Archaeal virus entry and egress

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Abstract

Archaeal viruses display a high degree of structural and genomic diversity. Few details are known about the mechanisms by which these viruses enter and exit their host cells. Research on archaeal viruses has lately made significant progress due to advances in genetic tools and imaging techniques, such as cryo-electron tomography (cryo-ET). In recent years, a steady output of newly identified archaeal viral receptors and egress mechanisms has offered the first insight into how archaeal viruses interact with the archaeal cell envelope. As more details about archaeal viral entry and egress are unravelled, patterns are starting to emerge. This helps to better understand the interactions between viruses and the archaeal cell envelope and how these compare to infection strategies of viruses in other domains of life. Here, we provide an overview of recent developments in the field of archaeal viral entry and egress, shedding light onto the most elusive part of the virosphere.

Keywords: archaeal virus; virus–host interaction; virus entry; viral egress; archaeal cell surface; infection mechanism

Introduction

Viruses are infectious agents that infect host cells to reproduce. Microbial viruses are the most abundant biological entities in the biosphere, outnumbering cellular life by at least a factor of 10, and can be found everywhere on earth (Suttle 2007, Roux et al. 2018). Consequently, viruses play an important role in the complex dynamics of ecosystems and are key drivers of evolution (Suttle 2013). Archaea, like bacteria and eukaryotes, are susceptible to viral infection. Archaeal viruses are significantly undersampled compared to bacterial and eukaryotic viruses. This is likely the consequence of multiple factors, such as challenges in cultivation of archaea (with many uncultured clades) and challenges in viral selection (as many archaea do not form lawns under laboratory conditions, limiting plaque-based assays). In addition, it is generally observed that the fraction of viruses with nonlytic infection cycles is higher in archaea compared to bacteria, thus also necessitating more labour intensive isolation methods, such as enrichment cultures. Archaeal viruses remain highly elusive, and few have been characterized in detail (Pietilä et al. 2014, Atanasova et al. 2015, Krupovic et al. 2018, Baquero et al. 2021b, Wirth and Young 2020).

Phylogeny and genome structure of archaeal viruses

Archaeal viruses are different from bacterial and eukaryotic viruses and have a unique set of viral morphologies. They are characterized by a high degree of sequence diversity and the functions of their gene products are largely unknown. To address this lack of knowledge, the study of viral isolates is of major importance (Prangishvili et al. 2017). Based on their morphology, archaeal viruses can be distinguished into those that are archaea-specific (unique types) and those that resemble viruses of other domains of life (i.e. cosmopolitan types). (Pietilä et al. 2014, Iranzo et al. 2016). Archaeal viruses show high diversity with over 45 classified families and 135 species recognized by the International Committee on Taxonomy of Viruses (https://ictv.global/). Metagenomics have also provided helpful tools in the identification of novel archaeal viruses. Culture-independent genomic studies on samples from extreme environments have uncovered several new archaeal virus genomes, such as those of six Asgard viruses (Medvedeva et al. 2022, Rambo et al. 2022, Tamarit et al. 2022). Furthermore, several viruses not belonging to any of the known families representing new virus types, have been uncovered in this manner (Laso-Pérez et al. 2023, Medvedeva et al. 2022, Molnár et al. 2020, Iranzo et al. 2016, Dávila-Ramos et al. 2019, Liu et al. 2019).

The known archaea-specific viruses, which often infect archaea of the Thermoproteota phylum, are morphologically highly diverse. They can be found in the shapes of coils (*Spiraviridae*), ovoids (*Guttaviridae*), spindles/lemons (*Bicaudaviridae*, *Fuselloviridae*, *Halspiviridae*, *Thaspiviridae*, and *Itzamnaviridae*), bottles (*Ampullaviridae*), rods (*Ahmunviridae* and *Clavaviridae*), spheres (*Globuloviridae*), and elipsoids (*Ovaliviridae*) (Häring et al. 2004, 2005b,c, Mochizuki et al. 2010, 2011, 2012, Wang et al. 2018, Kim et al. 2019, Laso-Pérez et al. 2023, Yeats et al. 1982, Martin et al. 1984, Nadal et al. 1986, Bath and Dyall-Smith 1998).

Furthermore, archaeal filamentous virions representing the families of *Rudiviridae* or *Lipothrixviridae* superficially resemble bacterial and eukaryotic filamentous single-stranded (ss) DNA and ssRNA viruses but have double-stranded (ds)DNA genomes (Krupovic et al. 2018). In fact, all the discovered archaeal viruses have a DNA genome, which is mostly double-stranded and either a linear or circular molecule. To date, only members of the *Spiraviridae* and *Pleolipoviridae* families are known to have ssDNA (Pietilä et al. 2009). Furthermore, archaeal RNA viruses are yet to be discovered, but metagenomic studies hint at their existence (Bolduc et al. 2012, Le Lay et al. 2023).

In contrast, most isolated viruses infecting members of the Euryarchaeota phylum, such as methanogens (Ngo et al. 2022) and haloarchaea (Roine and Oksanen 2011), are cosmopolitan viruses that resemble viruses of bacteria. This might in part be attributed to host cultivation and virus isolation bias. For instance, electron microscopy analyses of samples from haloarchaea rich environments, such as the Dead Sea and Lake Retba in Senegal, showed the most encountered virus to be spindle-shaped (Oren et al. 1997, Sime-Ngando et al. 2010).

Cosmopolitan archaeal viruses include head-tailed viruses with a siphovirus morphotype (the families: *Anaeroviridae*, *Druskaviridae*, *Graaviviridae*, *Haloferuviridae*, *Leisingerviridae*, *Madisavirida*, *Saparoviridae*, *Suolaviridae* and *Vertoviridae*), myovirus morphotype (*Hafunaviridae*, *Halomagnusviridae*, *Pyrstöviridae* and *Soleiviridae*), or podovirus morphotype (*Shortaselviridae*) (Pietilä et al. 2013a,b, Luk et al. 2014, Wolf et al. 2019, Liu et al. 2021, Pagaling et al. 2007, Atanasova et al. 2012, Sencilo et al. 2013). The similarity in appearance is reflected by their genomes, as their gene products are partially homologous to phage structural proteins, proteins involved in virion maturation and genome packaging (Krupovič et al. 2010). In addition, icosahedral internal membrane-containing archaeal viruses of the families *Turriviridae*, *Simuloviridae* and *Sphaerolipoviridae* resemble bacteriophages e.g. of the families *Corticoviridae* or *Tectiviridae* (Abrescia et al. 2012, Demina et al. 2017, Liu et al. 2023, Wirth et al. 2011). Despite the similar appearance of cosmopolitan archaeal viruses, around 80% of the archaeal viral genes encode for proteins for which no homologues exist within bacterial or eukaryotic viruses (Munson-Mcgee et al. 2018). Nonhomologous proteins could be part of unique interaction mechanisms between archaeal viruses and their hosts. Viral entry and egress mechanisms have been intensively studied in bacterial and eukaryotic viruses but remain largely elusive for archaeal viruses (Prangishvili et al. 2017, Baquero et al. 2021a).

The archaeal cell envelope as a barrier for viral entry

The cell envelope constitutes a major barrier for viral infection. Most viruses have to cross this barrier twice during its infection cycle; upon entry and egress. The archaeal cell envelope consists of ether-linked lipids with a glycerol-1-phosphate backbone, whereas, in general, bacteria and eukaryotes contain ester-linked lipids with a glycerol-3-phosphate backbone (Villanueva et al. 2021). Furthermore, archaea lack a peptidoglycan layer i.e. murein, which is an essential and almost ubiquitous cell wall component in bacteria (Vollmer et al. 2008). Instead of murein, many archaea are encapsulated by a paracrystalline protein surface layer (S-layer), consisting of one or two repeating (glyco)proteins (Ilk et al. 2011, Rodrigues-Oliveira et al. 2017). S-layer proteins, in contrast to bacterial peptidoglycan, can be highly diverse between species. In addition some archaeal cell envelopes contain pseudomurein, methanochondroitin, or several other cell-wall components (Albers and Meyer 2011).

Archaea express various filamentous surface structures, embedded within the S-layer. Most of these filaments that have been characterized are homologous to bacterial type IV pili (Chaudhury et al. 2018). A prominent example is the archaeal flagellum (archaellum), a rotary swimming propeller that consists of helically organized archaellins, which are structurally similar to type IV pilins (Makarova et al. 2016, Poweleit et al. 2016, Daum et al. 2017, Meshcheryakov et al. 2019, Gambelli et al. 2022). Type IV pili are used for initial cell contact by some bacteriophages (Tittes et al. 2021, Mahillon et al. 2023). Other archaeal cell surface filaments include type IV adhesive pili, type IV UV-inducible pili, and nontype-IV filaments, such as cannulae, hami, fimbriae, threads, and protein sheaths (Albers and Meyer 2011, Chaudhury et al. 2018, Klingl et al. 2019, Tittes et al. 2021, Gaines et al. 2022).

The structural organization of each virus particle has evolved to suit its purpose of genome protection, transport, host recognition and genome delivery (Poranen et al. 2002, Elois et al. 2023). The striking structural diversity of archaeal viruses indicates that their host recognition and entry mechanisms might be very diverse as well (Quemin and Quax 2015). On the other hand, it is an open question whether cosmopolitan archaeal viruses have similar entry and egress mechanisms as their bacterial or eukaryotic counterparts. The interaction of archaeal viruses with the host cell surface has been studied mainly for a selected set of viruses infecting members of the Thermoproteota and Euryarchaeota. As a consequence, only a few host receptors used for viral adsorption have been identified and only a handful of egress mechanisms have been described so far. Over the last decade, various studies using genetic approaches and imaging techniques, such as whole cell cryo-electron tomography (cryo-ET), have provided new insights into the shrouded archaeal virosphere (Quemin et al. 2020, Quemin et al. 2013, Daum et al. 2014, Li et al. 2022, Rambo et al. 2022). Here, we provide an update of the recent scientific developments of archaeal virus entry and release. Characteristics of viral families discussed in this paper can be found in Table S1 (Supporting Information).

Virus entry into the archaeal cell

The first step of the infectious cycle of an archaeal virus is its entry into the cell, which includes adsorption to the host cell and penetration of its envelope. The viral host range is determined by the successful viral recognition of host cell receptors (Maginnis 2018). Recognition depends on the characteristics of the host cell envelope, in terms of accessibility and number of receptors that allow the virion to adsorb to its host (Poranen et al. 2002). Once the first contact between a virus and a cell surface receptor has occurred, reversible binding takes place, followed by irreversible binding to the same or another cell surface receptor (Bertozzi Silva et al. 2016).

After virus adsorption, the virion undergoes an irreversible conformational change, which eventually causes the injection/release of the viral genome into the host cell (Molineux and Panja 2013). In bacteriophages, three main strategies of genome release into the host cell have been identified including (i) genome release through an icosahedral capsid vertex, (ii) genome injection with the virion capsid left on the cell envelope, and (iii) membrane fusion (Poranen et al. 2002). In comparison, information on archaeal viral entry is very scarce. However, recent studies have significantly increased the available knowledge and resulted in the identification of several archaeal viral receptors.
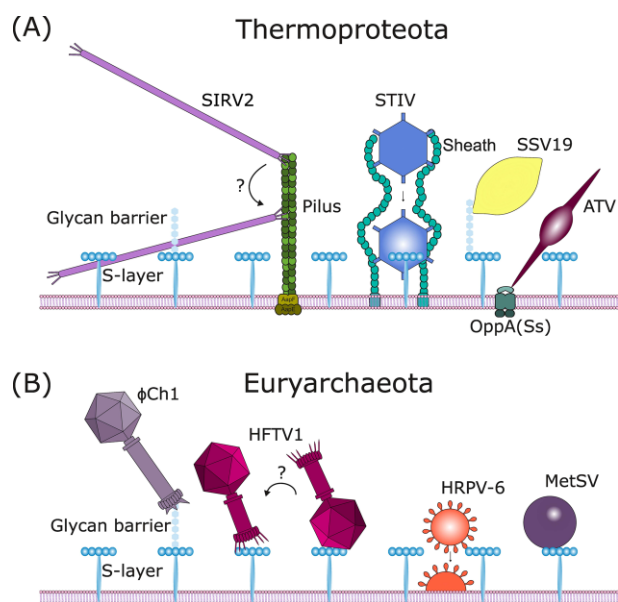
Figure 1. Schematic depiction of virus interactions with the archaeal cell envelope at virus entry. (A) Host receptors of viruses infecting Thermoproteota. (B) Host receptors of viruses infecting Euryarchaeota.

Filamentous surface structures as sites for archaeal virus host cell recognition

Archaeal filamentous surface structures can serve as (primary) attachment sites for archaeal viruses. For some filamentous and rod-shaped viruses, terminal structures have been shown to mediate contact between the capsid and host cell surface filaments (Quemin et al. 2013, Hartman et al. 2019, Rowland 2020). Acidianus filamentous virus 1 (AFV1) was reported to attach to host filaments via claw-like terminal structures (Bettstetter et al. 2003). The terminal claws on both ends of the rod-shaped virion seem to be functionally identical and transform to a closed conformation to keep the virus attached (Bettstetter et al. 2003). Similarly, filamentous *Sulfolobus islandicus* rod-shaped virus 2 (SIRV2) binds with its three terminal tail fibres to long surface filaments of *S. islandicus* (Fig. 1A). Each virion can attach to two filaments at a time (each at one terminus of the particle). In *Sulfolobus*, the receptor of SIRV2 was shown to be encoded by the sso3139–3141 operon, which is thought to include a part of the membrane-bound complex of the adhesive type IV pilus (Deng et al. 2014). Once the virus encounters the cell surface, the capsid appears to break up into fragments, possibly as a consequence of DNA injection (Quemin et al. 2013). It is unclear how the virus travels from the surface filaments to the host cell surface (Quemin et al. 2013). Albeit not observed yet, it is conceivable that pilus retraction upon virus binding may play a part in this process. The entry mechanisms for the filamentous tristomaviruses remain unknown, though it is possible that their terminal filaments are also involved in viral attachment (Rensen et al. 2016, Wang et al. 2020).

Sulfolobus turreted icosahedral virus (STIV) binds directly to surface filaments of unknown function by its turret protein C381 (Hartman et al. 2019). The turret proteins decorate the vertices of the icosahedral capsid and cryo-ET showed that single virions can interact with multiple host filaments. STIV 'petal' protein C557 is part of the vertex complex and has been proposed to be involved in virus–host attachment (Maaty et al. 2006, Khayat et al. 2010). C557 transiently binds the turret protein C381 of newly synthesized virions, blocking their ability to rebind to pili of the infected

cell post egress, and therefore acting de facto as a maturation factor (Hartman et al. 2019). To date, it is unknown how STIV moves via the filaments to the cell surface for genome delivery, but it has been hypothesized that entanglement with multiple pili might eventually lead to contact with the cell surface (Hartman et al. 2019).

Lemon-shaped fuselloviruses likely also use tail structures to bind the host receptor. In case of Sulfolobus spindle-shaped virus 9 (SSV9), type IV pili serve as receptors. Deletion of both pilin genes *pilA1* and *pilA2* encoding the major pilins of *the S. islandicus* adhesive pilus provides resistance towards SSV9 infection (Rowland 2020, Rowland et al. 2020). Nonetheless, SSV9 still adsorbs to the cell surface, suggesting that there is another secondary receptor involved in the entry of the virus. It is not yet known if other members of the *Fuselloviridae* also bind surface filaments. Since spindle-shaped viruses are generally equipped with long terminal tail structures (Palm et al. 1991, Hong et al. 2015, Prangishvili et al. 2018, Kim et al. 2021, Wang et al. 2022), it is conceivable that these are used to contact the host. For example, it has been suggested that Acidianus spindle-shaped virus 1 (ASV1) and *Sulfolobus* spindle-shaped virus 6 (SSV6) use thick, crown-like filament bundles to attach to their hosts. Nevertheless, evidence for this hypothesis is currently lacking, and the host cell receptors remain to be identified (Redder et al. 2009, Quemin and Quax 2015).

Viral binding to the archaeal cell surface

Several filamentous viruses have also been reported to directly interact with the cell surface instead of cell surface filaments. This is the case for Acidianus filamentous virus 2 (AFV2), which employs 'brush-like' filaments bundles for its primary host interaction (Häring et al. 2005a). Similarly, *S. islandicus* filamentous virus (SIFV) uses 'mop-like' extensions to bind the cell wall (Arnold et al. 2000). *Sulfolobus* spindle-shaped virus 19 (SSV19) approaches *Sulfolobus sp.* E11-6 via its tail, which contains a protein domain with high sequence similarity to the endo-mannase domain of *Bacteroides thetaiotaomicron*, suggesting an evolutionary relationship (Han et al. 2022). *Sulfolobus* species are known to possess a highly glycosylated S-layer (Rodrigues-Oliveira et al. 2017). Therefore, it is proposed that SSV19 binds to and degrades the mannose residues of the S-layer glycoproteins triggering genome delivery similar to bacteriophages, such as P22 (Han et al. 2022). It is noteworthy that no enzymes with hydrolytic activity for S-layer degradation were identified yet in archaeal viruses. Acidianus two-tailed virus displays a predicted AAA ATPase, which interacts with the oligopeptide binding protein OppA(Ss) during surface binding. OppA(Ss) is part of a putative ABC-type transporter system and is expressed on the cell surface of *Sulfolobus solfataricus* (Erdmann et al. 2011).

Archaeal viruses with a head–tail morphology share a common evolutionary history with tailed dsDNA bacteriophages (Hartman et al. 2019). Viral species from both groups of viruses are placed in the class *Caudoviricetes* (Krupovic et al. 2011, Turner et al. 2023). It is likely that the entry and genome injection mechanisms of tailed archaeal viruses resembles those of their tailed bacteriophage counterparts (Tittes et al. 2021). Usually, bacteriophage tail fibres establish the initial interaction with the host cell. It has been shown that in several bacteriophages, such as T-even bacteriophages, that changes in the tail adhesin coding genes influence the host range (Riede et al. 1987, Tétart et al. 1996, 1998, Trojet et al. 2011). Similarly, mutations in the tail fibre genes of the archaeal tailed hafunaviruses probably determine their broad host range, possibly allowing them to use different cell surface receptors as their binding sites (Liu et al. 2021).

The archaeal tailed virus ϕCh1 binds to sugar moieties on its haloalkaliphilic host *Natrialba magadii* via its tail fibres (Witte et al. 1997, Klein et al. 2012; Fig. 1B). Similarly, Haloferax tailed virus 1 (HFTV1) binds to the S-layer of its euryarchaeal host *Haloferax gibbonsii* (Schwarzer et al. 2023). HFTV1 appears to absorb to the cell surface in two ways, via its tail or via its icosahedral head. A 50% frequency of both binding modes suggests a possible sequence of binding events (Schwarzer et al. 2023). The sequence of *H.gibbonsii* LR2-5 escape mutants showed alterations in the gene *HfgLR_11210*, which encodes for one of the two S-layer proteins. Hence, this glycoprotein probably displays a binding site for HFTV1 (Schwarzer et al. 2023). Adsorption of HFTV1 to the host surface occurs very fast (1.8 $\times$ 10^{-9} ml/min), compared to other haloarchaeal viruses (Schwarzer et al. 2023). After an initial absorption via the head, the viruses are hypothesized to reorientate and adsorb via their tail fibres, as described for some tailed dsDNA bacteriophages (Bertozzi Silva et al. 2016, Schwarzer et al. 2023). In another study, the alteration of one of the two S-layer protein genes in *Halorubrum lacusprofundi* resulted in resistance to *Halorubrum*-tailed virus-deep lake variant 1 (HRTV1-DL1). Additionally, the adsorption competence of the virus was reduced, indicating that one of the altered S-layer protein genes encodes the primary receptor for adsorption (Mercier et al. 2023).

Tailless archaeal viruses with an icosahedral capsid possess vertex proteins (*Chaacviridae*, *Portogloboviridae*, *Simuloviridae*, *Skuldviridae*, *Sphaerolipoviridiae*, and *Turriviridae*) (Jäälinoja et al. 2008, Veesler et al. 2013, Demina et al. 2017, Santos-Pérez et al. 2019). In combination with spike proteins, they form the receptor binding complex that is involved in host cell recognition and attachment (Viney 2001). Haloarcula californiae icosahedral virus 1 (HCIV-1), Haloarcula hispanica icosahedral virus 2 (HHIV-2), and virus SH1 share multiple genes with high sequence similarity, but differ greatly in their genes encoding for vertex complexes (Jaakkola et al. 2012, Demina et al. 2016, 2017). For instance, SH1 and HHIV-2 with structurally different host-recognition complexes infect *H. hispanica* (Jaakkola et al. 2012), preventing development of host resistance towards infection, as described for other closely related viruses (Saren et al. 2005). The receptors for HCIV-1, SH1, and HHIV-2 attachment remain unknown (Jaakkola et al. 2012, Demina et al. 2016). Similarly, closely related turriviruses STIV and STIV2 have different vertex complexes for host recognition (Happonen et al. 2010).

The pleomorphic archaeal viruses (*Pleolipoviridae*) have a conserved block of colinear core genes including the gene encoding the spike protein (Pietilä et al. 2012, Demina and Oksanen 2020). The spike proteins are embedded in the virion membrane and protrude from the virion surface.These spike proteins are used to bind the host cell, resulting in membrane fusion (El Omari et al. 2019). *In vitro* virus–liposome fusion assays based on dequenching of fluorophore-labelled virions (Bignon et al. 2022) showed the interaction of Halorubrum pleomorphic virus 6 (HRPV-6) with the host S-layer and subsequent membrane fusion (Bignon et al. 2022). It was suggested that a protein with a PGF-CTERM sorting-domain in the S-layer might be the trigger for membrane fusion, but only in the presence of magnesium ions. Similarly, the spike protein VP5 of Halorubrum pleomorphic virus 2 (HRPV-2) acts as a trigger for membrane fusion (El Omari et al. 2019). The fusion can be triggered naturally or by heating, which might indicate that structural changes induced by partial denaturation might be the key for membrane fusion (El Omari et al. 2019).

Most identified viral receptors stem from *Sulfolobales* and haloarchaea. One exception is the methanogenic archaeal virus Methanosarcina spherical virus (MetSV), which attaches to its host *Methanosarcina mazei* via the S-layer or S-layer-associated proteins (Weidenbach et al. 2017, Gehlert et al. 2022).

Viral binding of archaeal receptors: what have we learned?

The euryarchaeal viral receptors that were identified recently are mainly the S-layer proteins (Hartman et al. 2019, Bignon et al. 2022, Han et al. 2022, Mercier et al. 2023, Schwarzer et al. 2023), while the majority of identified receptors for viruses infecting members of the Thermoproteota concern filamentous surface structures. Around 10% of bacterial viruses use filamentous surface structures as primary attachment sites (Zhang et al. 2020). Based on the limited number of currently identified archaeal virus receptors, the use of filamentous surface structures by archaeal viruses seems more pronounced in comparison with bacteriophages. However, this is mainly the case for viruses infecting members of the Thermoproteota phylum, whereas euryarchaeal viruses seem to prefer binding to the S-layer. If this division is a general trend, or based on the low number of identified receptors, will become clearer through the identification of more receptors.

In general, dedicated capsid structures, such as turrets or spikes at the archaeal virion surface are involved in host cell attachment as described e.g. for sphaerolipoviruses or pleolipoviruses (Demina et al. 2016, 2017, Pietilä et al. 2016, Bignon et al. 2022).

For the majority of archaeal viruses, it is still a mystery how they overcome the archaeal S-layer. Many bacteriophages harbour tail proteins that are responsible for cell wall degradation (Nobrega et al. 2018, Leprince and Mahillon 2023). Depolymerases degrade polysaccharide chains and ectolysins degrade peptidoglycan. For instance, *Escherichia coli* T4 phage spike protein gp5 is a lysozyme, which hydrolyses peptidoglycan (Arisaka et al. 2003). With the discovery of the cell-wall degrading enzyme in the structure of SSV19, this raises the question, if more archaeal virions have enzymatic activities to facilitate receptor binding on the host surface, as it is the case for various viruses of bacteria (Han et al. 2022).

Mechanisms of genome delivery

Once the virus has irreversibly adsorbed to the host cell surface, the genome is transferred into the cell cytoplasm. Enveloped viruses, including members of the family *Pleolipoviridae*, likely fuse with the host membrane and thereby release their genome directly into the cytoplasm (Pietilä et al. 2016, Bignon et al. 2022).

Nonenveloped virions typically remain on the outside of the host cell or are disassembled during genome delivery (Kalia and Jameel 2011, Xu and Xiang 2017). Several hypotheses on genome transfer exist for nonenveloped viruses with an internal lipid membrane. The tailless Enterobacteria phage PRD1 (PRD1) forms a membrane tube from its internal membrane vesicle, along which a linear dsDNA genome can be injected into the host cytoplasm (Peralta et al. 2013). Since the archaeal viruses SH1, HHIV-2, HCIV-1, and MetSV share a similar virion architecture, with an internal lipid membrane and a linear dsDNA genome, they conceivably could follow the same genome delivery strategy as PRD1 for genome delivery (Bamford et al. 2005, Gehlert et al. 2022). For STIV, the domain 2 of the turret protein C381 shows highest sequence similarity to the knob domain of the podophage HS1 needle tip, which is likely involved in DNA ejection. Hence, the C381 protein might be involved in the first adsorption step or genome release (Hartman et al. 2019).

The tailed archaeal viruses probably follow similar mechanisms for genome transfer as their bacterial counterparts. The viral genome is tightly packed to fit into the icosahedral capsid of the tailed bacteriophages (Kalia and Jameel 2011, Molineux and Panja 2013). The viral DNA packaging and release is controlled via the portal complex within the viral capsid. Conformational changes allow for the opening or closure of this portal (Molineux and Panja 2013). The packaged DNA is dehydrated, causing high osmotic pressure inside the capsid. Upon infection, the portal opens, and the viral genome is fully transferred by the ensuing hydrodynamic flow forces. However, according to the continuum model, a secondary force is required for complete ejection (Molineux and Panja 2013). For spindle-shaped His1 virus of *H. hispanica*, the dsDNA ejection was measured in single-molecule experiments using fluorescence microscopy. The DNA ejection velocity of 144 ± 72 kbp/s is comparable to phage DNA ejection velocities, such as T7 phage with 140 kbp/s (Kemp et al. 2004, Hanhijärvi et al. 2013). The ejection process could be induced by external osmotic pressure created by higher concentration of polyethylene glycol, magnesium, or sodium, supporting the continuum model (Hanhijärvi et al. 2013). Presumably upon receptor binding and subsequent initiation of genome release, the virion transitions from a spindle to a tubular structure. It has been proposed that the mechanics of this transformation facilitate genome ejection (Hong et al. 2015).

The mechanism of genome ejection for archaeal viruses binding to host surface filaments is yet unknown. However, comparable to bacteriophages, archaeal viruses might take advantage of the retraction force of pili or other cellular filaments to approach the cell surface, attach to it and deliver their genome. For instance, ssRNA phage MS2 infects *E. coli*, where the F-pilus retraction force brings the phage that initially binds to the side of the pili into close proximity of the cell surface. The complete retraction of the pilus leads to viral genome delivery (Harb et al. 2020).

Egress of progeny virus from the host cell

The final stage in the viral infection cycle is the release of newly synthesized virions from the host cell. Egress of viral progeny can either result in complete cell lysis or continuous production of virus particles without inflicting obvious harm on the host cell (Bettstetter et al. 2003, Svirskaitė et al. 2016). For archaeal viruses, only a few egress mechanism have been described in detail (Bize et al. 2009, Brumfield et al. 2009, Daum et al. 2014, Baquero et al. 2021a, Quemin et al. 2016, Liu et al. 2017, Wang et al. 2018). Most of the viruses with known egress mechanisms infect members from the order *Sulfolobales* from the phylum Thermoproteota. Nevertheless, these mechanisms are highly versatile and some display unique features that have not been observed among bacterial and eukaryotic viruses. In addition, the identification of gene products associated with viral progeny release is also in its infancy and almost no similarity has been observed in sequences compared to known proteins associated with viral release in bacteria and eukaryotes.

Viral release by virus associated pyramids

The best studied egress mechanism among archaeal viruses is egress by virus-associated pyramids (VAPs). This egress mechanism has been described only for archaeal viruses. Thus far, VAPs have been described for STIV (Brumfield et al. 2009), SIRV2 (Bize et al. 2009, Quax et al. 2011), ovoid-shaped ovalivirus SEV1 (Wang et al. 2018), and SIFV (Baquero et al. 2021a). All these viruses infect
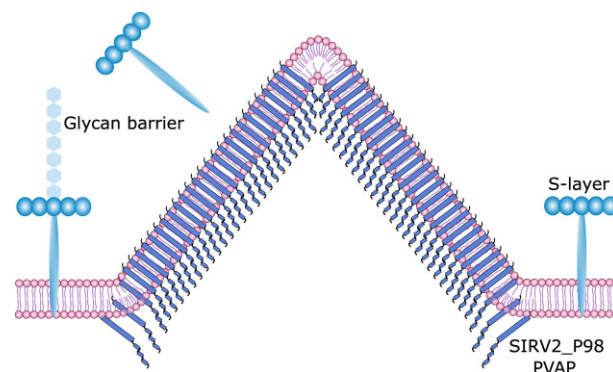

Figure 2. Schematic of potential VAP formation on a cell membrane by recruitment of PVAP subunits. PVAPs are depicted with their predicted N-terminal transmembrane domain and three C-terminal α-helices.

members of the hyper thermophilic and acidophilic *Sulfolobales* from the phylum Thermoproteota.

VAPs were first discovered and characterized in STIV and SIRV2 infected cells (Bize et al. 2009, Brumfield et al. 2009). In terms of morphology and sequence, these viruses are significantly divergent, however, their VAP-mediated egress mechanism is analogous. VAPs form large, usually 7-fold symmetric, hollow pyramidal-structures, which are embedded in the host cell membrane. These VAPs, which can be up to a few hundred nanometres in diameter, grow outward and penetrate the S-layer. At the final stage of the infection cycle, the leaflets of the VAPs open, thus forming apertures through which the progeny virus can egress from the host (Daum et al. 2014, Quax and Daum 2018). VAPs consist of hundreds of copies of a single, 10 kDa protein, dubbed protein of virus-associated pyramid (PVAP; protein C92 in STIV and P98 in SIRV2).

Lacking a predicted SEC signal sequence, PVAP is thought to be expressed as a soluble protein and to insert into the membrane upon a conformational change. The first step of VAP assembly likely entails the oligomerization of PVAP into a heptameric ring, which then recruits further subunits that assemble into the seven facets of the VAP (Fig. 2). Upon reaching a critical diameter of ~150 nm, the structure starts to open from the tip, leading to an unzipping of the seven seams, until the VAP has fully unfolded. The seven leaflets of an open VAP are usually curled outward, while in the closed VAP the facets are straight. This suggests that VAPs may, at least partially, be driven by mechanical tension, which is stored in the closed VAP and finally released during the unfolding process (Daum et al. 2014). Heterologous overexpression of PVAP in both *E. coli*- and yeast-yielded stable and intact pyramid structures. VAPs formed in the inner cell membrane of *E. coli* and in all intracellular membranes in the case of yeast. Although the VAPs appear to be able to assemble in different membrane environments, opening was not observed in the bacterial or eukaryotic hosts, suggesting that VAP opening may require a host-specific trigger (Quax et al. 2011, Daum et al. 2014).

Interestingly, VAPs from SEV1 have a 6-fold symmetry and produce apertures of around 250 nm in diameter, and are thus larger than those of STIV and SIRV2. However, the VAP protein subunit in SEV1 has so-far not been identified, likely due to low sequence homology (Wang et al. 2018). Large six-sided VAPs with a diameter of 220 nm, have also been observed for SIFV. In this case, the VAP is formed of multiple copies of the 10-kDa protein GP43. Heterologous overexpression of GP43 in *E. coli* yields stable pyramid structures in the membrane comparable to STIV and SIRV2

(Baquero et al. 2021a). Although pyramids are structurally similar, GP43 shares no homology with the PVAPs of STIV and SIRV2, nor with any proteins of SEV1. Homologous PVAP proteins have been discovered among all characterized lipothrixviruses of the genera *Betalipothrixvirus* and *Deltalipothrixvirus*, suggesting that this egress mechanism is conserved among these groups of viruses (Baquero et al. 2021b). In addition, six-sided pyramid structures have been observed on the surface of other species belonging to the phyla of Thermoproteota (Bize et al. 2008, Rensen et al. 2015). Induction of temperate viruses from *Pyrobaculum oguniense* cells by UV radiation-yielded six-sided pyramidic structures on their cell surfaces (Rensen et al. 2015).

Egress via VAPs was first believed to be a unique mechanism of viral release. However, several rudiviruses appear to encode for PVAP proteins. Furthermore, discovery of the VAPs with 6-fold symmetry speaks for a widespread egress strategy among Thermoproteota viruses or archaeal viruses in general. The lack of sequence homology between the PVAPs of the seven-sided and six-sided pyramids suggests a high diversity among putative proteins capable of forming VAPs in archaeal viruses. Thus, it is difficult to predict the egress mechanism based on the viral genome sequence. Nonetheless, the PVAPs discovered thus far do share common dominators such as having extensive α-helical content and an N-terminal transmembrane domain (Baquero et al. 2021b), which might help to identify novel PVAPs, potentially with different symmetries, in the future.

Viral egress by complete membrane disruption

When virus infection leads to a complete disruption of the host cell membranes, a decrease in turbidity of the infected host cell culture can be observed. In the case of viruses infecting members of Thermoproteota, a few viruses have been reported to release their progeny via cell lysis. Lysis induction by sulphur depletion of cultures infected by Thermoproteus tenax virus 1, 2, or 3 results in a decrease in turbidity (Janekovic et al. 1983). Cultures infected by Pyrobaculum filamentous virus 1 (PFV1) display growth retardation during infection. Furthermore, at later stages of the PFV1 infection cycle the host cell membrane appears to be slashed open by long straight 'cuts' (Rensen et al. 2016).

Archaeal viruses with head–tail morphology representing the class *Caudoviricetes* and infecting euryarchaeal host strains have been known since the mid-70s, before archaea were classified as a separate domain of life (Wais et al. 1975, Torsvik and Dundas 1980). In many cases, viral infection of archaeal head–tail viruses results in complete cell lysis, as demonstrated by a drop in optical density of the infected host cell culture and a concomitant release of viral progeny (Pietilä et al. 2013b, Svirskaitė et al. 2016, Schwarzer et al. 2023). As a result, progeny yield can be very efficient, leading to 10^{10}–10^{11} pfu/ml.

Many bacteriophages release progeny via cell lysis, which usually involves a typical holin–endolysin system (Young et al. 2000). Holins are small and diverse membrane proteins that share little sequence similarity (Saier and Reddy 2015). Typically located adjacent to the endolysin gene, holins feature at least one transmembrane domain along with a highly charged, hydrophilic C-terminal domain (Shi et al. 2012, Cahill and Young 2020). Holins initially accumulate harmlessly in the host's membrane until reaching a critical concentration, upon which they aggregate into rafts, collapsing the membrane potential. This collapse triggers a conformational change, which culminates in hole formation (White et al. 2011). Through these openings, endolysins pass the membrane to cleave murein (Young 2014).
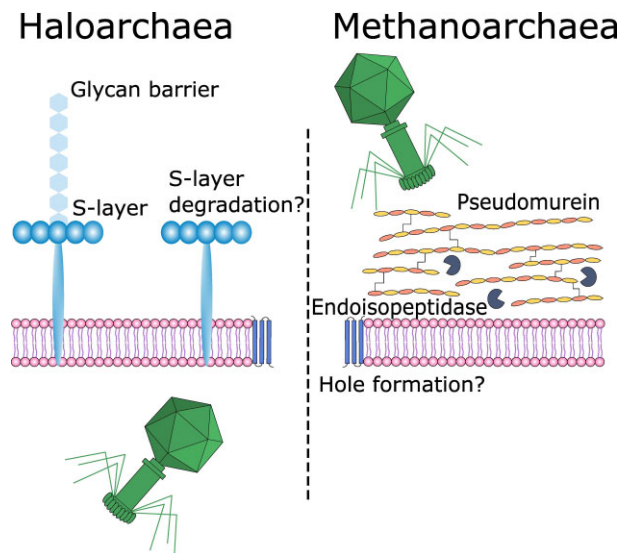

Figure 3. Hypothetical lytic egress mechanism of a head tail viruses infecting halo-(left) and methanogenic archaea (right) involving the use of a hole forming protein to perforate the hosts membrane in halo- and methanoarchaea.

Because archaea do not have a murein cell wall layer, a holin–endolysin system would not be effective. Nevertheless, a few species of *Methanothermobacter* and one of *Methanobrevibacter* possess a pseudomurein cell wall layer that is structurally different from bacterial murein. Pseudomurein is composed of *N*-acetyltalosaminuronic acid and *N*-acetyl-d-glucosamine linked by β-1,3 glycosidic bonds. Although murein and pseudomurein are functionally and structurally similar, they are believed to be the product of convergent evolution (Steenbakkers et al. 2006). Strikingly, pseudomurein is restricted to a few methanogenic archaea, whereas murein is highly conserved among bacterial species (Visweswaran et al. 2011). In contrast to bacterial cell wall hydrolases, cell wall hydrolyses in archaea are still very mysterious. For instance, pseudomurein is unsusceptible for several antibiotics that inhibit the synthesis or function of the peptide subunits of murein (Varnava et al. 2017). Furthermore, bacterial endolysins are ineffective in cleaving pseudomurein (Visweswaran et al. 2011).

However, pseudomurein degrading enzymes have been discovered in the genomes of a few archaeal viruses integrated in the genomes of methanogens. The defective prophages ΨM1 of *Methanothermobacter marburgensis* (Meile et al. 1989), ΨM100 of *Methanothermobacter wolfeii* (Luo et al. 2001), and φmru of *Methanobrevibacter ruminantium* (Altermann et al. 2018) are capable of autolysis of the methanogenic archaea. Endoisopeptidases PeiW (ΨM1) and PeiP (ΨM100) break the ε-isopeptide bond Ala-ε-Lys in the peptide chain of pseudomurein (Visweswaran et al. 2011). PeiR (φmru) is reported to have a similar activity although the pseudomurein peptide side chain of *M. ruminantium* has a different amino acid composition (Altermann et al. 2018).

It remains unclear how the intracellularly produced pseudomurein degrading enzymes pass the archaeal cell membrane (Quemin and Quax 2015) (Fig. 3). The identification of genes encoding pore-forming holins in archaeal virus genomes remains elusive, possibly due to the generally low sequence identity of holins genes. Nonetheless, possible holin homologues have been identified in a few species of archaea, but none have been functionally characterized (Saier and Reddy 2015).
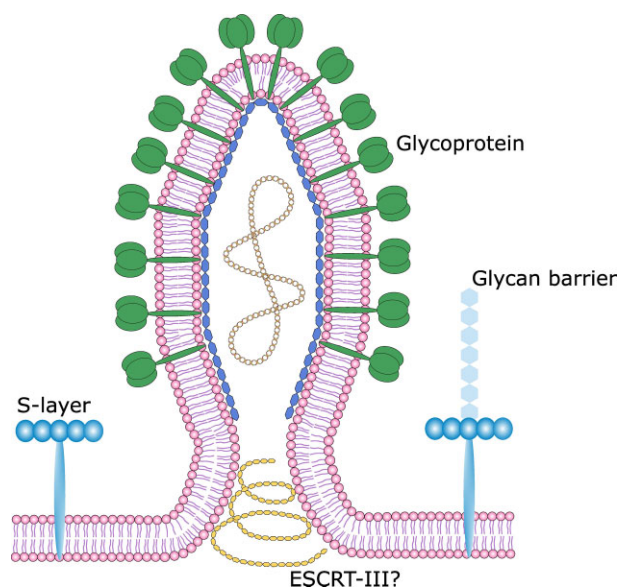
Figure 4. Schematic of budding by viruses infecting species of Thermoproteota. Depicted is the hypothesized involvement of the ESCRT mechanism in budding of the viruses, reminiscent of budding by some eukaryotic viruses.

Viral release without membrane disruption

Many archaeal viruses, especially viruses infecting members of the Thermoproteota, do not cause cell lysis at the final stage of the infection cycle. Instead, they are believed to be continuously produced, and leave the cell without disrupting the membrane (Bettstetter et al. 2003, Pina et al. 2014, Papathanasiou et al. 2019). Like bacteriophages, the life cycles of archaeal viruses can be very diverse (Mäntynen et al. 2021). Archaeal viruses often contain membranes, which are derived from host lipids (Roine and Bamford 2012, Atanasova et al. 2015, Attar 2016). Some viruses, for instance lipothrixviruses, are surrounded by an external lipid bilayer. Other viruses may have internal membranes underneath an icosahedral protein capsid, as in the case of sphaerolipoviruses or turriviruses (Arnold et al. 2000, Bettstetter et al. 2003, Kivelä et al. 2006, Brumfield et al. 2009, Zhang et al. 2012, Liu et al. 2018). Vesicle-like pleolipoviruses are only surrounded by a membrane (Pietilä et al. 2012). Pleolipoviruses establish nonlytic and persistent infection of the host cells, and virion progeny egress continuously. Egress presumably occurs through budding, although the underlying molecular mechanism has yet to be elucidated (Pietilä et al. 2012, Svirskaitė et al. 2016, Atanasova et al. 2018, Demina and Oksanen 2020, Bignon et al. 2022).

Egress for archaeal viruses by budding has been reported for spindle-shaped viruses infecting species of *Sulfolobus*. Upon egress, the viral nucleoproteins are emitted from the host through a rod-shaped intermediate structure protruding from the host surface. In this process, the SSV1 viral nucleoprotein complexes obtain their lipid envelope. The formation of the bud-neck by SSV1 resembles that of eukaryotic viruses such as Ebola or HIV (Noda et al. 2006, Sundquist and Kräusslich 2012). Scission occurs at the formed bud-neck separating the SSV1 virions from the cellular membrane (Quemin et al. 2016).

Several aspects of archaeal virus budding closely resemble that of eukaryotic viral budding (Fig. 4). In many cases, enveloped eukaryotic viruses make use of the endogenous ESCRT (endosomal sorting complexes required for transport) scission machinery, which is hijacked to cleave the membrane neck (Votteler and

Sundquist 2013). It is known that most archaea from the TACK and Asgard superphyla encode homologs of the eukaryotic ESCRT pathway (Samson et al. 2008, Frohn et al. 2022, Hatano et al. 2022). The ESCRT system plays an important role in the egress of Sulfolobus tengchongensis spindle-shaped virus (STSV2). Infected cells formed buds at one cell pole. When ESCRT-III deletion mutants (Δ*escrt-III-3*) are infected with STSV2, they are unable to form buds. Overexpression of ESCRT-III in uninfected cells results in cells with a morphology similar to budding of infected wild type cells. In addition, ESCRT appears to localize at the bud in STSV2 infected cells, compared to a more scattered distribution in uninfected cells (Liu et al. 2017).

ESCRT-mediated egress in various enveloped eukaryotic viruses shows striking similarities with egress of enveloped archaeal viruses (Schöneberg et al. 2016, Vietri et al. 2020). The involvement of the ESCRT mechanisms in budding of STSV2 suggests a conserved ESCRT-mediated egress mechanism for enveloped viruses in the archaeal domain. Nonetheless, budding has also been suggested as egress mechanisms for pleolipoviruses infecting haloarchaea that do not possess ESCRT homologs. These viruses could make use of yet unknown budding strategies, independent of the ESCRT machinery.

A recent publication demonstrated the involvement of a small GTPase in extracellular vesicle (EV) formation in *H. volcanii*. Small GTPase OapA (HVO_3014), a Ras superfamily GTPase, was found to be enriched in EVs of *H. volcanii*. OapA deletion mutants were unable to form EVs, whereas overexpression of OapA resulted in increased vesicle production. Furthermore, homologous proteins were identified across multiple lineages of archaea, especially Euryarchaea and DPANN (Mills et al. 2023). Given the demonstrated involvement of small GTPase OapA (HVO_3014) in EV formation in *H. volcanii*, it is possible to speculate that enveloped viruses could exploit this small GTPase to facilitate their budding process. Interestingly, other enveloped archaeal viruses such as lipothrixvirus SIFV, do not bud from the cell, but egress via VAPs. The origin of the viral lipid envelope is thus hitherto elusive.

Egress of archaeal viruses: what do we know?

The study of archaeal virus release mechanisms currently indicates three main modes of egress: (i) via VAPs, (ii) viral budding, or (iii) VAP-independent disruption of the cell membrane. VAP-based egress was initially thought to be confined to a small group of viruses. The recent discovery of six-sided VAPs highlights diversity among VAP-based egress mechanisms and points towards a more widespread use of this viral release mechanism.

In contrast, enveloped archaeal viruses are thought to egress by budding, reminiscent of ESCRT-mediated budding observed among some eukaryotic viruses. The study of STSV2 indicates a role for the ESCRT pathway and suggests that the virus hijacks the system for egress, although the proteins involved remain to be identified (Liu et al. 2017). Identification of these proteins would benefit predicting, which other viruses employ a budding egress mechanism.

A significant number of archaeal viruses, especially archaeal tailed viruses, causes total disruption of the cell envelope. This is atypical for both the VAP-based and the budding egress mechanism, and thus suggests that archaeal viruses employ at least one additional egress mechanism. The main players of this hypothetical egress system are still completely uncharacterized. Elucidating egress mechanisms of head tailed archaeal viruses would not only further our understanding of this elusive group of viruses but also shed light on the evolution of head tailed viruses.

Outlook

In recent years, our understanding of viral interaction with the archaeal cell envelope has greatly increased with the help of novel genetic and imaging tools. Novel egress mechanisms have been elucidated, and more are certain to follow, given that viral infection of archaeal head–tail viruses results in complete cell lysis.

To date, the great unknown of viral entry is the delivery of the viral genome into the archaeal host cell. While host attachment and viral egress of several models have been visualized in great detail with the help of cryo-ET, the genome delivery of most archaeal viruses remains obscure. The only exception is the recently revealed fusion mechanism of HRPV-6, which likely represents the mode of genome delivery of pleolipoviruses (Bignon et al. 2022).

Archaeal-tailed viruses likely eject their genome through the tail, similar to their bacterial counterparts, however, this still needs to be confirmed. Other genome delivery mechanisms of archaeal viruses remain unaddressed. In viruses, structure and function are ultimately linked. This raises the question if the high diversity of archaeal virion shapes reflects highly divergent genome delivery mechanisms among archaeal viruses. With the increased application of cryo-ET to the investigation of archaeal viruses, and the development of enhanced light microscopy and native conditions for several model archaea, this fascinating question will certainly be addressed in future.

Supplementary data

Supplementary data is available at *FEMSML* Journal online.

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