Contribution ID: **118** Type: **Talk from invited speaker**

Diverse responses of an archaeal CBASS

Wednesday, 7 October 2026 09:00 (30 minutes)

Many bacterial cyclic-oligonucleotide-based antiphage signalling systems (CBASS) defend against viral infections by depleting cellular NAD+ levels, eventually leading to dormancy or death. This abortive infection strategy is beneficial in stopping fast lytic infections, as cells die before spreading the virus to neighbouring cells. However, in chronic viral infections, which often occur in archaea, abortive infection could be detrimental, as the cost of immunity may outweigh that of infection. We study an archaeal CBASS that is expressed in the model species *Haloferax volcanii* DS2 and *Haloferax gibbonsii* LR2-5. We show that the system provides protection to *H. gibbonsii* against a lytic tailed haloarchaeal virus HFTV1 by depleting NAD+, and causing premature lysis. H-CBASS2 is also activated to a lower degree during infection with the chronic, non-lytic, virus HFPV-1, and promotes virus clearance after several passages without killing host cells. Cells that cleared the HFPV-1 infection became substantially more resistant to subsequent infections, due to accumulated mutations in envelope-associated proteins. Cell death by extreme NAD+ depletion only occurred after a very long incubation with HFPV-1. These findings suggest that the magnitude of H-CBASS2 response is somehow tuned to the infection type and can benefit the host during non-lytic infections, potentially explaining why such systems are relatively common in archaea.

Reference #1

Reference #2

Reference #3

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Session Classification: Novel anti-viral defence systems

Track Classification: Novel anti-viral defence systems