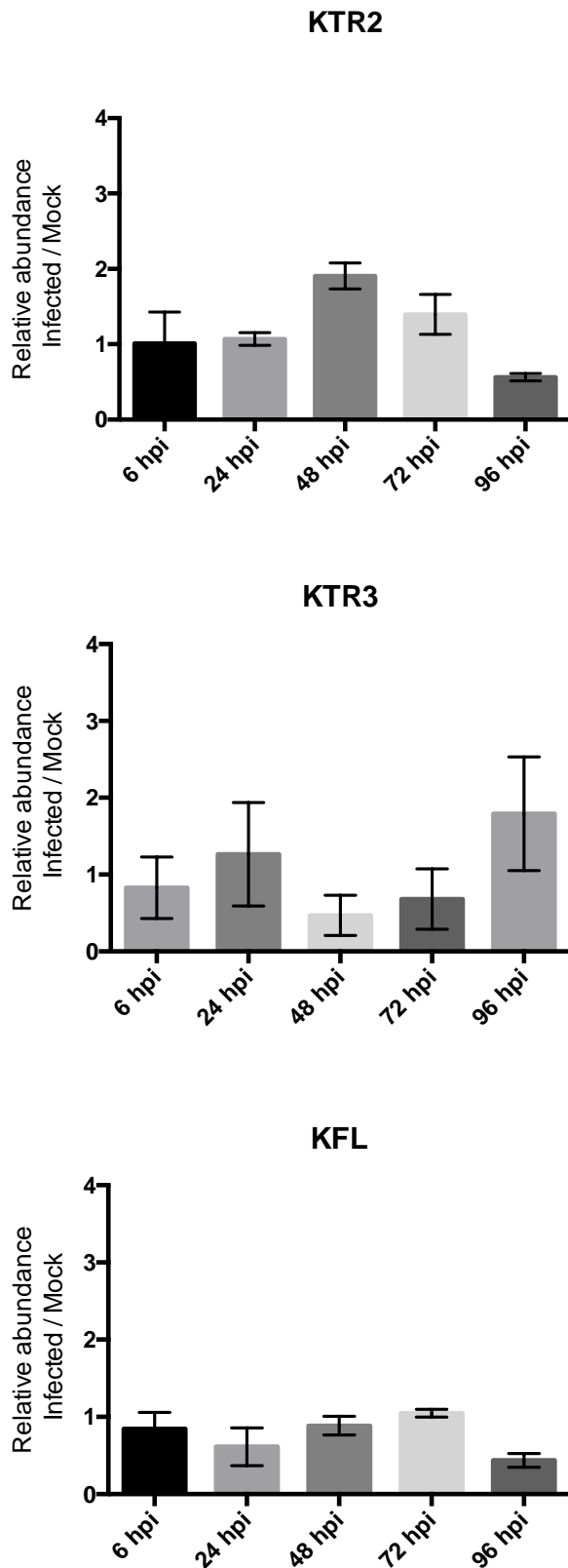
Figure S6: Transient expression of KTR2/3 in common bean hypocotyls and *C. lindemuthianum* disease assay scoring at 7 dpi.

A and B. Pictures of JaloEEP558 hypocotyls after agroinfiltration with pCambia0390 (A) or pCambia0390+KTR2/3 (B), and inoculation with *C. lindemuthianum* strain 100. As expected, no symptoms were observed at 7 dpi, since JaloEEP558 is resistant to strain 100.

C and D. Pictures of BAT93 hypocotyls after agroinfiltration with pCambia0390 (C) or pCambia0390+KTR2/3 (D), and inoculation with *C. lindemuthianum* strain 100. At 7 dpi, typical anthracnose symptoms were massively observed in (C) and only rarely in (D), where droplets of inoculum were deposited.

E. Example of symptoms scoring. When clear browning symptoms were observed, the inoculation spot was scored as susceptible (S), while when no or very limited browning was observed the inoculation spot was scored as resistant (R).
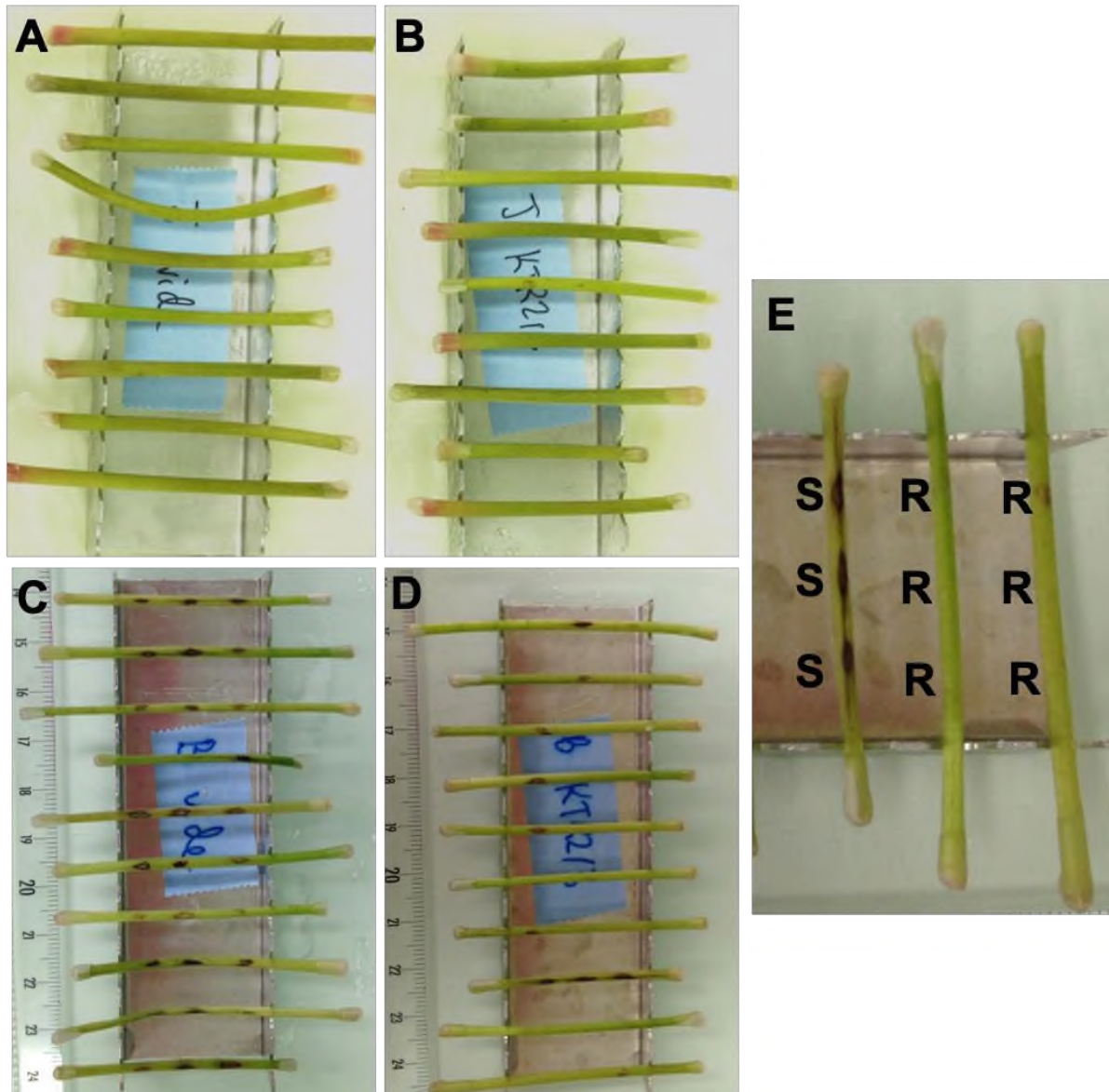
Table S1: List of primer sequences used in this study (RT-qPCR, Cloning).

Gene or amplification product name	Primer Name	Primer sequences (5' - 3')	Amplicon size (bp)	Reference
RT-qPCR primers				
Tested genes				
Phvul.001G243600 (KTR2)	RepetJalo1F	GTCCCACGCAGAAGCAAAAGC	153	This study
	KTR2q-R	GCCATAGCGAGTGAGAGTGCG		This study
Phvul.001G243700 (KTR3)	KTR3q-F	ATGCACAGGGGAATGGGATG	234	This study
	RepetJalo1R	TTGGGAGCAGTACCTGTTCGTG		This study
(KTR2/3)	KTR3q-F	ATGCACAGGGGAATGGGATG	279	This study
	KTR2q-R	GCCATAGCGAGTGAGAGTGCG		This study
Phvul.001G243800 (KFL)	KinaseFL-3F	CGGGATCCGATGCTGCTCGGGGAATAG	373	This study
	KinaseFL-3R	CGGGATCCCATTTCTCCAGCCAAAATACG		This study
Reference genes				
Insulin degrading enzyme (PvIDE)	IDE-F	GCAACCAACCTTTCATCAGC	156	(Borges et al. 2012)
	IDE-R	AGAAATGCCTCAACCCTTTG		(Borges et al. 2012)
Unknown 1 (PvUkn1)	Ukn1-F	ATTCCCATCATGCAGCAAAG	192	(Borges et al. 2012)
	Ukn1-R	AGATCCCTCCAGGTCAATCC		(Borges et al. 2012)
Unknown 2 (PvUkn2)	Ukn2-F	CCAATTCAACCATCCCTCAC	153	(Borges et al. 2012)
	Ukn2-R	AAACTCCTCTGCACCCTCAG		(Borges et al. 2012)
Actin-11 (PvAct11)	Act11-F	TGCATACGTTGGTGATGAGG	190	(Borges et al. 2012)
	Act11-R	AGCCTTGGGGTTAAGAGGAG		(Borges et al. 2012)
KTR2/3 amplification				
	RepetJalo1F	GTCCCACGCAGAAGCAAAAGC	3179 in JaloEEP558*	This study
	RepetJalo1R	TTGGGAGCAGTACCTGTTCGTG	1639 in BAT93 **	This study
	KTR2/3exp-F2	CGGGATCCGTCAAATTGGAGATTTTTACATAG	1563	This study
	KTR2/3exp-R	CGGGATCCCCAATTTGACTAGTACCGCCG		This study
Long Range PCR amplification				
LR1	LR1-F	AAGCGAAAAACACGCTCACT	9464	This study
	LR1-R	GTCCTATCTCACTCGCCCTTTAT		
LR2	LR2.2-F	GCAATTAGGGGAATGACACTGCT	7523	This study
	LR2.2-R	ACAAATACCCAAAGCCCTCTCCA		
LR3	LR3-F	TATCTGCGACCAAACAGTGC	10927	This study
	LR3-R	CCGAGGACAATTCACAAGGTA		

LR4	LR4-F	GACAGTGCCATAGCGAGTGA	9812	This study
	LR4-R	CTCCTTGCCCAGAACTTGAG		
LR5	LR5-F	GAGGAAGGAACCAAAGTCTAACAG	11943	This study
	LR5-R	GCCCTTCTTATTTTTGTCCCAAAC		
LR6	LR6-F	CTCTCCCAACCCAACTCCTGAA	8415	This study
	LR6-R	ACGGTGTCCTTGGTTCATGTCC		
LR7	LR7-F	GGTGGGTGCCTCCAATCAGT	7844	This study
	LR7-R	TTCTCCTCCAATCTCCTTCAACTC		

*: referred to as the 3.1kb band, **: referred to as the 1.7kb band

Supplemental Table S2: Common bean genotypes of various geographical origins tested for resistance to strain 100 of *C. lindemuthianum* and for the presence of *KTR2/3* encoding gene.

[a]Resistant = R; Susceptible = S; Ambiguous = -.

[b] size of the fragment amplified by PCR using RepetJalo1F/R

[c] in bold, underlined, genotypes for which *KTR2/3* gene was sequenced

[d] MA=MesoAmerican

The genotypes presenting a 3.1 Kb band possess KTR2/3, while the genotypes presenting a 1.7 Kb
band lack KTR2/3. The blue highlighting shows that all the genotypes presenting the 3.1 Kb band
(KTR2/3) are resistant to strain 100 of *C. lindemuthianum* and are cultivated Andean genotypes,
with the notable exception of the four Mesoamerican cultivars.

	Gene pool/ Country of origin	BC / ADP ID	Market class / Type	Genotype	Disease reaction to strain 100[a]	PCR[b,c]
Cultivated	Andean	ADP-1	Red mottle	ROZI KOKO	R	**~3.1 Kb**
Cultivated	Andean	ADP-10	Red	CANADA	R	~1.7 Kb
Cultivated	Andean	ADP-102	Purple speckled	Jesca	R	**~3.1 Kb**
Cultivated	Andean	ADP-33	Purple speckled	KIJIVU	R	~3.1 Kb
Cultivated	Andean	ADP-427	Light Red Kidney	Badillo	S	~1.7 Kb
Cultivated	Andean	ADP-526	Red mottle	CAL-143	-	~3.1 Kb
Cultivated	Andean	ADP-598	Dark Red Kidney	Charlevoix	R	~3.1 Kb
Cultivated	Andean	ADP-599	Dark Red Kidney	Isles	R	~3.1 Kb
Cultivated	Andean	ADP-602	Light Red Kidney	Sacramento	R	~3.1 Kb
Cultivated	Andean	ADP-608	Cranberry	UI-51	S	~1.7 Kb
Cultivated	Andean	ADP-610	Cranberry	G122	R	**~3.1 Kb**
Cultivated	Andean	ADP-611	Red mottle	Pompadour B	R	~1.7 Kb
Cultivated	Andean	ADP-612	Red mottle	ICA Quimbaya	R	**~3.1 Kb**
Cultivated	Andean	ADP-617	Cranberry	Red Rider	-	~3.1 Kb
Cultivated	Andean	ADP-621	Yellow	Jalo EEP558	R	**~3.1 Kb**
Cultivated	Andean	ADP-623	Light Red Kidney	Drake	R	~3.1 Kb
Cultivated	Andean	ADP-624	Cranberry	Dolly	R	~3.1 Kb
Cultivated	Andean	ADP-626	Dark Red Kidney	Montcalm	R	**~3.1 Kb**
Cultivated	Andean	ADP-634	Light Red Kidney	UC Red Kidney	R	~3.1 Kb
Cultivated	Andean	ADP-638	Dark Red Kidney	Red Hawk	R	DNA not available
Cultivated	Andean	ADP-639	Light Red Kidney	Chinook 2000	R	~3.1 Kb
Cultivated	Andean	ADP-640	White Kidney	Beluga	R	**~3.1 Kb**
Cultivated	Andean	ADP-641	Cranberry	Capri	S	~1.7 Kb
Cultivated	Andean	ADP-642	Cranberry	Taylor Hort.	S	~1.7 Kb
Cultivated	Andean	ADP-643	Cranberry	Cardinal	S	~1.7 Kb
Cultivated	Andean	ADP-645	White Kidney	Lassen	R	~3.1 Kb
Cultivated	Andean	ADP-646	Yellow	Myasi	S	~1.7 Kb
Cultivated	Andean	ADP-647	Light Red Kidney	Red Kanner	S	~1.7 Kb
Cultivated	Andean	ADP-648	Light Red Kidney	Red Kloud	S	~1.7 Kb
Cultivated	Andean	ADP-650	Light Red Kidney	K-42	R	~3.1 Kb
Cultivated	Andean	ADP-653	Dark Red Kidney	USDK-CBB-15	R	~3.1 Kb
Cultivated	Andean	ADP-655	Dark Red Kidney	Fiero	R	~3.1 Kb
Cultivated	Andean	ADP-656	Dark Red Kidney	Royal Red	R	~3.1 Kb
Cultivated	Andean	ADP-657	Light Red Kidney	Kardinal	R	**~3.1 Kb**
Cultivated	Andean	ADP-658	Light Red Kidney	Blush	R	~3.1 Kb
Cultivated	Andean	ADP-660	Cranberry	Krimson	R	~1.7 Kb
Cultivated	Andean	ADP-664	White Kidney	Silver Cloud	R	~3.1 Kb
Cultivated	Andean	ADP-665	White Kidney	USWK-CBB-17	S	~1.7 Kb
Cultivated	Andean	ADP-668	Cranberry	Cran-09	S	~1.7 Kb
Cultivated	Andean	ADP-672	Dark Red Kidney	CDRK	R	~3.1 Kb
Cultivated	Andean	ADP-676	Light Red Kidney	CELRK	R	~3.1 Kb
Cultivated	Andean	ADP-677	Cranberry	Etna	S	~1.7 Kb
Cultivated	Andean	ADP-683	Pink cranberry	Ind. Jamaica Red	S	~1.7 Kb
Cultivated	Andean	ADP-684	Dark Red Kidney	Majesty	R	**~3.1 Kb**
Cultivated	Andean	ADP-687	Light Red Kidney	Pink Panther	R	**~3.1 Kb**
Cultivated	Andean	ADP-7	Yellow	BUKOBA	S	~1.7 Kb
Cultivated	MA[d]	BC007	Great Northern	BelNeb-RR-1	S	~1.7 Kb
Cultivated	MA	BC016	Pinto	Bill Z	S	~1.7 Kb
Cultivated	MA	BC017	Pinto	Ouray	S	~1.7 Kb
Cultivated	MA	BC018	Pinto	Grand Mesa	S	~1.7 Kb
Cultivated	MA	BC020	Pinto	Montrose	S	~1.7 Kb
Cultivated	MA	BC025	Pinto	Arapaho	S	~1.7 Kb

Cultivated	MA	BC026	Small red	DOR 364	S	~1.7 Kb
Cultivated	MA	BC063	Black	Black Magic	S	~1.7 Kb
Cultivated	MA	BC069	Black	Blackhawk	S	~1.7 Kb
Cultivated	MA	BC070	Pinto	Sierra	S	~1.7 Kb
Cultivated	MA	BC073	Pinto	Aztec	S	~1.7 Kb
Cultivated	MA	BC074	Navy	Huron	-	~1.7 Kb
Cultivated	MA	BC075	Black	Raven	R	**~3.1 Kb**
Cultivated	MA	BC077	Navy	Newport	R	**~3.1 Kb**
Cultivated	MA	BC079	Pinto	Kodiak	S	~1.7 Kb
Cultivated	MA	BC080	Great Northern	Matterhorn	S	~1.7 Kb
Cultivated	MA	BC084	Black	Phantom	R	**~3.1 Kb**
Cultivated	MA	BC085	Black	Jaguar	R	**~3.1 Kb**
Cultivated	MA	BC086	Navy	Seahawk	S	~1.7 Kb
Cultivated	MA	BC088	Black	Zorro	-	~1.7 Kb
Cultivated	MA	BC089	Pinto	Santa Fe	S	~1.7 Kb
Cultivated	MA	BC110	Pinto	Topaz	S	~1.7 Kb
Cultivated	MA	BC120	Pinto	La Paz	S	~1.7 Kb
Cultivated	MA	BC121	Pinto	Baja	S	~1.7 Kb
Cultivated	MA	BC137	Great Northern	Beryl R	S	~1.7 Kb
Cultivated	MA	BC139	Great Northern	Sapphire	S	~1.7 Kb
Cultivated	MA	BC141	Small red	Garnet	S	~1.7 Kb
Cultivated	MA	BC142	Pink	ROG 312	S	~1.7 Kb
Cultivated	MA	BC160	Pink	UI-537	S	~1.7 Kb
Cultivated	MA	BC161	Pinto	Common Pinto	S	~1.7 Kb
Cultivated	MA	BC168	Pinto	UI-196	S	~1.7 Kb
Cultivated	MA	BC172	Black	UI-906	S	~1.7 Kb
Cultivated	MA	BC177	Pinto	UI-111	S	~1.7 Kb
Cultivated	MA	BC178	Pinto	UI-114	S	~1.7 Kb
Cultivated	MA	BC180	Great Northern	BelNeb-RR-2	S	~1.7 Kb
Cultivated	MA	BC185	Great Northern	GN#1Sel27	S	~1.7 Kb
Cultivated	MA	BC186	Great Northern	GN Harris	S	~1.7 Kb
Cultivated	MA	BC190	Great Northern	Starlight	S	~1.7 Kb
Cultivated	MA	BC191	Great Northern	Emerson	R	~1.7 Kb
Cultivated	MA	BC193	Great Northern	ABC-Weihing	S	~1.7 Kb
Cultivated	MA	BC195	Great Northern	ABCP-8	S	~1.7 Kb
Cultivated	MA	BC196	Great Northern	Chase	S	~1.7 Kb
Cultivated	MA	BC209	Pinto	AC Pintoba	S	~1.7 Kb
Cultivated	MA	BC215	Black	A-55	S	~1.7 Kb
Cultivated	MA	BC220	Great Northern	JM-24	S	~1.7 Kb
Cultivated	MA	BC222	Pinto	Quincy	S	~1.7 Kb
Cultivated	MA	BC223	Pinto	Burke	R	~1.7 Kb
Cultivated	MA	BC225	Pinto	JM-126	S	~1.7 Kb
Cultivated	MA	BC227	Pinto	Pindak	S	~1.7 Kb
Cultivated	MA	BC228	Pinto	Nodak	S	~1.7 Kb
Cultivated	MA	BC229	Pinto	Holberg	S	~1.7 Kb
Cultivated	MA	BC231	Pinto	Othello	S	~1.7 Kb
Cultivated	MA	BC232	Pinto	NW590	S	~1.7 Kb
Cultivated	MA	BC235	Pinto	USPT-WM-1	S	~1.7 Kb
Cultivated	MA	BC236	Pinto	USPT-CBB-1	S	~1.7 Kb
Cultivated	MA	BC238	Pinto	USPT-ANT-1	S	~1.7 Kb
Cultivated	MA	BC243	Small red	USRM-20	S	~1.7 Kb
Cultivated	MA	BC266	Pink	6R-42	S	~1.7 Kb
Cultivated	MA	BC268	Pink	USWA-61	S	~1.7 Kb
Cultivated	MA	BC271	Small red	Rojo Chiquito	S	~1.7 Kb
Cultivated	MA	BC273	Black mottle	Orca	S	~1.7 Kb
Cultivated	MA	BC278	Pink	Viva	S	~1.7 Kb
Cultivated	MA	BC279	Pink	Roza	S	~1.7 Kb
Cultivated	MA	BC280	Pink	Harold	S	~1.7 Kb
Cultivated	MA	BC297	Great Northern	GN9-4	S	~1.7 Kb
Cultivated	MA	BC306	Navy	Avalanche	S	~1.7 Kb
Cultivated	MA	BC307	Black	Eclipse	R	~1.7 Kb
Cultivated	MA	BC358	Great Northern	Orion	S	~1.7 Kb
Cultivated	MA	BC375	Pink	Yolano	S	~1.7 Kb
Cultivated	MA	BC383	Pinto	Apache	R	~1.7 Kb
Cultivated	MA	BC384	Pinto	Fiesta	S	~1.7 Kb
Cultivated	MA	BC386	Pinto	Buster	S	~1.7 Kb
Cultivated	MA	BC387	Pinto	Medicine Hat	S	~1.7 Kb

Cultivated	MA	BC393	Navy	Avanti	S	~1.7 Kb
Cultivated	MA	BC-030	Small white	Morales	not tested	~1.7 Kb
Cultivated	MA	BC-258	Small white	NW_395	not tested	~1.7 Kb
Cultivated	MA	BC-260	Small white	USWA_50	not tested	~1.7 Kb
Cultivated	Andean		Cranberry	G19833	R	~1.7 Kb
Cultivated	Andean			MDRK	R	~3.1 Kb
Cultivated	Andean			AFN	S	~1.7 Kb
Cultivated	Andean			Aiguille verte	S	~1.7 Kb
Cultivated	Andean			La victoire	S	~1.7 Kb
Cultivated	Andean/MA			Widusa	S	~1.7 Kb
Cultivated	MA			Mex222	R	~1.7 Kb
Cultivated	MA			AB136	S	~1.7 Kb
Cultivated	MA			BAT93	S	~1.7 Kb
Cultivated	MA			Cornell 49242	S	~1.7 Kb
Cultivated	MA			DOR364	S	~1.7 Kb
Cultivated	MA			Michelite	S	~1.7 Kb
Cultivated	MA			Mz	S	~1.7 Kb
Cultivated	MA			PI 207261	S	~1.7 Kb
Cultivated	MA			To	S	~1.7 Kb
Cultivated	MA			Tu	S	~1.7 Kb
Cultivated				Rosinha	R	~1.7 Kb
Cultivated	Andean			AND277	R	~3.1 Kb
Cultivated	Andean			Kaboon	R	~3.1 Kb
Cultivated	Andean			Perry Marrow	R	~3.1 Kb
Cultivated	Andean			Black Valentine	S	~1.7 Kb
Cultivated				Castelluccisa	S	~1.7 Kb
Cultivated	Andean			Corel	S	~1.7 Kb
Cultivated				Degli Ortolani	S	~1.7 Kb
Cultivated				Fagiolo del Purgatorio	S	~1.7 Kb
Cultivated	MA			G2333	S	~1.7 Kb
Cultivated				Va la vacca	S	~1.7 Kb
Cultivated	MA		Pink	Sutter Pink	S	~1.7 Kb
Wild	Colombian			G24404	R	~1.7 Kb
Wild	Mexico			G11051	R	~1.7 Kb
Wild	Guatemala			G19908	R	~1.7 Kb
Wild	Honduras			G50722	R	~1.7 Kb
Wild	Ecuador			G23582	S	~1.7 Kb
Wild	Peru			G23422	R	~1.7 Kb
Wild	Argentina			G19898	R	~1.7 Kb
Wild	Bolivia			G23442	S	~1.7 Kb
Wild	Argentina			G7469	R	~1.7 Kb
Wild	Peru			G12856	R	~1.7 Kb
Wild	Argentina			G19888	S	~1.7 Kb
Wild	Argentina			G19891	R	~1.7 Kb
Wild	Argentina			G19897	S	~1.7 Kb
Wild	Argentina			G19898	-	~1.7 Kb
Wild	Argentina			G19901	S	~1.7 Kb
Wild	Argentina			G21199	S	~1.7 Kb
Wild	Peru			G23420	S	~1.7 Kb
Wild	Peru			G23421	R	~1.7 Kb
Wild	Bolivia			G23444	R	~1.7 Kb
Wild	Bolivia			G23445	S	~1.7 Kb
Wild	Peru			G23455	S	~1.7 Kb
Wild	Argentina			G19902	R	~1.7 Kb
Wild	Argentina			ANP 1053	-	~1.7 Kb
Wild	Argentina			G19892	-	~1.7 Kb
Wild	Argentina			G21194	-	~1.7 Kb
Wild	Peru			G23419A	S	~1.7 Kb
Wild	Mexico			G11056	S	~1.7 Kb
Wild	Costa Rica			G23418	-	~1.7 Kb
Wild	Mexico			G23429	S	~1.7 Kb
Wild	Colombia			G23462	S	~1.7 Kb
Wild	Mexico			G24378	S	~1.7 Kb
Wild	Mexico			G24571	-	~1.7 Kb
Wild	Mexico			G24572A	-	~1.7 Kb
Wild	Mexico			PI325677	S	~1.7 Kb
Wild	Mexico			G12873	R	~1.7 Kb

Wild	Mexico			PI417770	R	~1.7 Kb
Wild	El Salvador			PI201013	S	~1.7 Kb
Wild	Mexico			G12879	-	~1.7 Kb
Wild	Mexico			86	S	~1.7 Kb
Wild	Ecuador			G23726	R	~1.7 Kb
Wild	El Salvador			G21245	-	~1.7 Kb