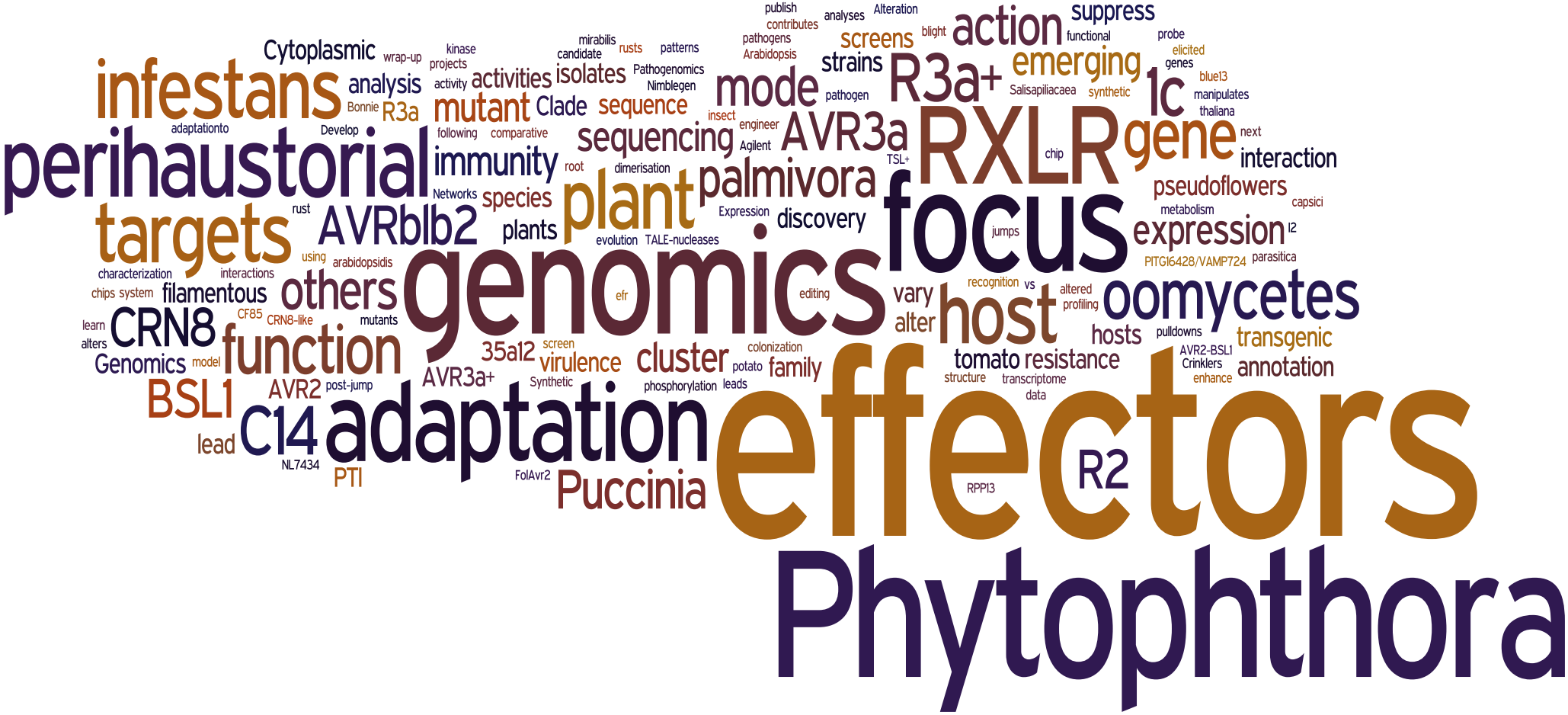


Keeping up with the Plant Destroyers

Sophien Kamoun

@kamounlab

Kamoun Lab @ TSL

Sophien Kamoun
@KamounLab

"Medecine can cure you one day but plants save your life everyday" #TwitterWisdom #plantsci /via @CristobalUauy

Crop losses due to fungi and oomycetes (filamentous plant pathogens)

Crop *Host species*	2009/2010 harvest (million tonnes)	Calories per 100g flour (un-cooked)	Disease/Pathogen and variation in % losses	Loss of food* for *x* million over 1 year, given diet of 2,000 calories per day

TOTAL: Could feed 596 – 4,287 million mouths *per annum***

Fisher *et al*. 2012

TOTAL: Could feed 596 – 4,287 million mouths *per annum***

Armed and Dangerous

These fungi, weeds, and viruses are among the more serious biological threats to food security—so researchers are working hard on countermeasures

BIG 7

RICE BLAST

Pest: *Magnaporthe oryzae*
Crops: Rice, 50 species of grasses and sedges

POTATO BLIGHT

Pest: *Phytophthora infestans*
Crops: Potatoes; also tomatoes and other solanaceous crops

BLACK SIGATOKA

Pest: *Mycosphaerella fijiensis*
Crops: Bananas, plantains
Whereabouts: This fungus, first detected in Fiji in 1964, is now found in 100 countries in the Americas, Africa and South Asia.

WHEAT STEM RUST

Pest: *Puccinia graminis* Ug99
Crop: Wheat

ASIAN SOYBEAN RUST

Pest: *Phakopsora pachyrhizi*
Crops: At least 31 legume species, notably soybeans

Filamentous plant pathogens (fungi and oomycetes) cause destructive plant diseases

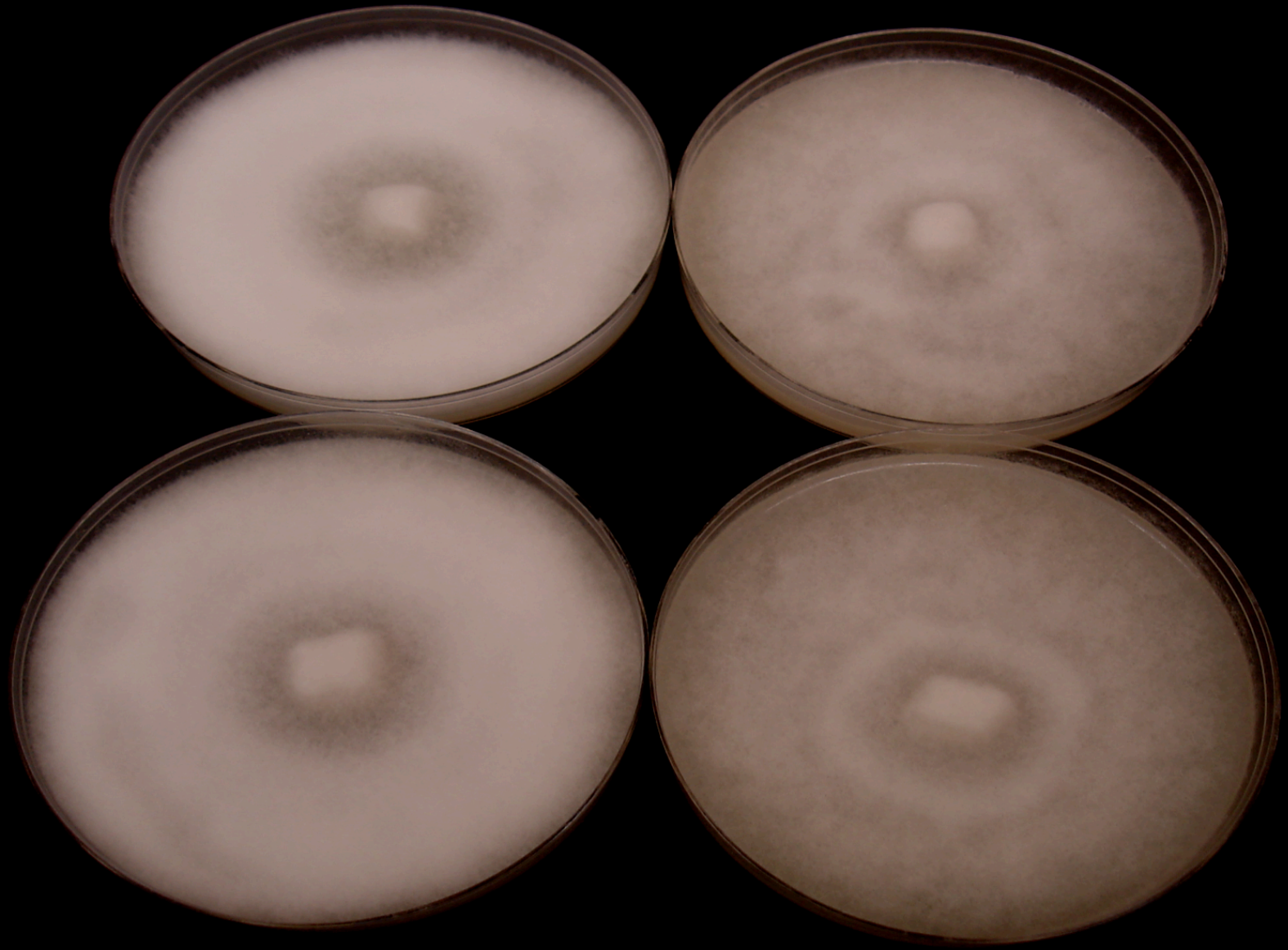
- **Filamentous pathogens** (fungi and oomycetes) cause most destructive diseases of plants
- **Highly adaptable -** can rapidly overcome plant resistance
- **Large population sizes**; mixed asexual and sexual reproduction


The Irish potato famine pathogen *Phytophthora infestans* causes potato blight

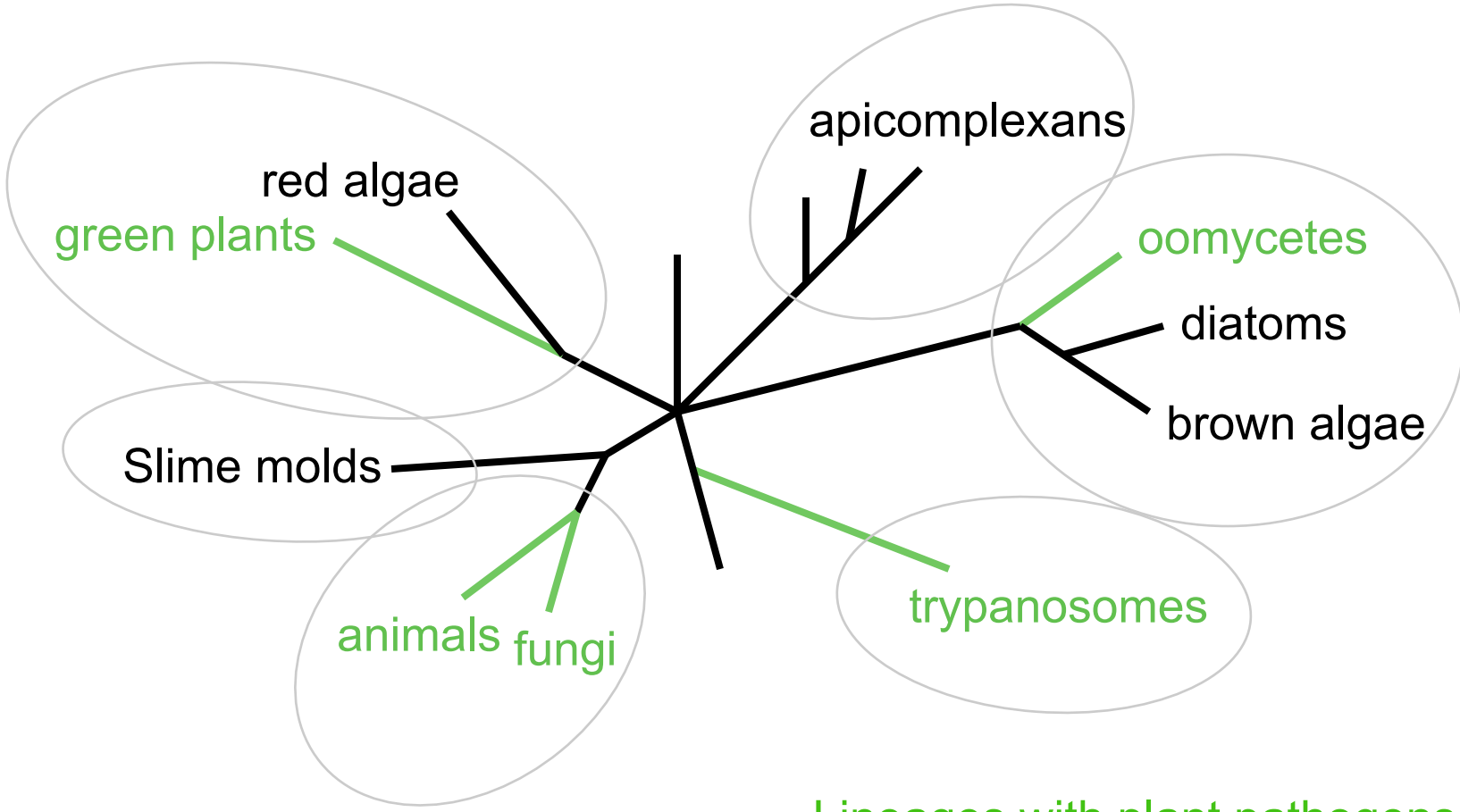
Phytophthora is Greek for “plant-destroyer”

Infection of potato plants
by *Phytophthora infestans*

Phytophthora: fungus-like oomycetes



Oomycetes are heterokonts - related to brown algae and diatoms



Lineages with plant pathogens in green

Adapted from Baldauf, Science (2003)

Phytophthora is an oomycete not a fungus

Oomycetes form an ancient eukaryotic lineage

- may have been parasitic ~300 million year ago
- present in the 407 million year-old Rhynie Chert, an ecosystem of plants, fungi and oomycetes

Christine Strullu-Derrien and Paul Kenrick
@ Natural History Museum

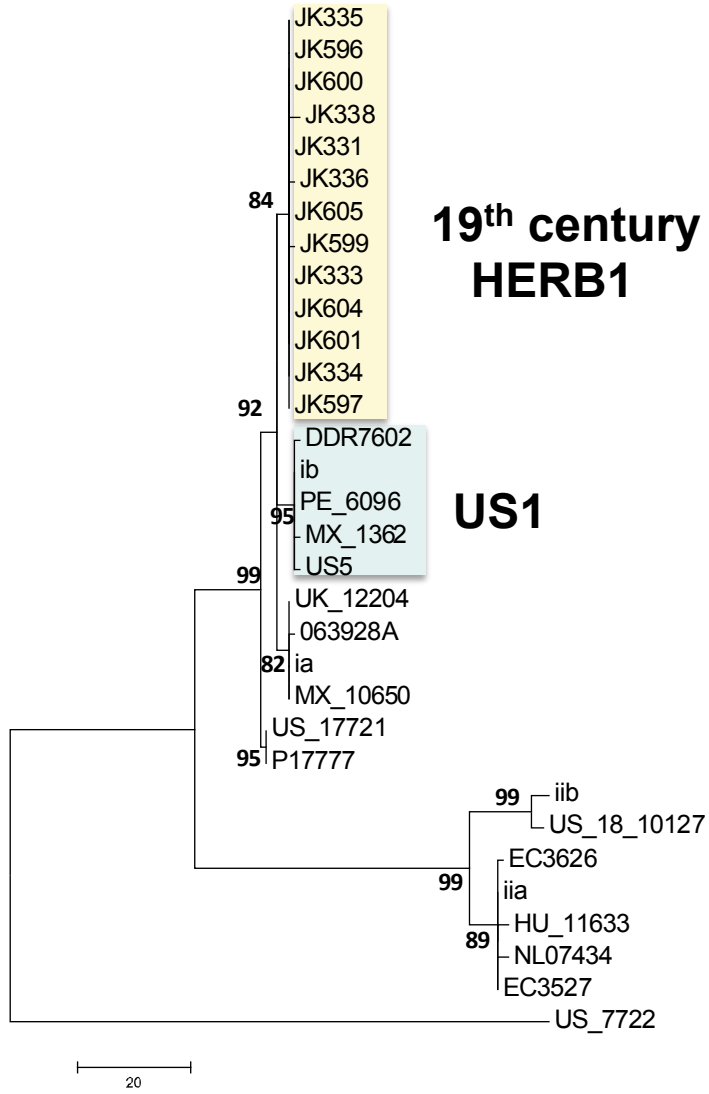

The Irish potato famine pathogen *Phytophthora infestans* causes potato blight

“Out of Mexico” – migration paths of *P. infestans*

Yoshida *et al*., 2013, eLife; 2014 PLOS Pathogens

w/ Johannes Krause, Marco Thines, Detlef Weigel and Hernan Burbano

“Genome archaeology” reveals HERB1 – the *P. infestans* lineage that triggered the Irish potato famine



19th century HERB1

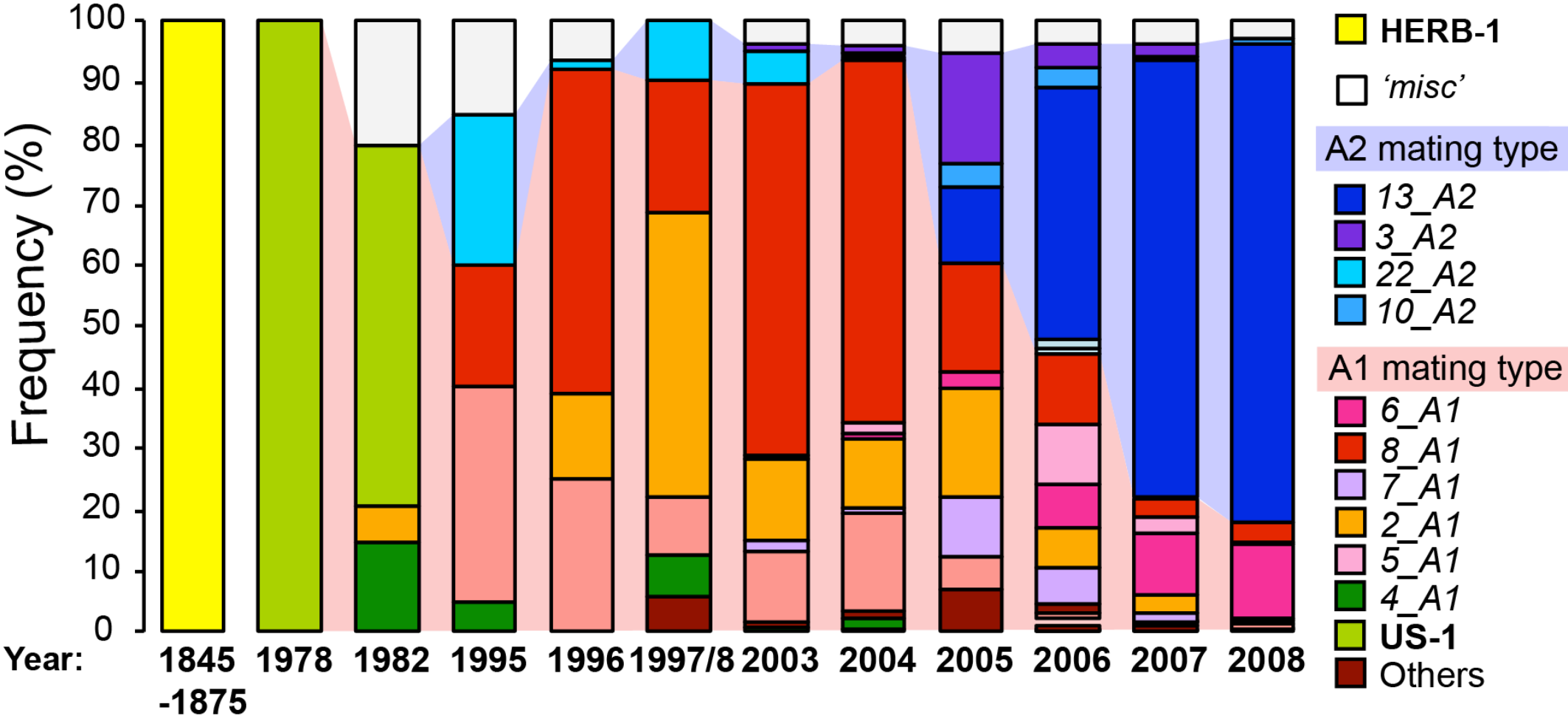
US1

Yoshida *et al*., 2013, eLife

w/ Johannes Krause, Marco Thines, Detlef Weigel and Hernan Burbano


Phytophthora Infestans Herb-1

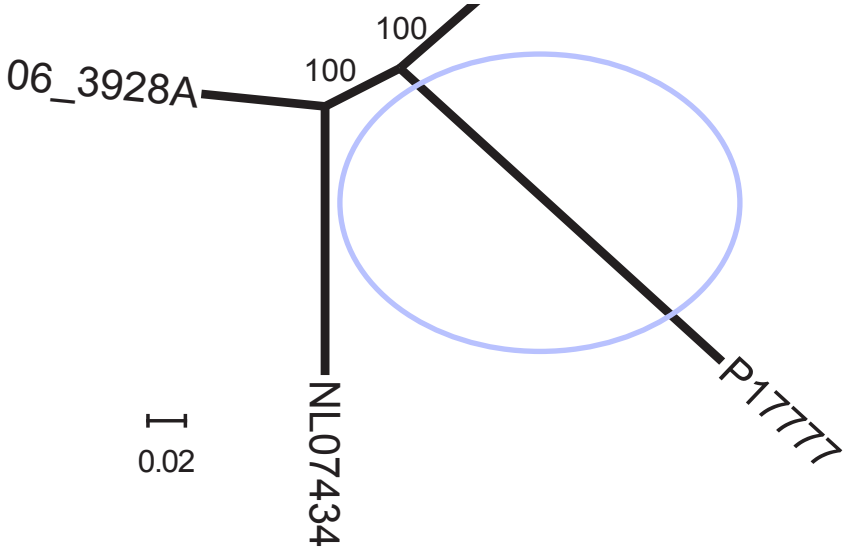
Attack of the clones - rise and fall of *Phytophthora infestans* asexual lineages in Britain

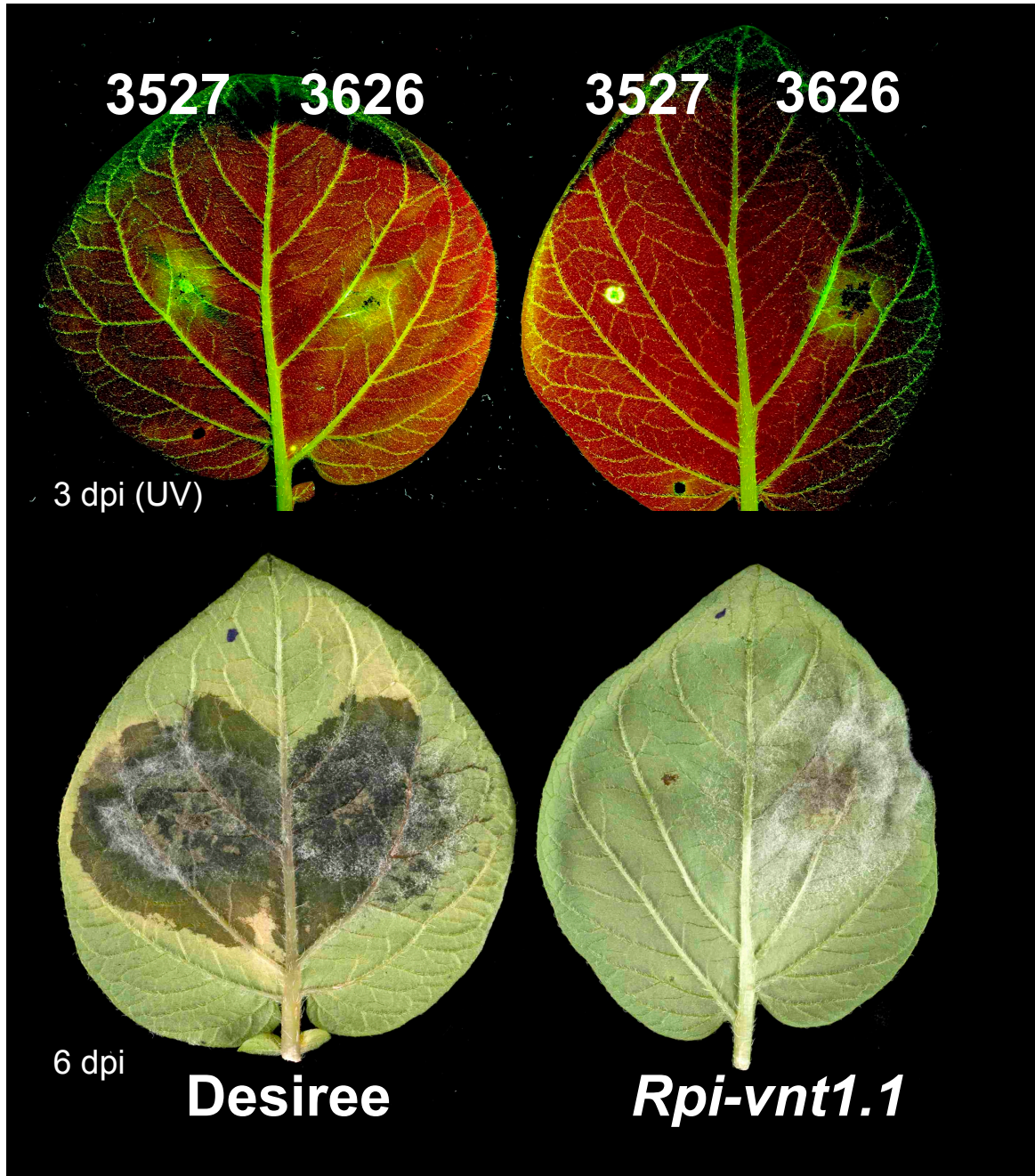


Yoshida *et al.*, 2014 PLOS Pathogens

Adapted from David Cooke, James Hutton Inst.

Evolution of virulence in the EC1 clonal lineage





Collaboration with Matthieu Pel and Vivianne Vleeshouwers

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Collaborate | EDIT

P_infestans_collection_places_2013

Collection trip was in 2013 in Colombia and Ecuador

Unlisted · Open Collaboration · 645 views
Created on Mar 20 · By · Updated Mar 21

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- E1
  Machachii,Potato fields,-00° 32' 21.4", -078° 36' 25.7", 3210m,Solanum tuberosum, Super Chola
- E2
  Machachii,Potato fields,-00° 32' 17.3", -078° 36' 03.3"", 3165m,Solanum tuberosum, Super Chola
- E3
  Chaupi,Potato fields,-00° 37' 24.6", -078° 36' 09.6", 3466m,Solanum tuberosum, Super Chola
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- E4
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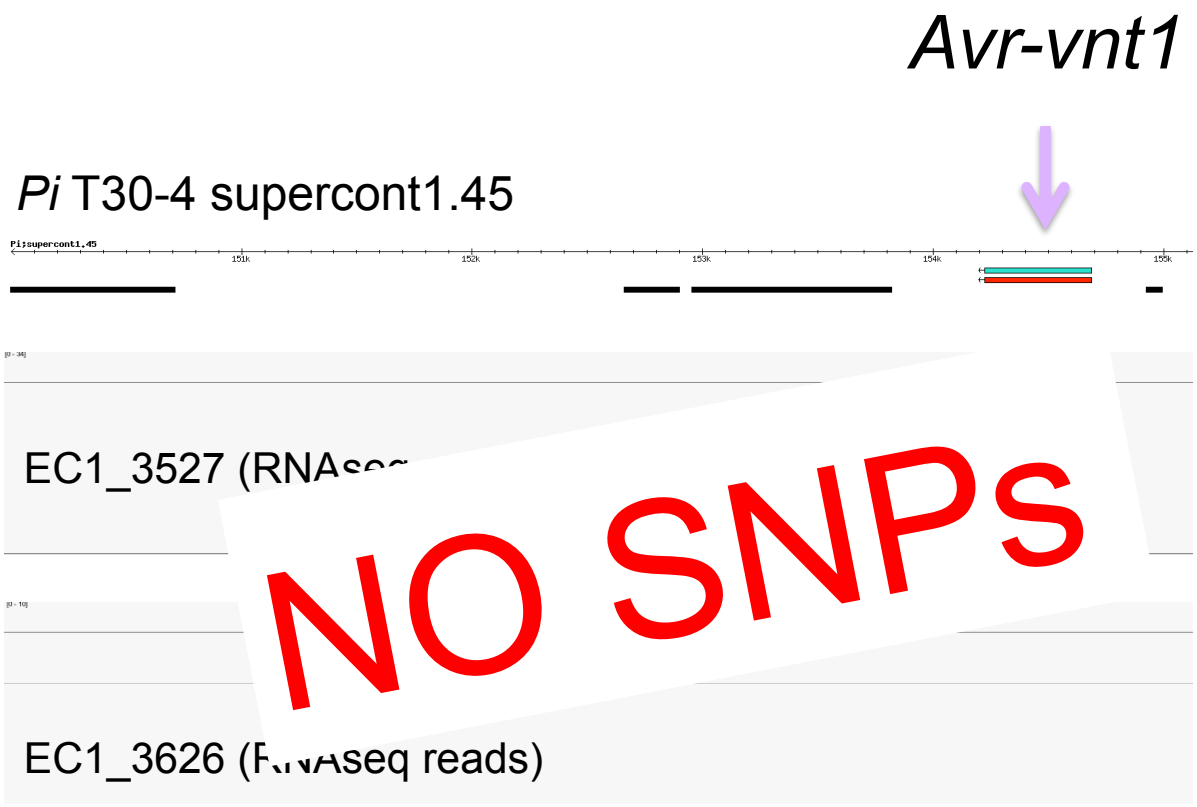
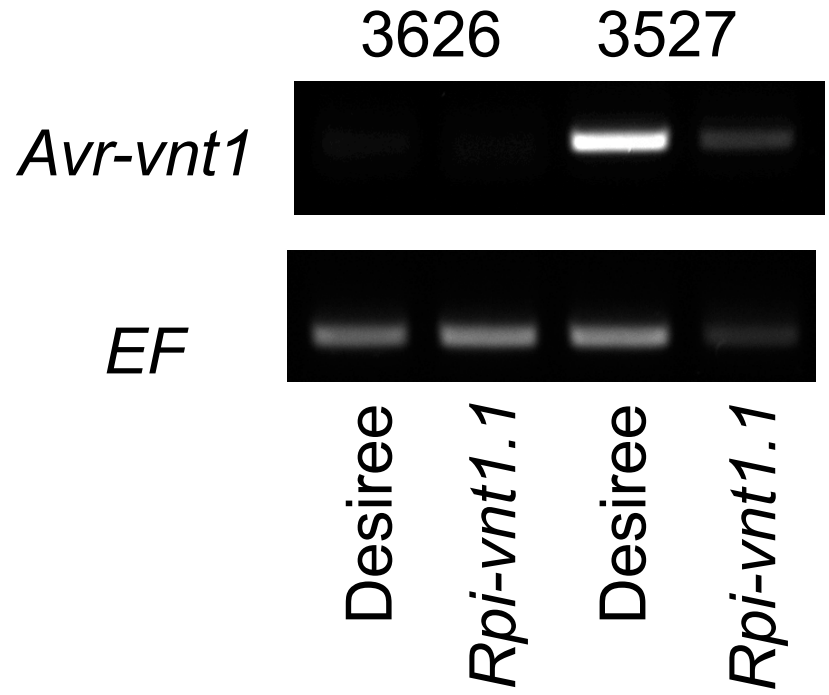
- E12

Kentaro Yoshida with collaborators in Ecuador, Colombia and Peru

Avr-vnt1 gene silencing resulted in gain-of-virulence





RT-PCR by Ricardo Oliva & Kamil Witek

Kentaro Yoshida, Marina Pais, Liliana Cano
Collaboration with Matthieu Pel and Vivianne Vleeshouwers

Attack of the Clones

Fungi have long been seen as the least interesting pathogens, but two catastrophes in the animal world have changed that view

WHEN *NATURE* RECENTLY ACCEPTED A review co-authored by Sarah Gurr, the plant pathologist from the University of Oxford in the United Kingdom sent the journal a self-produced image to consider for its cover. It shows a fungus looking like one of those colossal, menacing tripods from H. G. Wells's *War of the Worlds*, stalking through a field, with bats, frogs, and toads fleeing before it in a crazed panic. "Fungal Wars of the World," Gurr called it.

The picture didn't make it, but many scientists agree with its message: Fungi have now become a greater global threat to crops, forests, and wild animals than ever before. They have killed countless amphibians, pushing some species to extinction, and they're threatening the food supply for billions of people. More than 125 million tons of the top five food crops—rice, wheat, maize, potatoes, and soybeans—are destroyed by fungi every year.

Like other infectious agents, fungi benefit from a combination of trends, such as increased global travel and trade, new agricultural practices, and perhaps global warming. But they have several unique features researchers say—including the way the can switch from asexual to sexual reproduction—that enable them to exploit thes opportunities particularly effectively.

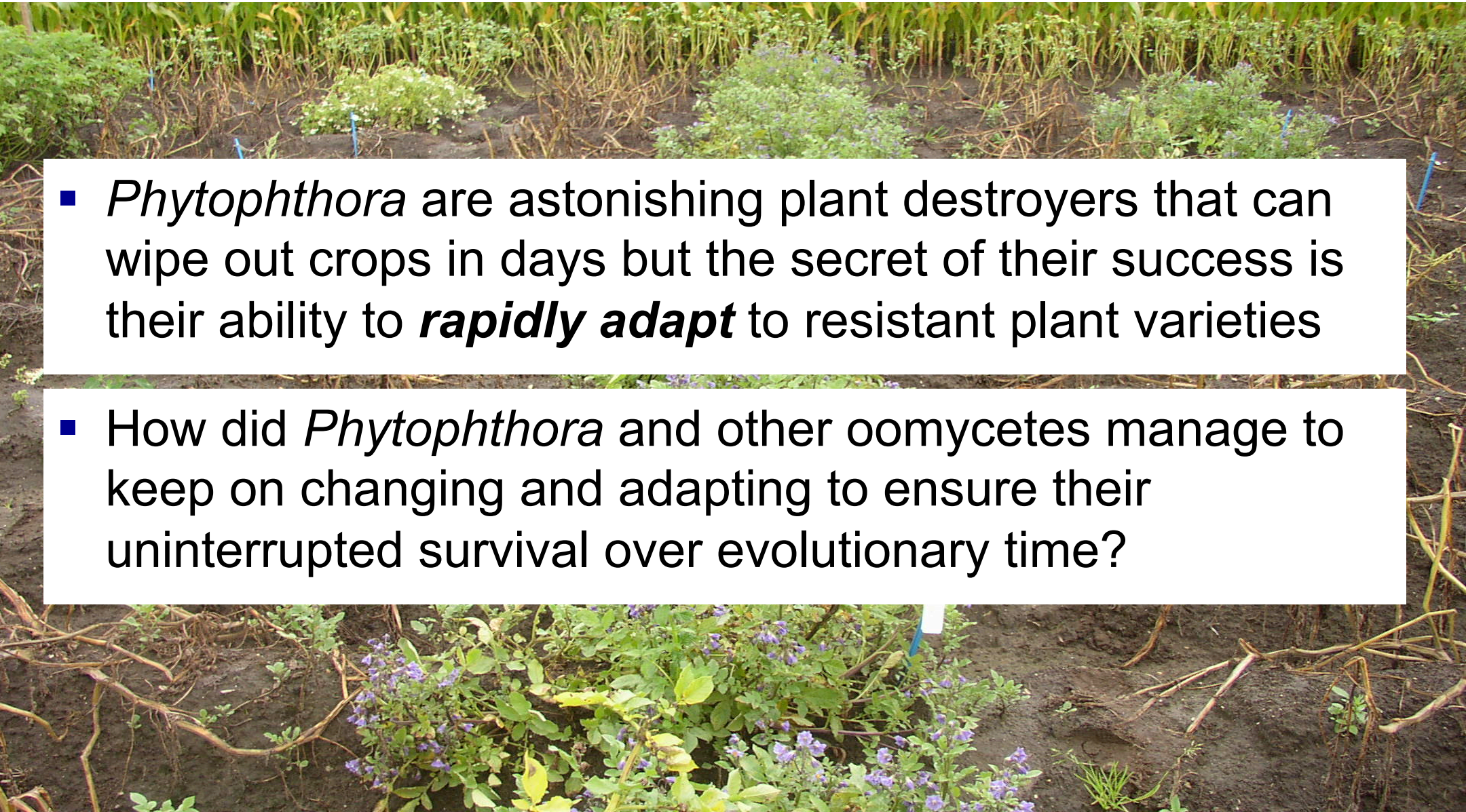
The *Nature* paper, published in April, wa in part a cry for attention; its authors say th world isn't fully aware of the dangers an should invest more in countermeasures. Fo decades, fungal diseases have been over shadowed by bacteria and viruses. "Ther are probably 50 or 100 bacterial experts fo every fungal expert," says Bruce McDonald a plant pathologist at the Swiss Federa Institute of Technology in Zurich. "Ther has always been a sense that fungi are no that important," adds microbiologist Artur Casadevall of Albert Einstein College o Medicine in New York City.

That has begun to change only ver recently, thanks in part to some highly pub licized animal die-offs. "A few years ago people just scoffed when you thought a fun gus had killed an animal such as a bat," say Gudrun Wibbelt, a veterinary pathologist a

Published by AAAS

- How can pathogen clones evolve rapidly?
- How does asexual reproduction affect genome evolution?
- Do pathogens require sexual reproduction?
- What's the role of humans in pathogen evolution?

Why the misery? Why are oomycetes the scourge of farmers worldwide?



- *Phytophthora* are astonishing plant destroyers that can wipe out crops in days but the secret of their success is their ability to ***rapidly adapt*** to resistant plant varieties

- How did *Phytophthora* and other oomycetes manage to keep on changing and adapting to ensure their uninterrupted survival over evolutionary time?

The genome sequence of *Phytophthora infestans*

with Brian Haas, Mike Zody, and Chad Nusbaum
@ Broad Institute

BROAD INSTITUTE

NSF

NRI
Knowledge For Tomorrow's Solutions

Pathogenomics is an emerging field of plant pathology

2005

young rice seedlings, whole plants often die, whereas spread of the disease to the stems, nodes or panicle of older plants results in nearly total loss of the rice grain[2]. Different host-limited forms of *M. grisea* also infect a broad range of grass species including wheat, barley and millet. Recent reports have shown that the fungus has the capacity to infect plant roots[7].

Here we present our preliminary analysis of the draft genome sequence of *M. grisea*, which has emerged as a model system for understanding plant–microbe interactions because of both its economic significance and genetic tractability[1,2].

Acquisition of the *M. grisea* genome sequence

The genome of a rice pathogenic strain of *M. grisea*, 70-15, was sequenced through a whole-genome shotgun approach. In all, greater than sevenfold sequence coverage was produced, and a

marker. In addition, 19 scaffolds (65% of genome assem
contained more than one marker and could
the map. The ends of chromosomes were ident
repeat motif (TTAGGG)*n*. Thirteen telomeric sequences
placed at the ends of scaffolds, of which six could be placed at
ends of chromosomes, whereas the remainder were associated
unanchored scaffolds (Supplementary Table S2). Genome cove
was estimated by aligning 28,682 *M. grisea* expressed sequence
(ESTs), representing genes expressed during a range of deve
mental stages and environment
94% of the ESTs were aligned
many of these ESTs being from d

The gene content of a plant pa

Within the *M. grisea* genome, 1

2006

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Jocelyn K. C. Rose,[24] Yasuko Sakihama
Chantel F. Scheuring,[18] Brian M. Smith
Trudy A. Torto-Alalibo,[1] Joe Win,[9] Zhan
Daniel S. Rokhsar,[2,7] Jeffrey L. Boore[2,

Draft genome sequences have been dete
the sudden oak death pathogen *Phytoph*
species share the kingdom Stramenopila
presence of many *Phytophthora* genes o
ancestry for the stramenopiles. Comparis
and diversification of many protein famil

2009

(KTH), School of Biotechnology, AlbaNova University Centre, Stockholm SE-10691, Sweden. [12]Department of Plant Pathology and Plant-Microbe Biology, Cornell University
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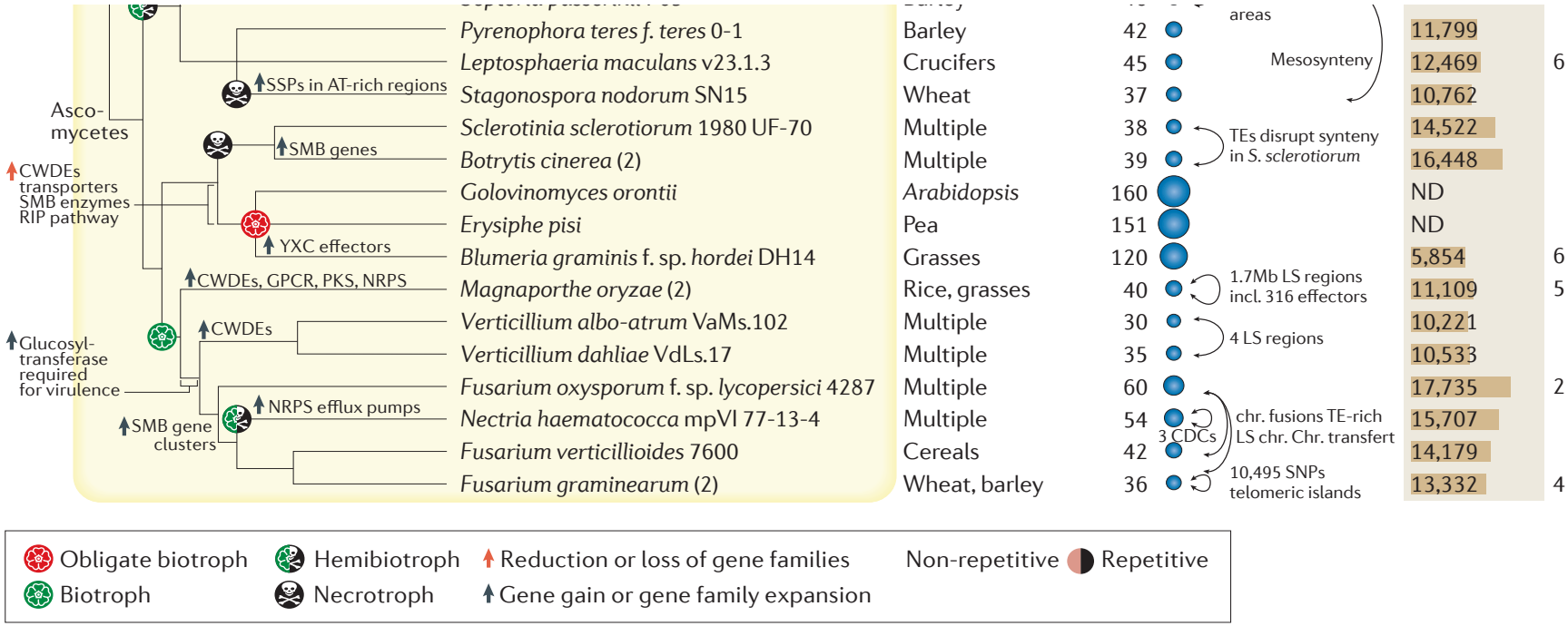
University of Dundee (at SCRI), Invergowrie, Dundee DD2 5DA, UK. †Present addresses: 454 Life Sciences, Branford, Connecticut 06405, USA (C.D.K.); Virginia Bioinforn
Institute, Virginia Polytechnic and State University, Blacksburg, Virginia 24061, USA (T.T.-A.); Biomedical Diagnostics Institute, Dublin City University, Dublin 9, Ireland (K
*These authors contributed equally to this work.



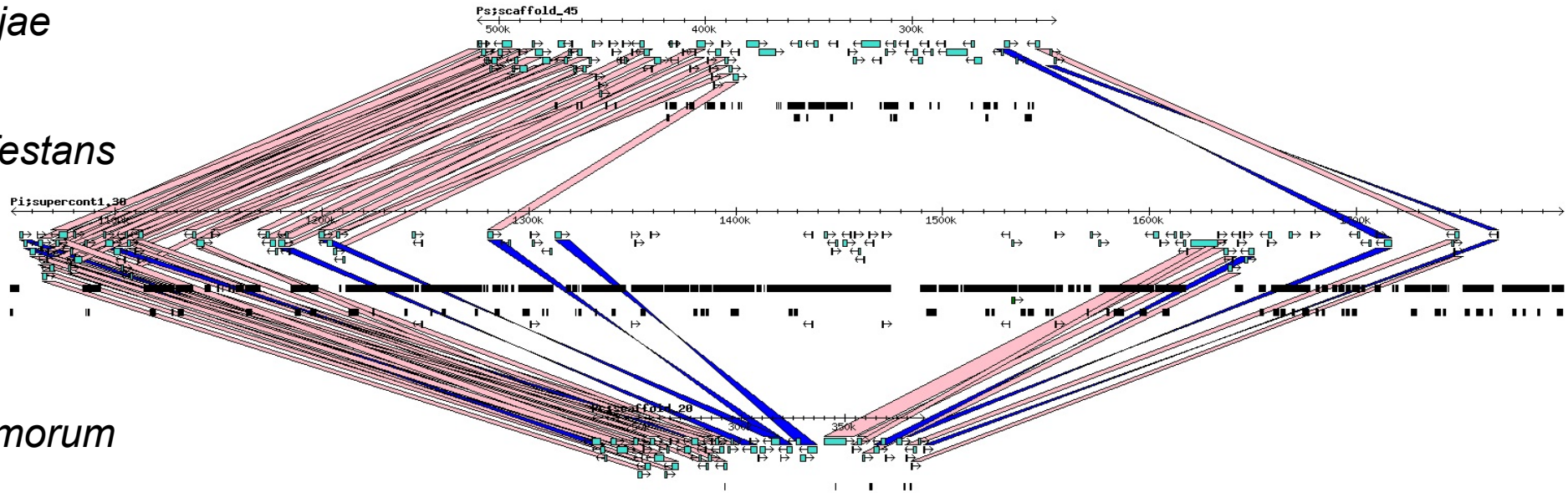
Raffaele and Kamoun, 2012
Nature Reviews Microbiol

Phytophthora infestans genome architecture - repeat-rich and gene-poor loci interrupt colinear regions

P. sojae

P. infestans

P. ramorum



B. Haas, S. Kamoun *et al.* Nature, 2009

Genomes of host-specific filamentous plant pathogens – *the bigger the better!*

- Typically, larger genomes than non-parasitic relatives
- Extreme repeat-driven expansions in distinct lineages:
  - *Phytophthora infestans*: 240 Mb, 74% repeats
  - Rust fungi: 68-100 Mb, 45% repeats
  - Powdery mildew fungi: 120-160 Mb, 65% repeats
- In sharp contrast to many parasites and symbionts that tend to evolve small compact genomes

Reduction and Compaction in the Genome of the Apicomplexan Parasite *Cryptosporidium parvum*

The genome of *Tetranychus urticae* reveals herbivorous pest adaptations

At 90 megabases *T. urticae* has the smallest sequenced arthropod genome.

Parasites genomes are often considered to be "reduced" or "degenerate," but exactly what do these terms mean? How various are the forces that affect genome size and density, and how do their effects differ in different parasites?

Extreme genome reduction in symbiotic bacteria

John P. McCutcheon[1] and Nancy A. Moran[2]

Sequence and genetic map of *Meloidogyne hapla*: A compact nematode genome for plant parasitism

Charles H. Opperman[a,b,c], David M. Bird[a,b], Valerie M. Williamson[d], Dan S. Rokhsar[e], Mark Burke[a], Jonathan Cohn[a], John Cromer[a], Steve Diener[a,f], Jim Gajan[a], Steve Graham[a], T. D. Houfek[a,g], Qingli Liu[d,h], Therese Mitros[i], Jennifer Schaff[a,j], Reenah Schaffer[a], Elizabeth Scholl[a], Bryon R. Sosinski[k,l], Varghese P. Thomas[d], and Eric Windham[a]

- Why is bigger better in filamentous plant pathogens?
- Which evolutionary tradeoffs counterbalance the cost of the larger genomes?

Fungal mimicry of plants - “fake” flowers triggered by the rust *Puccinia monoica* on *Boechera stricta*

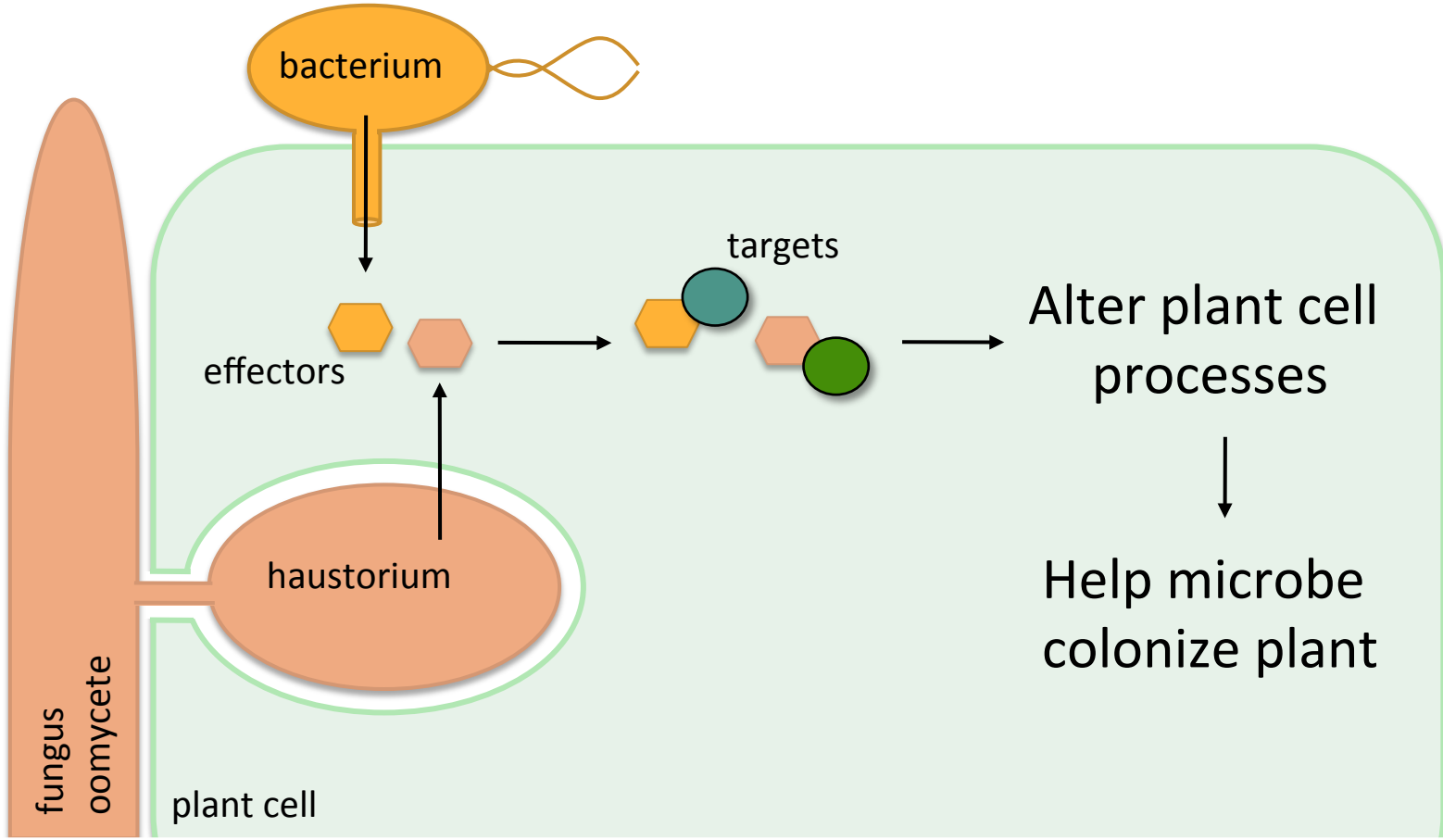
rust infected plant
with pseudoflowers

healthy plant with
normal flowers

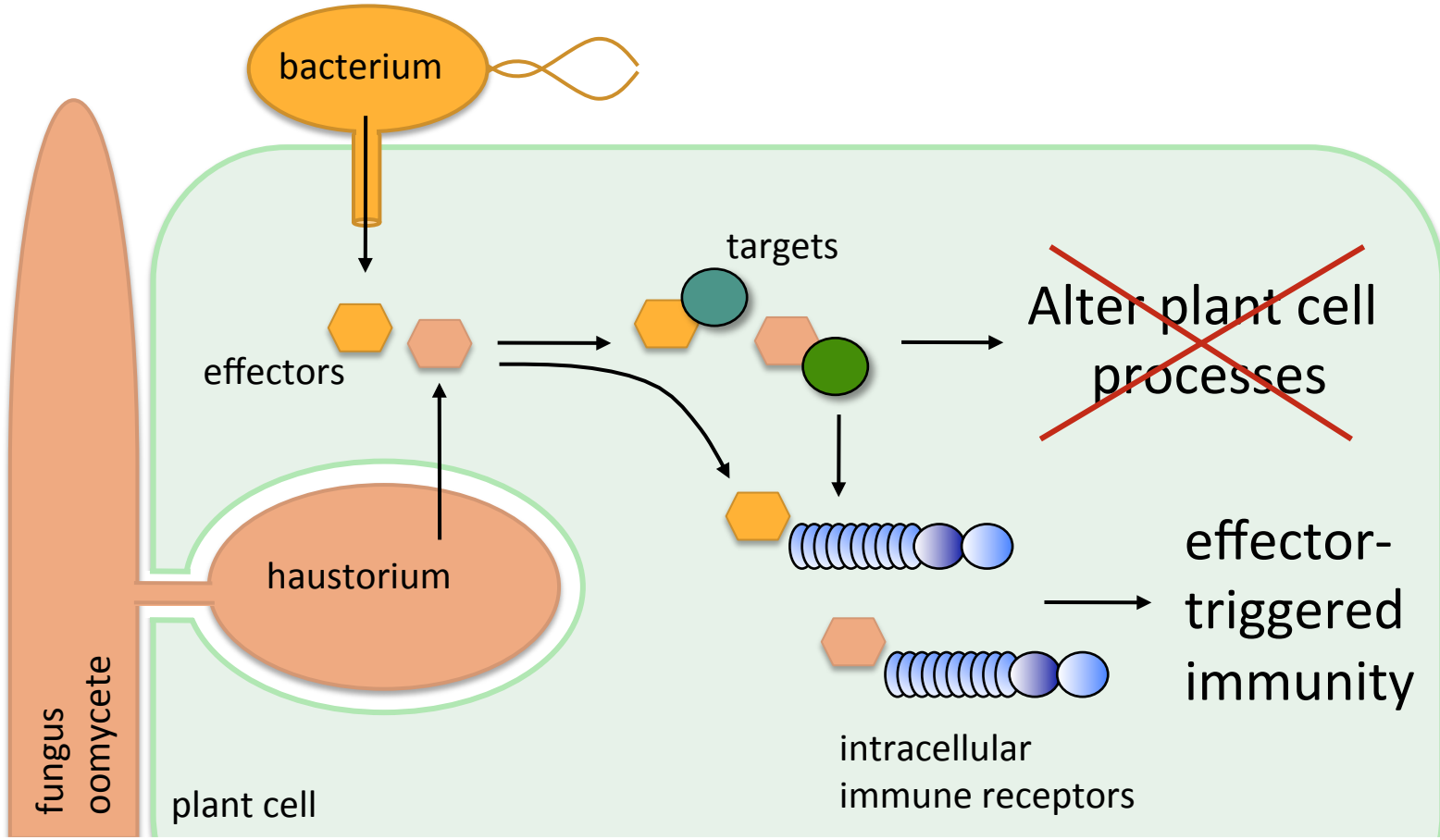
Effectors – secreted pathogen molecules that perturb plant processes

- **Effectors** – described in parasitic bacteria, oomycetes, fungi, nematodes, and insects
- Encoded by genes in pathogen genomes but function in (inside) plant cells – **operate as plant proteins**
- **Target of natural selection** in the context of coevolutionary arms race between pathogen and plant
- **Current paradigm** – effector activities are key to understanding parasitism

Microbes alter plant cell processes by secreting a diversity of effector molecules

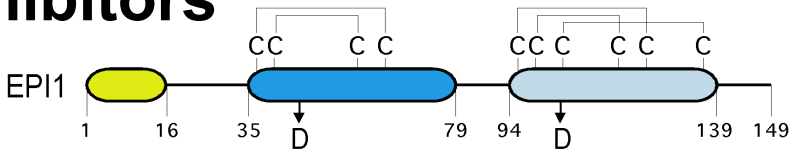


Some effectors “trip the wire” and activate immunity in particular plant genotypes



The diverse effectors of *Phytophthora infestans*

Protease inhibitors



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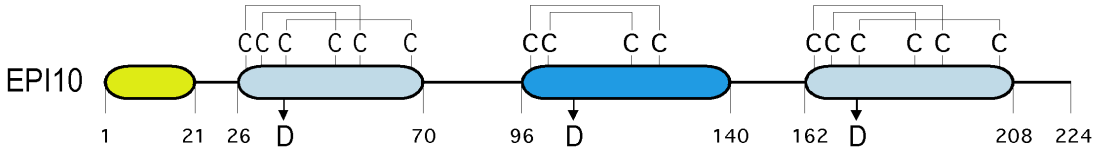
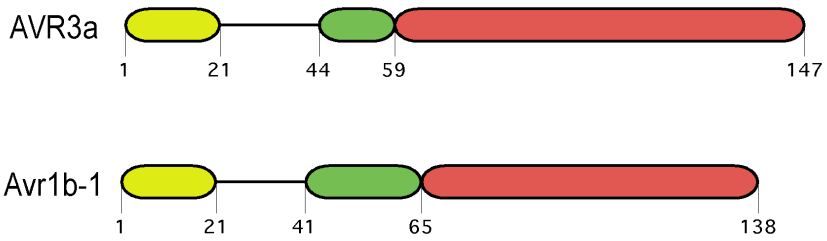
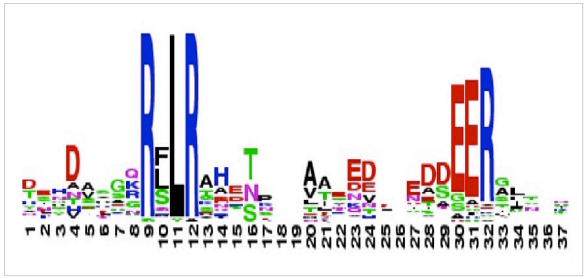


Apoplastic

RXLR

Host-translocated





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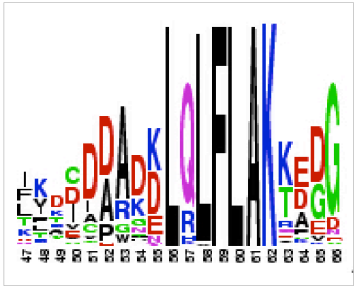
Crinklers



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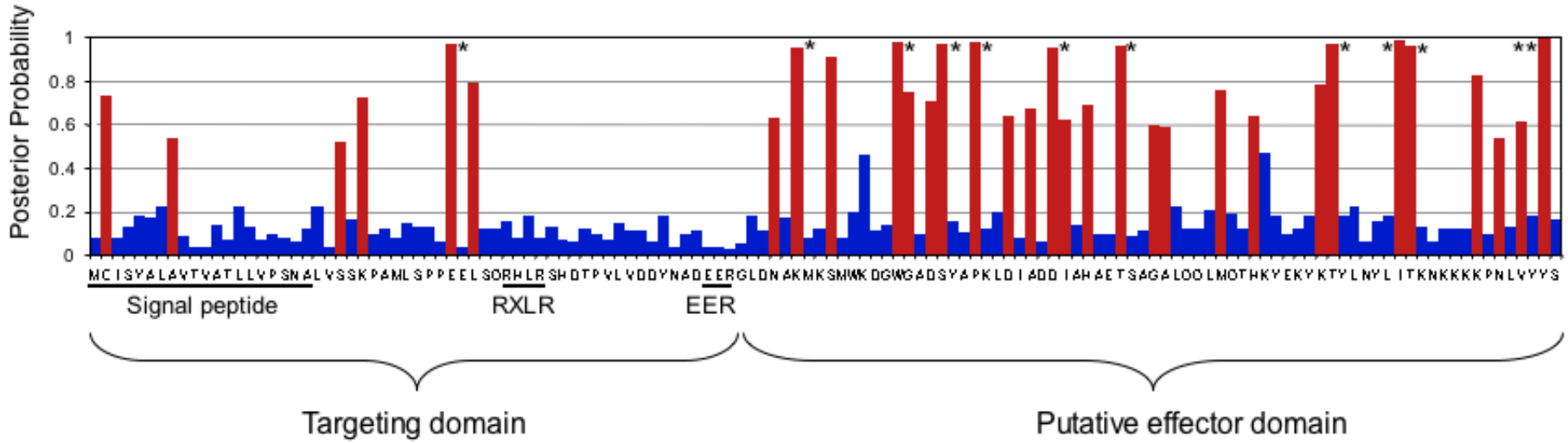
Positive selection has targeted the C terminal domain of RXLR effectors (ML method in paml)

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                                20                  40                  60                  80                 100                 120
PiPGG1_3  MRISYALTVTVATLLVPSNALVNSKPAMLSPPGEPSQRHLRSHDTPVLVDDYNADEERGLDKAAMKTMWEDGMSAAGYAKKLGITDKIALAEKSAGVLQQLMQTRRYEKYQQYLNYLAKKNKKKKPDLIYLS
PiPGG1_4  ........................T.......E.....................................................T.......S.....................................
PiPGG1_1  .C.....A.............S.K.......E.L.........................N.K..S..K..WG.DS..P..D.A.D..H..T...A.......HK....KT.....IT.......N.V.Y.
PiPGG1_2  .......A.............S.K.......E.........................N.K..S..K..W..DN..P....A.D..H..T...A....T..HK....KT.....IT.....Q.N.V.Y.
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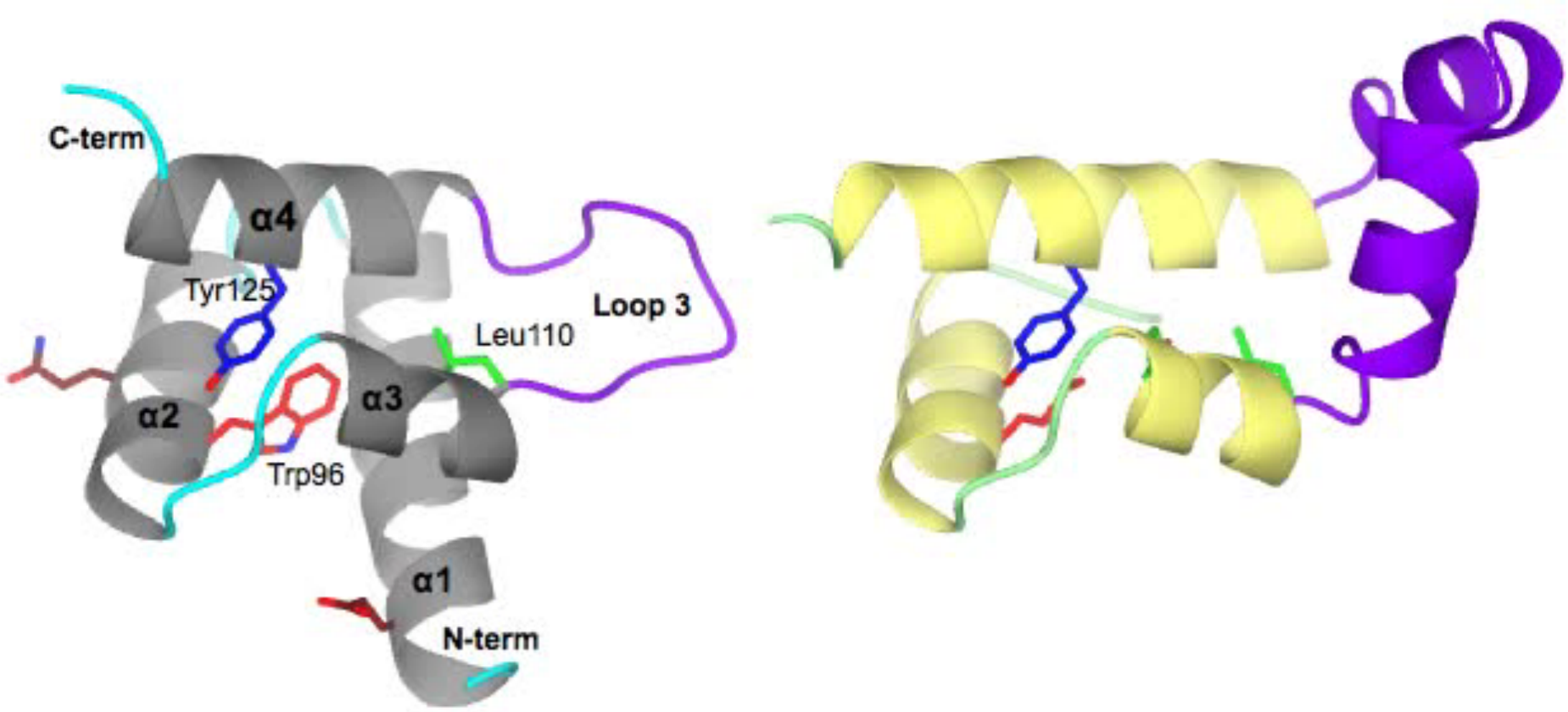
- Consistent with the view that RXLR effectors are modular

Win et al. Plant Cell, 2007

RXLR effector proteins have conserved but adaptable structures



Mark Banfield Lab @ John Innes Centre

Boutemy et al. JBC 2011

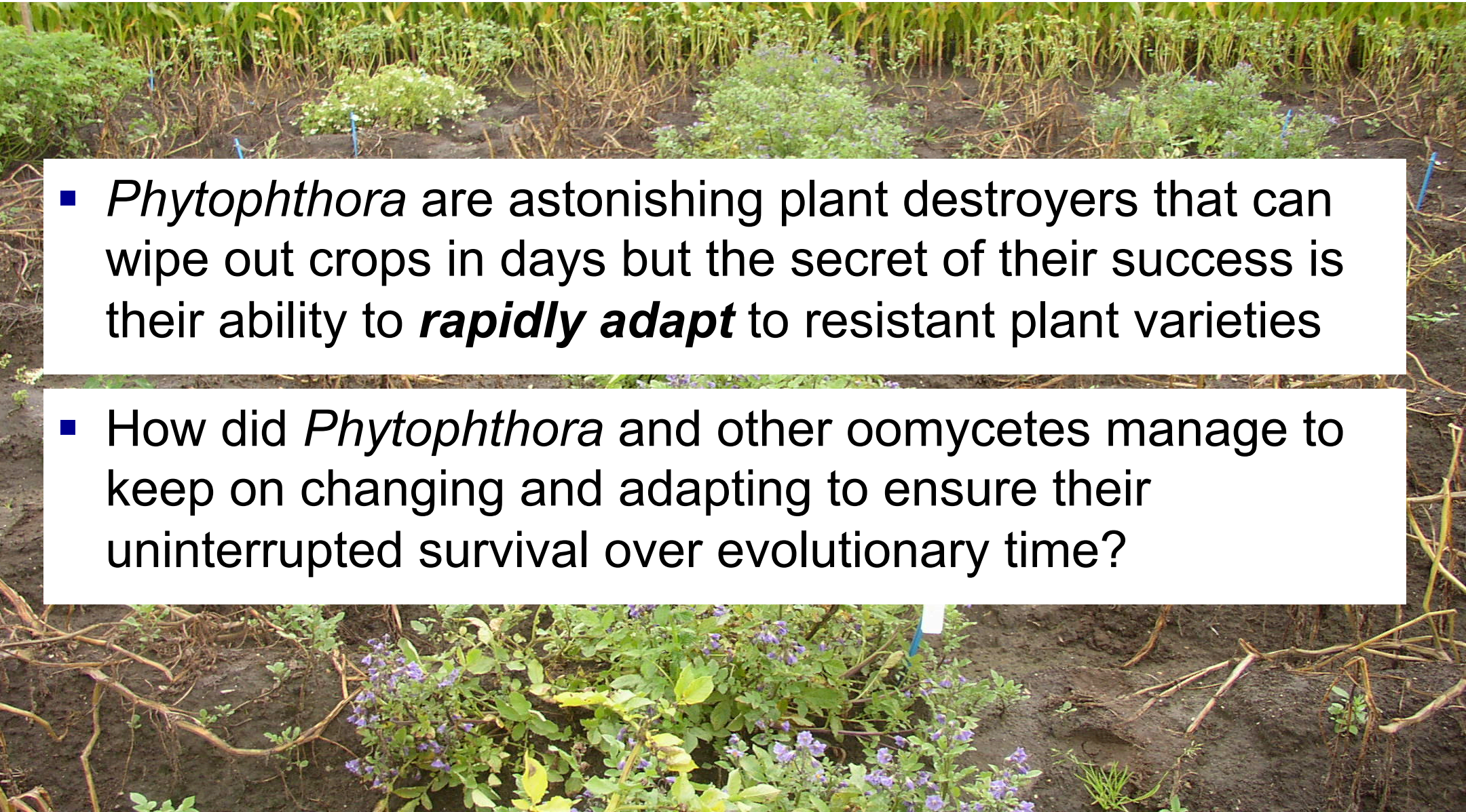
Win et al. PLoS Pathogens 2012

WY-fold of *Phytophthora* RXLR-WY effectors

- Insertion/deletions in loop regions between α-helices
- Extensions to the N- and C-termini
- Amino acid replacements in surface residues
- Tandem domain duplications
- Oligomerization

  - A structural template for rapid biochemical diversification?

Why the misery? Why are oomycetes the scourge of farmers worldwide?



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Phytophthora infestans genome architecture - repeat-rich and gene-poor loci interrupt colinear regions

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P. infestans

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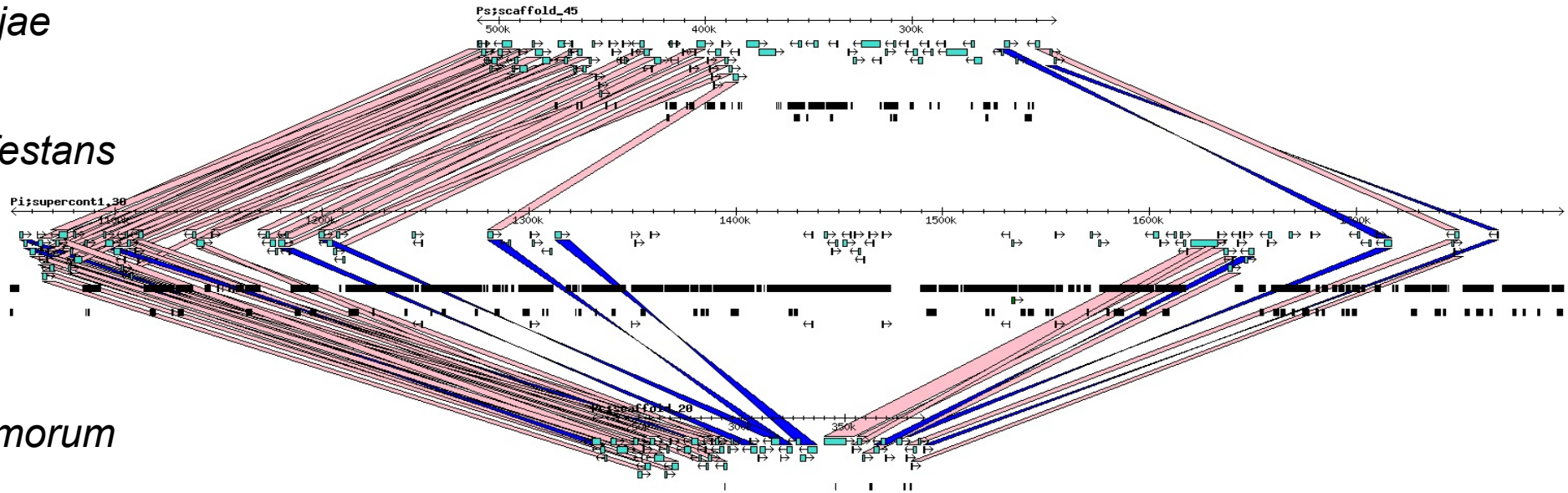

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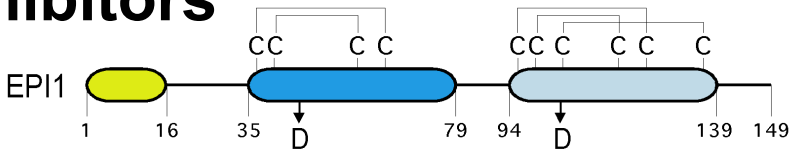
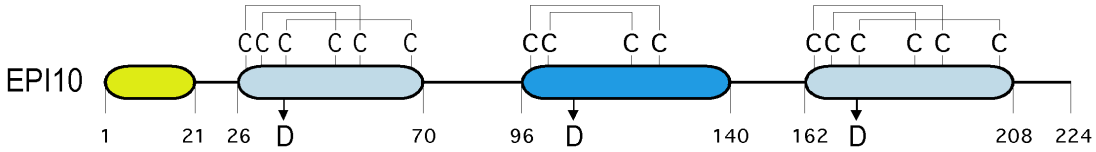
The diverse effectors of *Phytophthora infestans*

Protease inhibitors



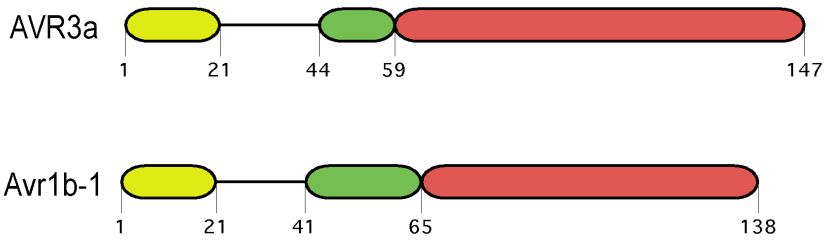
~38

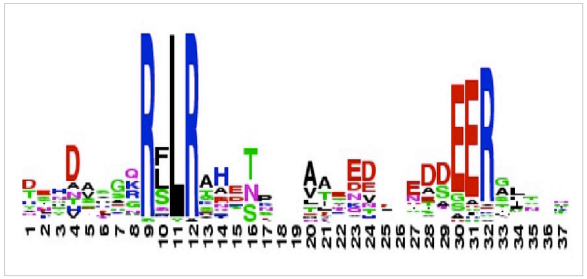


Apoplastic

RXLR

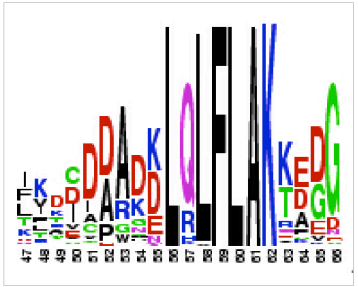
Host-translocated





~550

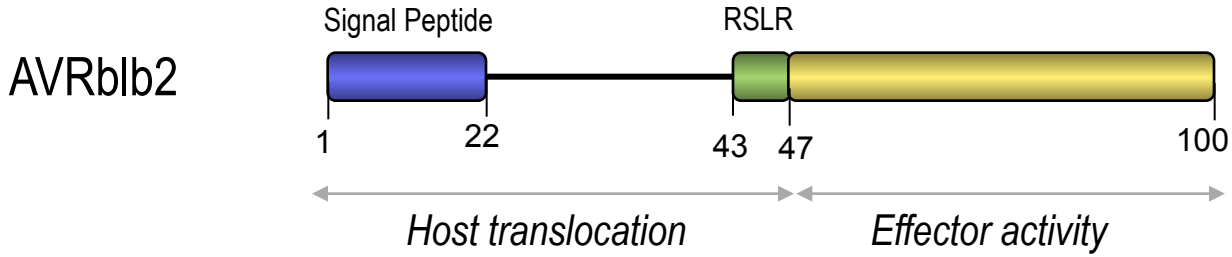
Crinklers



~200

~250ψ

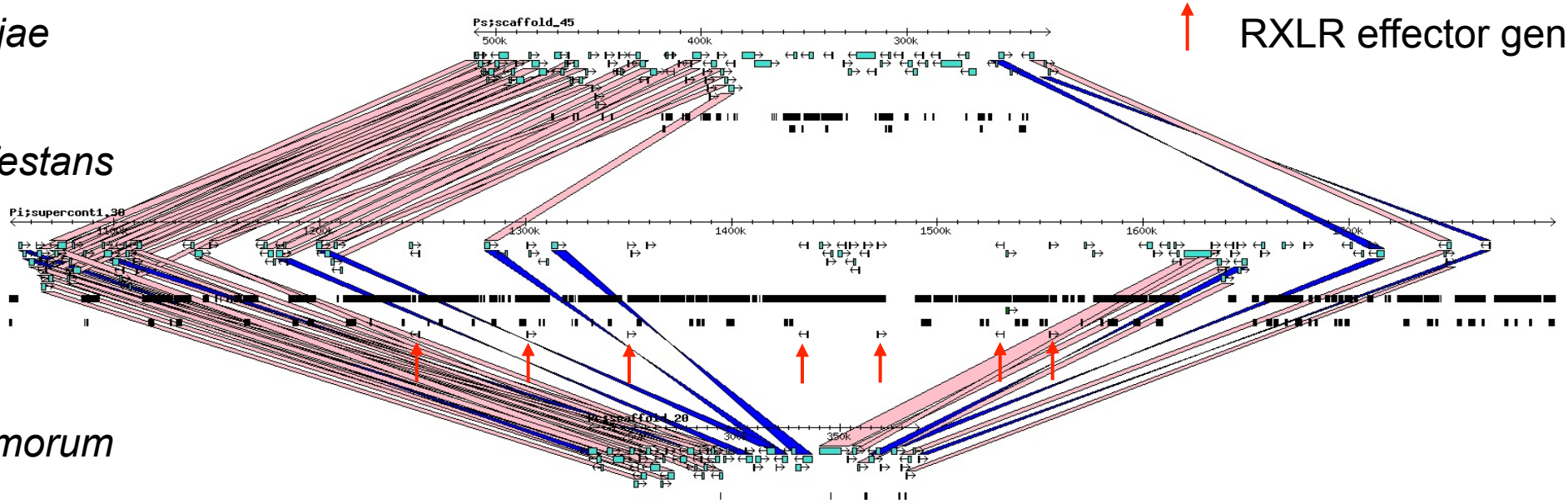
Phytophthora infestans effectors typically occur in the expanded, repeat-rich and gene-poor loci



P. sojae

P. infestans

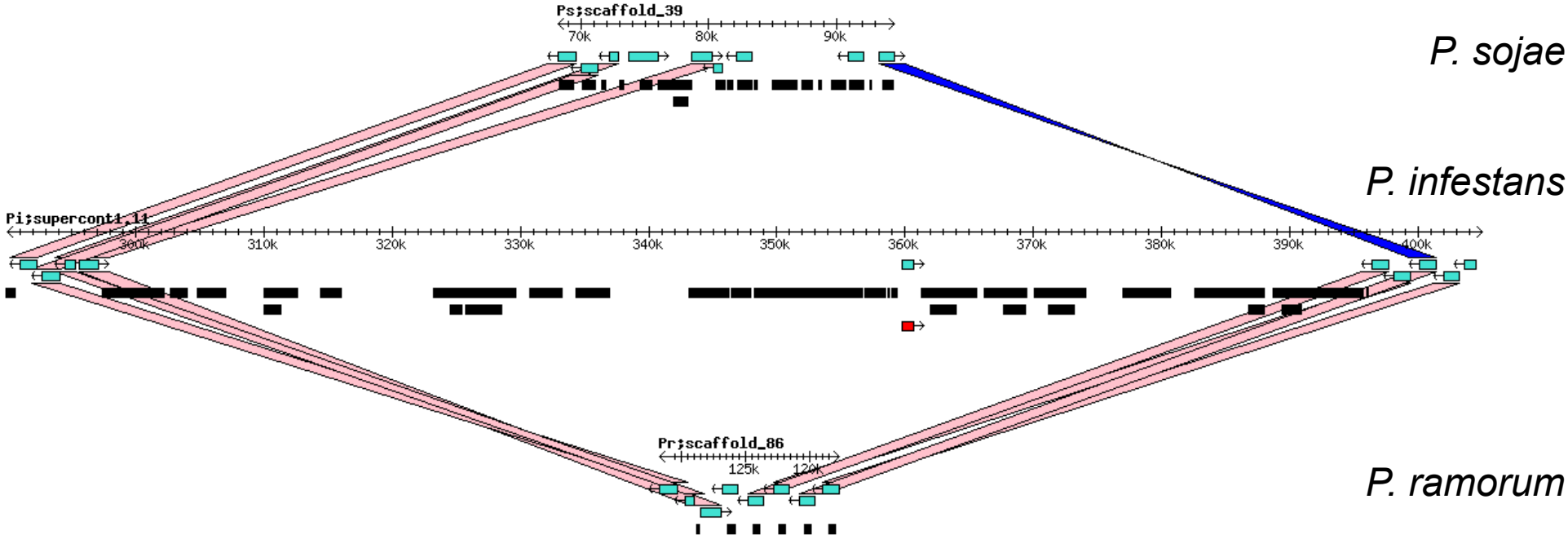
P. ramorum



B. Haas, S. Kamoun *et al.* Nature, 2009

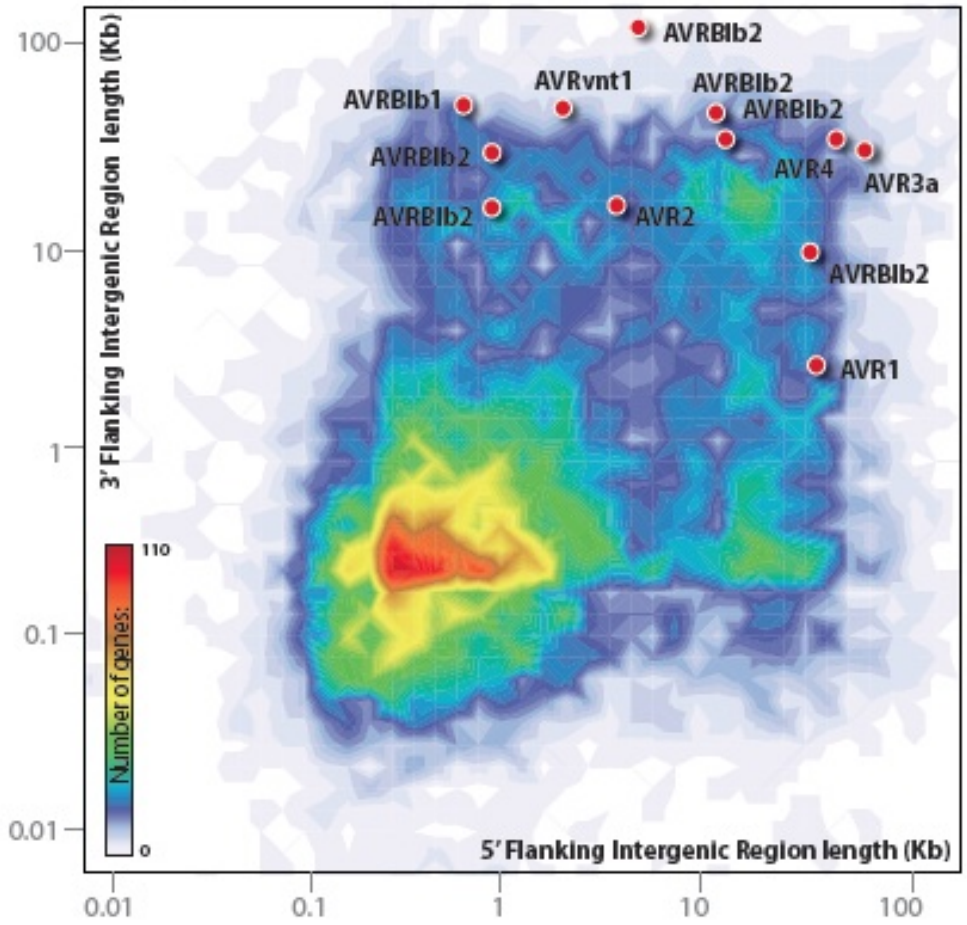
The “two-speed genome” of *P. infestans* underpins high evolutionary potential

- Gene-sparse regions of genome show highest rates of structural and sequence variation, signatures of adaptive selection
- Gene-sparse regions underpin rapid evolution of virulence (effector) genes and host adaptation

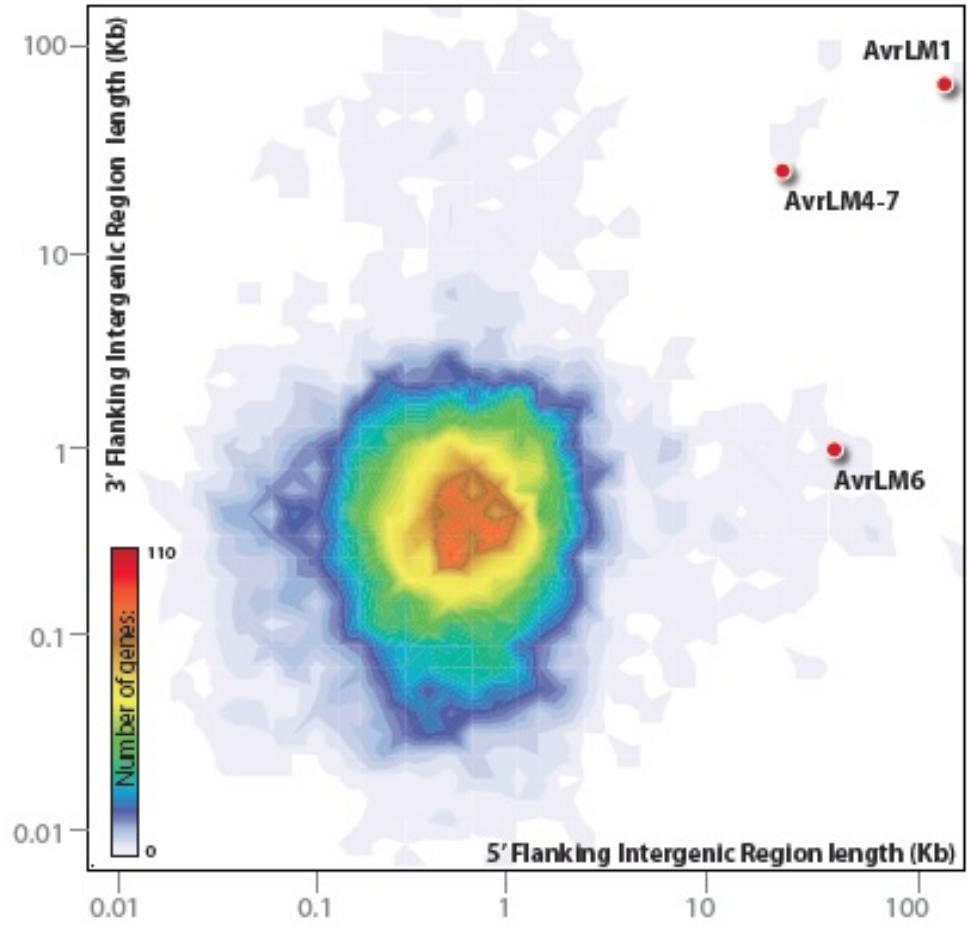


Oomycete and fungal plant pathogens independently evolved **“two-speed” genomes**

***Phytophthora infestans* >>> Oomycete**

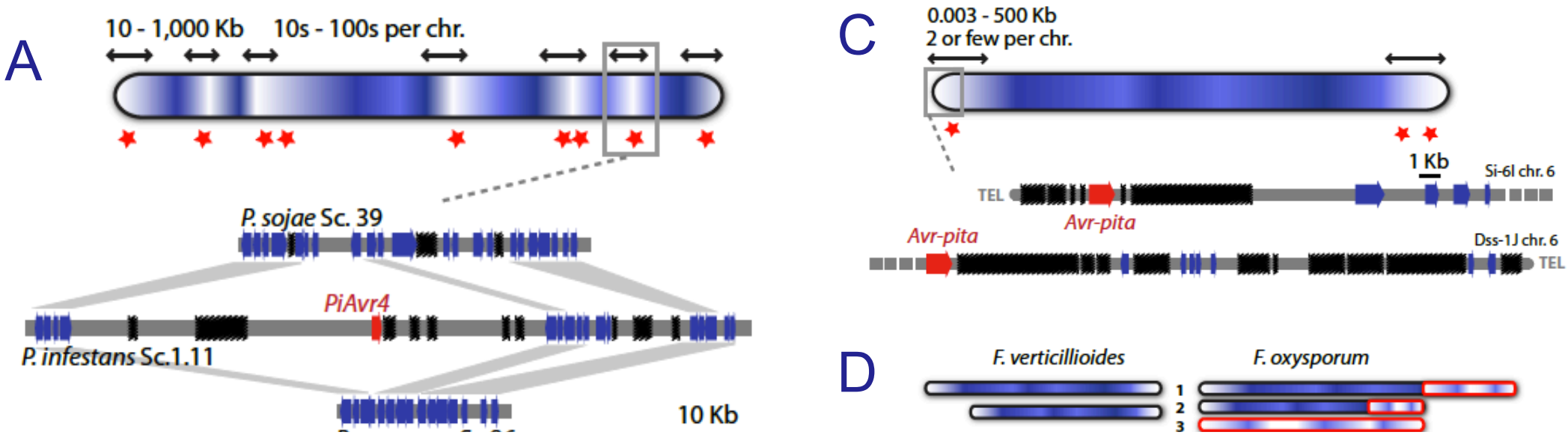


***Leptosphaeria maculans* >>> Fungus**



Sylvain Raffaele

Genome biology: the peculiar architecture of filamentous plant pathogen genomes



- Effector genes populate specific (repeat-rich) compartments of filamentous pathogen genomes

- Repeat-rich genome compartments contribute to the emergence of new virulence traits > “two-speed genome”

The “two-speed” genome per JBS Haldane *ca*. 1949

Haldane, J.B.S. (1949). Disease and evolution. La Ricerca Scientifica, 19, 2–11.

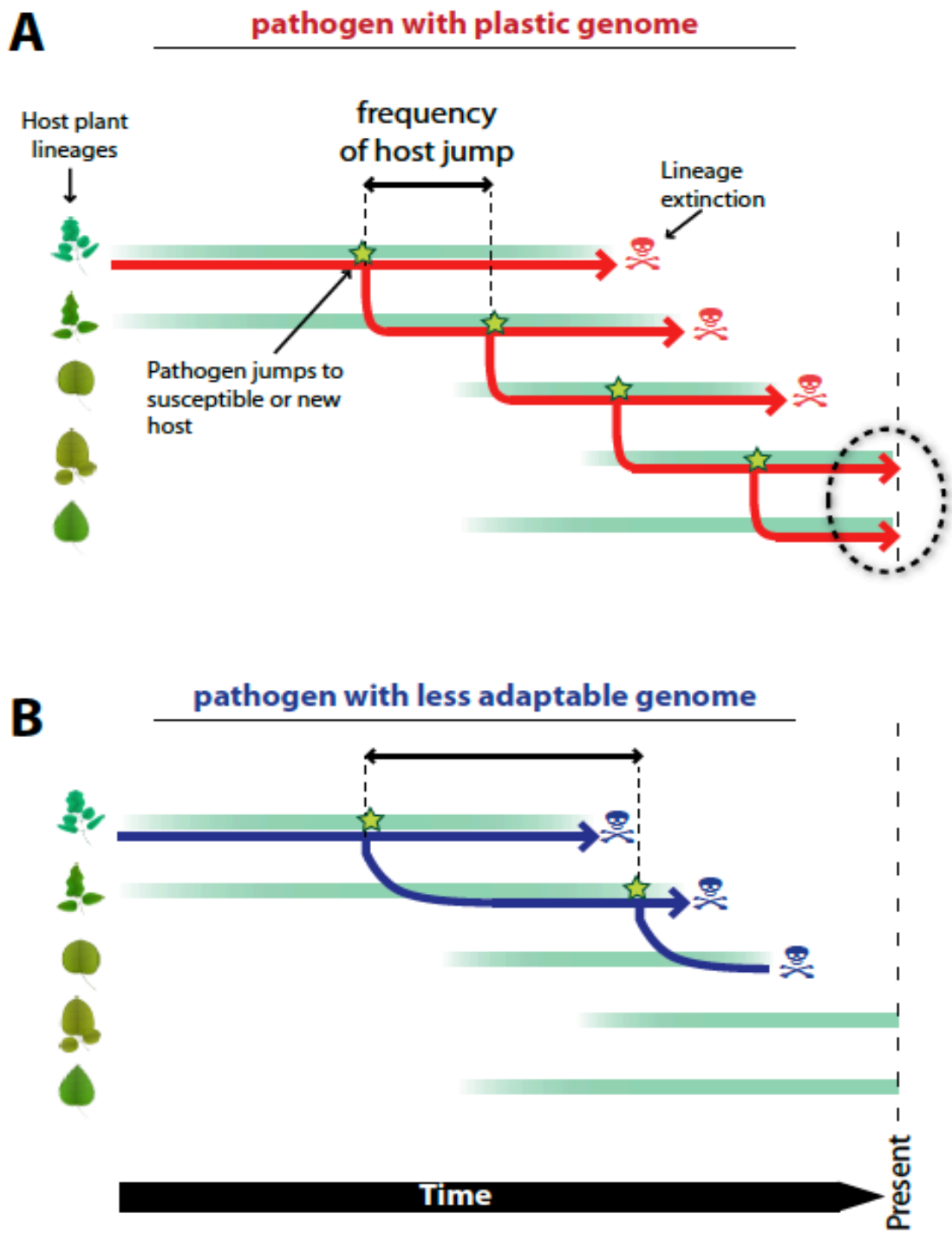
- “…it would be advantageous for a species if the genes for biochemical diversity [in disease resistance] were particularly mutable, provided that this could be achieved without increasing the mutability of other genes whose mutation would give lethal or sublethal genotypes.”

How does the two-speed genome accelerate evolution? …drive new virulence traits?

- Structural genome variation – *increased genome instability and structural variation, deletions, duplications etc.*
- Horizontal gene/chromosome transfer – *mobile effectors*
- Increased local mutagenesis – *RIP mutation leakage*
- Epigenetics – *heterochromatin leakage?*
- …more to be discovered

Why the "two-speed" genome? Jump or die!

- Lineages with less adaptable genomes suffer higher extinction rates, thus a macroevolutionary disadvantage
- Lineages that have adaptable genomes end up dominating the biota
- **Clade selection** – opposes short term advantages conferred by smaller compact genomes

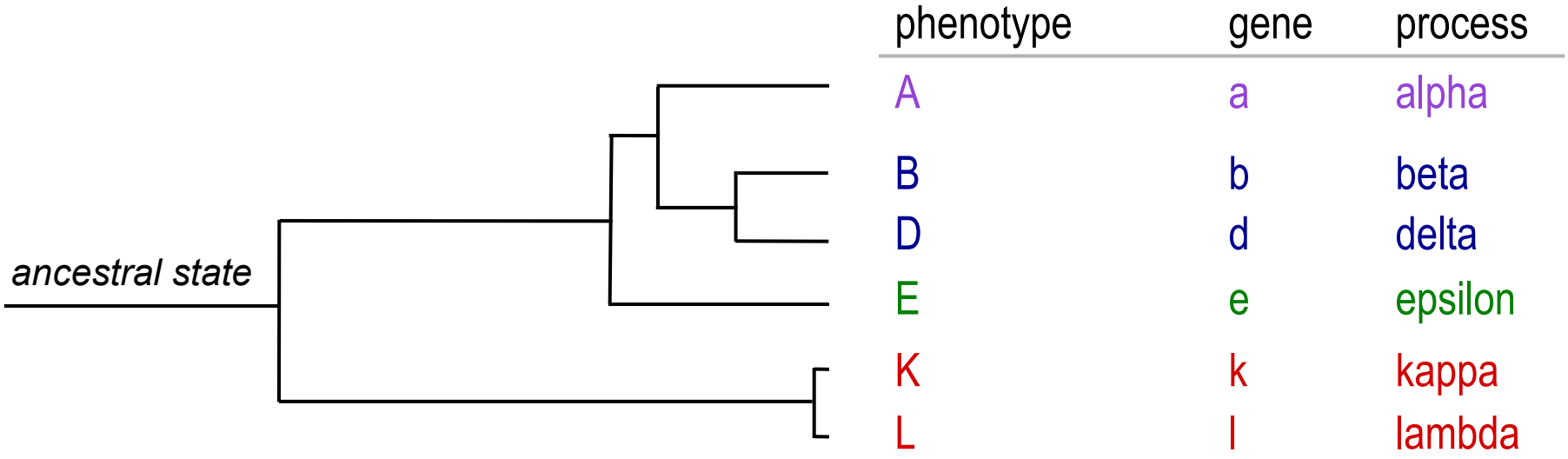


Raffaele and Kamoun, Nature Reviews Microbiol 2012

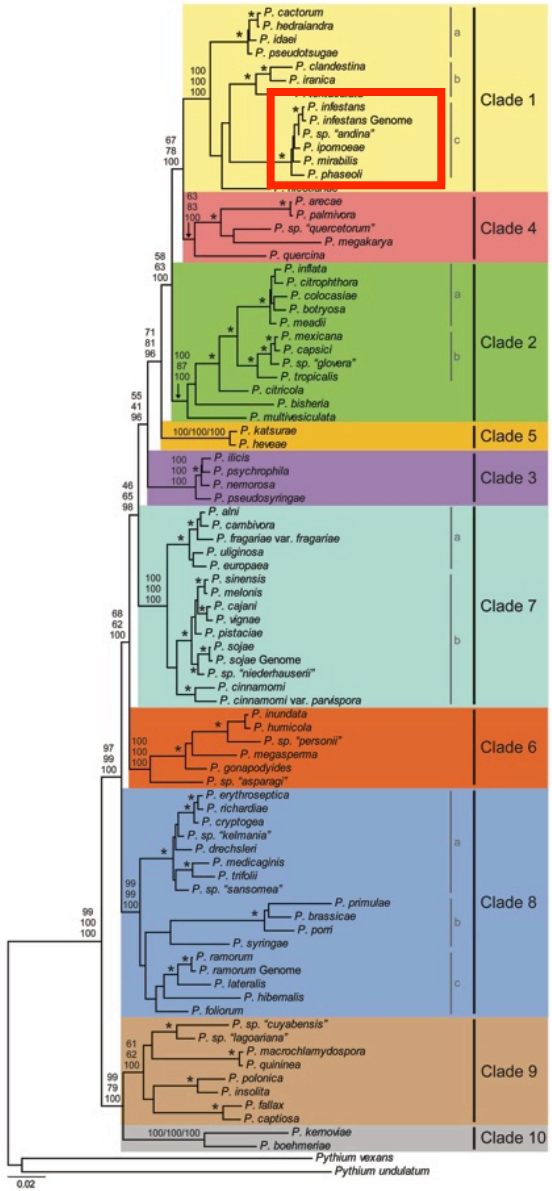
GC Wlliams (1992) Oxford Uni Press

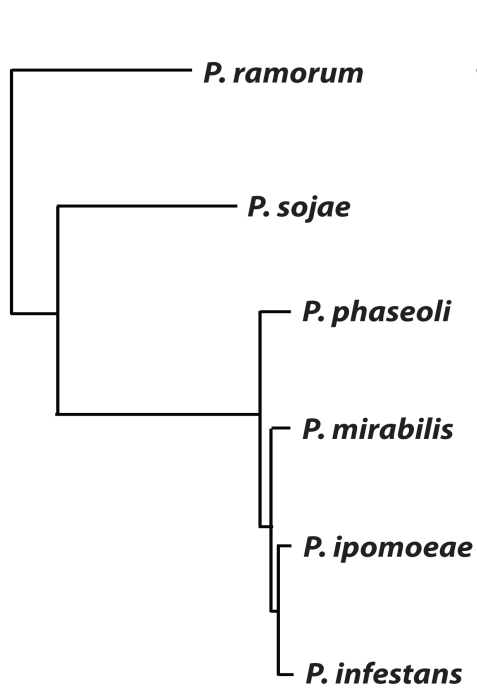
#EvoMPMI – Evolutionary Plant-Microbe Interactions

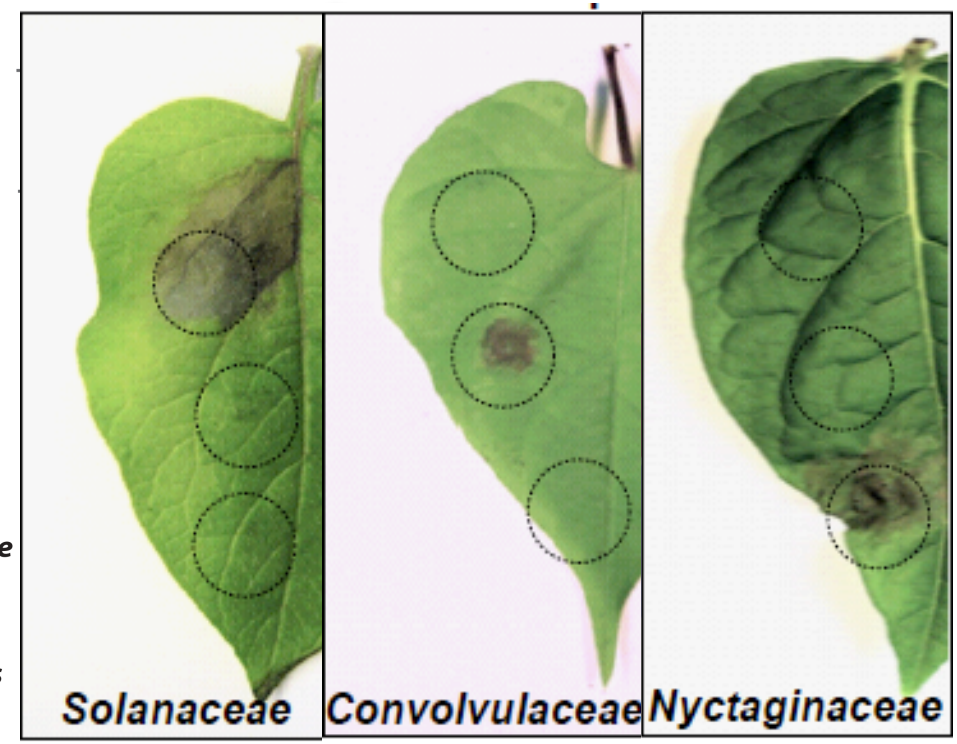
- Comparative studies within a *phylogenetically and ecologically robust framework* to test *specific hypotheses* about how evolution has tweaked mechanisms of pathogenicity and immunity
- Alternative to genetic approaches – genetic screens have taken place in nature throughout evolution



Species in the *Phytophthora infestans* lineage (clade 1c) evolved by host jumps



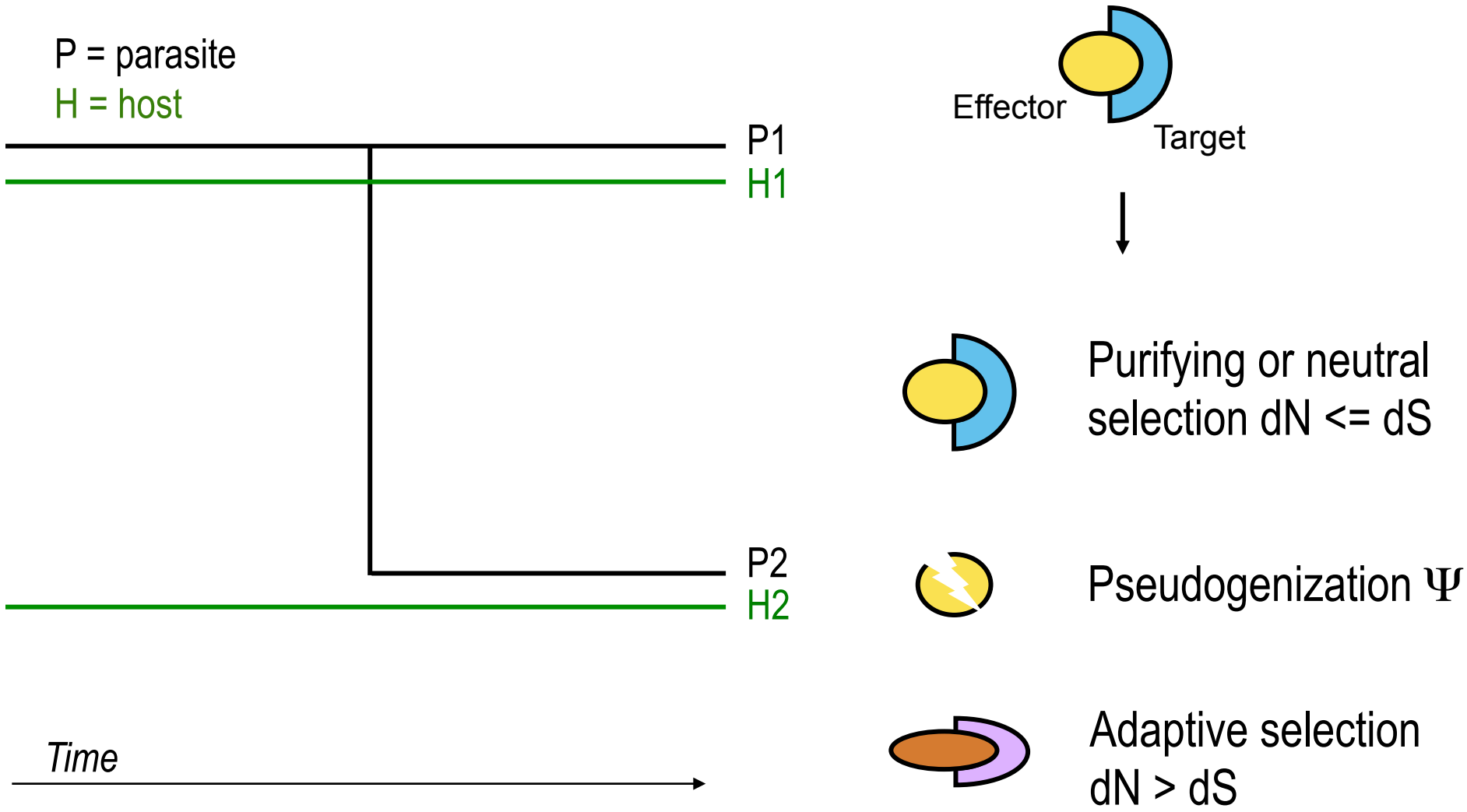




- Diverged ~1300 years ago; 99.9% identical in ITS
- Three species naturally co-occur in Toluca, Mexico
- Specialized on their respective hosts

multilocus phylogeny of *Phytophthora* from Blair et al. 2008 Fungal Genet Biol

Host jumps must have a dramatic impact on effector evolution

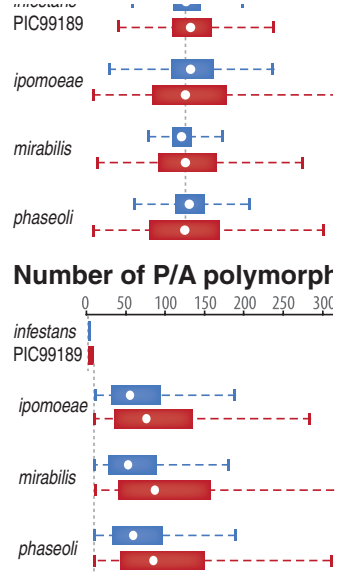


Impact of host jumps on genome and effector evolution

and positive selection. These loci are enriched in genes induced in planta, implicating host adaptati in genome evolution. Unexpectedly, genes involved in epigenetic processes formed another class of evolving residents of the gene-sparse regions. These results demonstrate that dynamic repeat-rich g compartments underpin accelerated gene evolution following host jumps in this pathogen lineage.

Phytophthora infestans is an economically important specialized pathogen that causes the destructive late blight disease on *Solanum* plants, including potato and tomato. In central Mexico, *P. infestans* naturally co-occurs with two extremely closely related species, *Phytophthora ipomoeae* and *Phytophthora mirabilis*, that specifically infect plants as diverse as morning glory (*Ipomoea longipedunculata*) and four-o'clock (*Mirabilis jalapa*), respectively. Elsewhere in North America, a fourth related species, *Phytophthora phaseoli*, is a pathogen of lima beans (*Phaseolus lunatus*). Altogether these four *Phytophthora* species form a very tight clade of pathogen s that share ~99.9% identity in their ribosoma internal transcribed spacer regions (*1*). Ph netic inferences clearly indicate that species *Phytophthora* clade 1c [nomenclature of (*2*)] e through host jumps followed by adaptive s ization on plants belonging to four differe tanical families (*2*, *3*). Adaptation to thes plants most likely involves mutations in th dreds of disease effector genes that populate poor and repeat-rich regions of the 240–me pair genome of *P. infestans* (*4*). However, c ative genome analyses of specialized sister s

dN/dS in genes from gene-de regions (GDRs) and gene-sparse regions (GSRs). Sta ical significance was assessed by unpaired *t* test assuming unequal variance (CNV, dN/dS); assuming equal variance (SNP frequency); or by Fisher's exact test (P/A) (•$P < 0.1$; ***$P < 10^{-4}$). Whiskers show first value outside 1.5 times the interquartile range. (**B**) Distribution of polymorphism in *P. mirabilis* and *P. phaseoli* according to local gene density (measured as length of 5′ and 3′ flanking intergenic regions, FIRs). The number of genes (P/A polymorphisms) or average values (CNV, SNP, dN/dS) associated with genes in each bin are shown as a color-coded heat map.

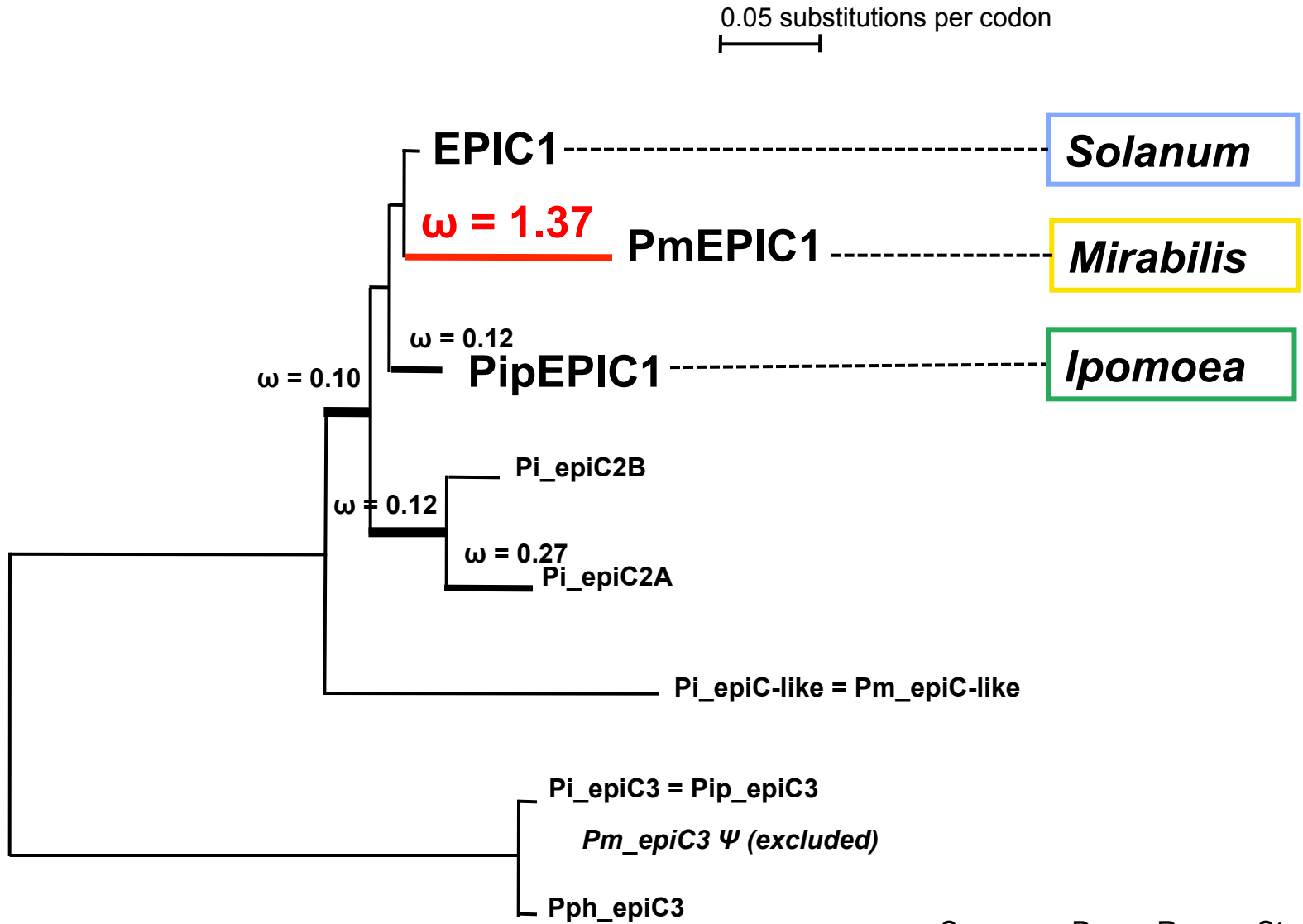


- We detected 345 *in planta* induced genes under positive selection in *P. mirabilis* relative to *P. infestans* (Raffaele *et al*. 2010)
- What is the biochemical basis of adaptive selection? → *P. mirabilis* protease inhibitor PmEPIC1 on *Mirabilis jalapa*

Evolution of EPIC1 protease inhibitor effector family

Suomeng Dong, Remco Stam,
Liliana Cano et al. Science 2014

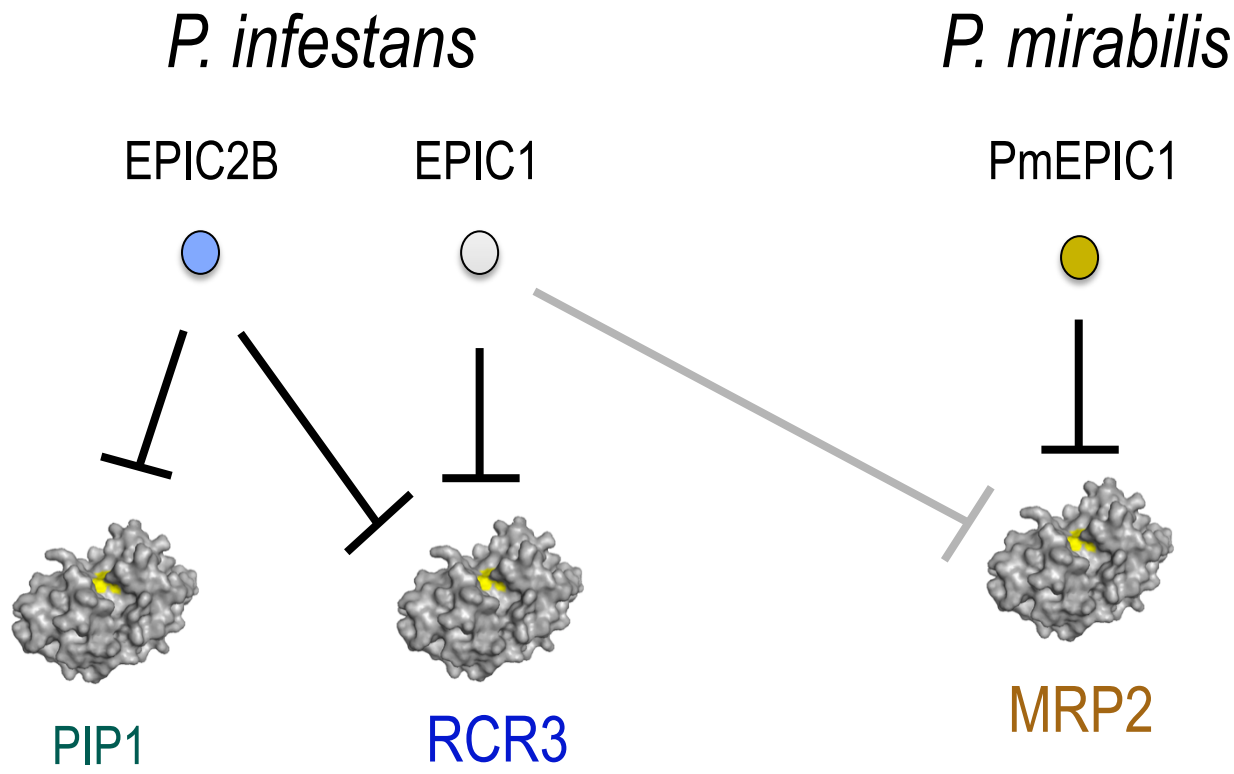
Protease inhibitor PmEPIC1 is under positive selection



ω = nonsynonymous (dN)/ synonymous (dS)
ω ratio calculated per branch using two-ratio model of codeml

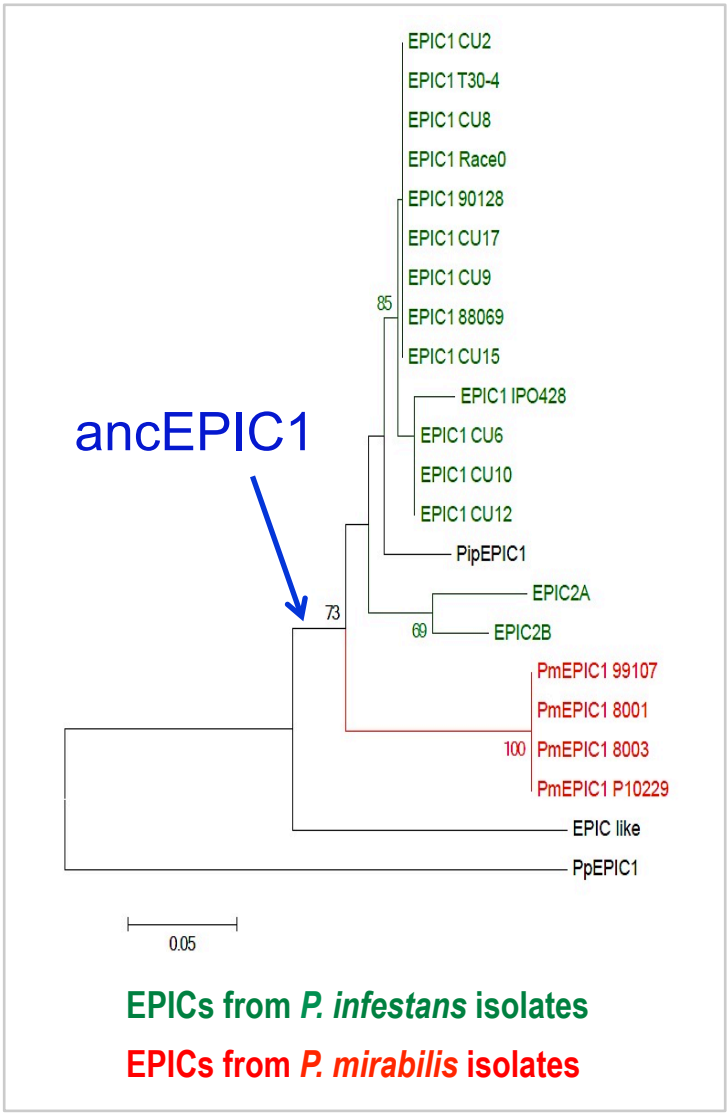
Suomeng Dong, Remco Stam,
Liliana Cano et al. Science 2014

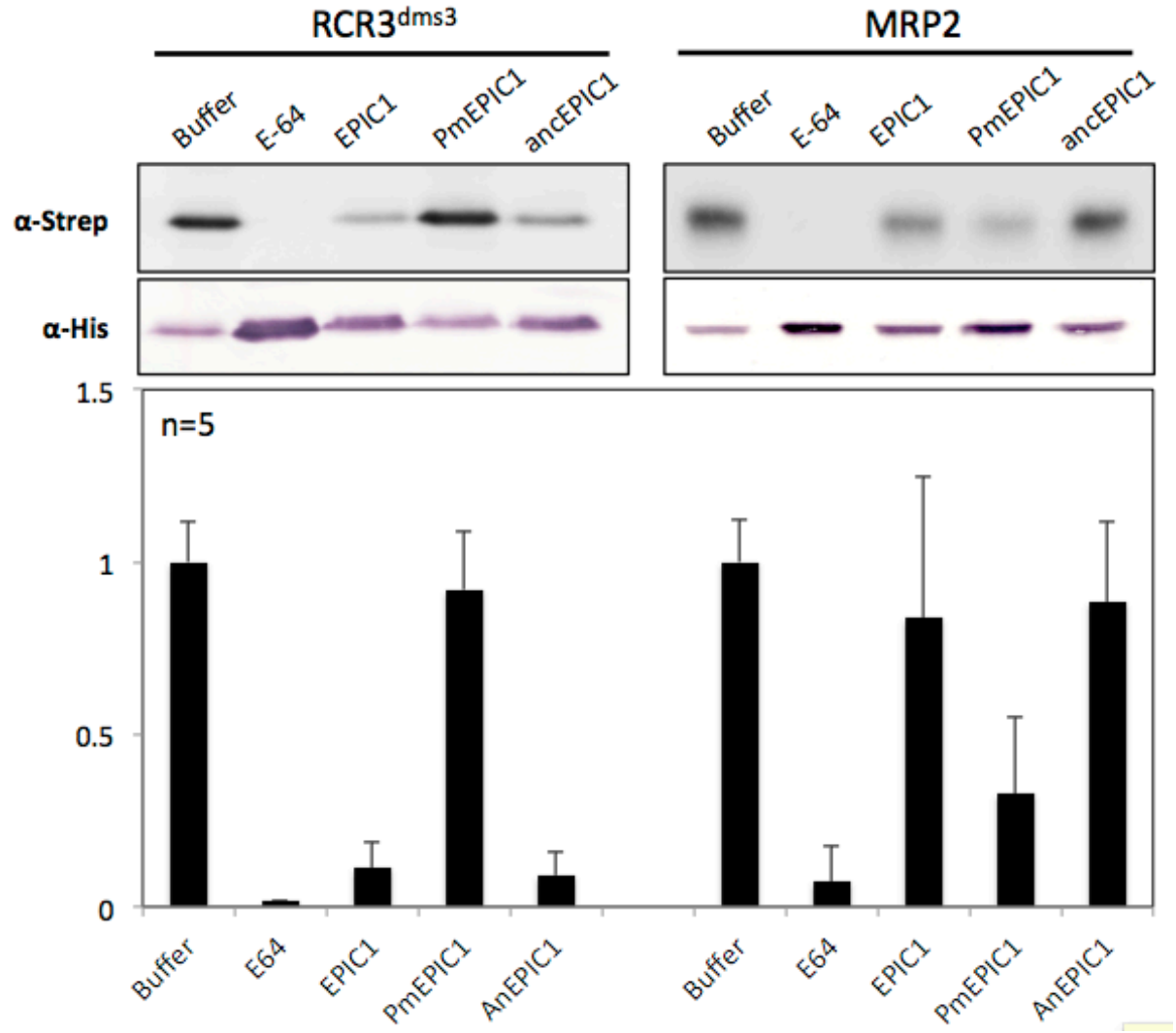
Effector adaptation and specialization to host target following jump?



Suomeng Dong, Remco Stam,
Liliana Cano et al. Science 2014

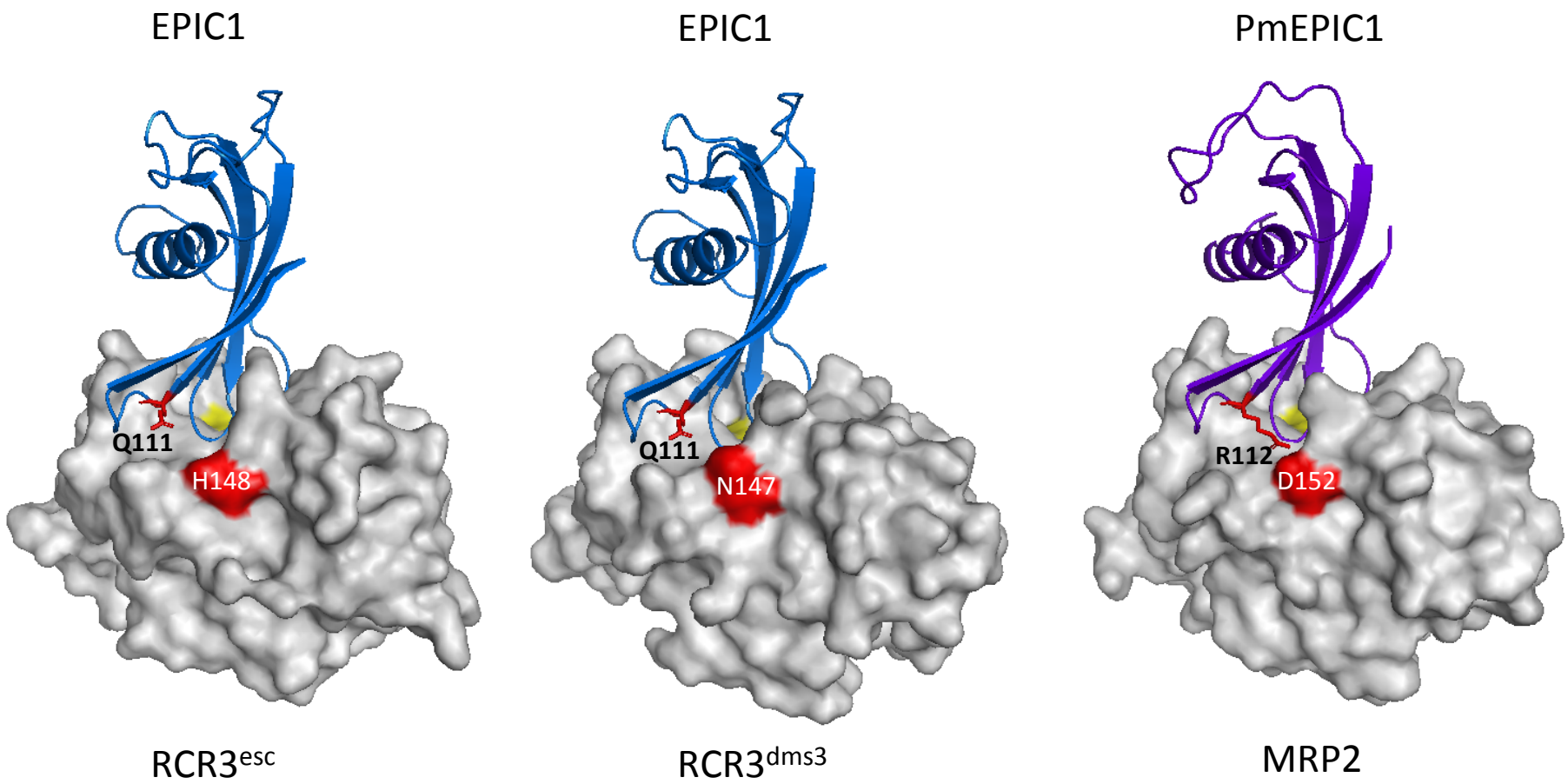
A resurrected ancestral EPIC1 can inhibit potato RCR3 but not Mirabilis MRP2





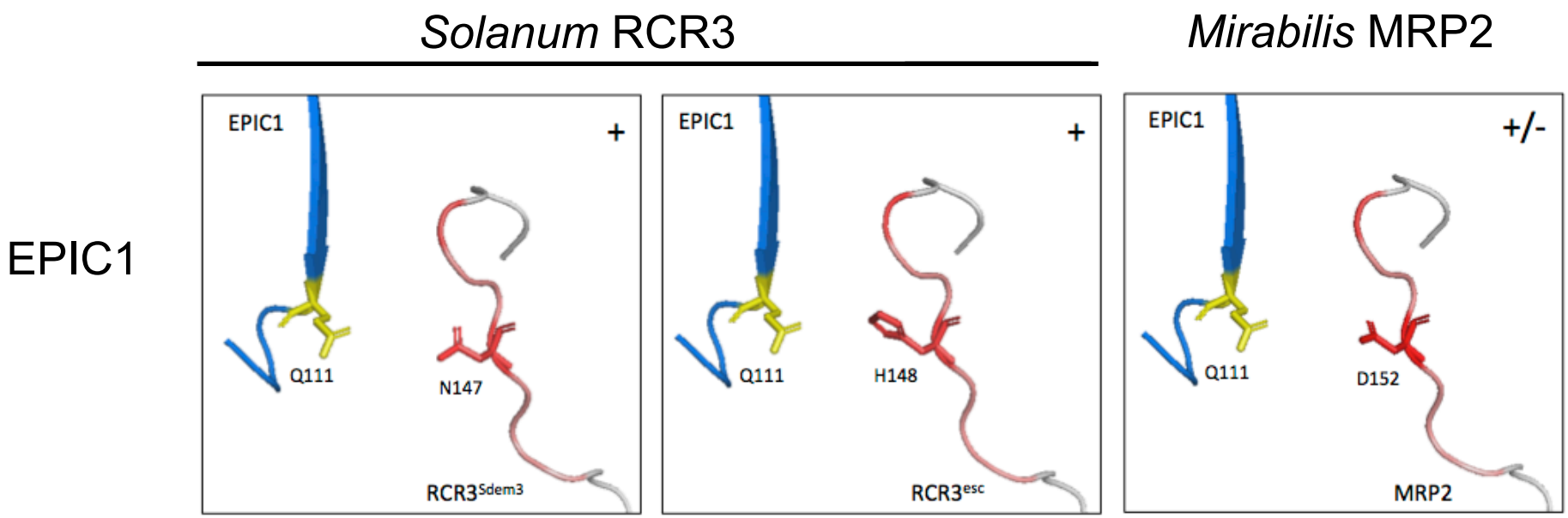
Suomeng Dong, Kentaro Yoshida

EPIC1/RCR3 predicted contact residues are polymorphic



Suomeng Dong with Mark Banfield and Renier van der Hoorn

Single residues in EPIC1 and RCR3 affect inhibition



- Gln to Arg substitution in PmEPIC1 increased MRP2 inhibition but carries a **trade off**, impairs RCR3 inhibition

- **Antagonistic pleiotropy** - an effector that evolved higher activity on new target performs poorly on the ancestral host; leads to specialization!

Mo Farah challenges Usain Bolt to a charity race over an intermediate distance of 600 or 800 metres

Double Olympic champion Mo Farah has challenged Usain Bolt, the world's fastest man, to a charity race over an intermediate distance.