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MARINE MICROBIOLOGY

Algal virus boosts nitrogen uptake in the ocean

Marine viruses are major sources of mortality in ocean microbial communities and, consequently, are thought to have important roles in influencing nutrient cycles. In addition to causing cell death by predation, viruses have been hypothesized to influence marine microbial ecosystems through manipulating host metabolism during infection. In a recent study, Monier *et al.* identified a host-derived ammonium transporter in the genome of an algal virus that increases the rate of nitrogen uptake by infected cells and changes how the host accesses diverse nitrogen sources during infection.

To investigate potential influences of viruses on host nitrogen uptake, the authors searched all available viral genomes for the presence of nitrogen transporter genes and found a single viral protein that was putatively involved in direct nitrogen uptake. The transporter was identified in the genome of OtV6, a large double-stranded DNA (dsDNA) virus in the *Phycodnaviridae* family. OtV6 infects *Ostreococcus tauri*, a unicellular green alga, which is the smallest known free-living non-symbiotic eukaryote on Earth, and a common member of phytoplankton communities. The viral protein sequence shares sequence and predicted structural homology with members of the Amt/Mep/Rh superfamily of proteins that transport ammonium ions across membranes and was thus named viral ammonium transporter (vAmt) by the authors.

Next, the authors confirmed that the vAmt-encoding gene was expressed during OtV6 infection by carrying out reverse transcription PCR (RT-PCR) on RNA that was isolated from infected and uninfected *O. tauri* cells, and observed that vAmt was only expressed in infected cells. To establish whether vAmt is a functional ammonium transporter, the vAmt-encoding gene was cloned and transformed into a *Saccharomyces cerevisiae* mutant that lacks native ammonium transporters. The authors found that the yeast mutant showed increased growth in medium that contained ammonium as the sole nitrogen source compared with the untransformed control, which is consistent with the hypothesis that vAmt is a functional ammonium transporter. Furthermore, the mutants that were transformed with the vAmt-encoding gene had a higher rate of uptake of radiolabelled methylammonium at low concentrations than host-encoded transporters, which suggests that vAmt could alter nitrogen uptake dynamics during OtV6 infection.

To test whether vAmt expression during OtV6 infection altered ammonium uptake rates, the authors measured radiolabelled methylammonium uptake over 16 h and observed statistically significant increases in uptake in infected cells and a depletion of ammonium concentrations 8 h post-infection. The authors also investigated the range of alternative nitrogen substrates of vAmt in *S. cerevisiae* and saw significant increases in levels of respiration when three alternative nitrogen sources were tested (D,L-α-aminobutyric acid, glucuronamide and D-mannosamine), indicating that OtV6 infection could enable the host to access diverse nitrogen sources, which would provide a nutritional benefit to the host.

To investigate the ancestry of the vAmt-encoding gene, a phylogenetic tree using protein sequences from the Amt/Mep/Rh superfamily was constructed and showed that the viral transporter is closely related to a family of *Ostreococcus* spp. ammonium transporters, leading the authors to hypothesize that the vAmt-encoding gene is derived from the host, probably through a host-to-virus horizontal gene transfer event.

In summary, these data suggest that the viral manipulation of host nutrient acquisition confers a fitness advantage to viruses and leads to changes in the physiology and ecology of phytoplankton, and consequently, marine nutrient cycles.

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> “OtV6 infection could enable the host to access diverse nitrogen sources”

ORIGINAL ARTICLE Monier, A. *et al.* Host-derived viral transporter protein for nitrogen uptake in infected marine phytoplankton. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1708097114 (2017)

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