

Genome of Irish potato famine pathogen decoded

Findings suggest a “two-speed” genomic strategy to outwit plant hosts

Norwich, UK – A large international research team has decoded the genome of *Phytophthora infestans*, the notorious organism that triggered the Irish potato famine in the mid-19th century and also threatens this year’s potato and tomato crops.

Published in the September 9 online issue of the journal *Nature*, the study reveals that the organism boasts an unusually large genome size. It is more than twice that of closely related species and has an extraordinary structure. These features appear to enable the rapid evolution of certain genes, particularly ones involved in plant infection.

“Our findings suggest a ‘two-speed’ genome, meaning that different parts of the genome are evolving at different rates,” said Professor Sophien Kamoun, head the Sainsbury Laboratory in Norwich, and co-lead author.

The pathogen can quickly adapt to new plant hosts, attacking even genetically resistant potatoes that have been painstakingly bred to fend off infection. How it can adapt so rapidly to these immune potatoes has long puzzled scientists.

“This two-speed genome structure might enable *P. infestans* to rapidly adapt to host plants while the core set of genes for the basic biology of the organism remain conserved.” said Kamoun.

P. infestans has re-emerged as a pervasive enemy of potato and tomato production, causing worldwide losses from blight exceeding £4 billion a year. Potatoes are the staple food in many countries and the most important non-cereal crop in the world.

In recent years, potato growers in the UK have been forced to increase spraying by up to 30 per cent. In Ireland, some have described this season as the worst year for blight in living memory. *P. infestans* also threatens tomato crops across much of the US.

“This pathogen has an exquisite ability to adapt and change, and that’s what makes it so dangerous,” said Dr Chad Nusbaum, co-director of the Genome Sequencing and Analysis Program at the Broad Institute of MIT and Harvard. “We now have a comprehensive view of its genome, revealing the unusual properties that drive its remarkable adaptability. Hopefully, this knowledge can foster novel approaches to diagnose and respond to outbreaks.”

Long considered a fungus, *P. infestans* is now known to be a member of the oomycetes or “water molds”, which are more closely related to the malaria parasite than to fungi. The pathogen thrives in cool, wet weather, and can infect potatoes, tomatoes and other related plants, causing a “late blight” disease that can decimate entire fields in just a few days.

To understand the genetic basis for the pathogen’s success, the researchers, led by scientists at the Sainsbury Laboratory and the Broad Institute, decoded the *P. infestans* genome. They compared it to the genomes of two close relatives: *P. sojae*, which infects soybeans, and *P. ramorum*, which prefers oak and other trees.

One of the most striking findings to emerge from these comparisons is the fact that the genome size is two and a half to four times the size of its relatives’.

But perhaps even more surprising than the genome’s large size is the source of its added bulk. There are not more genes *per se*, but a huge amount of repetitive DNA. In fact, this type of DNA accounts for about 75% of the entire *P. infestans* genome.

“Such a large amount of repetitive DNA is pretty surprising, since there is a metabolic cost to maintain it,” said Nusbaum. “As a genome biologist, I have to wonder how the organism benefits from having it.”

The researchers gained some key insights into the potential advantages of carrying this glut of repetitive DNA. They made three critical observations:

- The *P. infestans* genome is comprised of alternating repeat-rich (and gene-poor) regions and gene-dense regions;
- The gene-dense regions are shared among other *Phytophthora* species, preserved over million years of evolution, whereas the repeat-rich regions are undergoing relatively rapid changes;
- The repeat-rich regions contain fewer genes compared to other genomic regions. But those genes they do contain are ones that play crucial roles in plant infection.

Co-lead author Brian Haas, Manager of Genome Annotation, Outreach, Bioinformatics, and Analysis at the Broad Institute, says “In contrast to the well-conserved regions where most genes are found, the repeat-rich regions change rapidly over time, acting as a kind of incubator to enable the rapid birth and death of genes that are key to plant infection. As a result, these critical genes may be gained and lost so rapidly that the hosts simply can’t keep up.”

Critical genes, known as “effector” genes, can perturb plants’ normal physiology, enabling the pathogen to establish a foothold.

In subsequent paper to be published in *Plant Cell*, Kamoun and colleagues identify the function of 16 effector genes, some of which suppress the host plant’s immunity allowing the pathogen to colonise.

However, other effectors can trigger plants’ immune responses, making them prime targets for combating *P. infestans* infection.

These findings not only expand the catalogue of known *P. infestans* effector genes, they also highlight a critical subset of genes undergoing rapid turnover. Further studies of these genes will foster a deeper understanding of plant infection and help identify potential targets for fighting back.

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Paper cited:

Hass *et al.* Genome sequence and analysis of the Irish potato famine pathogen *Phytophthora infestans*. *Nature* DOI 10.1038/nature08358

About the Sainsbury Laboratory

The Sainsbury Laboratory (TSL) is a world-leading research centre focusing on making fundamental discoveries about plants and how they interact with microbes. TSL is evolving its scientific mission so that it not only provides fundamental biological insights into plant-pathogen interactions, but also delivers novel, genomics-based, solutions which will significantly reduce losses from major diseases of food crops, especially in developing countries.

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