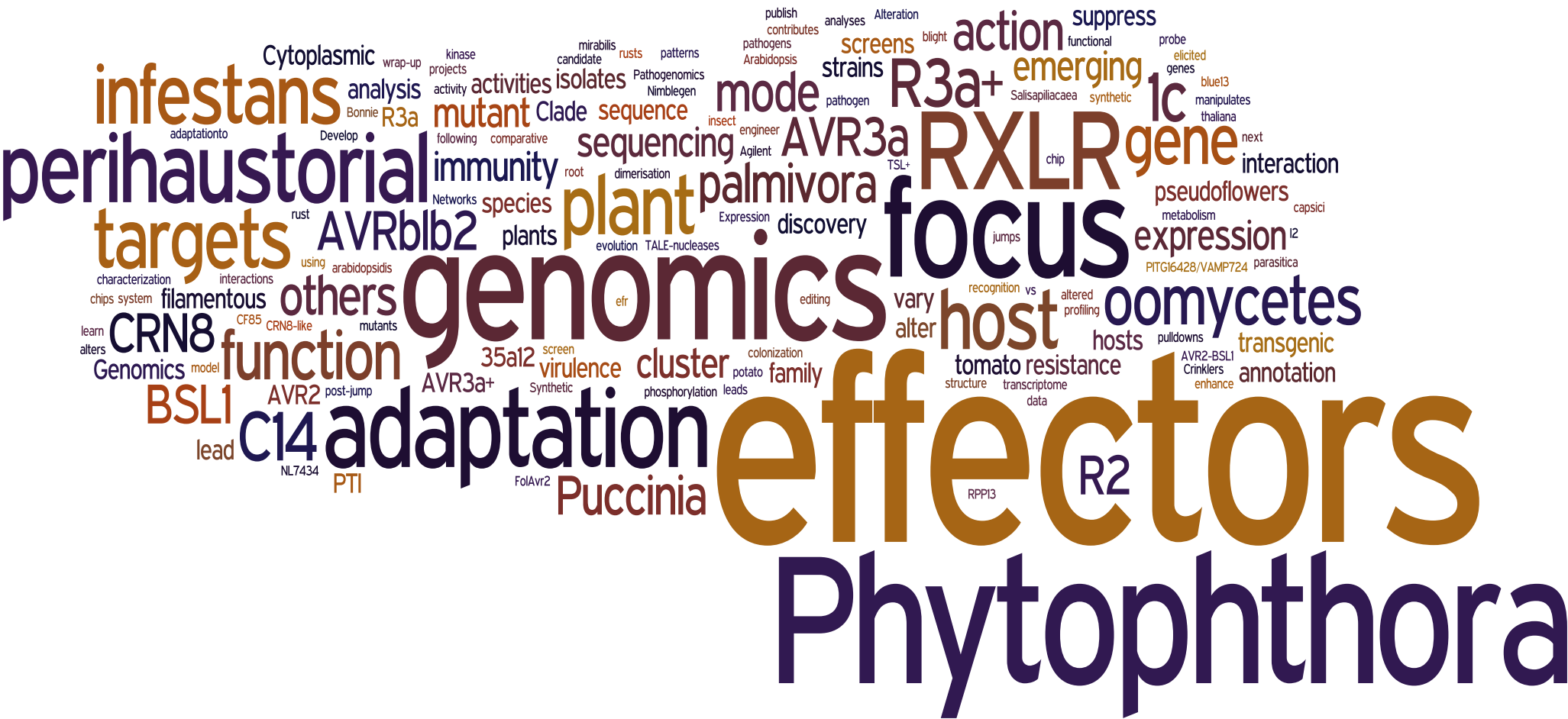




Keeping up with the Plant Destroyers

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

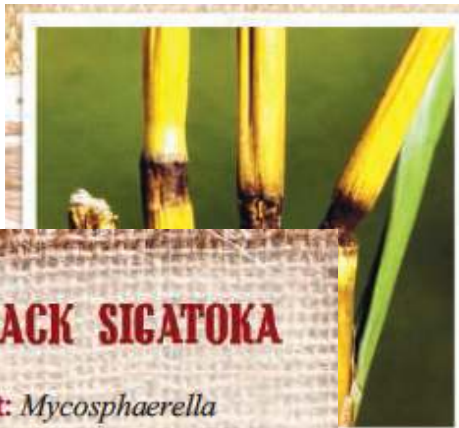


BLACK SIGATOKA

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.



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



ASIAN SOYBEAN RUST

Pest: *Phakopsora pachyrhizi*

Crops: At least 31 legume species, notably soybeans



WHEAT STEM RUST

Pest: *Puccinia graminis* Ug99

Crop: Wheat

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

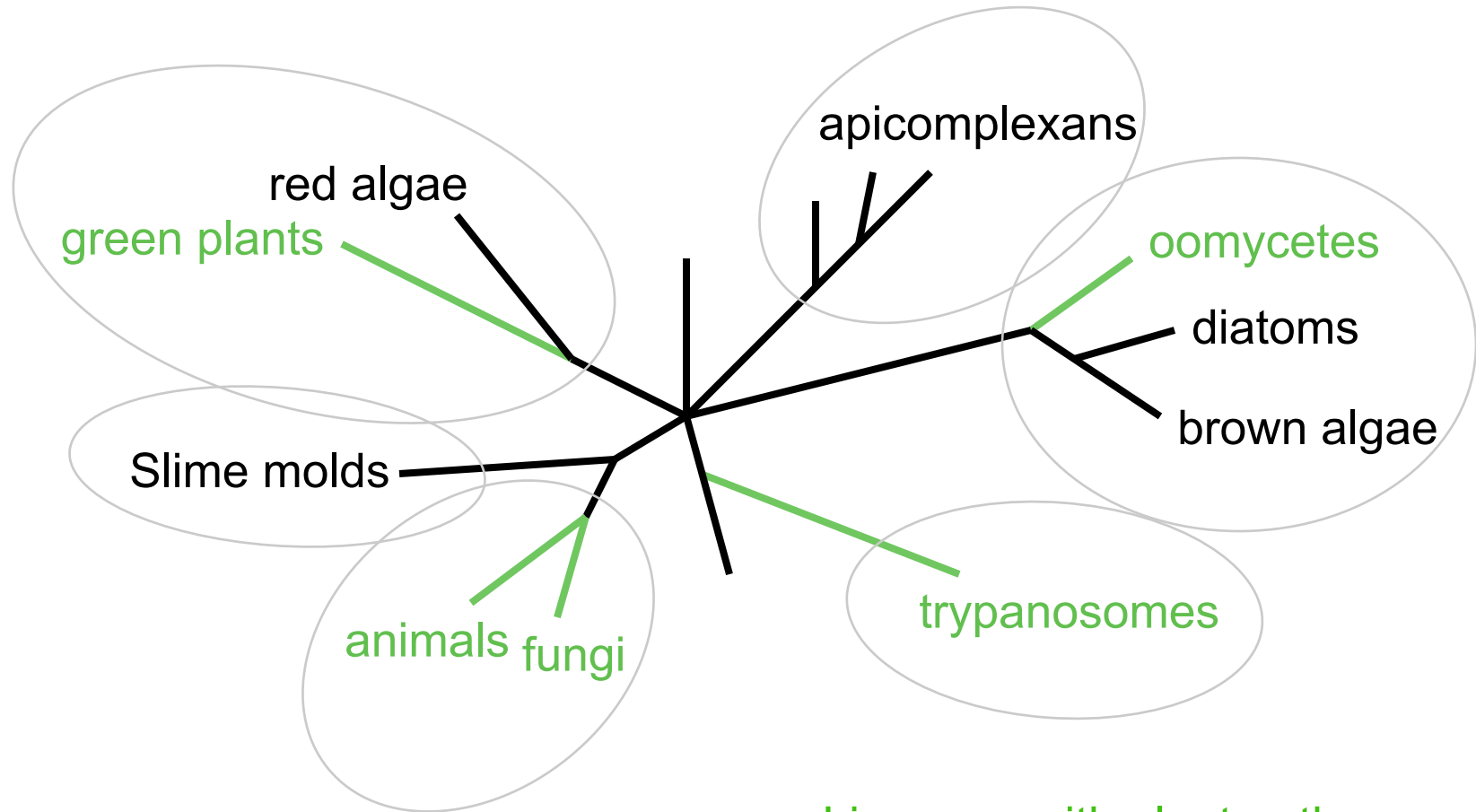
Phytophthora is Greek for “plant-destroyer”

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



Sophien Kamoun
@KamounLab

I am holding a 300 million year old oomycete. 300 million years!!! How cool is that?



RETWEETS
21

FAVORITES
15



8:08 PM - 29 Jan 2014

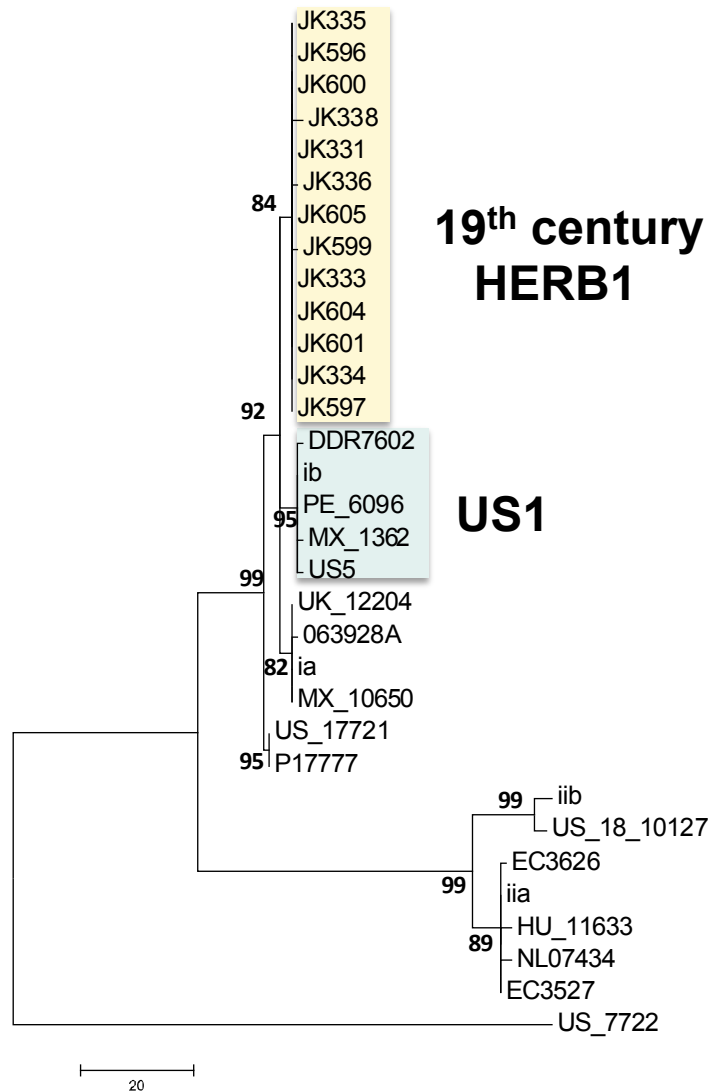
The Irish potato famine pathogen *Phytophthora infestans* causes potato blight



“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



Yoshida *et al.*, 2013, eLife

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

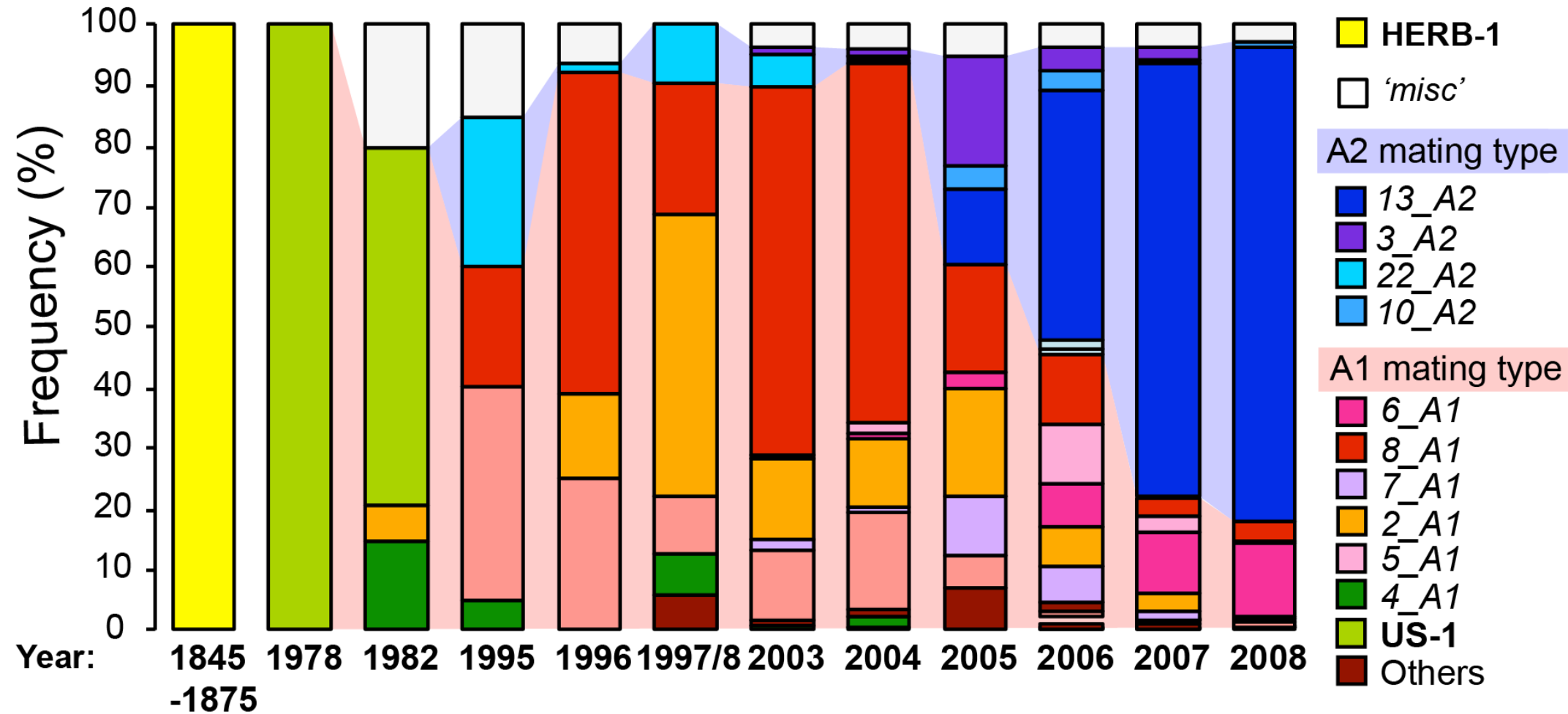




Phytophthora Infestans Herb-1



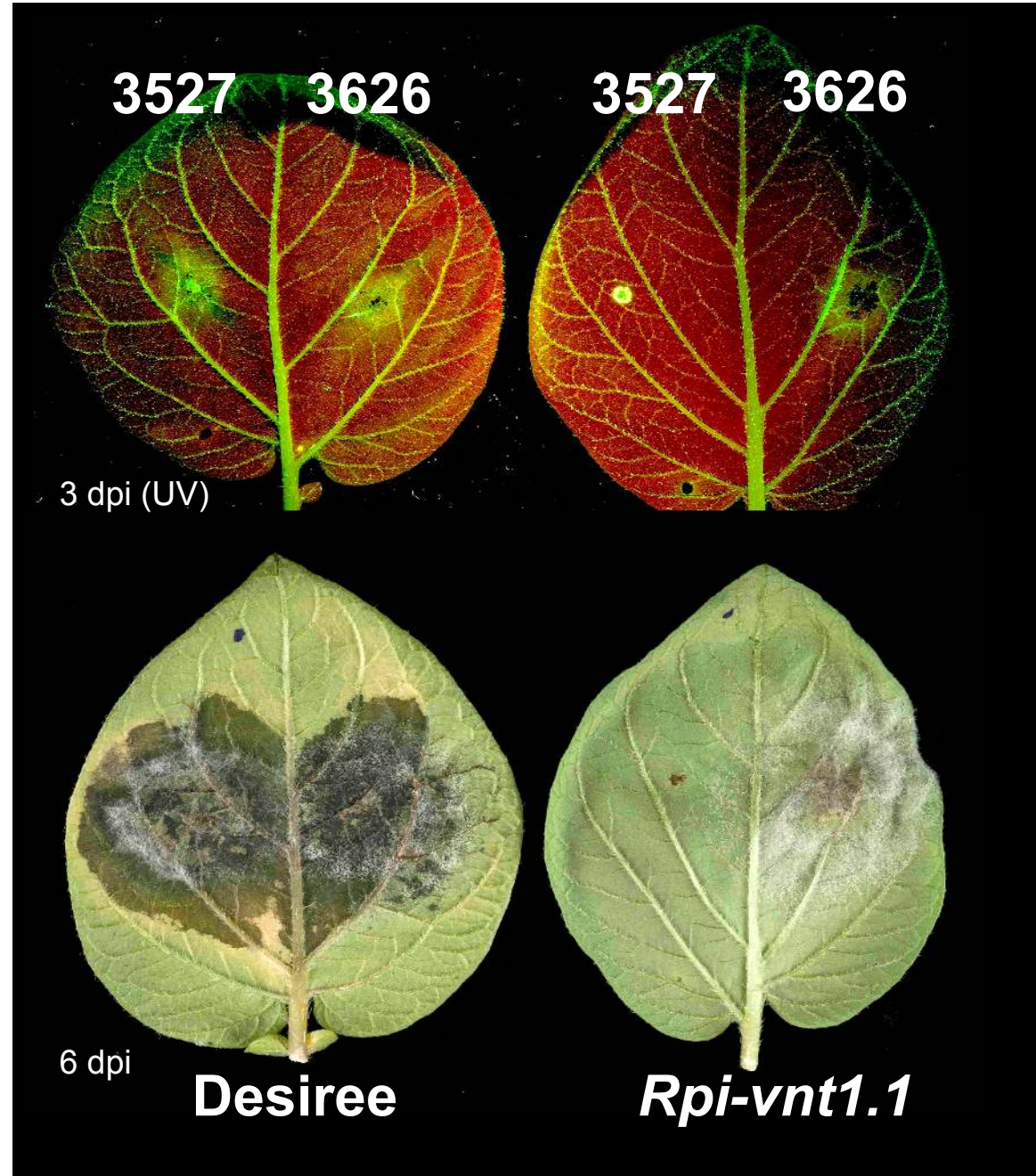
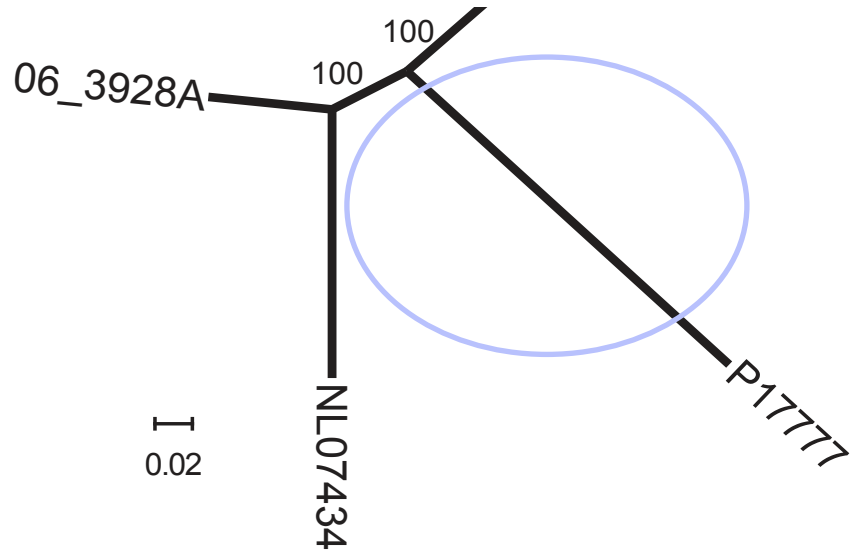
Attack of the clones - rise and fall of *Phytophthora infestans* asexual lineages in Britain



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

Get directions My places

Save to My Places **EDIT**

Collaborate

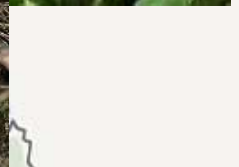
P_infestans_collection_places_2013

Collection trip was in 2013 in Colombia and Ecuador

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

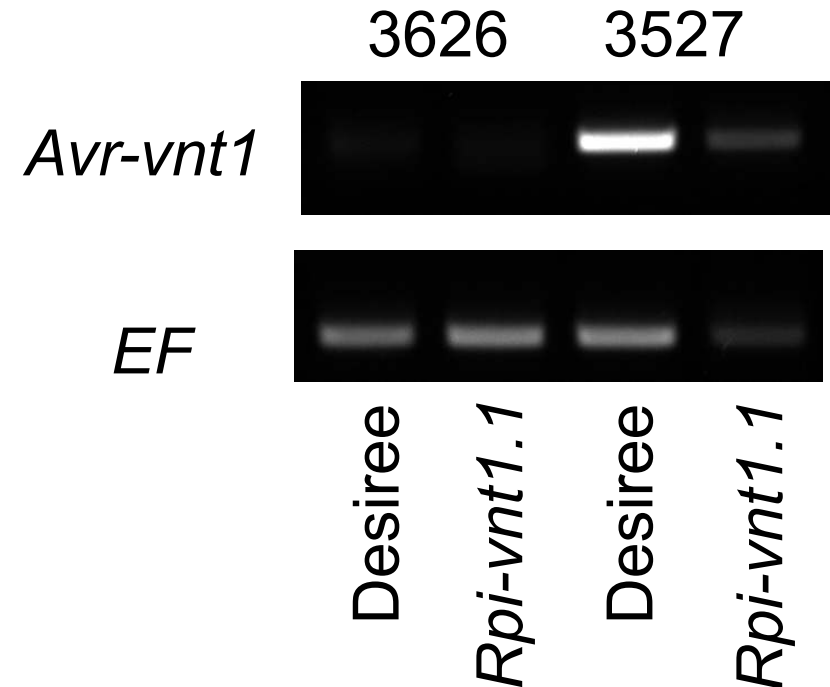
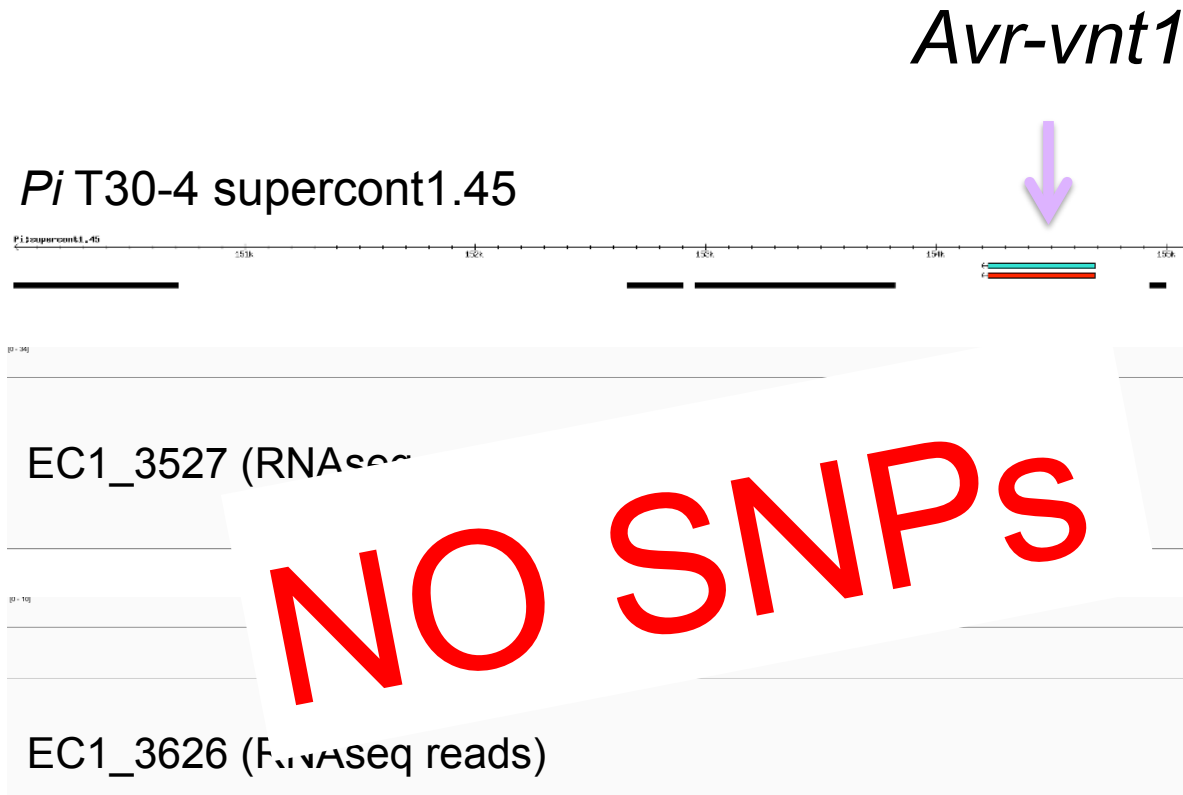
[Rate this map](#) · [Write a comment](#) · [KML](#)

-  **E1**
Machachi, Potato fields, -00° 32' 21.4", -078° 36' 25.7", 3210m, Solanum tuberosum, Super Chola
-  **E2**
Machachi, 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
-  **DSCF0184.JPG**
Date: Mar 5, 2013, 12:47 PM Number of Comments on Photo: 0 View Photo
-  **E4**
-  **E5**
-  **E6**
-  **E7**
-  **E8**
-  **E9**
-  **E10**
-  **E11**
-  **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 warm-

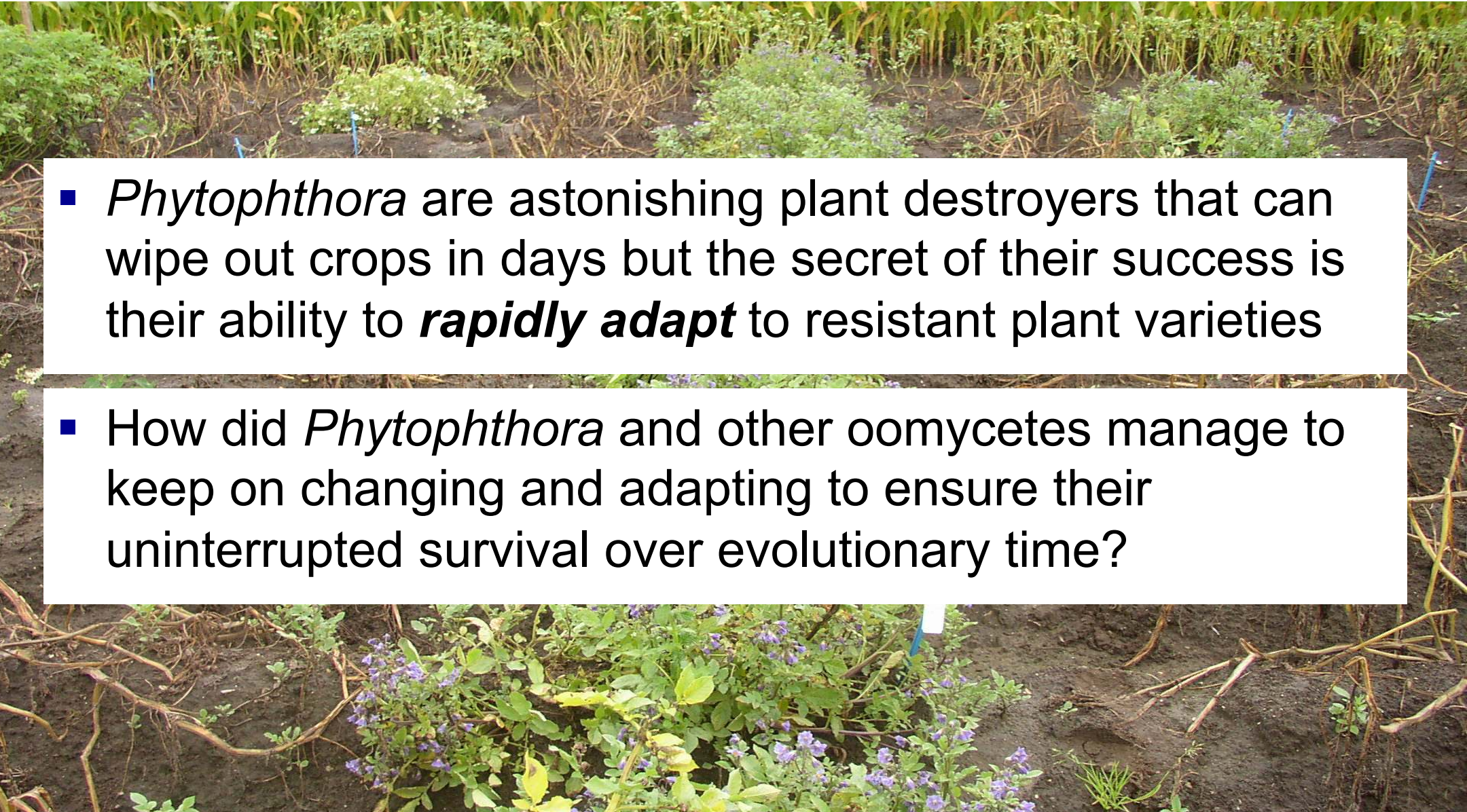
ing. But they have several unique features, researchers say—including the way they can switch from asexual to sexual reproduction—that enable them to exploit these opportunities particularly effectively.

The *Nature* paper, published in April, was in part a cry for attention; its authors say the world isn't fully aware of the dangers and should invest more in countermeasures. For decades, fungal diseases have been overshadowed by bacteria and viruses. "There are probably 50 or 100 bacterial experts for every fungal expert," says Bruce McDonald, a plant pathologist at the Swiss Federal Institute of Technology in Zurich. "There has always been a sense that fungi are not that important," adds microbiologist Arturo Casadevall of Albert Einstein College of Medicine in New York City.

That has begun to change only very recently, thanks in part to some highly publicized animal die-offs. "A few years ago people just scoffed when you thought a fungus had killed an animal such as a bat," says Gudrun Wibbelt, a veterinary pathologist at

- 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



Pathogenomics is an emerging field of plant pathology

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 grain². 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 roots⁷.

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 assembly) contained more than one marker and could thus be oriented on the map. The ends of chromosomes were identified by the telomeric repeat motif (TTAGGG)*n*. Thirteen telomeric sequences were placed at the ends of scaffolds, of which six could be placed at the ends of chromosomes, whereas the remainder were associated with unanchored scaffolds (Supplementary Table S2). Genome coverage was estimated by aligning 28,682 *M. grisea* expressed sequence (ESTs), representing genes expressed during a range of developmental stages and environments. 94% of the ESTs were aligned, many of these ESTs being from d

The gene content of a plant pathogen
Within the *M. grisea* genome, 1

2005

2006

980

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NATURE | VC

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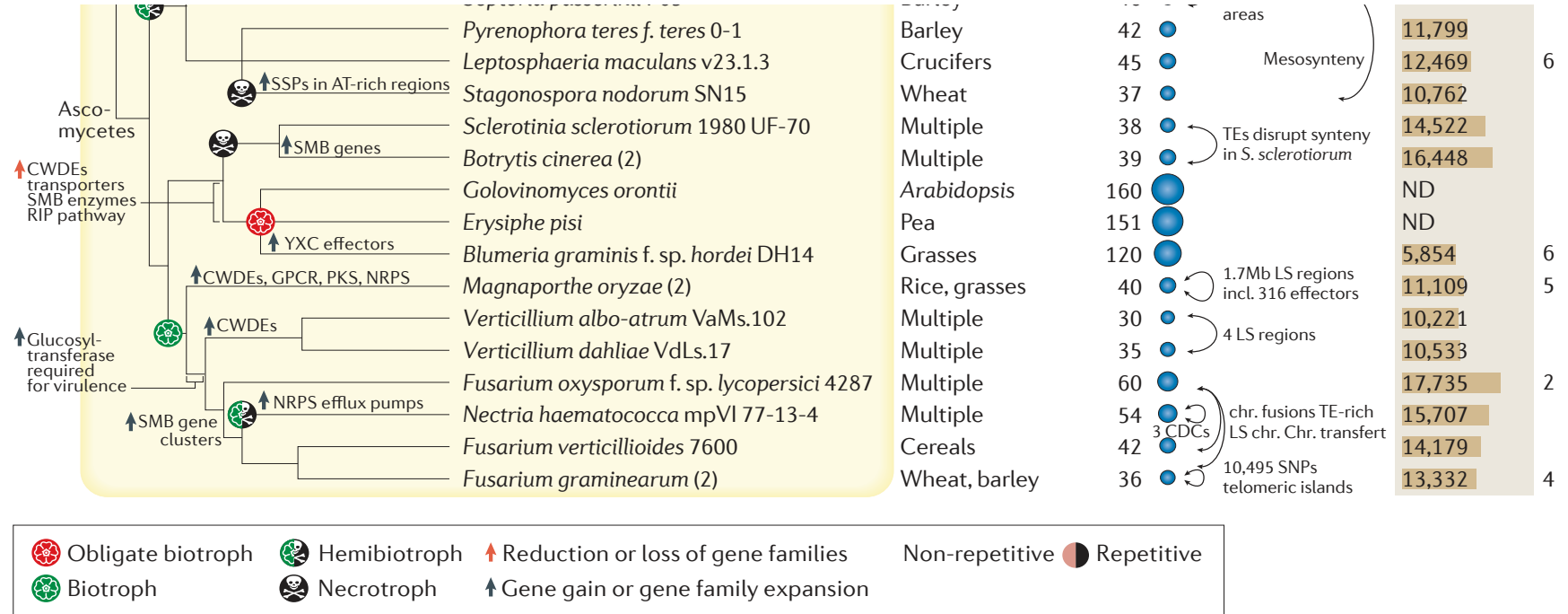
Paul F. Morris,²⁴ Vipaporn Phantumarat,²⁴ Jocelyn K. C. Rose,²⁴ Yasuko Sakihama,²⁴ Chantel F. Scheuring,¹⁸ Brian M. Smith,¹⁸ Trudy A. Torto-Alalibo,¹ Joe Win,⁹ Zhan Daniel S. Rokhsar,^{2,7} Jeffrey L. Boore^{2,1}

Draft genome sequences have been determined for the sudden oak death pathogen *Phytophthora ramblensis*. The draft genome sequences share the kingdom Stramenopila and the presence of many *Phytophthora* genes of common ancestry for the stramenopiles. Comparison of the draft genome sequences with those of other stramenopiles reveals diversification of many protein families.

2009

(KTH), School of Biotechnology, AlbaNova University Centre, Stockholm SE-10691, Sweden. ¹²Department of Plant Pathology and Plant-Microbe Biology, Cornell University, New York 14853, USA. ¹³Center for Genome Research and Biocomputing and Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon 97331, USA. ¹⁴Biology Department, Lafayette College, Easton, Pennsylvania 18042, USA. ¹⁵Plant Molecular Sciences Faculty of Biomedical and Life Sciences, Bower Building, University of Glasgow G12 8QQ, UK. ¹⁶USDA-ARS, Dale Bumpers National Rice Research Center, Stuttgart, Arkansas 72160, USA. ¹⁷Department of Molecular Biology, Massachusetts General Hospital, Boston, Massachusetts 02114, USA. ¹⁸Delaware Biotechnology Institute, University of Delaware, Newark, Delaware 19711, USA. ¹⁹Department of Plant Pathology, North Carolina State University, Raleigh, North Carolina 27695, USA. ²⁰USDA-ARS, Beltsville, Maryland 20705, USA. ²¹Department of Plant and Soil Sciences, University of Delaware, Newark, Delaware 19716, USA. ²²Department of Entomology and Plant Pathology, University of Tennessee, Knoxville, Tennessee 37996, USA. ²³Institute for Genome Sciences and Policy, University of Maryland School of Medicine, Baltimore, Maryland 21201, USA. ²⁴Department of Biochemistry, Vanderbilt University School of Medicine, Nashville, Tennessee 37232, USA. ²⁵The College of Wooster, Department of Biology, Wooster, Ohio 44691, USA. ²⁶Department of Biological Sciences, Bowling Green State University, Bowling Green, Ohio 43403, USA. ²⁷University of Aberdeen, School of Medical Sciences, College of Life Sciences and Medicine, Institute of Medical Sciences, Foresterhill, Aberdeen AB25 2ZD, UK. ²⁸Medical and Phytopathology Laboratory, Los Andes University, Bogotá, Colombia. ²⁹Institute of Genetics and Molecular Medicine, University of Edinburgh, Cancer Research Centre, Victoria General Hospital, Edinburgh EH4 2XU, UK. ³⁰J. Craig Venter Institute, Rockville, Maryland 20850, USA. ³¹Department of Plant Sciences, Tel Aviv University, Tel Aviv 69978, Israel. ³²Department of Chemistry, Laboratory of Genetics, Laboratory for Molecular and Computational Genomics, University of Wisconsin Biotechnology Center, University of Wisconsin-Madison, Madison Wisconsin 53706, USA. ³³University of Hohenheim, Institute of Botany 210, D-70593 Stuttgart, Germany. ³⁴Division of Plant Science, College of Life Sciences, University of Dundee (at SCRI), Invergowrie, Dundee DD2 5DA, UK. †Present addresses: 454 Life Sciences, Branford, Connecticut 06405, USA (C.D.K.); Virginia Bioinformatics Institute, Virginia Polytechnic and State University, Blacksburg, Virginia 24061, USA (T.T.-A.); Biomedical Diagnostics Institute, Dublin City University, Dublin 9, Ireland (K.R.). *These authors contributed equally to this work.





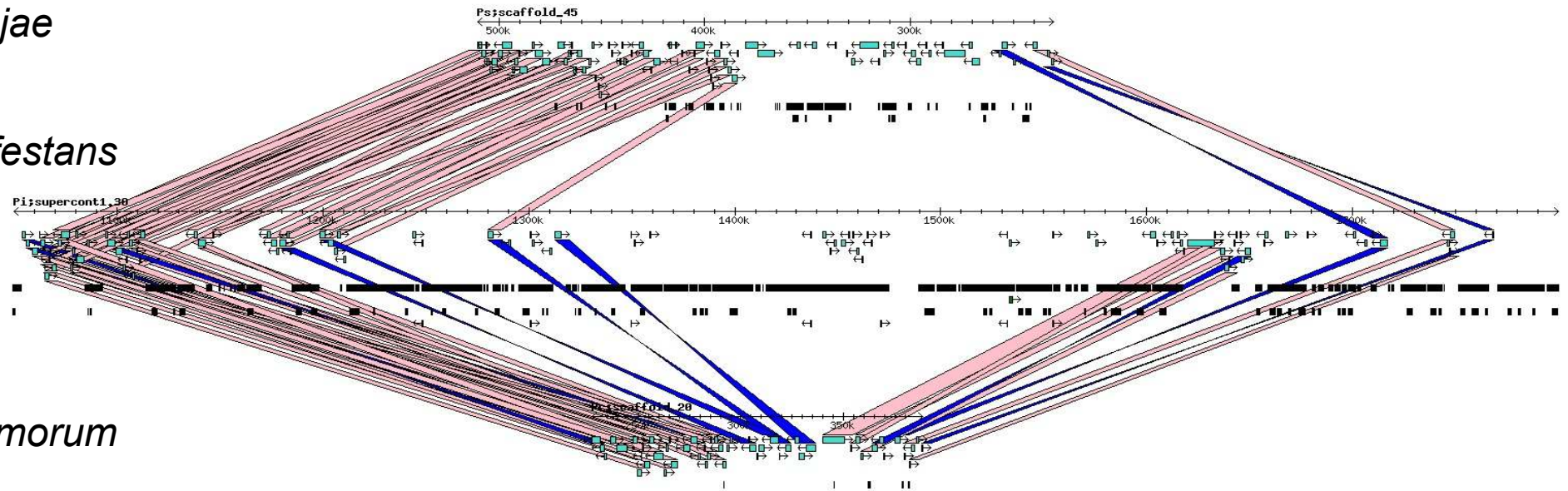
Nature Reviews | Micro

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

P. sojae

P. infestans

P. ramorum



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. McCutcheon¹ and Nancy A. Moran²

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^a, 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 Mitrosⁱ, 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 *Boechea stricta*



rust infected plant
with pseudoflowers

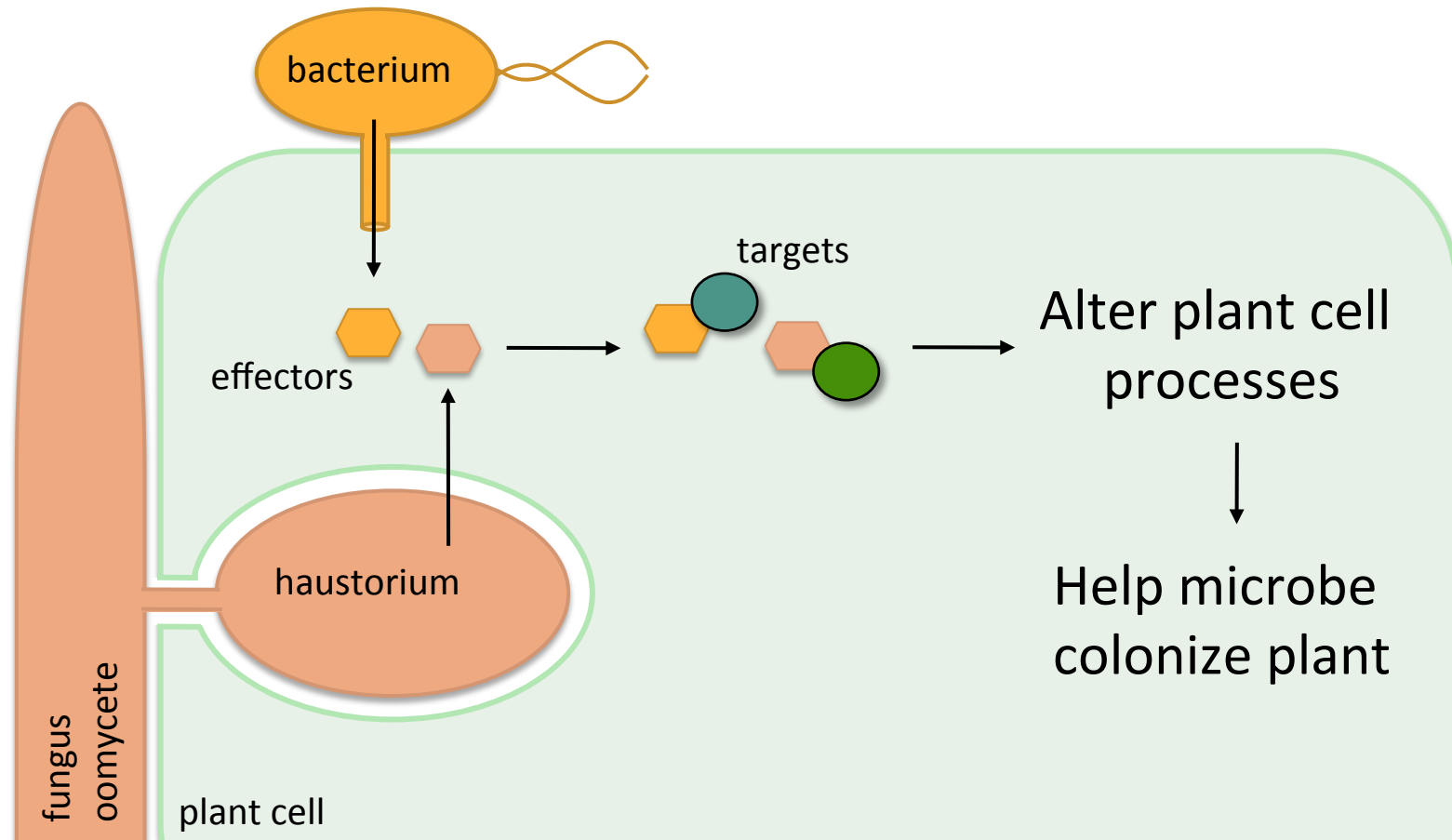


healthy plant with
normal flowers

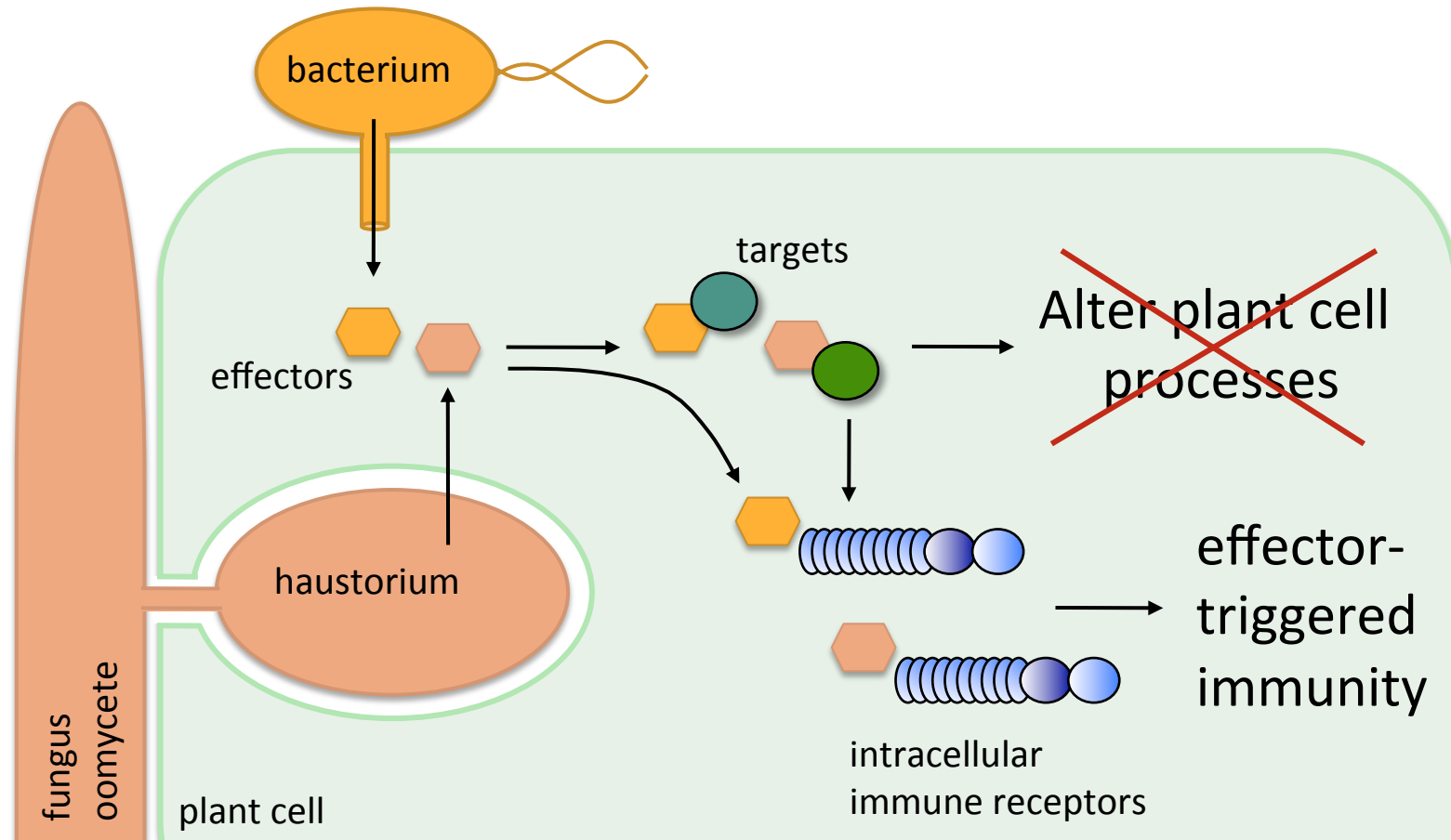
Effectors – secreted pathogen molecules that perturb plant processes

- ➡ **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

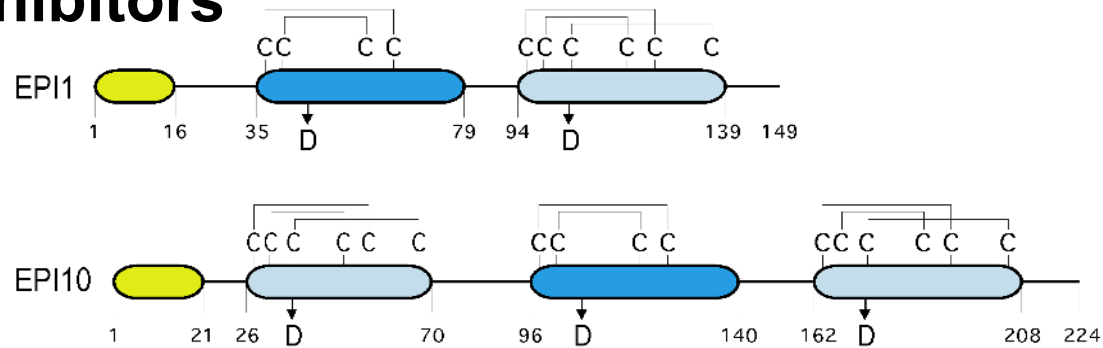


Some effectors “trip the wire” and activate immunity in particular plant genotypes



The diverse effectors of *Phytophthora infestans*

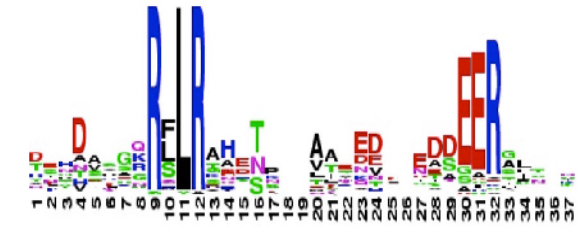
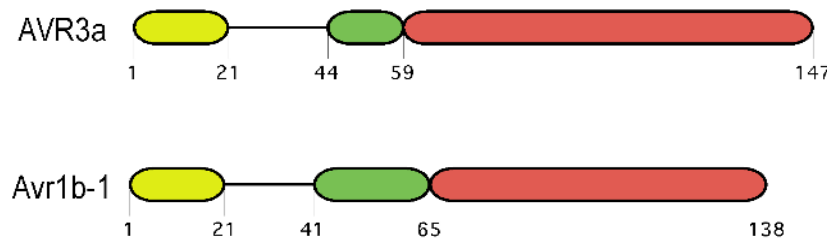
Protease inhibitors



~38

Apoplastic

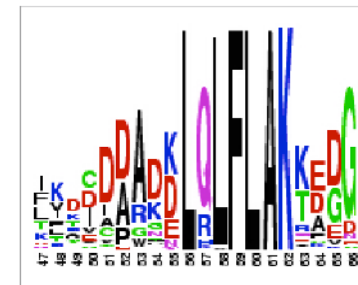
RXLR



~550

Host-translocated

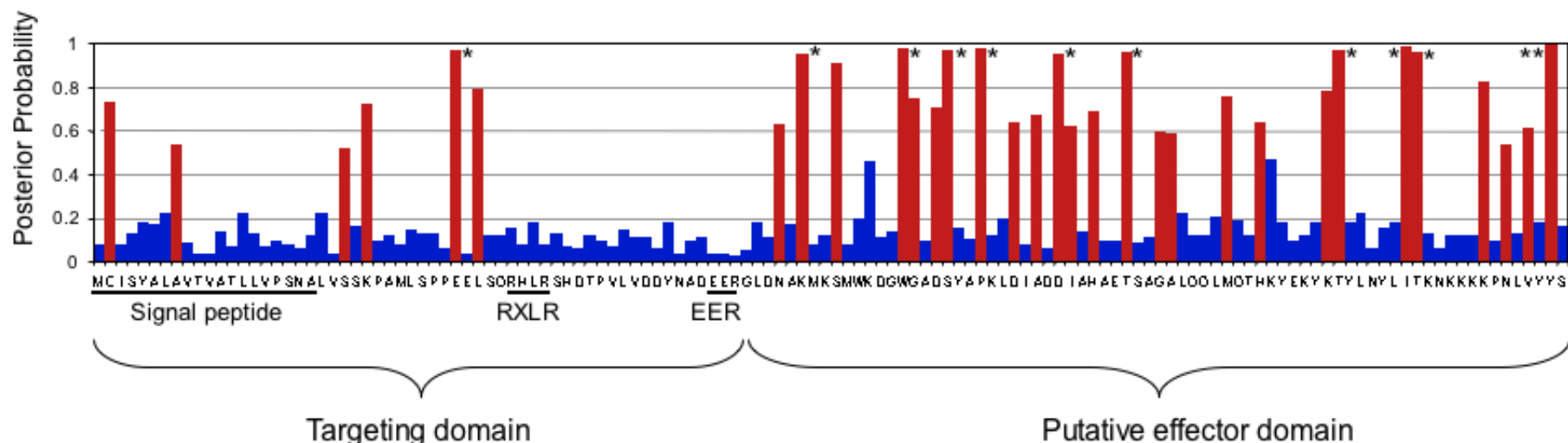
Crinklers



~200

~250 ψ

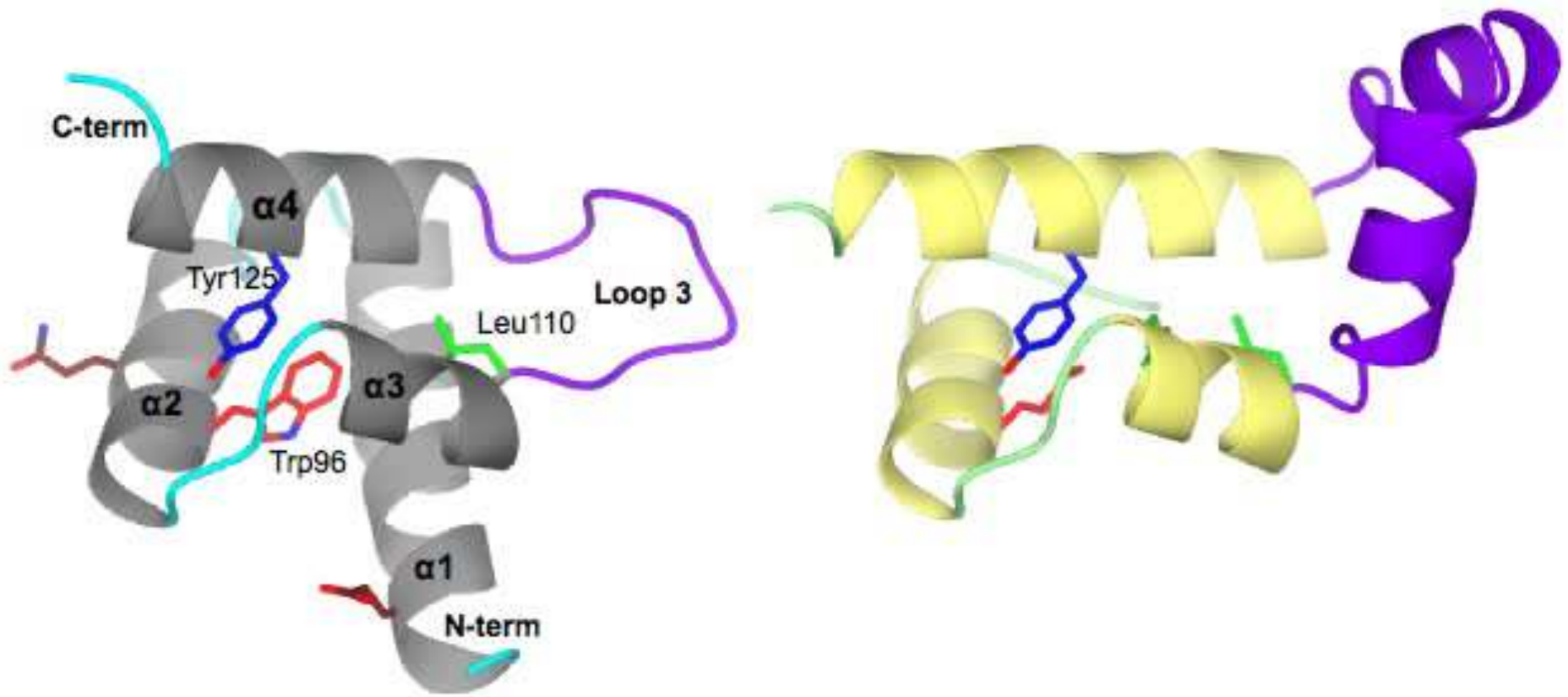
Positive selection has targeted the C terminal domain of RXLR effectors (ML method in paml)



20 40 60 80 100 120
 PiPGG1_3 MRISYALTVTATLLVPSNALVNSKPAMLSPPGEPSSQRHLRSHDTPVLVDDYNAD^{GLD}KAAMKTMWEDGMSAAGYAKKLGITDKIALAEKSAGVLQQLMQTRRYEKYQYLYNLAKNNKKKKPDLIYLS
 PiPGG1_4T.....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_2A.....S.K.....E.....N.K..S..K..W..DN..P...A.D..H..T...A....T..HK....KT.....IT.....Q.N.V.Y.

- Consistent with the view that RXLR effectors are modular

RXLR effector proteins have conserved but adaptable structures

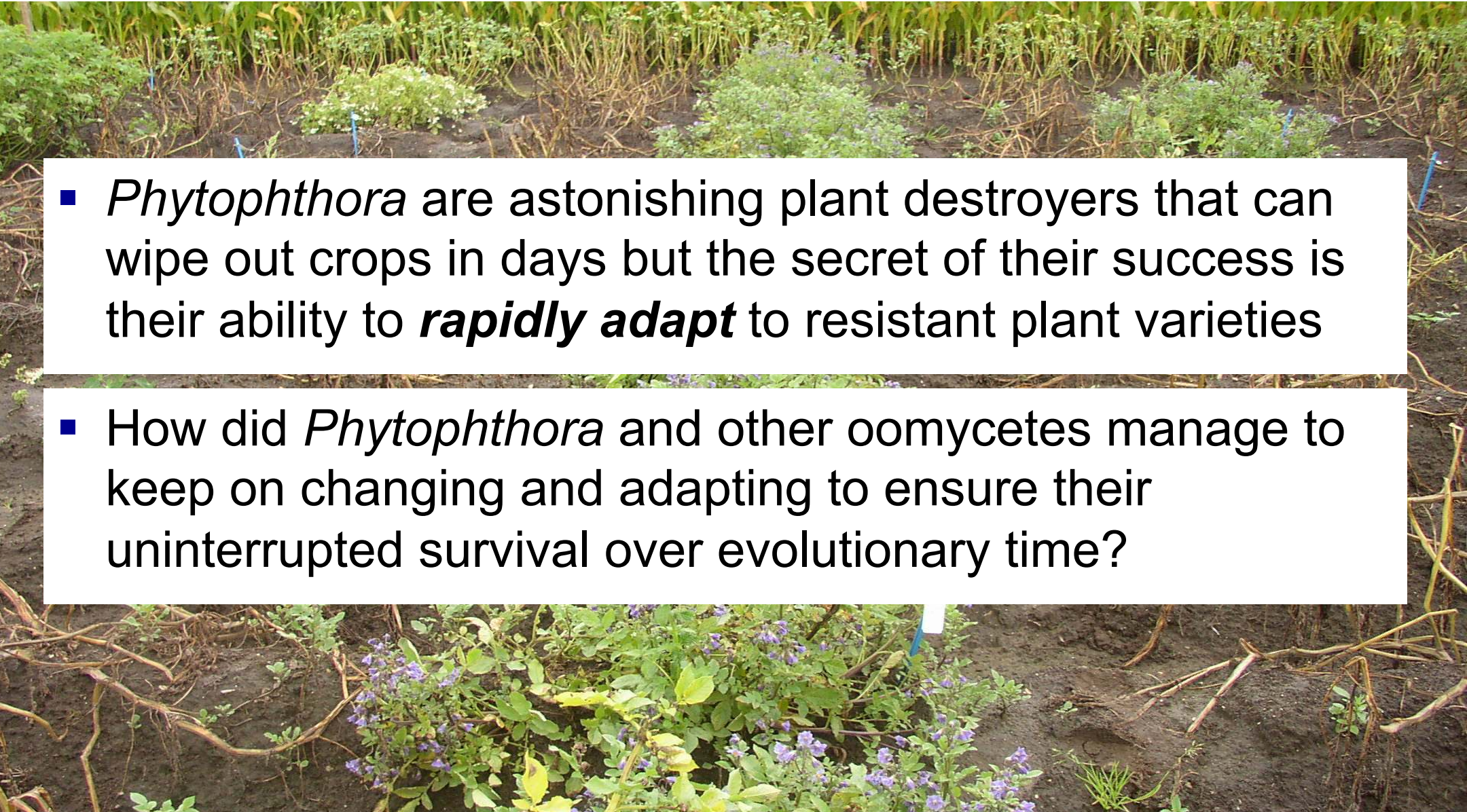


WY-fold of *Phytophthora* RXLR-WY effectors

- ➡ 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?

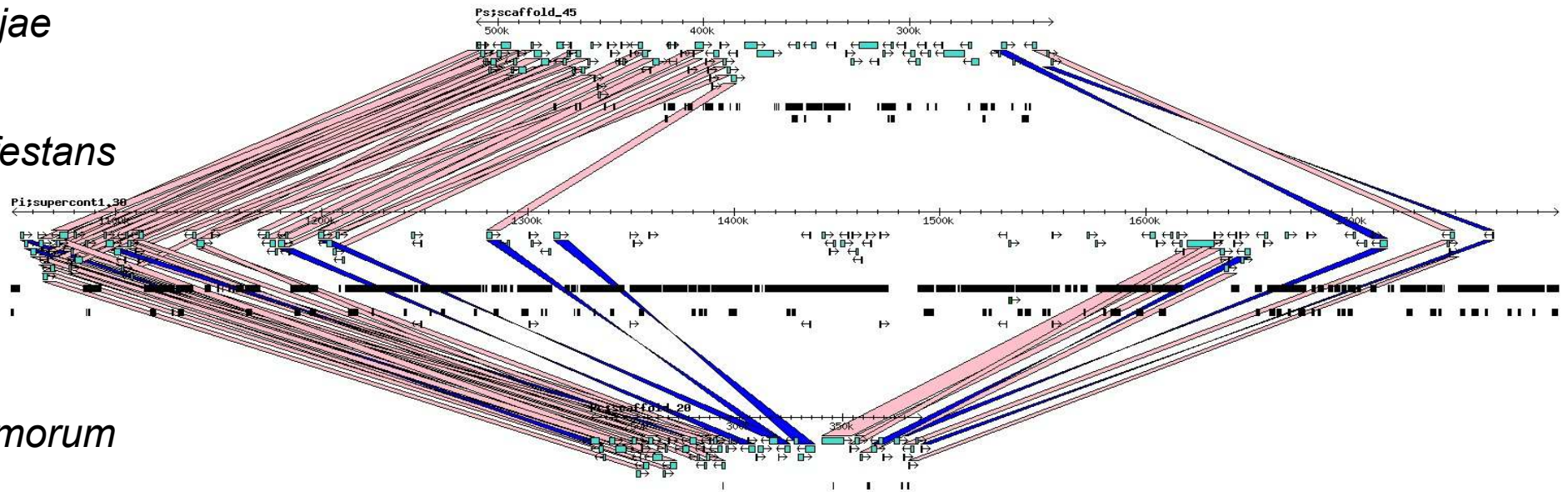
- 
- *Phytophthora* are astonishing plant destroyers that can wipe out crops in days but the secret of their success is their ability to ***rapidly adapt*** to resistant plant varieties
 - How did *Phytophthora* and other oomycetes manage to keep on changing and adapting to ensure their uninterrupted survival over evolutionary time?

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

P. sojae

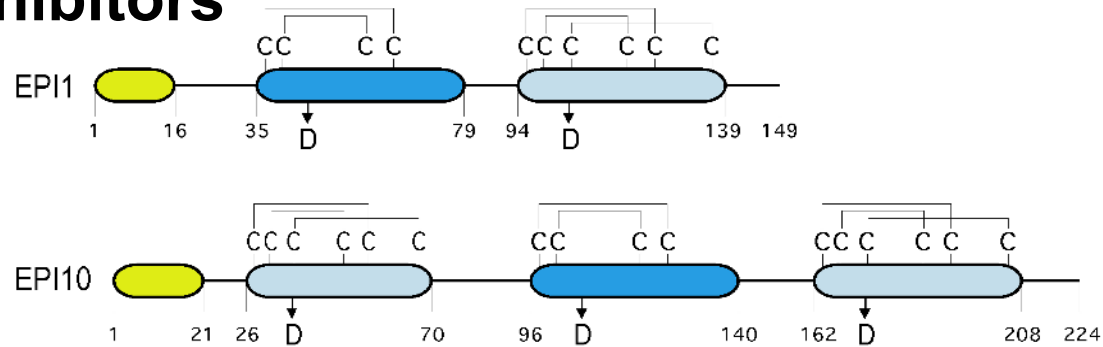
P. infestans

P. ramorum



The diverse effectors of *Phytophthora infestans*

Protease inhibitors

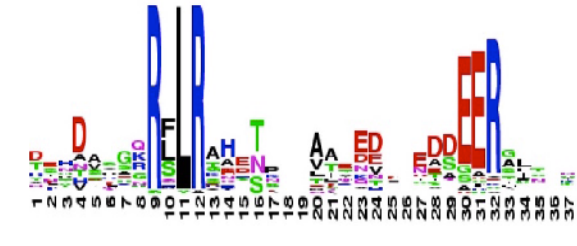
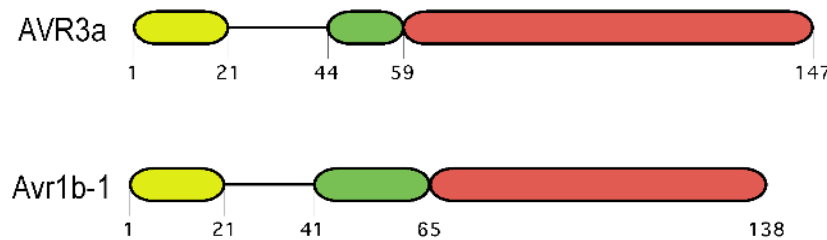


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Apoplastic

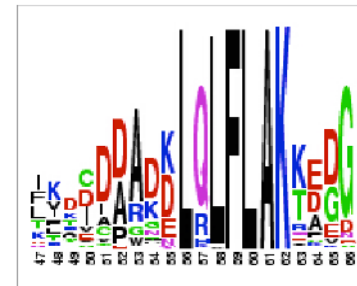
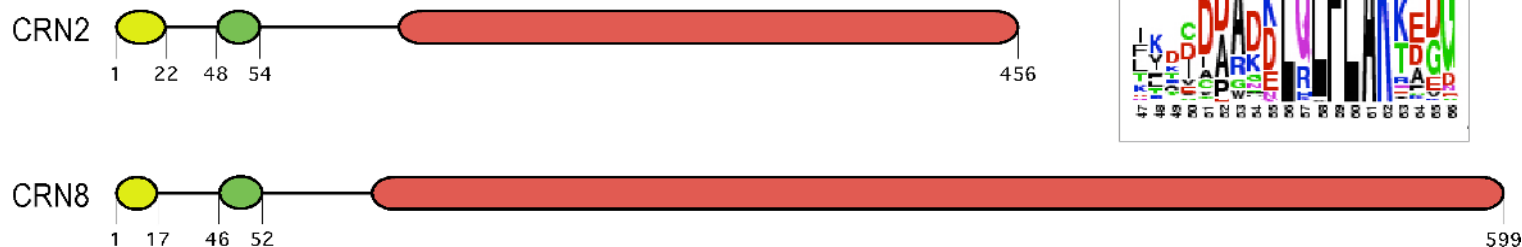
RXLR

Host-translocated



~550

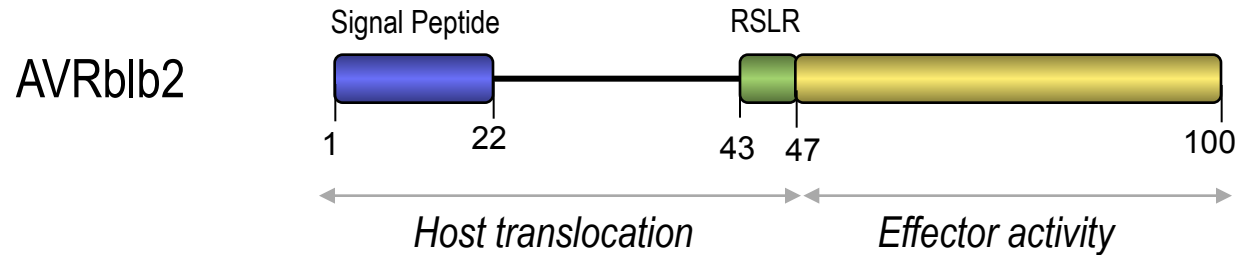
Crinklers



~200

~250 ψ

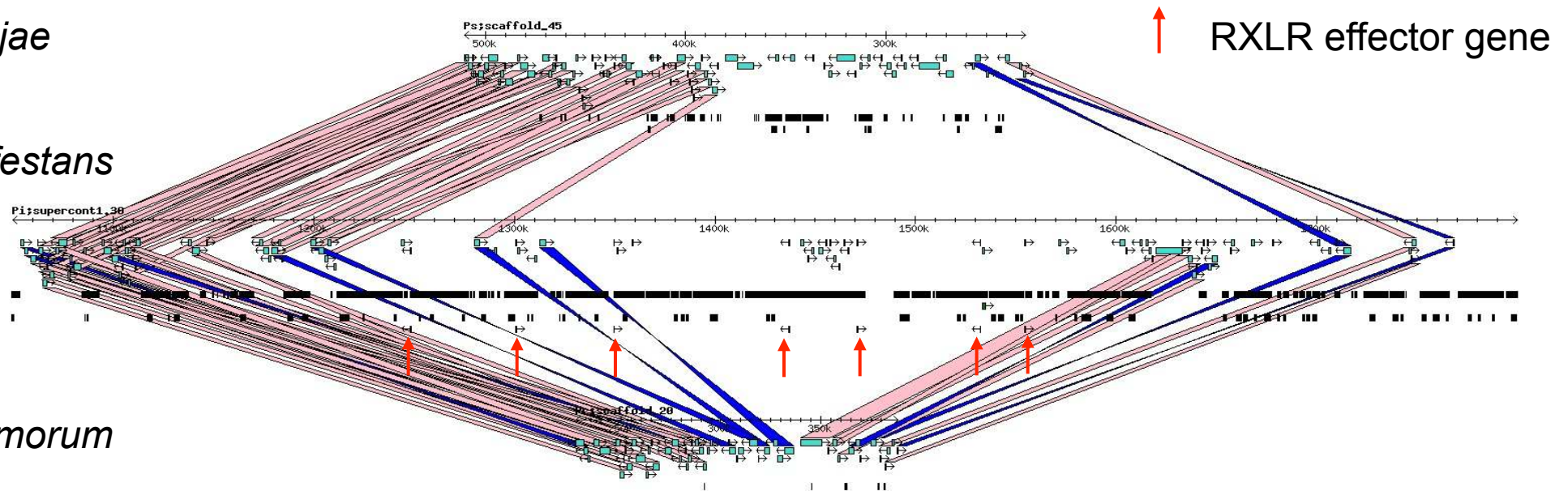
Phytophthora infestans effectors typically occur in the expanded, repeat-rich and gene-poor loci



P. sojae

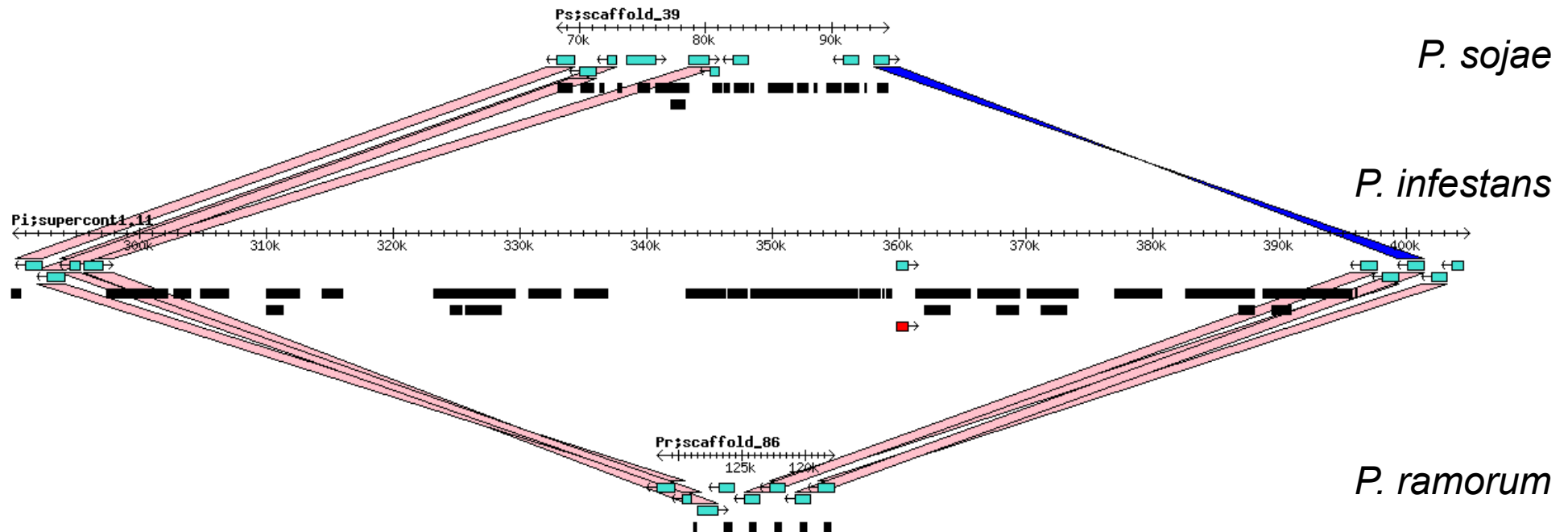
P. infestans

P. ramorum



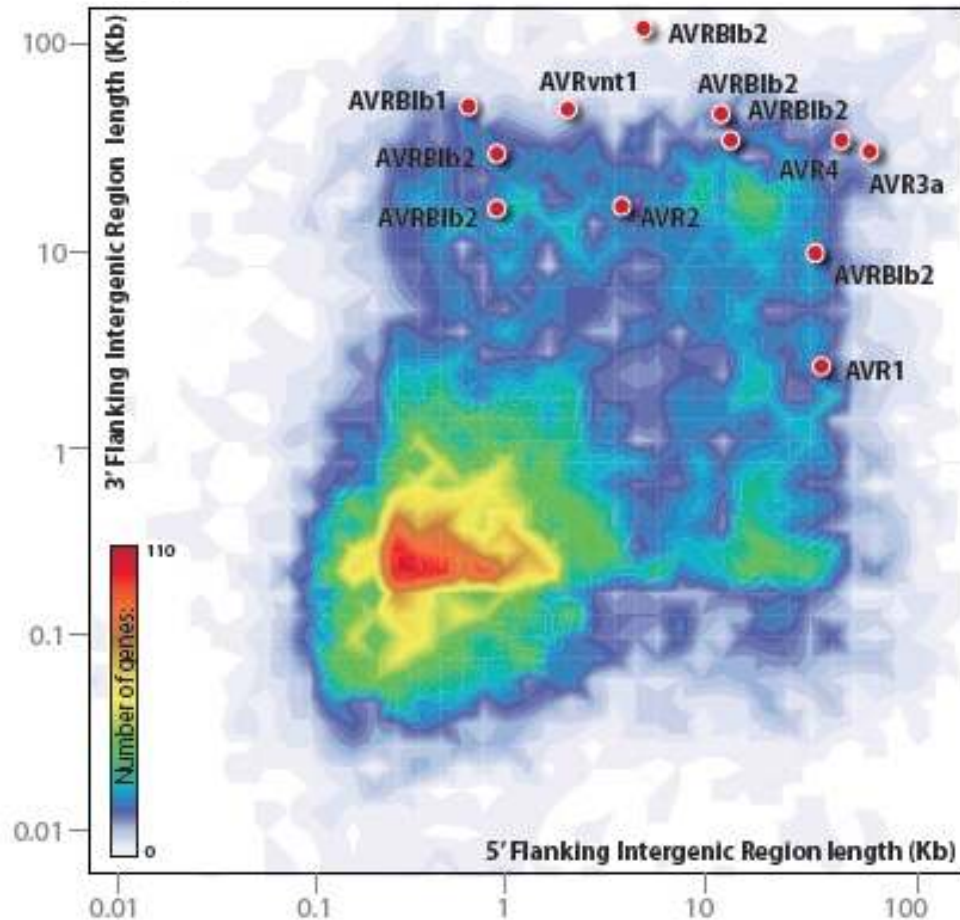
The “two-speed genome” of *P. infestans* underpins high evolutionary potential

- 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

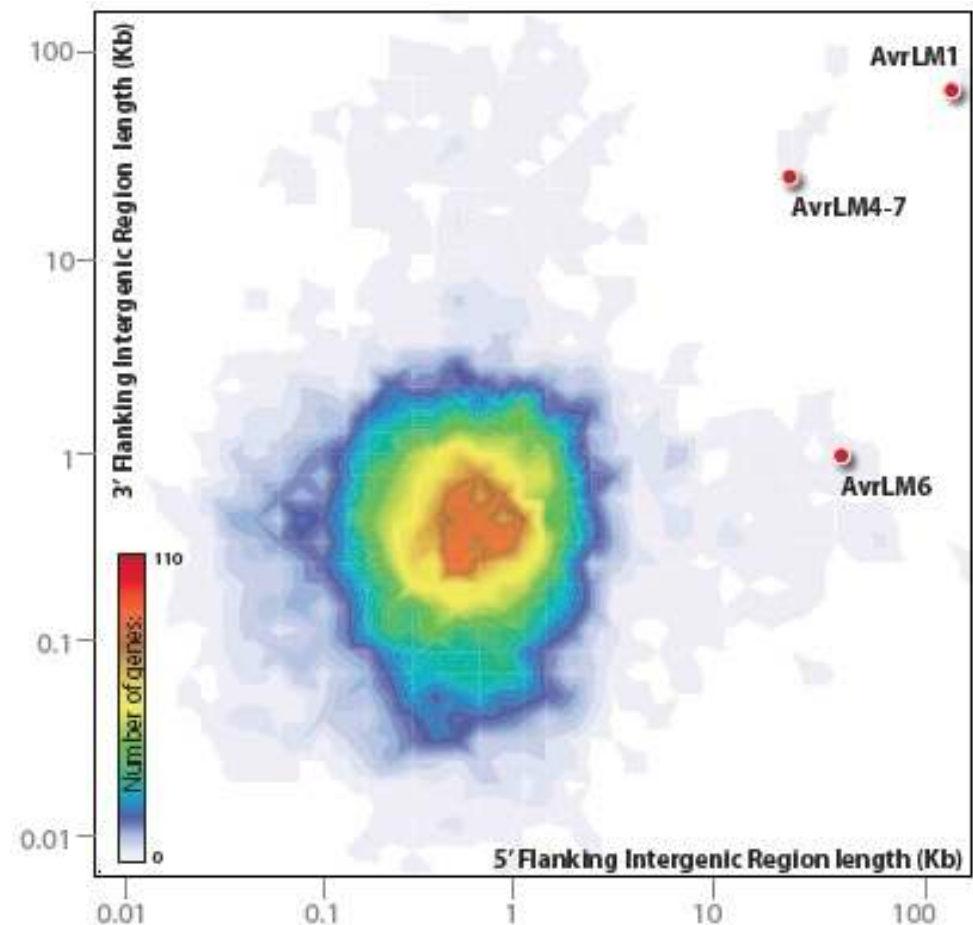


Oomycete and fungal plant pathogens independently evolved “**two-speed**” genomes

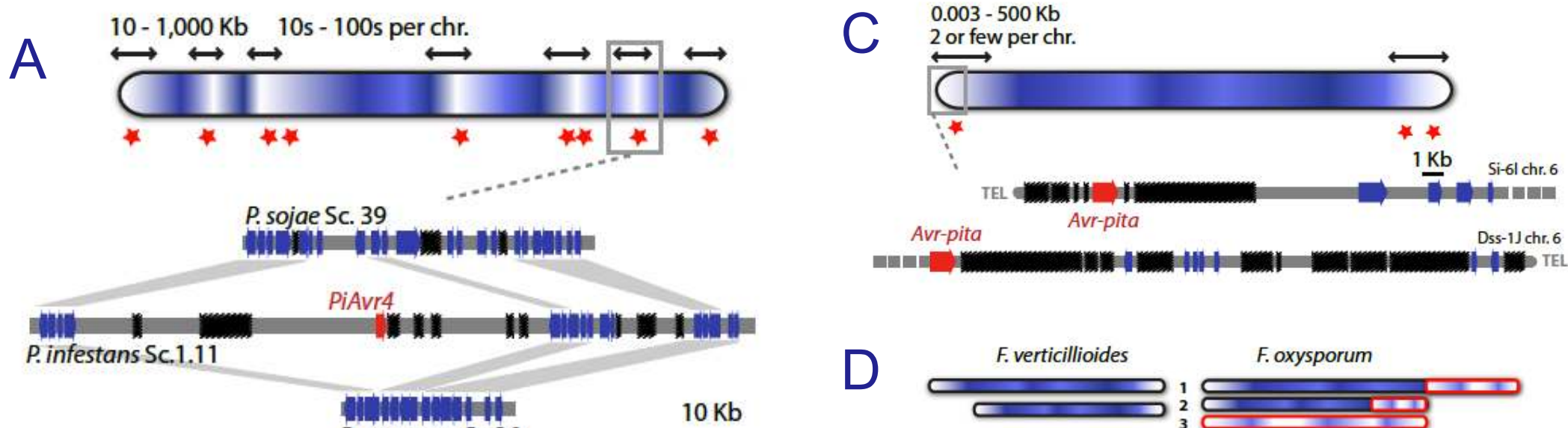
***Phytophthora infestans* >>> Oomycete**



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



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.

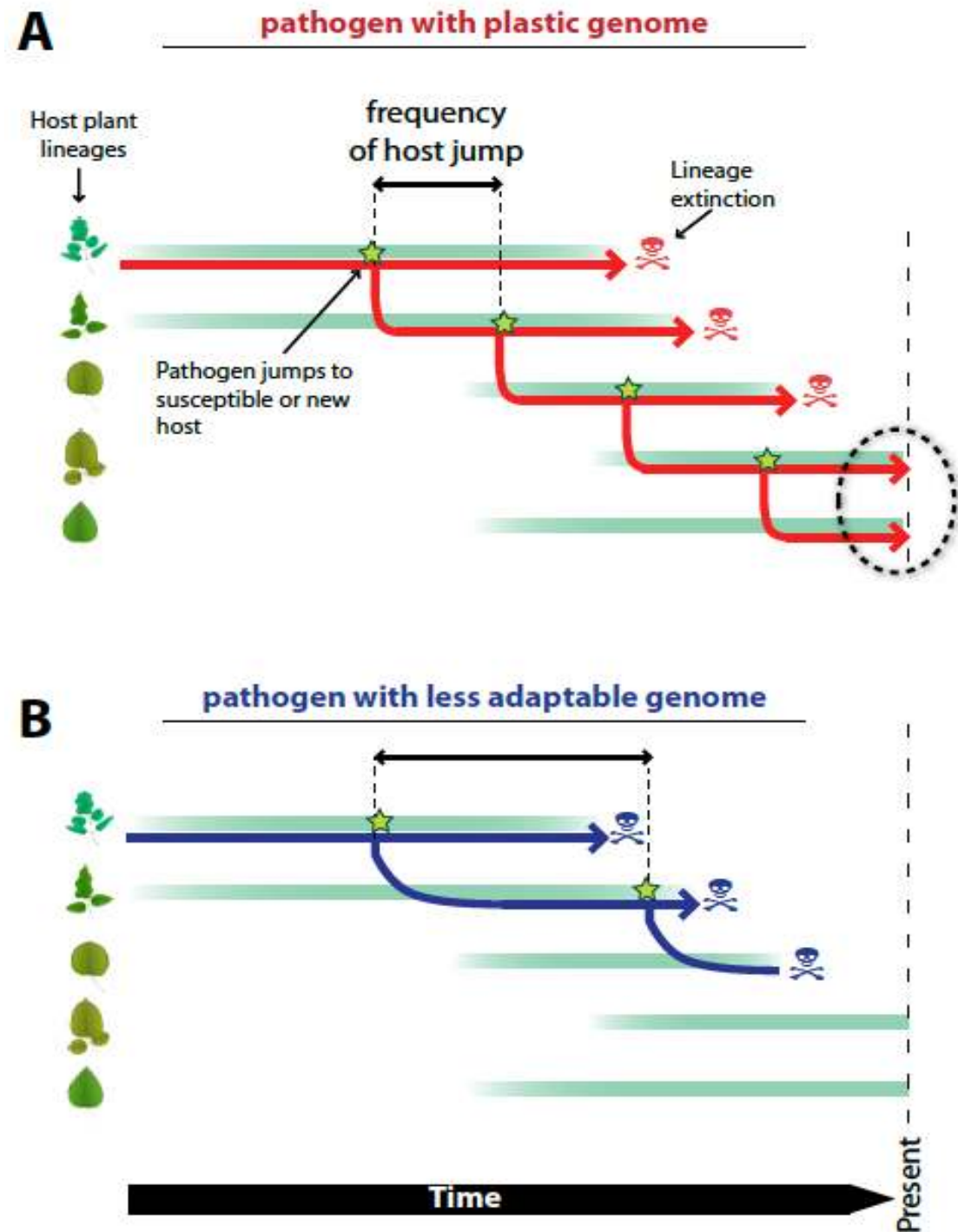
- “...it would be advantageous for a species if the genes for biochemical diversity [in disease resistance] were particularly mutable, provided that this could be achieved without increasing the mutability of other genes whose mutation would give lethal or sublethal genotypes.”

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!

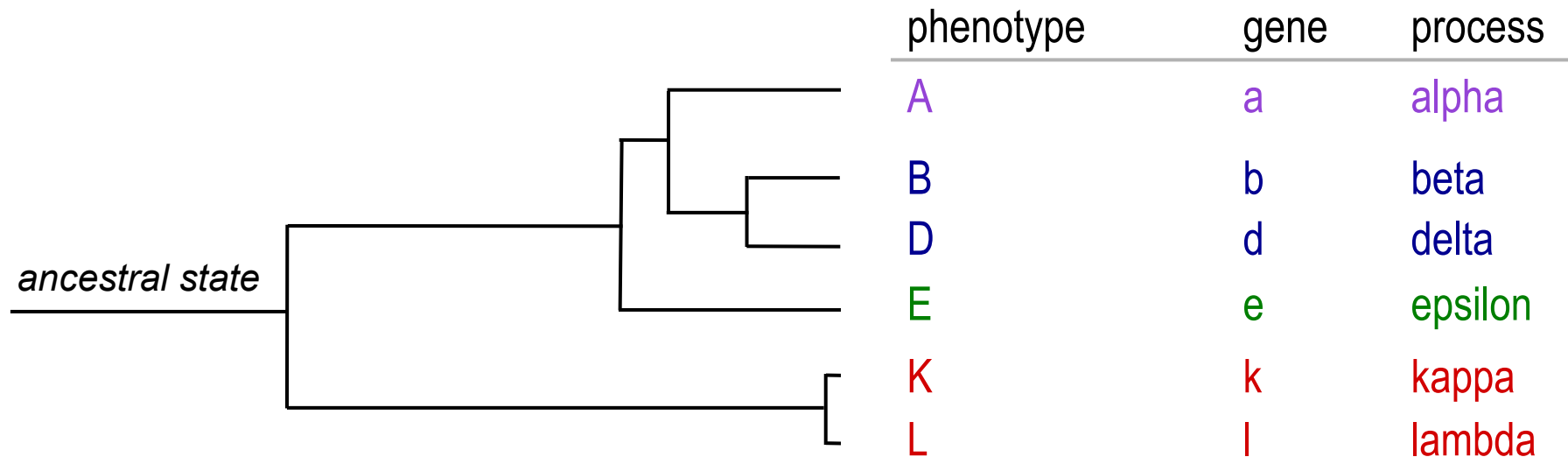
- Lineages with less adaptable genomes suffer higher extinction rates, thus a macroevolutionary disadvantage
- Lineages that have adaptable genomes end up dominating the biota
- **Clade selection** – opposes short term advantages conferred by smaller compact genomes



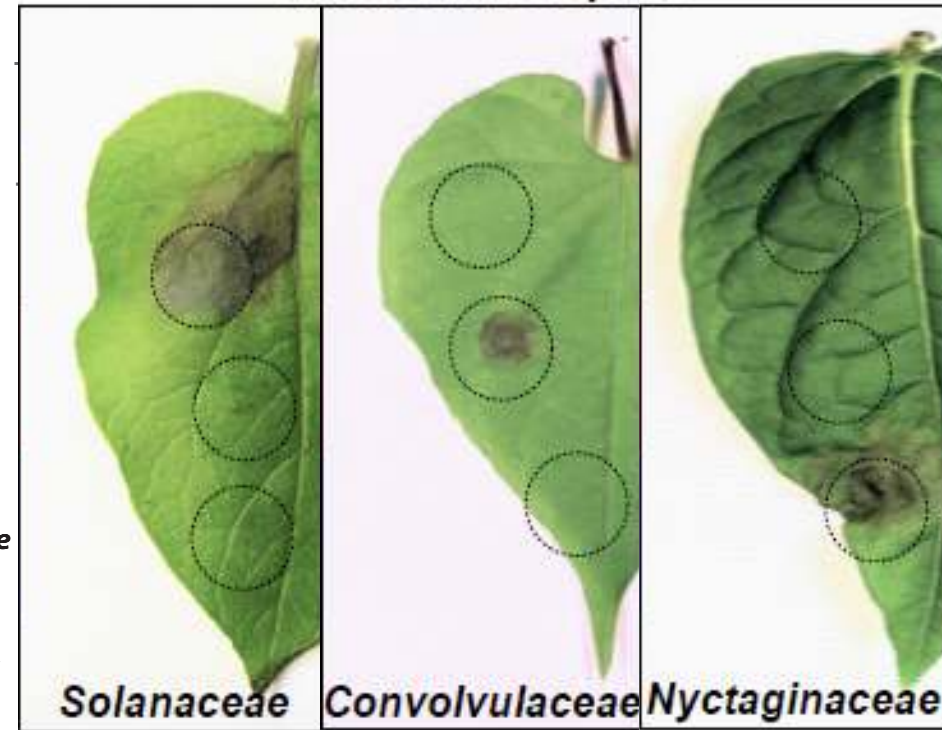
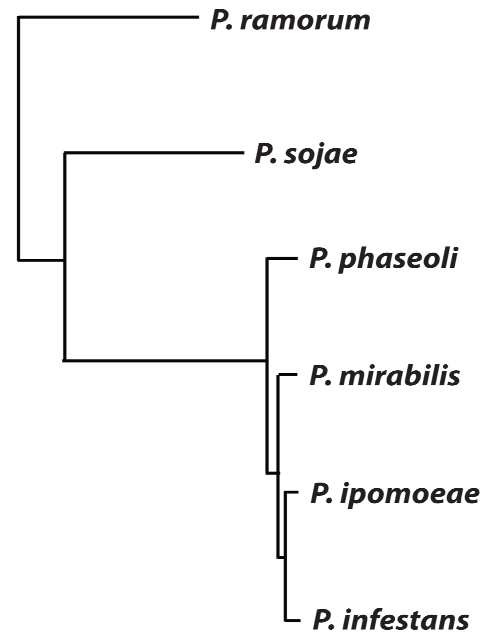
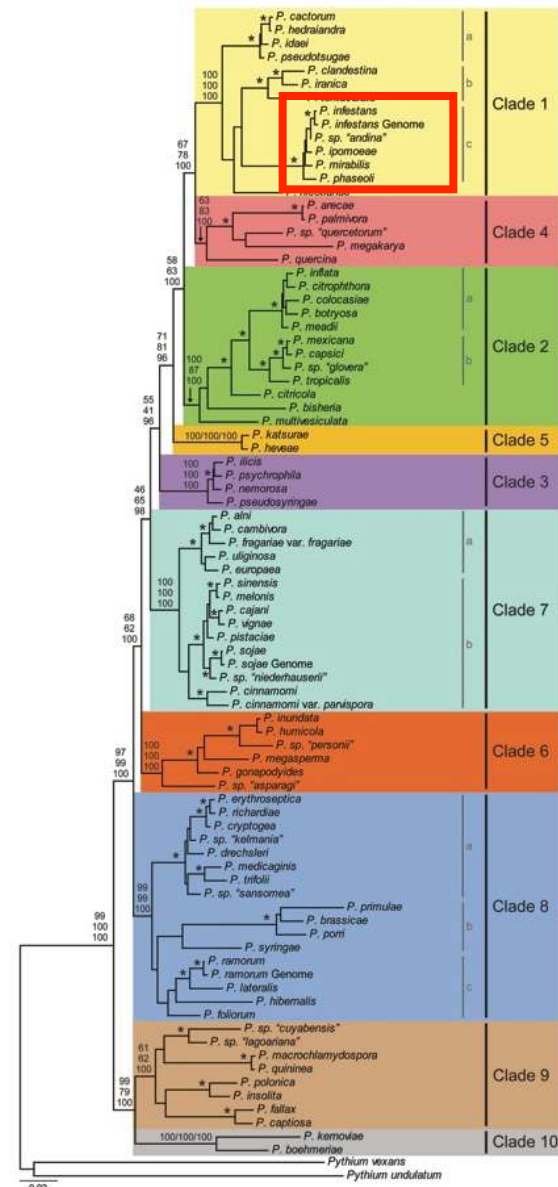


#EvoMPMI – Evolutionary Plant-Microbe Interactions

- ➡ 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

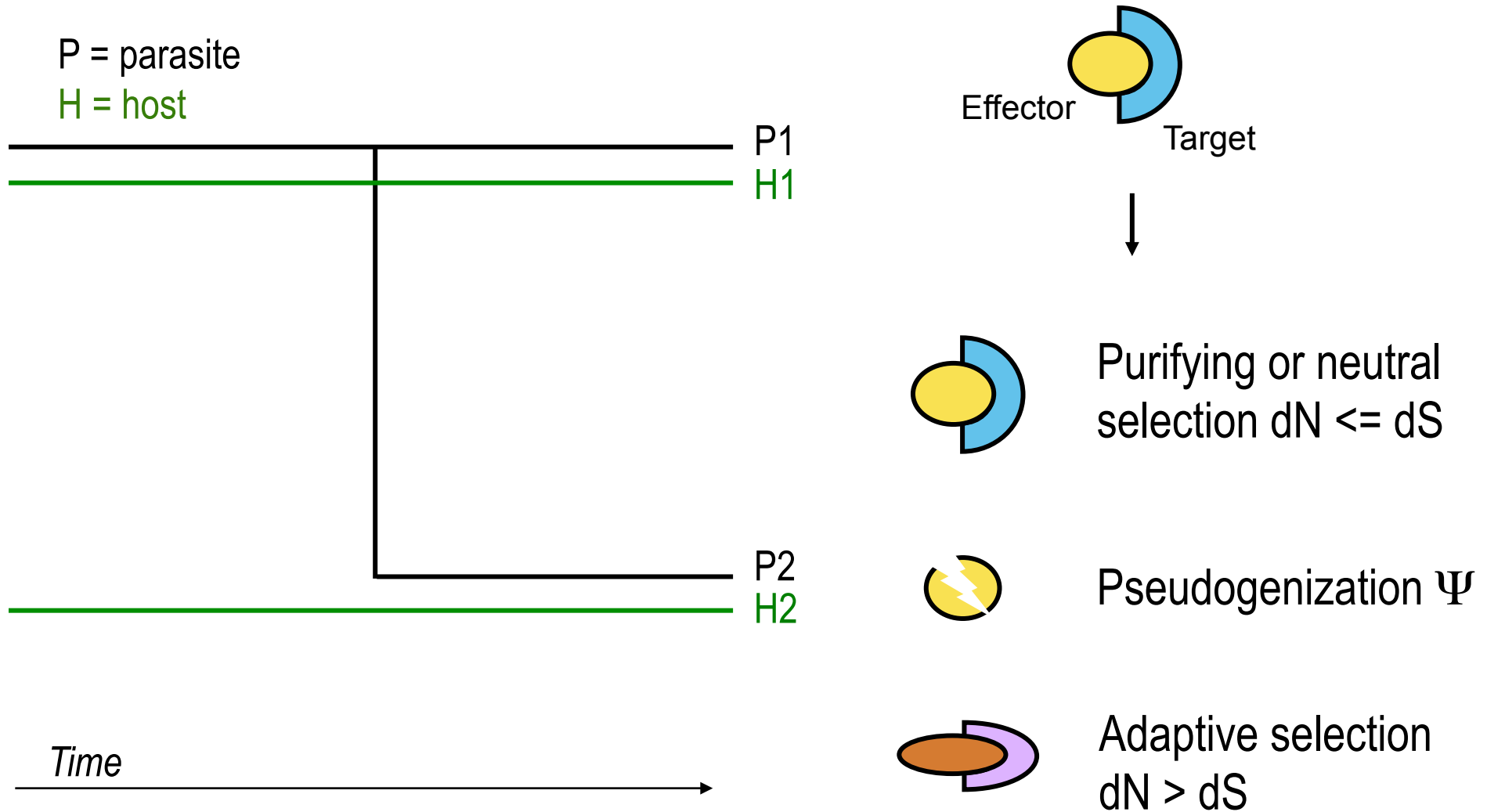


Species in the *Phytophthora infestans* lineage (clade 1c) evolved by host jumps



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

Host jumps must have a dramatic impact on effector evolution



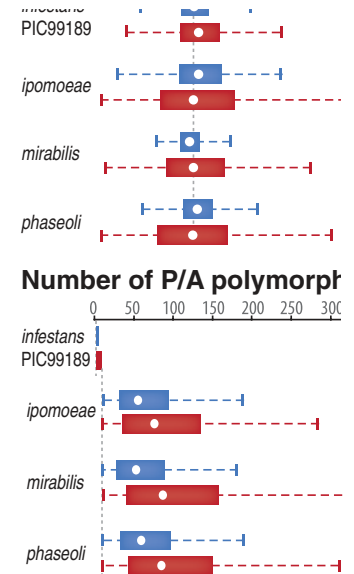
Impact of host jumps on genome and effector evolution

and positive selection. These loci are enriched in genes induced in plants, implicating host adaptation 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 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* spe-

cies form a very tight clade of pathogens that share ~99.9% identity in their ribosomal internal transcribed spacer regions (ITS). Phylogenetic inferences clearly indicate that species *Phytophthora* clade 1c [nomenclature of (2)] emerged through host jumps followed by adaptive specialization on plants belonging to four different botanical families (2, 3). Adaptation to these plants most likely involves mutations in hundreds of disease effector genes that populate poor and repeat-rich regions of the 240-Mbp pair genome of *P. infestans* (4). However, comparative genome analyses of specialized sister s-

dN/dS in genes from gene-dense regions (GDRs) and gene-sparse regions (GSRs). Statistical 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.



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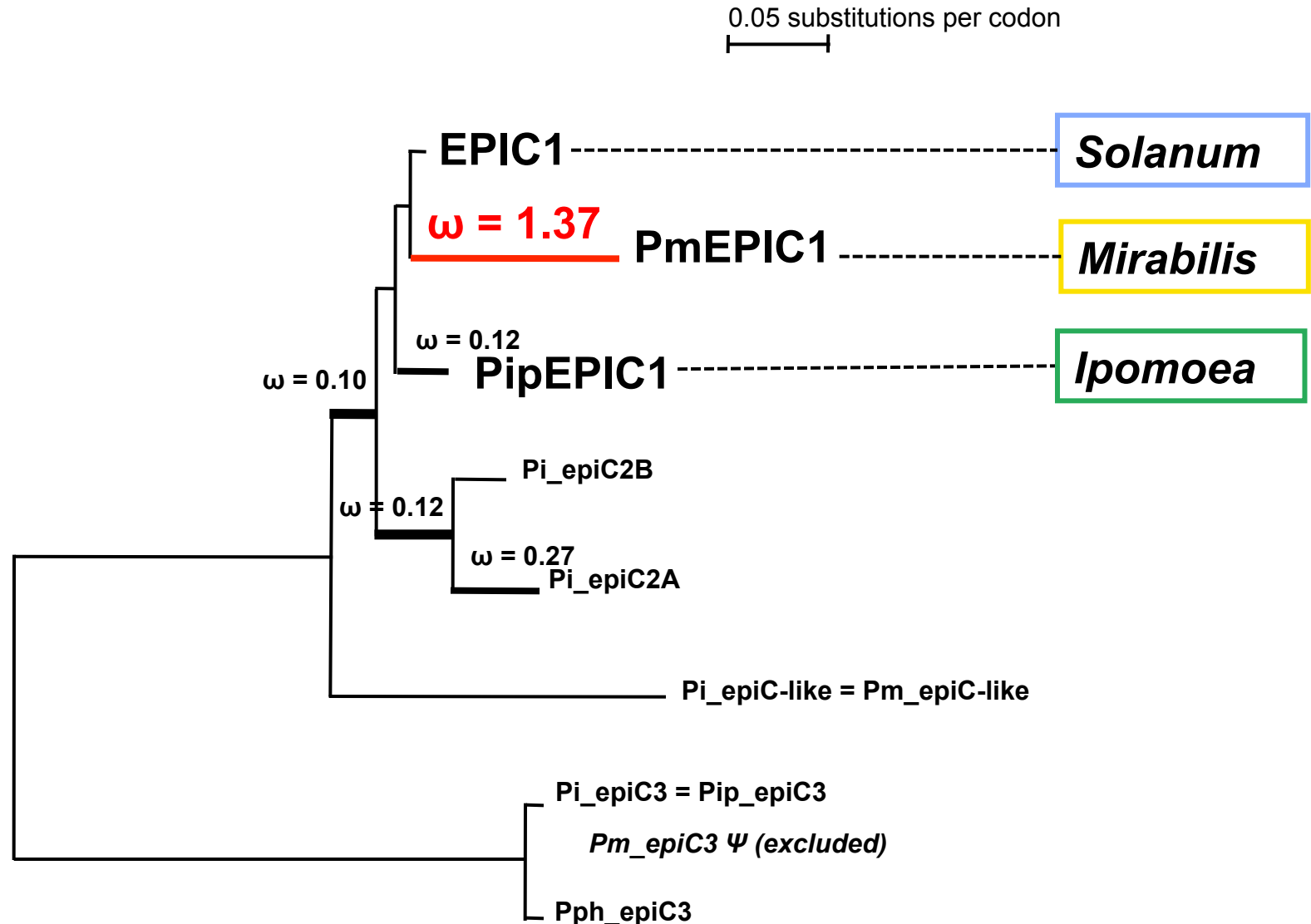
www.sciencemag.org SCIENCE VOL 330

- 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

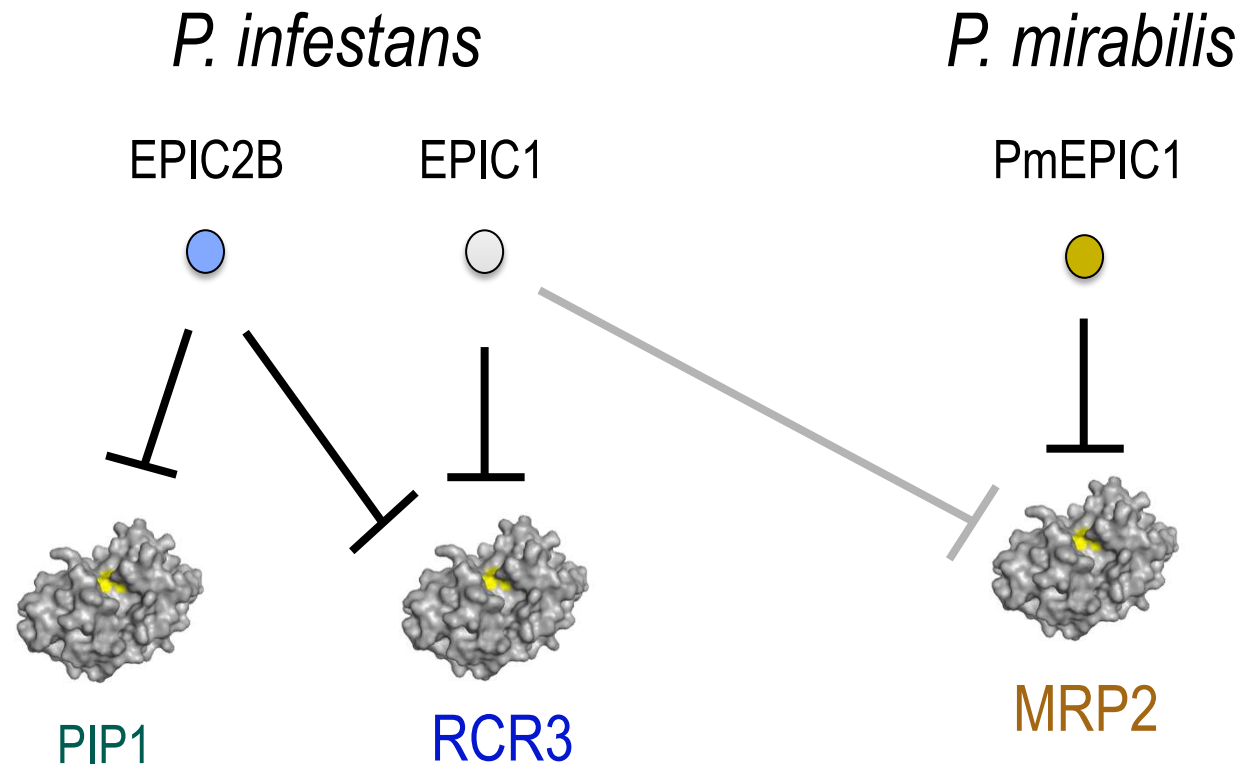
Protease inhibitor PmEPIC1 is under positive selection



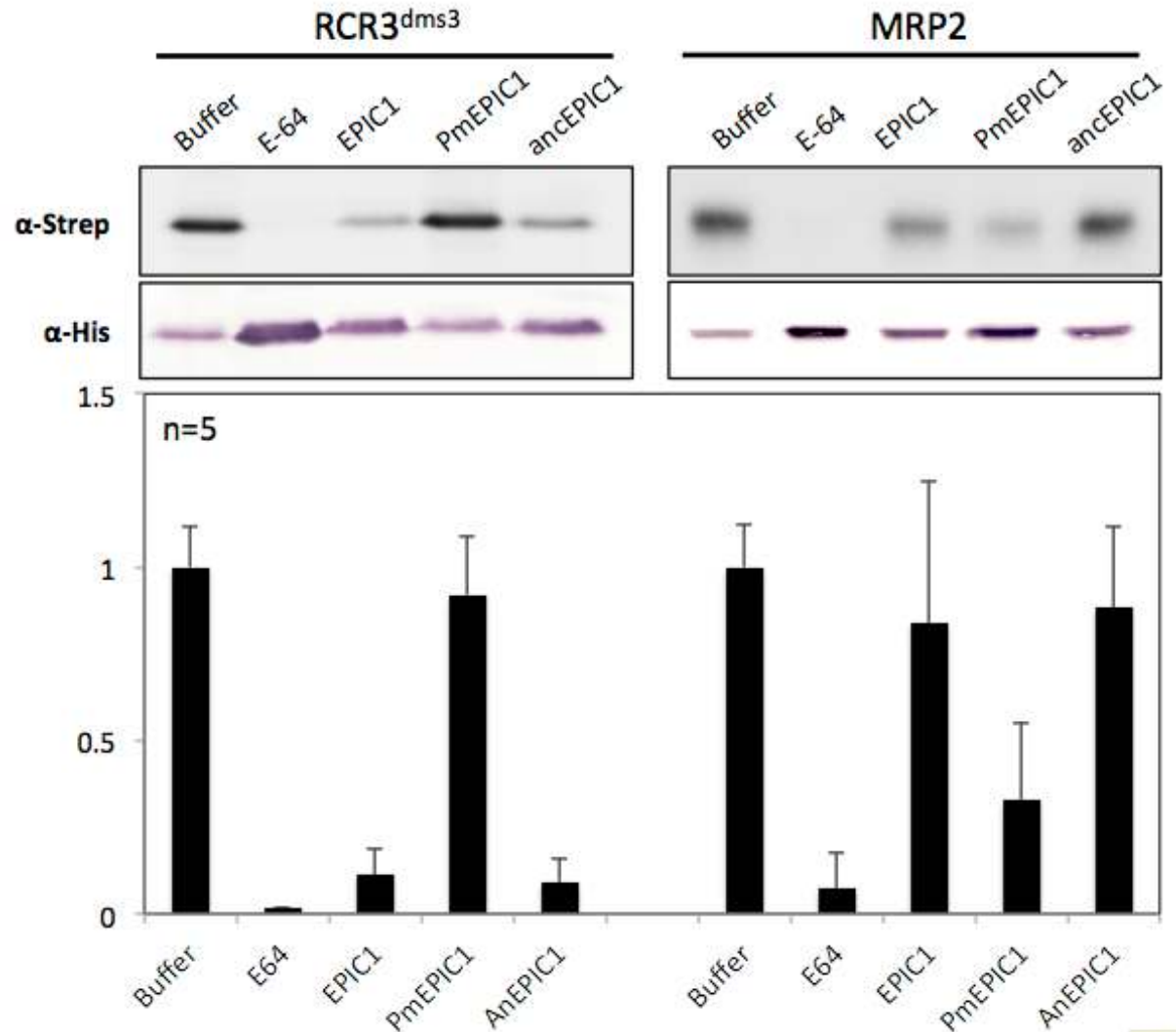
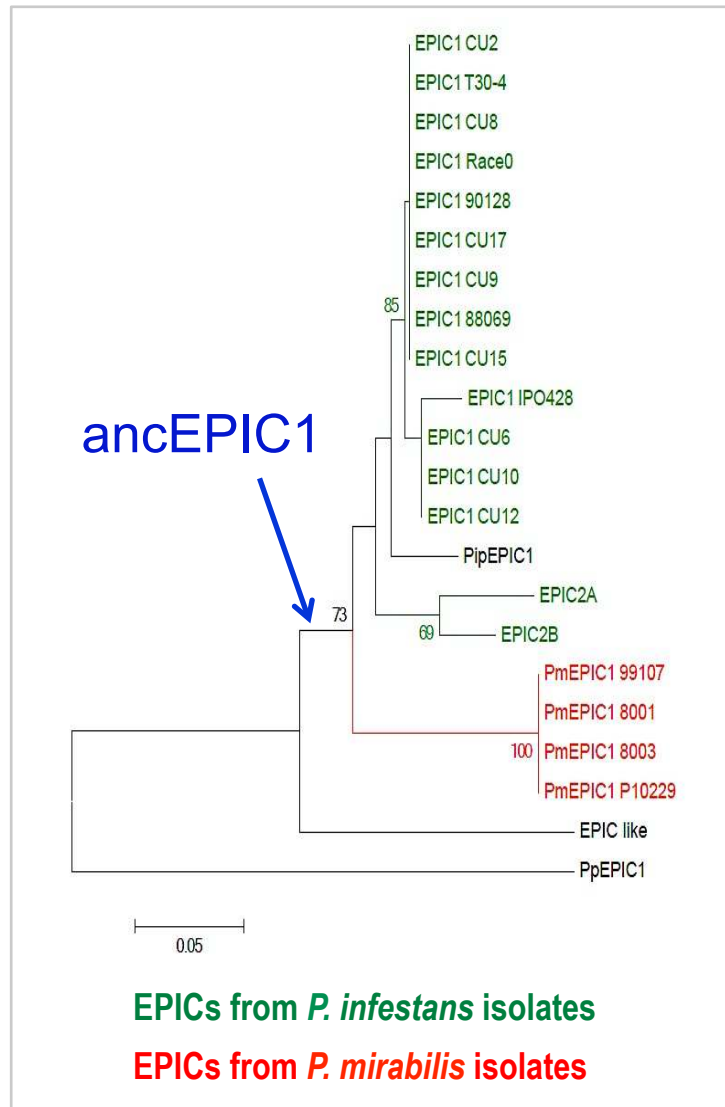
ω = nonsynonymous (dN)/ synonymous (dS)
 ω ratio calculated per branch using two-ratio model of codeml

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

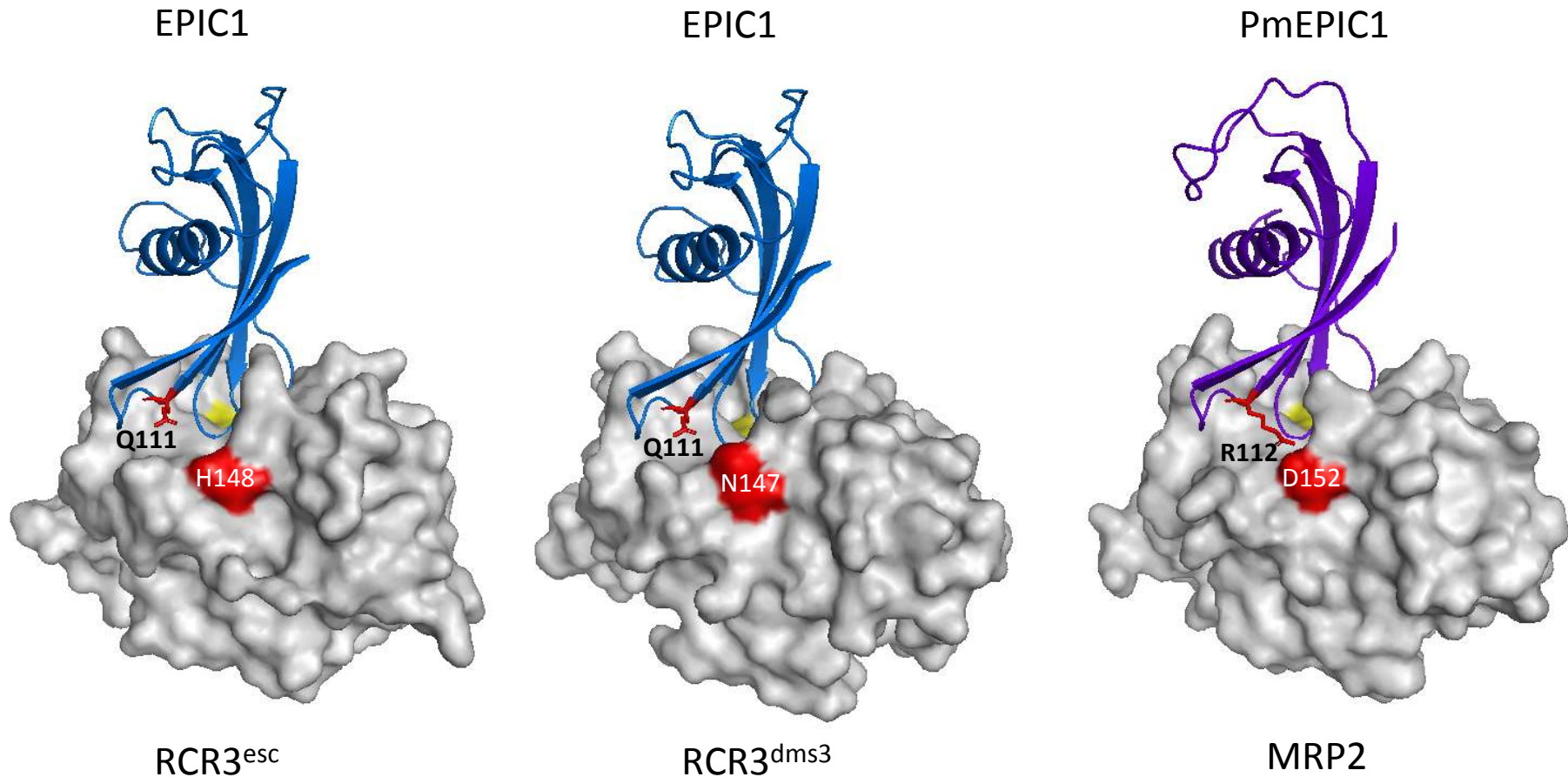
Effector adaptation and specialization to host target following jump?



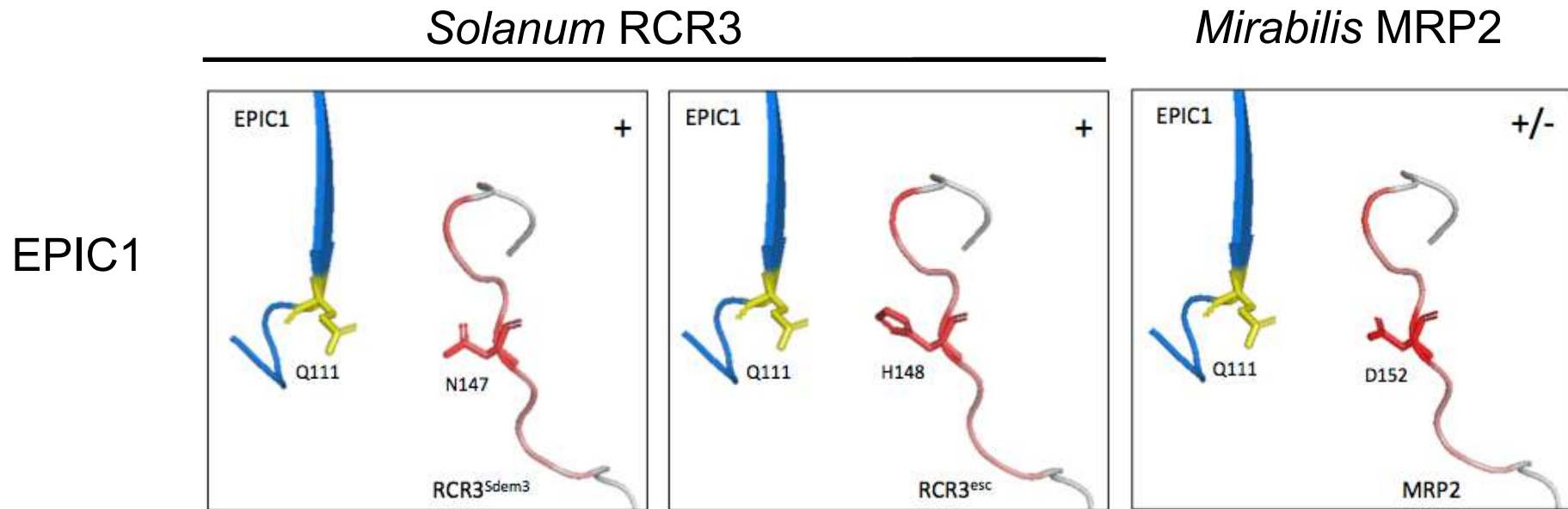
A resurrected ancestral EPIC1 can inhibit potato RCR3 but not Mirabilis MRP2



EPIC1/RCR3 predicted contact residues are polymorphic



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.

