Pmk1, which was previously shown to be crucial for appressorium development and pathogenicity. Following invasion of the first rice epidermal cell, the authors inhibited Pmk1, which prevented the invasion of adjacent epidermal cells, as indicated by the observation that the first infected cell became filled with fungal hyphae. This suggests that Pmk1 is crucial for cell-to-cell spread and invasive growth. RNA sequencing (RNA-seq) analysis revealed that the expression of 1,457 fungal genes was altered in cells in which Pmk1 was inhibited compared with cells in which Pmk1 activity was not suppressed. A subset of genes that were positively regulated by Pmk1 encode fungal effectors implicated in the suppression of plant immunity, including several Bas effectors, which might function at cell wall crossing sites. Indeed, Bas2–GFP and Bas3–GFP were not expressed in fungal cells in which Pmk1 was inhibited. Moreover, Pmk1 inhibition was accompanied by increased reactive oxygen species-dependent callose deposition.

The RNA-seq analysis also revealed that morphogenetic regulators are downregulated in the fungus during Pmk1 inhibition, including factors that regulate the dynamics of septins and filamentous (F)-actin, which form a ring-shaped structure at the base of the appressorium to promote the penetration of the rice leaf. Septin mutants did not penetrate the rice leaf efficiently, and in cases in which penetration was successful, the mutants had a decreased ability to spread between rice cells, which is consistent with a role for septins in cell-to-cell invasion by regulating the constriction of invasive hyphae of *M. oryzae*.

In sum, the results of this study reveal that the MAPK pathway controls plant tissue invasion by M. oryzae through the control of hyphal constriction and the expression of effectors to suppress plant immunity.

Andrea Du Toit

ORIGINAL ARTICLE Sakulkoo, W. et al. A single fungal MAP kinase controls plant cell-to-cell invasion by the rice blast fungus. *Science* **359**, 1399–1403 (2018)

tissue tropism with their mammalian counterpart; members of the genus *Hepacivirus* were found in the liver, whereas members of the *Picornaviridae*, *Calciviridae* and *Astroviridae* families were found in the gut.

The authors constructed phylogenetic trees that revealed the evolutionary relationships between the newly identified viruses and found that the phylogenetic history of the RNA viruses mirror that of their hosts over long evolutionary timescales, which implies that RNA viruses followed a similar evolutionary trajectory as their vertebrate hosts, co-evolving with them for millions of years. Their proposed evolutionary trajectories for RNA viruses were calibrated and supported by comparisons using dated orthologous endogenous retroviruses. Although they found that each vertebrate class is dominated by a specific set of RNA viruses, some of the viruses were found to infect multiple hosts, which indicates that in addition to co-divergence, cross species transmissions had regularly occurred and were sustained throughout evolutionary history.


Credit: Citizen of the Planet/Alamy Stock Photo

In summary, this study expands our understanding of RNA virus evolution, but with millions of animal species on Earth that have not been surveyed, we are only beginning to understand the diversity and evolution of these viruses.

Ashley York

ORIGINAL ARTICLE Shi, M. et al. The evolutionary history of vertebrate RNA viruses. *Nature*. https://doi.org/10.1038/s41586-018-0012-7 (2018)

IN BRIEF

PARASITE BIOLOGY

A dormant state within cells

Chagas disease, which is caused by *Trypanosoma cruzi*, is an important health concern owing to the lack of effective drug treatment for chronic infection. A recent study found that although drug treatment rapidly reduced the numbers of *T. cruzi* in vivo, treatment failed to completely clear the infection. The authors went on to investigate the possible involvement of dormant or non-replicating forms of *T. cruzi* to explain treatment failure. Indeed, a small fraction of amastigotes (which are the intracellular, non-motile forms of the parasite) was non-replicating during acute and chronic infections in mice. Non-replicating amastigotes differentiated into trypomastigotes, which exited host cells and infected new host cells, where they converted back into replicating amastigotes, with a small number of the progeny again arresting replication. Finally, dormant amastigotes were resistant to extended drug treatment and could re-establish an infection.

ORIGINAL ARTICLE Sánchez-Valdéz, F. J. et al. Spontaneous dormancy protects *Trypanosoma cruzi* during extended drug exposure. *eLife* **2018**, e34039 (2018)

VIRAL INFECTION

At the end of KSHV's tether

The latent viral genomes of Kaposi's sarcoma-associated herpesvirus (KSHV), known as episomes, are maintained as non-integrated circular genomes that are attached to host chromatin via tethers that comprise homodimers of latency-associated nuclear antigen (LANA) that bind with their carboxyl termini to episomal terminal repeats and with their amino termini to host chromatin. However, the architecture of these tethers in cells was not well understood. Here, the authors used stochastic optical reconstruction microscopy to visualize single tethers in cells that were latently infected with KSHV and to reveal the nanoarchitecture of full-length KSHV tethers. They showed that the folding of the viral chromatin is intrinsic to the viral episome, is independent of the cellular environment and mimics that of active chromatin. Moreover, LANA dimers are arranged in ordered clusters that are projected outward from the terminal repeat region of the viral genome.

ORIGINAL ARTICLE Grant, M. J. et al. Superresolution microscopy reveals structural mechanisms driving the nanoarchitecture of a viral chromatin tether. *Proc. Natl Acad. Sci. USA* https://doi.org/10.1073/pnas.1721638115 (2018)

PARASITE DEVELOPMENT

Assessing chromatin accessibility

How stage-specific gene transcription is orchestrated during the blood stages of the *Plasmodium falciparum* life cycle has been unclear. The authors of this study directly profiled chromatin accessibility using the assay for transposase accessible chromatin sequencing and identified ~4,000 regulatory regions in the *P. falciparum* genome that become accessible during the intraerythrocytic stages. The chromatin accessibility pattern positively correlated with the abundance of the respective mRNA transcripts. The stage-specific regulatory regions were enriched in consensus binding sequences for transcription factors belonging to the *P. falciparum* AP2 family, and DNA pull-down assays showed that members of this family as well as other histone-binding proteins bind to these regions.

ORIGINAL ARTICLE Toenhake, C. G. & Fraschka, S. A.-K. et al. Chromatin accessibility-based characterization of the gene regulatory network underlying *Plasmodium falciparum* blood-stage development. *Cell Host Microbe*. https://doi.org/10.1016/j.chom.2018.03.007 (2018)