Literature study of virus size, burst size, latent period and genome size across different lytic eukaryotic and prokaryotic virus groups- an overview of traits and possible trade-offs

R.M. Prathibha Nilakshi Ranasinghe

Thesis submitted in partial fulfilment of the requirements for the degree

Master of Science in Microbiology

Department of biology, University of Bergen

November 2019

ACKNOWLEDGEMENT…….

First, I would like to express my deepest gratitude to my supervisors, prof. *Gunnar Brathbak* and Dr. *Selina Våge*, for accepting me as their student, for being so sympathetic about my situation and for their endless patience with me, when all the given deadlines swiftly passed by. Thankful to both of your advices, feedback and assistance, finally after 42 years, my dream came true. Specially for Selina, thank you so much for correcting each and every tedious, silly writing errors in every page in my draft. Your comments and corrections shaped my thesis greatly.

Then, I must acknowledge *Beate Ulrikke Rensvik* / Study leader, Department of Biological Sciences at University of Bergen, for giving me a second chance to continue my education. I would also greatly appreciate *Kathrine Brosvik Thorsen* at HR division for granting me study leave, thanks to her, I got this valuable opportunity for MSc education. Then, I have to say how grateful I am to *Solveig Thorkildsen*, at biodiversity group, who is my supervisor at work. I am indebted to her for her support, understanding and lenience towards me during these past months. Otherwise I could never balance my role as a single mother together with work and education. I have to thank *Chandana Nissanka*, for his sacrifices and I just want to say that I have not forget any of them. Then I remind my mother filled with gratitude, for continuously reminding me that age is not an obstacle to fulfil my goal of higher education, which she never had the opportunity. Finally, even though I never got any support other than driving me crazy, I have to mention my boys, since at last, now I have time to spend with them!

ABSTRACT

Viruses are directly and indirectly involved in important animal, soil and oceanic ecosystem processes via their influence on microbial life (Krishnamurthy et al., 2016). Their astonishing abundance and genetic diversity have attracted various fields of research interests. Influence of marine viruses on their host and subsequent costs can be categorized into ecological, biological, geological and chemical effects (Fuhrman, 1999).

We need more understanding of the factors and processes, which drive variation in viral traits across various taxonomic groups. There are trade-offs associated with central events (such as adsorption, host range, replication and persistence) of phage life histories, which deter synchronised enhancement of fitness traits. Several trade-off types between viral traits have been suggested. For example, reproduction and survival trade-offs, specificity and generality trade-off, virulence trade-off and further, virus trade-offs relating to host traits are also discussed.

The aim of this study is to collect literature data on growth parameters (traits) for different viruses, to see if there is evidence of trade-offs between traits. To answer this research question, I gathered published data on viral traits as; latent period (LP), burst size (BS), genome size (GS), virion size (VS), host range and various other information about different virus (dsDNA, ssDNA, dsRNA and ssRNA) and phage groups (*Siphoviridae*, *Myoviridae and Podoviridae*) and categorized them. Graphs between each trait pair were made using those data, for each virus category (for e.g. BS vs LP, BS vs GS, BS vs VS, LP vs GS, LP vs VS and GS vs VS etc). Knowledge of viral traits and trade-offs can be incorporated into models to better understand the role of viruses in processes of global significance.

I could not find any strong general trends between any of the traits except weak positive relation between (BS vs LP), (VS vs GS) for dsDNA viruses of eukaryotes, weak negative relation between (BS vs LP) for ssRNA viruses of eukaryotes, weak to moderate positive relation between (BS vs LP), (BS vs host: virus genome ratio), (VS vs GS), weak negative relation between (LP vs host growth rate) for cyanophages and for archaeal viruses; totally 6 trait pairs were tested and four pairs displayed weak to moderate positive relation.

It was not wise to pool marine, fresh water and other viruses from different environments, since those fundamentally exclusive environments force their inhabitants (including viruses) to choose unique trade-offs, independent of their phytogenic or systematic assemblage. It is reasonable to conclude that “the search for trade-offs should concentrate on species that co-occur” (Litchman and Klausmeier, 2008)

TABLE OF CONTENTS

List of figures

List of tables

1. Introduction

1.1 Viruses are everywhere

Viruses have a distinctive and profound role for diversity on a global scale (Suttle, 2007). They are present in every habitat such as ; soil (Kuzyakov and Mason-Jones, 2018, Williamson et al., 2007), rhizosphere (Appunu and Dhar 2008), human gut phageome (Manrique et al., 2017) (Manrique et al., 2016), human body (Barr, 2017) ; even in extreme environments (polar aquatic (Yau and Seth-Pasricha, 2019), Antarctic soil (Zablocki et al., 2014), deep-sea hydrothermal vents (Ortmann and Suttle, 2005) high temperature environments (Jacob et al., 2018), terrestrial hot springs (Zablocki et al., 2018), (Rachel et al., 2002), deserts (Fancello et al., 2013) and corals (Correa et al., 2013).

They infect all organisms, from bacteria to whales (Munn 2006), even parasitizing viruses itself as virophage (Bernard La et al., 2008, Bekliz et al., 2016). Being the most abundant and diverse "biological entities" (Breitbart et al., 2007) on the planet (Edwards and Rohwer, 2005), counting up to 10 million particles per millilitre (10^{7}) in the ocean (Breitbart et al., 2007), together with their extraordinary ability to infect every lineage in the tree of life, viruses are in a unique position to influence biological, geological and ecological processes.

Viruses, which infect bacterial hosts, are named **bacteriophages** or phages. They score the highest abundance among all the virus types , possess the highest proportion of global genetic diversity and are referred to as " biological dark matter" (Youle et al., 2012)

Even though Bacteriophages were discovered approximately 100 years ago by Felix d'Herelle (Chibani-Chennoufi et al., 2004) , it took another half a century to first describe the marine viruses (Spencer, 1955). It took yet another 3 decades until a Norwegian research group discovered the sheer numerical abundance of marine viruses (Bergh et al., 1989) . This discovery opened a door to a golden era of marine virus research. Since then, virus research has become a crucial part in biotechnology and ecology.

1.2 Abundance and diversity of marine viruses

Much of the genetic diversity of phages has yet to be discovered (Angly et al., 2006). Between 65-95% metagenomic sequences of marine viruses are idiosyncratic and shows no resemblance to known sequences (Mya et al., 2002). Wilhelm and Suttle, (1999) estimated that there are over 10^{31} viruses in the ocean alone. To better understand their numerical power, if we align

marine viruses (roughly assuming 50 nm diameter each) the line will be 400000 light years long , 16 times longer than our galaxy, which is calculated to be 25000 light years (Weinbauer and Rassoulzadegan, 2004). Apart from their abundance, it is estimated that there are 3000-7000 types of viruses in 200 L of sea water (Mya et al., 2002). In addition, they exert an enormous selective pressure on rest of the living counterparts (Jaakkola et al., 2012).

Over the last few decades our perspective on viruses has altered dramatically. We are passed the era of considering viruses as plain infectious, dangerous "fragments of life". Multi-disciplinary evidence suggest that we must revise the position of viruses and they are essential members of our planet (Le Romancer et al., 2007, Forterre, 2010).

1.3 Ecological role of viruses in microbial mortality, abundance, diversity and community structure

Lytic viruses can change the aquatic microbial and algal species composition (diversity), community composition, population dynamics (Bratbak et al., 1993) and succession by viral induced mortality process (Suttle, 1994, Wommack and Colwell, 2000, Suttle, 2005, Hennes and Simon, 1995). At any given time, half of the marine microbial population is virus infected (Hurwitz et al., 2013). Due to these viral infection and lysis processes, marine ecosystems face continuous structural and functional changes (Weinbauer and Rassoulzadegan, 2004) . This manipulation begins at individual cell level affecting the cellular physiology and expand up to multiple population, community and ecosystem level processes (Weitz and Wilhelm, 2012).

In the oceans, 90% of the biological carbon derives from bacterioplankton (bacteria and archaea) (Wilhelm and Suttle, 1999). Bacteriophages can lyse approximately 50% of marine prokaryotes daily (Proctor and Fuhrman, 1990, Breitbart et al., 2007). However, Suttle, (1994) has estimated the lysis percentage to be 10-20% of the bacterial community. Half of the global primary production comes from photosynthesis by phytoplankton (Brown et al., 2006). Two three percent of aquatic primary production can be lost by viral mediated lysis of phytoplankton. Algal bloom termination of *Emiliania huxleyi* caused by viral mortality is a classic example (Bratbak et al., 1993, Bratbak et al., 1996).

According to the proposed "killing the winner" hypothesis, lytic phages exert density dependent regulation of numerically and competitively dominant bacterial species (Thingstad and Lignell, 1997, Thingstad, 2000). This provides space for competitively inferior species to evolve, facilitates coexistence of diverse species thus enhance the diversity and maintain the

species richness (Weinbauer and Rassoulzadegan, 2004). “r” and K selection of viruses and their hosts is also an important factor in diversity maintenance in marine milieu (Suttle, 2007) Further, antagonistic coevolutionary “arms race” between viruses and their host is another “diversity-generating mechanism” driven by strain- specific viruses (Thingstad et al., 2014 and references therein, Xue and Goldenfeld, 2017). In this way, virus- induced selective microbial mortality alter the abundance of individual species, which leads to modification of community structure and composition (Bouvier and Del Giorgio, 2007)

1.4 Role of viruses in biogeochemical nutrient cycles

Viruses are the nanoscale drivers of the global -scale processes (Brussaard et al., 2008).Viral lysis of phytoplankton affect the nutrient (Wilhelm and Suttle, 1999) and carbon cycles in the sea (Weitz and Wilhelm, 2012, Weitz et al., 2015 , Jover et al., 2014). It interrupts the conventional nutrient and carbon flow to the upper trophic levels (Zimmerman et al., 2019). This has been discussed in the light of concepts like “microbial loop” or “viral shant” in aquatic food webs (Azam et al., 1983, Suttle, 2007, Fuhrman, 1999, Wilhelm and Suttle, 1999, Fuhrma and Suttle, 1993, Bratbak et al., 1994, Thingstad et al., 1993), and “biological carbon pump” (Jiao et al., 2010, Suttle, 2007).

Viral lysis of prokaryotes and eukaryotes releases the dissolve organic matter into the aquatic environment. The amount of fixed carbon which enter into the microbial loop at various trophic levels, through viral lysis, is estimated to be 5-25%. Dissolved organic carbon, phosphorus , nitrogen, iron and selenium become bioavailable after viral lysis of phytoplankton (Gobler et al., 1997). Then, those released components are taken up by bacteria. This can affect water chemistry.

1.5 Ecological viewpoint of virus- host interactions

Viruses facilitate diversity through horizontal (lateral) **g**ene **t**ransfer (HGT) (Canchaya et al., 2003). Bacteriophages also promote bacterial evolution by several ways (Penadés et al., 2015); HGT create divers microbial gene repertoires (Touchon et al., 2017), resistance and applies substantial selective pressure by altering the physical and genetic makeup of bacterial populations (Thingstad et al., 2014). Further, viruses can enhance their host by carrying “host genes (auxiliary metabolic genes)” for e.g. photosynthesis genes in cyanophages (Breitbart et al., 2007). The study of virus ecology has now reached its “third age” (Mann, 2005) due to recognition of their potential as genetic reservoirs in aquatic habitats (Frost et al., 2005, Paul

and Sullivan, 2005), gene therapy vectors (Robbins and Ghivizzani, 1998), phage therapy (Karin et al., 2018) and phage mediated spread of virulence and antibiotic resistance genes (Brown-Jaque et al., 2015). Further, viral lysis can exert a short term stimulatory effect on ecosystems by increasing the productivity of non-targeted hosts (Weitz et al., 2015). Instead of classical pair-wise approach to study the virus-host interactions, network level approach of "*virus-host interaction networks*" has recently been successfully introduced (Weitz et al., 2013, Flores et al., 2011).

After contemplating details of how viruses affect the global ecosystems in different ways, it is essential to understand what **factors affect growth, survival, dynamics of the viruses and host-virus interactions in the environment**. Viruses totally depend on their host for growth and replication, so all the factors affecting the host's growth will affect viruses indirectly as well. Temperature, water, hydrostatic pressure, ultraviolet radiation (sunlight), photosynthetic active radiation, ionic environment, salinity or freshwater and seawater mixing , oxygen, pH, organic and inorganic particles and dissolve substances, nutrients, grazing by heterotrophic nanoflagellates (HNF), host morphology and availability have been described as factors affecting the virus decay and infectivity (Fig. 1a and 1b) (Mojica and Brussaard, 2014, Weinbauer, 2004, Heldal and Bratbak, 1991, Parada et al., 2007, Wei et al., 2019). The viral pool can be altered by several ways; by changing host dynamics (host morphology, susceptibility, environmental fluctuations), by altering life cycle strategies and reproductive traits (LP, BS) or by various impacts from the surrounding (pollution, grazing, sinking etc. (Fig.1b). All these factors and processes are interconnected, and they can alter every stage of the viral life cycle (Fig.2).

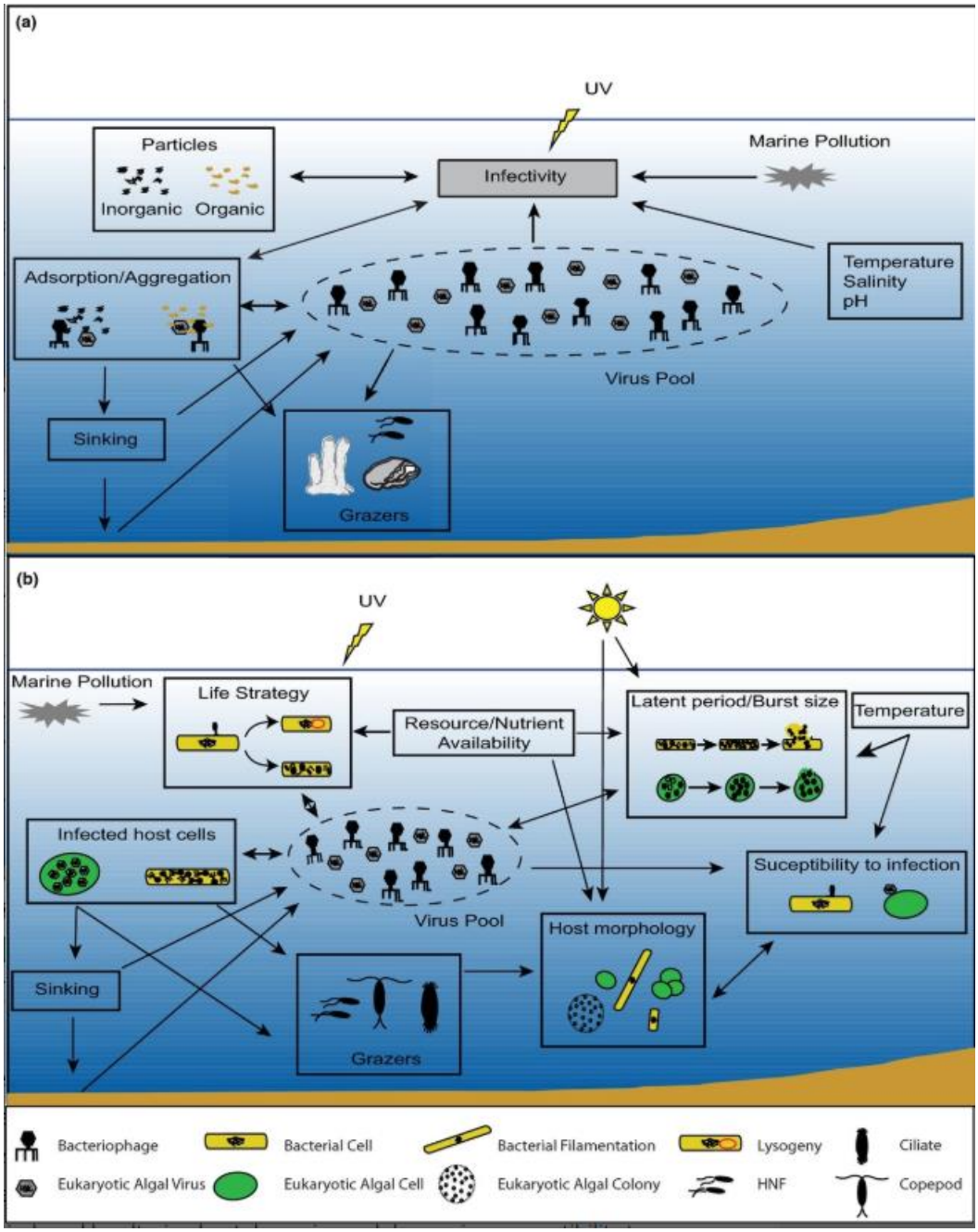

Fig. 1 Environmental factors affecting viral dynamics and virus–host interactions. **(a)** A variety of environmental factors contribute to inactivation or removal of free virus particles from the virus pool, which lower the host encounter and infection possibilities. For e.g. adsorption to inorganic or organic particles, aggregation, sinking and fed by grazers are among the removal factors, while ultraviolet radiation, temperature, pH, salinity fluctuations and marine pollutions cause inactivation of viruses. **(b)** Graphical overview of impact of various factors and processes on viral pool. See text. (Figure obtained from (Mojica and Brussaard, 2014, P.496).

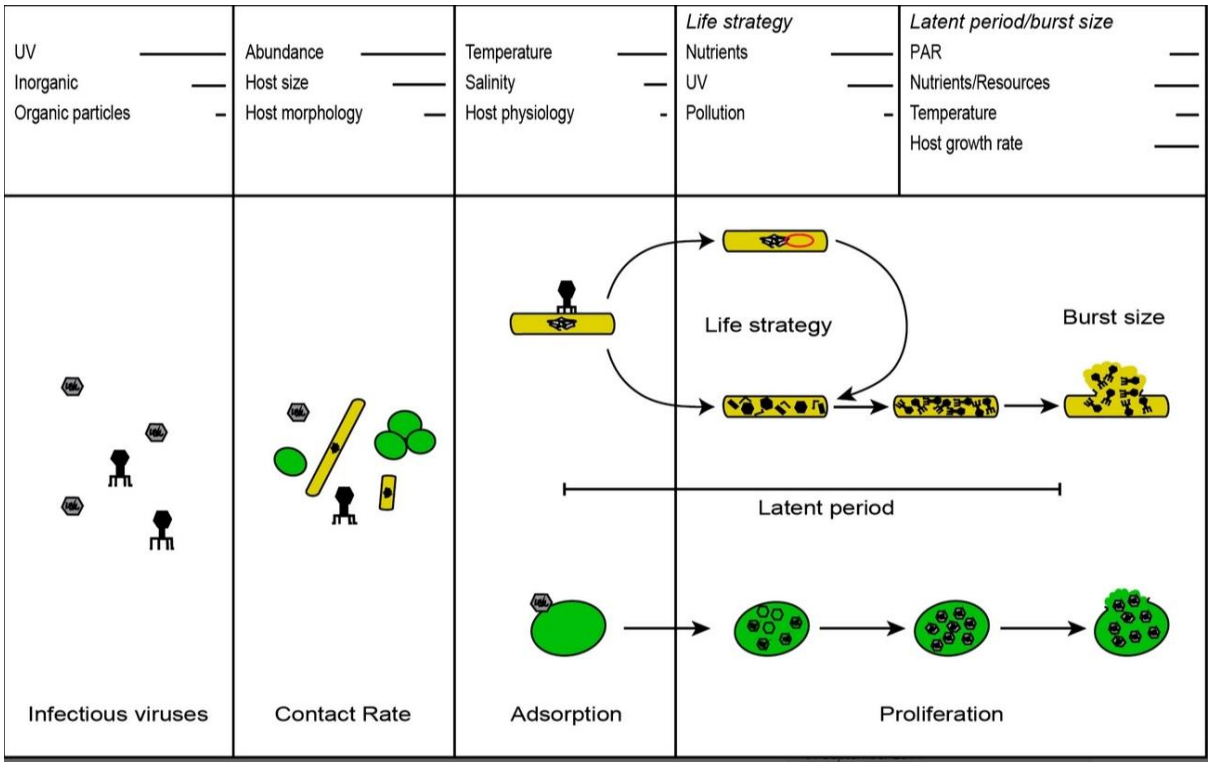

Fig. 2 Illustration of various life cycle stages of the viruses and impact of environment on them. Horizontal bars show the quantity of information available on impact of each environmental parameter on virus life cycle steps. obtained directly from ((Mojica and Brussaard, 2014), P.509) .

1.6 Morphological, structural and genetic diversity of viruses

~ 96%

As in every other life form, viruses come in various shapes and sizes. Within an infectious virus particle or a "virion", the viral nucleic acids are enclosed by a protein coat, in addition by some other layers too. Archaeal viruses and bacteriophages together make the virus group named prokaryote viruses. Their genetic material can be single -stranded (ss) or double- stranded (ds) deoxyribonucleic acid (DNA) or ribonucleic acid (RNA). Out of 6196 phages and 88 archaeal viruses, which have been described morphologically so far, are icosahedral tailed phages and belonged to 3 families; siphoviridae, myoviridae or podoviridae. Only 3.7 % take other forms as polyhedral, pleomorphic or filamentous (Ackermann and Prangishvili, 2012). Members of pleomorphic virus families have dsDNA genomes and a range of forms; two-tailed (*Bicaudaviridae*), bacilliform, fusiform, spherical, bottle (*Ampullaviridae*), lemon shape (*salterprovirus*, *Fuselloviridae*) and droplet

shape (*Guttaviridae*) and without capsid (*Plasmaviridae*) (Fig.3A). Many of pleomorphic viruses and some filamentous shape *Lipothrixviridae* members possess lipid envelops (Ackermann and Prangishvili, 2012). Bradley, (1967) introduced a virus classification based on nucleic acid type and gross morphology. Bradleys group A phages, correspond to the novel phage family *Myoviridae*, group B (=*Siphoviridae*), C (= *Podoviridae*), D (= *Microviridae*, ssDNA), E (=*Leviviridae*, ssRNA) and group F (= *Inoviridae*, ssDNA, long filamentous and short rods) (Ackermann and Prangishvili, 2012).

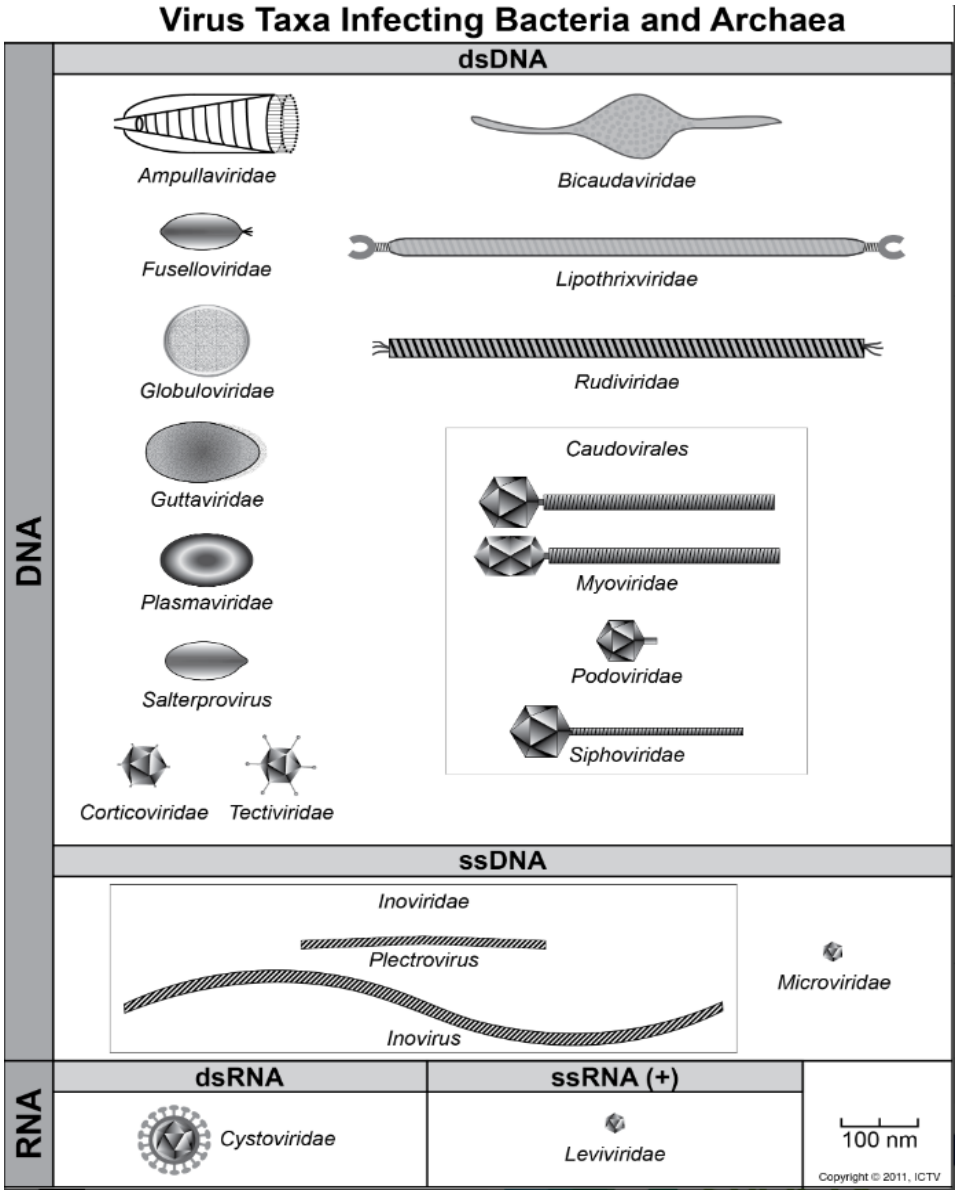

Fig. 3A

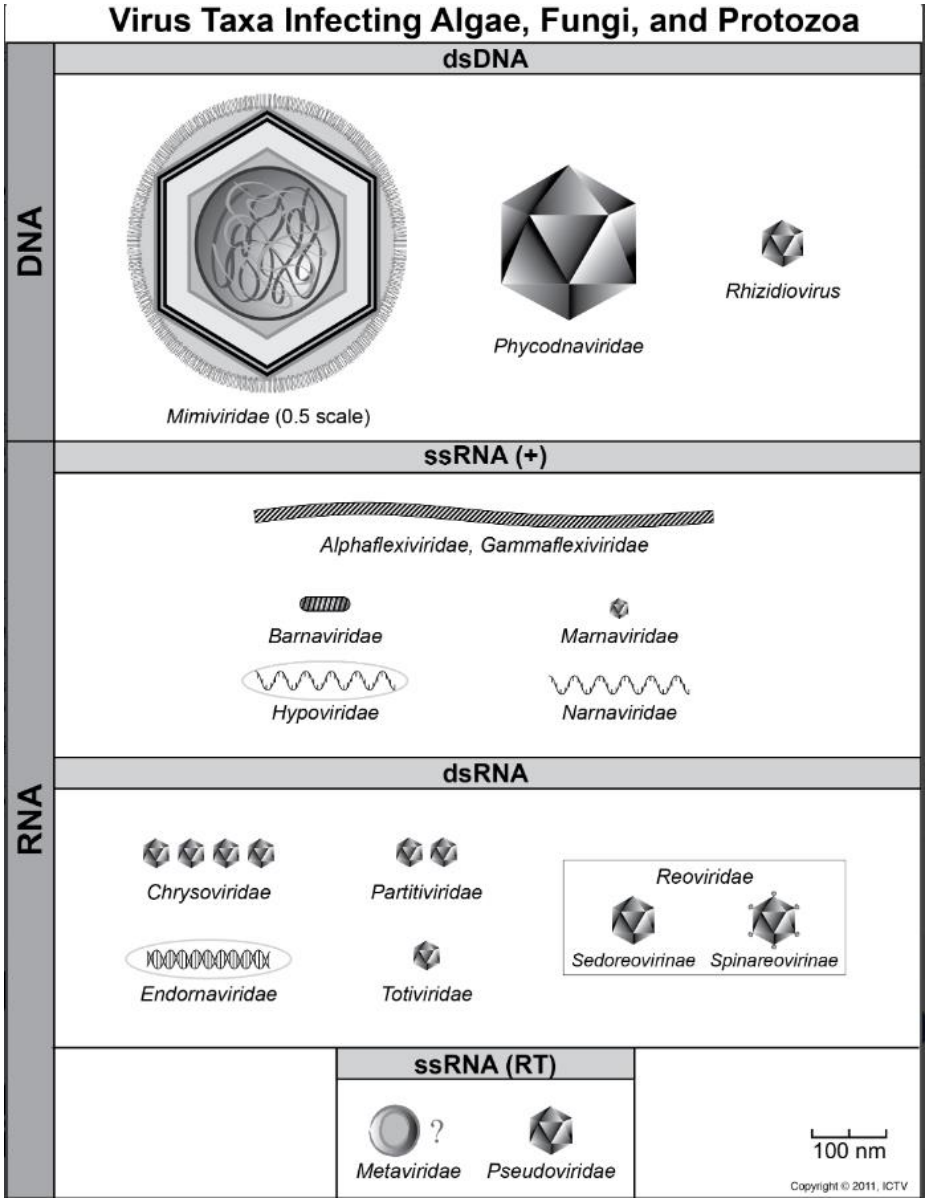

Fig. 3(B)

Fig.3 Morphology of virus taxa infecting **(A) bacteria and archaea**. **(B) algae, fungi and protozoa.** (Obtained from the ICTV report on virus taxonomy (ICTV, 2019b)

1.7 Virus Taxonomy

Viruses can be differentiated based on their morphology, infection strategy, genome architecture or based on their host range (King et al., 2011, Hyman and Abedon, 2010, Abedon and Murray, 2013).

True viruses (except viroids and virusoids) can be classified into Domain- Akamara (acellular infectious agents), Kindom- Euwiria (true viruses), Phylum- Deoxyribovira (DNA viruses) or Ribovira (RNA viruses), Class- dsDNA /ssDNA/dsRNA/ +ssRNA/-ssRNA. The international

Committee for the Taxonomy of Viruses (ICTV, 2019a) , determines taxonomic levels from order to species (Weinbauer, 2004).

Here, I only give some description on the virus families and groups I encountered in the present literature study. Some of the family names given in the literature were out of date according to present taxonomy and hence could not fit in, e.g. *Styloviridae*

dsDNA viruses

The (ICTV) 9th Report (King et al., 2011) has classified dsDNA viruses into 25 families. Out of those 25 families, I have gathered data on *Siphoviridae, Myoviridae, Podoviridae, Mimiviridae, Tectiviridae, Rudiviridae* and *Phycodnaviridae* families in the present study.

1.7.1 Prokaryotic dsDNA virus families

Order-Caudovirales, contains tailed prokaryotic dsDNA virus families **including archaeal viruses**. Icosahedral heads or prolate capsids are characteristic to this order. Pleomorphic, polyhedral and filamentous viruses are not yet classified into orders (Ackermann and Prangishvili, 2012). There are five families in Order-Caudovirales; *Siphoviridae, Myoviridae and Podoviridae, Ackermannviridae* and *Herelleviridae.* **Family-** ***Siphoviridae*** has long, noncontractile tails (Fig. 3 A). Tail length is usually twice as large than the head diameter, with some exceptions (Table.6.1-6.5). This is the largest prokaryote virus family (Ackermann and Prangishvili, 2012). Due to host specificity and narrow host range, siphoviruses tend to switch for lysogenic cycle under the low host availability (Šulčius et al., 2015). **Family-*Myoviridae*** members has contractile tails. e.g. T4. **Family-*Podoviridae*** has short tails (Fig. 3 A). There are currently four genera in the family, namely N4-like, T7-like, P22-like and F 29-like. Narrow host range seems to be a common trait among many podoviruses while myoviruses generally show the broadest range (Sullivan et al., 2003, Suttle, 2005).

1.7.2 Eukaryote dsDNA virus families

Phycodnaviridae Family

Characteristic features of this family can be listed as; infect wide range of eukaryotic microalgal hosts, both in marine and freshwater milieu, ubiquitous, large dsDNA genome (160 to >560kb) according to (Wilson et al., 2009). However some literature states the genome size as 180- 560 kb (Van Etten et al., 2002). Typical virion size is > 100 nm diameter, cytoplasmic assembly site, and polyhedral symmetry (Fig.3B) without external membrane (Sandaa et al., 2001 and references therein). Six genera are included in the family; Chlorovirus,

Coccolithovirus, Prymnesiovirus, Prasinovirus, Phaeovirus, and Raphidovirus (Wilson et al., 2009, 2011). Within the species barrier, some genera demonstrate a broader host range (e.g. prymnesioviruses, raphidoviruses and coccolithoviruses).

Out of the 7 families listed within ssDNA viruses, namely *Anelloviridae, Circoviridae, Geminiviridae, Inoviridae, **Microviridae**, Nanoviridae, and Parvoviridae*, I found only one example (family-***Microviridae***) in this thesis (Fig.3A), a phage, infecting marine photoheterotrophic alphaproteobacteria *Citromicrobium bathyomarinum* (Zheng et al., 2018).

In the ICTV (King et al., 2011), there is a proposed **new ssDNA virus Genus, Bacilladnavirus, (**Family- unassigned). The colony forming diatom, *Chaetoceros salsugineum,* is the host for this new virus species (CsalDNAV01).

1.7.3 RNA viruses and RNA bacteriophages

To date, there are only few RNA bacteriophages described in the literature (Krishnamurthy et al., 2016), compared to the vast amount of information available on DNA phages. The same applies to the RNA viruses infecting algae and diatoms, particularly there is only one report of ds RNA virus (MpRNAV) infecting phytoplankton host (*Micromonas pusilla*) in the literature (Brussaard et al., 2004). However, Krishnamurthy et al., (2016) found high RNA bacteriophage diversity globally, across a range of habitats including the first ever report of RNA bacteriophages, which infects gram positive bacteria. There are only 2 RNA bacteriophage families that were identified, namely ; family- *Leviviridae* (ssRNA) with 4 species so far and family *Cystoviridae* (dsRNA) with one described species (Krishnamurthy et al., 2016). The ICTV listed 8 families of **dsRNA viruses** and I found one family-***Reoviridae*** in this study (Fig.3B), infecting marine algae, *Micromonas pusilla* LAC 38 (Brussaard et al., 2004).

The ICTV 9th report (King et al., 2011) has divided ssRNA viruses into 2 groups namely; negative sense and positive sense. None of the negative sense ssRNA viruses include in this literature survey. They are causative agents of important human diseases (King et al., 2011) and infect arthropods, vertebrates and plants too (Jun-Hua et al., 2015).

Positive sense ssRNA viruses have at least 30 families and some unassigned groups (King et al., 2011). Out of them, order: Picornavirales, family: *Marnaviridae* (Fig.3B), **Genus: Bacillarnavirus,** comprise of eukaryote diatom viruses with linear ssRNA genomes such as; Rhizosolenia setigera RNA virus (RsetRNAV (RsRNAV) (Nagasaki et al., 2004),

CtenRNAV01(Shirai et al., 2008), CtenRNAV type II (Kimura and Tomaru, 2015), Chaetoceros socialis f. radians RNA virus (CsfrRNAV) (Tomaru et al., 2009b), Guinardia delicatula RNA virus (GdelRNAV) (Arsenieff et al., 2019), Nitzschia reversa RNA virus (NitRevRNAV) (Toyoda et al., 2019),

1.7.4 Cyanophages

Cyanophages are the group of bacteriophages, that infect cyanobacterial hosts. Their characteristic features are; dsDNA genome, head tail morphology, found in both marine and freshwater habitats and categorized under the 3 bacteriophage families described earlier in this study; *Siphoviridae, Myoviridae and Podoviridae*. Most of the marine cyanophages belong to family- *Myoviridae* while majority of freshwater isolates belongs to the other two families (Xia et al., 2013).

1.7.5 Archaeal viruses " not archaeal phages" (Abedon and Murray, 2013)

Viruses infecting the third domain of life (archaea), mostly have nothing in common with bacteriophages (Forterre, 2010). Even though, some studies have extensively discussed the similarities and contrast between the two groups (Pietila et al., 2014). Currently there are 17 families of archaeal viruses (Krupovic et al., 2018). Same as bacteriophages and algal viruses, archaeal viruses also have prominent role in carbon and nitrogen cycling through lysis of their hosts (Danovaro et al., 2016, Danovaro et al., 2017).

Compared to bacteriophages, archaeal viruses show astonishing morphological diversity and unique features, which are not observed in other viruses of eukaryotes or prokaryotes (Fig.3A) (Ackermann and Prangishvili, 2012, Dellas et al., 2014). They are mainly spindle-shaped, spherical or round, bottle-shaped, coil-shaped, droplet-shaped but some of them show characteristic head and tail morphology as well (Fig.3A) (Krupovic et al., 2018 and references therein). Viruses of the two archaeal phyla; Euryarchaeota and Crenarchaeota have distinguishable features. Viruses of Euryarchaeota (methanogens and extreme halophile hosts) have head and tail morphology, resemble bacteriophages and assigned to *Myoviridae, Podoviridae* and *Siphoviridae*. In contrast, viruses of extreme thermophiles (Crenarchaeota) show above described uncommon morphotypes and display little similitude to eukaryote viruses or bacteriophages (Bath and Dyall-Smith, 1998, Prangishvili and Garrett, 2005, Dellas et al., 2014).

Members of Euryarchaeota viruses can be either tailed viruses or polyhedral, pleomorphic or filamentous. Members of the Crenarchaeota viruses are classified into 8 families (Ackermann and Prangishvili, 2012). Thus far, the majority of all isolated viruses infecting archaea possess dsDNA genomes and only two families have ssDNA genomes (Prangishvili et al., 2017). Some archaeal viruses (except tailed viruses) have distinctive replication strategies and exclusive virus release mechanisms delineated with their host phylum (Bize et al., 2009). The majority of Crenarchaeal viruses are nonlytic (Dellas et al., 2014 and references therein). They continuously produce progeny viruses while host cell remains viable and release them by budding mechanisms without lysis of the host (Ackermann and Prangishvili, 2012). This chronic infection form is clearly different from the lysogenic pathway. Virus of euryarchaea are lytic (Dellas et al., 2014 and references therein).

1.8 Life cycle strategies of viruses

The virus life cycle can be lytic, lysogenic, pseudolysogenic or chronic infections (Fig.4).

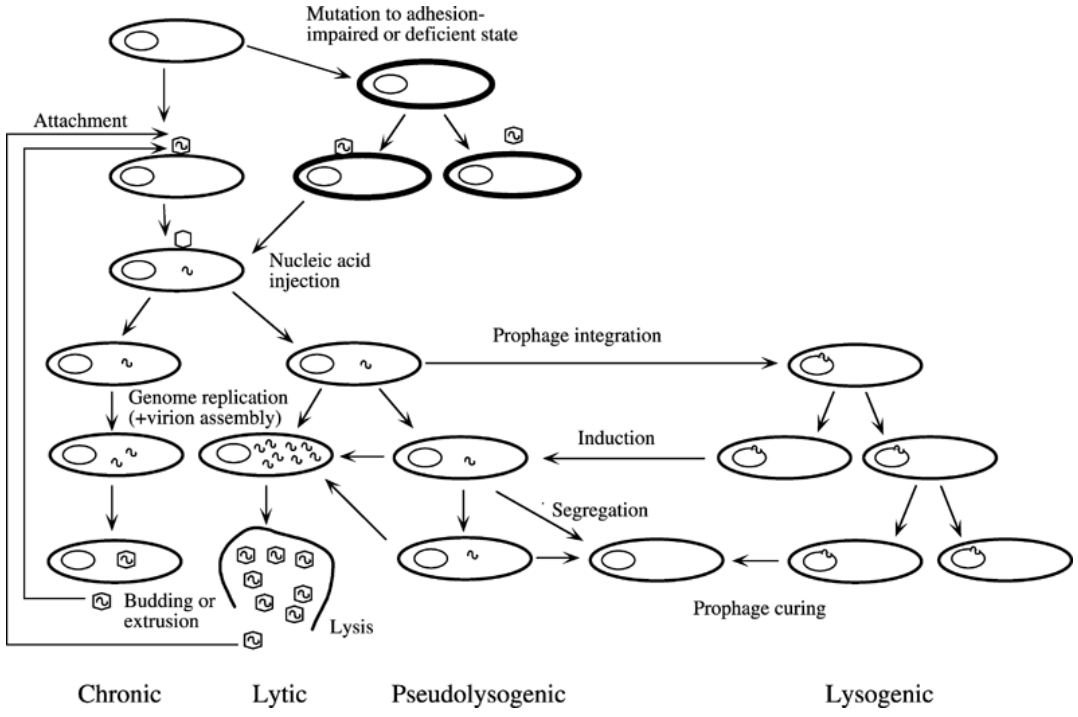

Fig. 4. Types of viral life cycles. (Obtained from Weinbauer, 2004) p. 131.

In the **lysogenic cycle**, also known as temperate cycle, the genome of the lysogenic phage usually continue to exist in the host as a dormant stage (prophage form) either by merge with host genome or as a plasmid and replicates together with the host for several generations until

the lytic cycle is activated by external factors (Weinbauer, 2004). (Fig. 4). Lysogenic cycle is more complex than lytic cycle (Sinha et al., 2018).

Chronic infection can be described as continuous release of newly synthesized virions from the host cell without host cell lysis. This progeny release can occur via extrusion or budding (Weinbauer, 2004). An example are archaeal viruses (Bath et al., 2006, Bath and Dyall-Smith, 1998). **Pseudolysogeny** form of life cycle also called persistent infections or carrier state (Fig.4), means that phage multiplication only occurs in a portion of the population (Weinbauer, 2004). An example is archaeal virus SIRV1, (Sulfolobus islandicus rod-shaped virus) (Prangishvili et al., 1999). Here, I have focused on lytic viruses and phages. Other than the effect of lytic viruses, the significance of lysogenic pathway is equally influential in marine environment as well as in every other environment. Lytic and lysogenic lifestyle of phages can be interpreted as survival strategies. When host bacteria are abundant in the surrounding environment, phages can enter lytic cycle, where the host lysis results in completing the virus life cycle. On the other end, during low host density or low host growth rate (poor growth conditions), lysogeny might be advantageous over lysis. During low host abundance, increased prophage induction has been observed (Long et al., 2007). However, this hitherto accepted paradigm has challenged by the new idea of "piggyback the winner", where high host abundance favours lysogeny (Knowles, 2016). Single stranded DNA viruses and RNA viruses do not have the ability to choose a lysogenic path (Stahl et al., 2015).

Lytic cycle and virus replication

Replication of **lytic phages** takes place over several steps (Fig 4), (i) phage encounter a bacterium which is susceptible for adsorption, (ii) irreversible **attachment**/adsorb of virion, (iii) inject its nucleic acid into host cell followed by host uptake of virion nucleic acid, (iv) virus take control of the cell's replication and protein synthesis mechanisms. Soon after the **injection** step, the eclipse phase of the virus life cycle begins. During the eclipse period, synthesis of new viral coat proteins, early enzymes and viral nucleic acid (by host machinery) takes place. (v) virions mature during the post-eclipse phase, where those newly synthesized viral genomes **assemble** with their capsids. As shown in Fig. 5, infectious virion units increased drastically within the host cell during this maturation period vi) release of virion at the end of maturation (Hyman and Abedon, 2009). During the rise period, **cell lysis** occurs , followed by mature phages being release into the environment, where free phages or virions can be detected

(Weinbauer, 2004). (vii) diffusion-delimited time period, where virions look for a susceptible host cell in the surrounding milieu to adsorb.

All these steps in the virus life cycle (adsorption period, virion attachment and nucleic acid injection, LP and virion release of virions) (Hyman and Abedon, 2009) contribute to determine the generation time of virus. For many bacteriophages, the replication cycle can take 20-60 minutes, while it is much longer in most animal viruses and algal viruses. There are several release mechanisms, which are dependent on the type of virus, namely; excretion (extrusion), budding and cell lysis.

At low multiplicity of infection or the number of viruses co-infecting one host cell (m.o.i.), a single phage may infect a single host cell, while at high m.o.i., **superinfection (**"re-infection with a homologous phage") can happen (Parada et al., 2006 and references therein). **Superinfection** can lead to "lysis inhibition" (delayed cell lysis) and hence extension of LP which eventually increase BS as well (Abedon, 1999). The third type of infection pattern, "co-infection" whereas, two or more viruses infect one host at the same time.

1.9 Viral traits

Trait based approach to study ecology, including marine viral ecology, has gain interest as an alternative to the species based approach (Litchman and Klausmeier, 2008, Record et al., 2016). Traits can be defined as "characteristics of organisms that link processes at the individual level to population-, community-, or ecosystem-level processes" (Record et al., 2016). There are traits, specific to every life event; reproduction, ontogeny, growth and defence. They are usually taxon-transcending and finally regulate fitness (Record et al., 2016). Traits are typically interconnected and relationships between and among them frequently discernible as trade-offs (Litchman and Klausmeier, 2008). Evolution of traits aims to optimize the viral fitness (Edwards and Steward, 2018).

Primary traits (characteristics), which regulate the virus- host dynamics for lytic viruses are **genome size of the virus, latent period and burst size**. In addition, capsid size, structure, genome type, host range, virulence, transcription control ability, entry and release mechanisms, replication and assembly site are also contributing (Record et al., 2016). On the other hand morphology (structure), genome size, growth conditions and growth rate (physiological status) of the host are the prime selective forces for viral traits and their evolution (Bratbak et al., 1998, Edwards and Steward, 2018b). Trait plasticity mainly depend on environment and taxonomic division (Litchman and Klausmeier, 2008).

1.9.1 Latent period

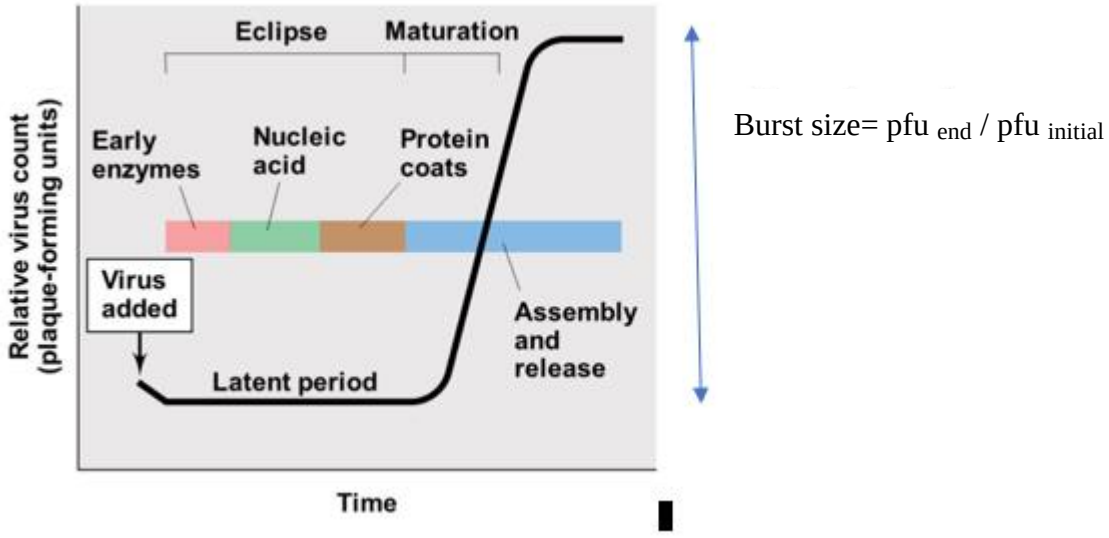

Fig. 5 "Schematic illustration of different phases of the **one step growth curve of virus** replication. Eclipse phase starts following adsorption of virions to the host cell. The eclipse and early maturation phases together make latent period of viral infection. Latent period calculated from the time point that the virion attaches to the host and/ or uptake of nucleic acid. Latent period extent through eclipse and post eclipse phases and end with host lysis, liberating the newly synthesise viral progeny. Virulent, lytic viruses show all those steps in their life cycle" (obtained from Stahl et al., 2015).

The latent period of the virus life cycle commences from the virus adsorption to the host cell and ends upon the host cell lysis. During this infection phase, extracellular or free phages are not detectable (Weinbauer, 2004). One step growth curves can be used to calculate LP and BS for a given phage host system (Middelboe et al., 2010) (Fig. 5). The time duration between the infection (virus addition) and the first surge in viral titre represent the latent period (Fig.5). Triggering of cell lysis involves physiological as well as environmental factors (Young, 1992).

G**rowth rate, genotype and generation time of the host** has an impact on latent period of the virus (Zhang and Jiao, 2009, (Edwards and Steward, 2018b, Proctor et al., 1993, Guixa-Boixareu et al., 1996). Using phage T4 and *Escherichia coli* K- 12 as a model, Nabergoj et al., (2018) elucidated, a decline of latent period up to a limiting value with increasing host growth rate.

Some studies have pointed out that there is a connection between available **phosphate and nitrate** levels (environmental conditions) and viral production and latent period (Zimmerman et al., 2019 and references therein, (Maat and Brussaard, 2016, Wilson et al., 1996, Bachy et al., 2018). However, it is not clear that this is an indirect impact by altered host growth rate or

a direct impact (i.e., growth limiting factor, lack of phosphate to nucleic acid synthesis) (Zimmerman et al., 2019). However, after established the apparent dependence of viral traits on host physiology, it should also note that the sensitivity of this dependence is variable with each virus-host system , underlying cell biology and the factors itself which cause the change in host growth rate (Zimmerman et al., 2019, Bachy et al., 2018). Some studies investigate **irradiance** effects on latent period and viral production (Baudoux and Brussaard, 2008, Piedade et al., 2018).

1.9.2 Burst size

Burst size (BS) of viruses can be defined as the number of progeny viral particles released per host cell into the extracellular environment upon cell lysis (Weinbauer, 2004). This depends on the type of virus as well as the host cell (host genotype). It could vary from few to several thousand virions. It is a key parameter in epidemics, population dynamics and viral ecology (Parada et al., 2006). BS calculation is important when evaluating the virus induced mortality of hetero- and autotrophic, prokaryotic and eukaryotic micro-organisms and viral shunt process in aquatic food webs (Fuhrman, 1999). The ratio between, the increase in viral titre: the decrease in host cell concentration during a certain time interval is used to calculate the BS.

$$BS = (V_{max} - V_{min}) / (H_{max} - H_{min})$$

Where, V_{min} and V_{max} are the minimal and maximal viral concentrations, respectively, and H_{min} and H_{max} , the minimal and maximal host abundances (Arsenieff et al., 2019).

Most of the literature investigated in this study has used one step growth curves (Ellis and Delbrück, 1939) to characterize virus life cycle (Fig.5). One step means only a single infection cycle is permitted, without allowing re-infections.

Methods commonly used to enumerate and estimate of burst size are transmission electron microscopy (TEM) using whole cells (Heldal and Bratbak, 1991) or thin sections, TEM together with streptomycin treatment, plaque forming assay using double layer agar method, one step growth curves, epifluorescence microscopy methods, most probable number assay and flow cytometry. All approaches have their inherent advantages and disadvantages (Weinbauer, 2004). Novel methods as phage FISH and gene ELISA (Dang et al., 2015) are also introduced recently. Studies based on phage host systems have used “one step growth curve experiments” (Weinbauer, 2004), while studies on environmental samples have mostly used TEM to count visibly virus infected cells since for natural communities one step growth curve method cannot

be applied (Parada et al., 2006). Those studies with environmental samples and TEM approach, estimate minimum (average BS from all infected host cells in a sample) and maximum burst size values (use only the cells, which are fully packed with newly synthesized virions to estimate the BS) (Weinbauer, 2004). In minimum BS scenario, there is a possibility that the number of virions within host cell might still increase before cell burst. On the other hand, in maximum BS situation there is a likelihood that some host cells will burst before they fully packed with progeny virions (Weinbauer, 2004).

Host and virus species, m.o.i. (Van Etten et al., 1983, Bratbak et al., 1998), host's abundance and metabolic activity (growth rate), cellular physiology, host cell size and age , bacterial production, host growth rate associated parameters (i.e. temperature, nitrate, phosphate), amount and activity of ribosomes, trophic status of the natural systems, various environmental parameters, e.g. salinity, temperature, availability and composition of the substrate and depth of the water (Weinbauer and Hoefle, 1998) are among the factors identified to affect the BS (Middelboe, 2000, Parada et al., 2006, Mathias et al., 1995). Some studies have reported BS variation among different host morphotypes (Weimbauer and Peduzzi, 1994). Nabergoj et al., (2018) demonstrated a positive linear correlation between burst size and host growth rate using phage T4 and Escherichia coli K- 12 as a model. New virus progeny production needs protein, RNA and DNA synthesis by host, which is directly depend on host's growth rate. Thus, higher host growth rate contributes to higher viral production rate as well (Edwards and Steward, 2018 and references therein). Moreover, virus infection does not inactivate the metabolic activity of the host until lysis. This is a strategy helps to increase BS since it is essential to keep the host metabolically active for continuous production of the viral progeny until burst of the cell (Parada et al., 2006 and references therein). "Ratio of infectious virions per host cell (m.o.i.), plays an important role in infection kinetics by facilitating the encounter rate between virions and hosts" (Brown and Bidle, 2014). BS and m.o.i. has a negative correlation. This is related to "phage induced-lysis from without" phenomenon (Brown and Bidle, 2014). It is postulated that virus and host genome size ratio is an indicator of BS and capsid size is related to the viral genome size (Record et al., 2016 and references therein). Therefore, if given similar condition, smaller viruses can produce more copies (increased burst size) than larger viruses. BS together with latent period is an indication of metabolic efficiency of virus replication *(*Record et al., 2016).

For a given virus-host system, optimizing latent period and burst size is a necessity for the most effective viral spread and life strategy (Parada et al., 2006). This balance between LP and BS is controlled by various biotic, abiotic, intrinsic and extrinsic factors.

1.9.3 Virion size and genome size of the viruses

Even though, size is a first order factor for organisms which determine their position within ecosystem function and structure, its importance as a virus trait is still unclear (Record et al., 2016). Virus size might be significant for host encounter (contact rate), virus production or survival, however, it has not been found evidence that larger viruses infect larger size host or vice versa (Record et al., 2016). Both virion size and host cell size influence the BS, because the number of newly assembled virions, which can be packed inside the host cell is depend on the above two traits. Moderate plasticity of virion size and morphology has been observed (Parada et al., 2006).

Virion head (capsid) size can be as small as 22 nm (*Chaetoceros socialis* f. radians- SssRNA viruses) (Tomaru et al., 2009a)) to 310 nm (Prymnesium kappa virus RF01 (PkV RF01) (Johannessen et al., 2015) or even up to 900nm (dsDNA Sulfolobus islandicus rod-shaped virus) (Prangishvili et al., 1999). Genome sizes of the viruses range from smallest reported of 4.36 kb (vB_Cib_ssDNA_P1- (host-*Citromicrobium bathyomarinum*)(Zheng et al., 2018) to "giruses" with genomes from 300 kb and up to 1259 kb. These large viruses belong to the Mimiviruses, phycodnaviruses and Marseillevirus (Van Etten et al., 2010, Colson et al., 2012).

1.10 Bacteriophage life history traits and associated trade-offs

Biological trade-offs are basic principal of life history theory. This thesis focuses on trade -offs between viral traits. "A trade-off is a relationship between the magnitudes of two (or more) quantitative traits such that changes in the net benefits derived from one imply opposite changes in net benefits derived from the other(s)" (Saeki et al., 2014). In nature, evolutionary, ecological dynamics and life histories are moulded and challenged by trade-offs. Type of life cycle, burst size (fecundity), latent period, reproduction rate (combination of burst size and latent period), adsorption rate, virion stability (survival outside the host), genome size, morphology and size of the capsid, host range have been identified as key traits in life history trade-offs (Zimmerman et al., 2019).

There are trade-offs associated with central events (such as adsorption , host range, replication and persistence) of phage life histories which deter synchronised enhancement of fitness traits

(Goldhill and Turner, 2014, Keen, 2014). These life history trade-offs of viruses have significant influence on bio-geo-chemical processes, dynamics in epidemiology, viral diversity and many more aspects including bio-engineering of viruses (Goldhill and Turner, 2014).

Pleiotropic genes control more than one trait; therefore, they can influence multiple phenotypes (traits). Trade-offs between traits can originate as a result of pleiotropy, since optimizing of one trait, can jeopardize another (McGee et al., 2014). Mutations regulate the fitness variation across variety of environments. Antagonistic trade-offs of such pleiotropic mutations are the driving force survival under stressful or changing environmental conditions (Dessau et al., 2012).

Due to their smaller genome size, overlapping genes and multifunctional proteins, viruses specially face this antagonistic pleiotropy (McGee et al., 2014). However, when selection pressure acts favourable for multiple traits at the same time, **payoffs** can have observed. For example increased growth rate together with increased stability for temperature and pH (McGee et al., 2014).

1.10.1 Reproduction and survival trade-offs

Several trade-off types between viral traits have been suggested. For example, **reproduction and survival trade-offs**; when burst size increase , it will cost the virus through increased decay rate (reduce survival) and vice versa (De Paepe et al., 2006). When there is a selective pressure from the environment in the form of stress factors, it has been shown that RNA virus select reproductive fitness trade-off for enhanced "thermal and structural stability of a viral enzyme" (Dessau et al., 2012). It has been suggested that this reproduction, survival trade-off is interceded by another trade-off in viral lytic enzymes; stability vs activity (Goldhill and Turner, 2014). Some studies however, did not find a trade-off between reproduction and survival (McGee et al., 2014). Still it is difficult to find mechanisms to reason this particular virus trade-off, since the extracellular and intracellular environments are very different for viruses (Goldhill and Turner, 2014).

Furthermore, García-Villada and Drake, (2013) have found decreased viability (lifespan) of phage particles as a trade-off for increased fecundity in coliphage Qß, under the experimental selection. Here in this example, higher fecundity has attained by decreasing the resource (mainly time) expenditure per virion. So, in other words, reduced latent period, increased burst size and reduced survival are the interconnected traits here (García-Villada and Drake, 2013). De Paepe et al., (2006) found evidence for strong positive correlation between mortality and

multiplication rate in coliphages. Here they used average burst size: average latent period ratio as multiplication rate. Multiplication rate can be used as a proxy for fecundity (García-Villada and Drake, 2013).

Genome organization of viruses also suffer trade-offs. E.g. packaging density of genome and capsid strength (Record et al., 2016). There is a limited available space inside the capsid to package the viral genome. When the genome become larger, there is not enough space in the capsid, in contrast, when there is too little genetic material inside the capsid, there is not enough pressure to eject the genome into host, thus infection initiation fails. So, there must be trade-offs, counterweighing the advantages of possessing a larger genome.

Record et al., (2016) have discussed the trade-offs with lysogeny to lysis switching (**virulence trade-off**) in detail, under the changing environmental factors as light, temperature and nutrients. Further, this trade-off can be governed by virus replication rate, burst, host-virus encounter and host and virus mortality (Record et al., 2016).

1.10.2 Specificity and Generality trade-off (host range) or negative correlation between reproductive rate and host range

Here I recap the hypothesis presented by Record et al., (2016). They have identified virus genome size, burst size and morphology as traits related to this host range trade-off. There should be a trade-off cost for being a generalist and to have a broader host range. A virus must possess variety of tactics to defeat the defence mechanisms of multiple hosts, to be a generalist. To achieve this, a broad host range virus should equip with a larger genome with high functionality compared to a narrow host range virus (a specialist). To produce progeny with a lager genome requires more resources from the host, which will impair the replication rate. Being a generalist within a highly diverse host community brings advantages, so the virus can infect diverse hosts and replicate. In this scenario, slow rate of viral population increase is the cost (trade-off) to bear in exchange of gaining access to larger group of hosts. In contrast, when the host population diversity is low, there is not any added advantage in trade-off for broader host range, so it is reasonable to trade for rapid population increase rate in return with narrow host range. There is some data for positive correlation between host range size and genome size for lytic viruses and negative correlation for lysogenic viruses. However, sometimes none of the correlations were observed (Record et al., 2016).

Moreover, there is a strong negative correlation between multiplication rate and both host range, persistence (Keen, 2014). This is comparable to offspring production and offspring quality trade-off in larger organisms (Keen, 2014).

Record et al., (2016 and references therein) has proposed affiliation between higher burst size and broader host range. Moreover, Wang, (2006) found that higher burst size relates to longer latent period. Then again, there is a connection between burst size, host range and burst size with genome size too (Record et al., 2016 and references therein). This indicates that several traits can be encompassed in a trade-off. Size related morphological traits (head, tail diameter and length) show positive relation with host range. Need of additional resources to make larger size viruses, may be the reason for this trade-off (Record et al., 2016).

1.10.3 Trade-off between latent period and burst size

Balance between the latent period and burst size is a fine decision to make to attain the optimal life strategy (Parada et al., 2006).

Best prolific lysis timing of the host (in other words duration of latent period) , or whether to select lytic pathway or not lyse the host at all (lysogenic pathway) is a trad-off, which include factors as multiplicity of infection, host density and quality (Abedon et al., 2003), size of the host cell,, growth rate of the host (Dennehy and Wang, 2011) , amount of viral genome inside the host, viral production rate (Wang et al., 1996) and amount of susceptible host outside (Goldhill and Turner, 2014 and references therein). To reach this optimum lysis timing trade-off, a 'decision' must be made, assessing what is the balance between minimum latent period and maximum burst size. Here, the underlying mechanism for this trade-off is simply the "time" (Goldhill and Turner, 2014). Assuming linear increase of viral progeny inside the host with time, longer latent period favours the increased burst size (reproduction). In contrast, shorter latent period, lower the viral progeny release but at the same time facilitate swift launch of new infection cycles (Wang, 2006, Wang et al., 1996) **.** Phages with intermediate burst times shows the highest fitness **(**Wang, 2006**).** Lysis timing has been observed to be an evolvable trait. Under experimental conditions, when there is a high abundance of susceptible hosts, latent period become shorter. In contrast, at low host density, latent period tend to be longer (Abedon et al., 2003). In general terms, it is safe to state that the optimum lysis timing trade-off is applicable for all obligate lytic viruses (Goldhill and Turner, 2014).

The nature of the viral genome, environmental stress factors and host range deciding proteins can affect the virus trade- offs (Goldhill and Turner, 2014). How these environmental stress

factors affect trade-offs is an understudied question. There has been demonstrated lowered reproduction, fecundity (decreased burst size) and lesser thermo-stability as adaptation costs (= trade-offs) under experimental conditions with selective pressures such as; heat shock, low pH, prolonged transmission (Goldhill and Turner, 2014 and references therein). Nonetheless, some studies did not show trade-offs to environmental stress factors (McGee et al., 2014). Various mechanisms engaging adaptive mutations contribute to theses survival trade-offs under the environmental stress (Goldhill and Turner, 2014).

1.10.4 Virus trade-offs relating to host traits (resistance to infection and virulence)

Virus traits and host traits are closely connected (Record et al., 2016), since virus genome is produced by the host. For that reason, virus trade-offs and host traits are also closely linked, for e.g. "competitive and defensive traits in hosts" (Record et al., 2016). Further, it has been reported increase in viral BS with host cell size (volume) (Weinbauer and Hoefle, 1998, Weimbauer and Peduzzi, 1994).

There are several other viral trade-offs have been presented in the literature; between stability and virulence, replication fidelity and immune escape, slower reproduction at cost of strong (stable) capsid (survival) and fecundity/longevity trade-off (Heineman et al., 2012).

Nevertheless, viruses infecting eukaryotes can hold both extreme stability and high virulence which has given a name "curse of the pharaoh" (Goldhill and Turner, 2014).

1.11 Aim of the current study and Hypotheses

A lot of data on burst size, latent period, host and viral properties are available but scattered in different fields of the literature and need to be assembled and systemized to draw generalized conclusions on traits and trade-offs across virus groups. Such collection and cataloguing will reveal any knowledge gaps and aid to identify future research needs in those areas.

The objective of this study was to

- Collect literature data on growth parameters (traits) for burst size, virion (capsid) size, latent period, genome size (of host and virus) for different virus groups.
- Based on the literature study, corroborate whether metadata reveal trade-offs in different virus groups;
    - ds/ss DNA , ds/ss RNA viruses

    - Siphoviridae, myoviridae and podoviridae bacteriophages
    - Between each pair of traits; BS vs LP, BS vs GS, BS vs VS, LP vs GS , LP vs VS and GS vs VS
- Research questions scrutinized in this study:
    - Are there any significant relationships between traits in various virus groups?
    - Are there any identifiable or distinguishable traits within virus groups?
    - Can we extract general knowledge from these data that can be used in models to understand the role of virus? For example, are there trade- offs (some models assume trade-offs, others don't)

Guided by previous work, here in this study, I am trying to combine published research data and available theories to comprehend viral trait diversity. As a first step. I compile published studies to search variation in BS, LP, GS and VS across diverse virus groups. Then I tried to find correlations between these traits. I have generated the following hypotheses based on the available knowledge on viral traits and trade-offs:

a) There is a positive correlation between LP and BS
b) There is a positive correlation between LP and GS
c) There is a positive correlation between LP and VS
d) There is a negative correlation between BS and GS
e) There is a negative correlation between BS and VS
f) There is a positive correlation between VS and GS
g) There is a negative relationship between LP and host growth rate
h) There is a positive relationship between BS and host: virus genome size ratio
i) There is a positive relationship between BS and host cell volume
j) There is a relationship between latent period and host: virus genome size ratio

I tried to find any patterns of trait variation (is there any or not, and if there are trends, are they robust or weak?). Finally, I tried to connect trends with responsible mechanisms.

2. Methods: Data synthesis

Compilation of viral traits

Here in this literature survey study, I searched for published literature from various databases connected to Oria UiB (library data base at university of Bergen), using key word combinations and sometimes through cited work from publications. Around 200 publications, containing both burst size and latent period data, were selected. By reviewing literature, I gathered information over 250 virus-host systems infecting unicellular hosts from both ecological and clinical studies. Results data are summarized in (Table 3-13 in Appendix).

All selected literature was summarized in a descriptive pivot table, with raw data gathered on virus traits; burst size, latent period, genome size, nucleic acid type (dsDNA/ssDNA/dsRNA/ssRNA) , virion size (capsid and tail measurements) and host characteristics for various viruses and phages infecting both prokaryotic and eukaryotic hosts, across wide range of environments. I also recorded the virus name, host species and taxon (cyanobacteria, diatom, chlorophyte, dinoflagellate etc) location and source of virus isolation, environment (freshwater, marine, soil etc.). I also include the method used for BS estimation (TEM, FCM, free virion count or count of plaque forming (infectious) units) together with temperature, m.o.i, and other special experimental conditions utilized for one step growth curves.

Then I categorized them into various groups, eukaryotic viruses into (dsDNA, ssDNA, dsRNA and ssRNA), bacteriophages into families (*Siphoviridae*, *Myoviridae* and *Podoviridae* and diverse other families). To test a variety of hypothesized correlations between viral traits, graphs were made using above data, between each trait pair for each virus category for e.g. BS vs LP, BS vs GS, BS vs VS, LP vs GS, LP vs VS and GS vs VS etc.

When a paper gives a value range for a trait, the mean value was calculated and used in graphs. I looked for any prominent relationships between traits within various virus groups and compared virus groups for any identifiable or distinguishable traits among or specific to them. **Why select those traits; BS, LP, GS and VS?** The reason for focusing on the above traits is that, they are the key quantitative viral traits as well as there is abundance of data on those traits are available in literature, scattered and waiting to be collected and categorized.

Prokaryote viruses categorized again into smaller groups, based on host characteristic. Such as; fresh water, marine, cyanobacterial, archaeal, phytopathogenic, halophilic, thermophilic,

psychrophilic, enteric, lactic acid producing, Roseobacter clade and phages of rhizosphere bacteria etc.(Ackermann and Prangishvili, 2012) has also used same system of grouping viruses after their host, since prokaryote viruses are basically “host-genus-specific”, and typically species-specific and even strain-specific. However, there are some polyvalent phages, which are capable of infecting hosts from unalike genera or species, for e.g. phages of enterobacteria (Hamdi et al., 2017) and viruses of brown algae (Short, 2012).

Out of the dimensions given by the references, head (capsid) diameter was taken to represent the virion size. When there are data for both capsid diameter and capsid length, the larger value was selected as virion size considering its impact on capsid volume which provide the room for viral genome.

Psychrophilic bacterial phages were removed from the analysis, since their extremely prolonged latent periods (which might had affected by temperature) masks any other existing correlations within the particular groups. When there is first and second burst data from one step growth curve experiments for e.g. (López-Cuevas et al., 2011), only first burst values were taken into pivot table.

3. Results and discussion of the results

The literature compilation gathered data on ~50 eukaryote virus strains, out of that, 28 were dsDNA, 9- ssDNA, 1- dsRNA, 10- ssRNA and 4 unclassified viruses of eukaryotes. Apart from eukaryote viruses, there were ~ 140 bacteriophage varieties, including 51 *Siphoviridae* members, 52 *Myoviridae*, 29 *Podoviridae*, 2 unclassified phages, 3 *Cystoviridae* (dsRNA), one each from *Tectiviridae* (dsDNA) and *Microviridae* (ssDNA), see Table 3-13 in the appendix. In addition, there were 13 archaeal viruses and 43 cyanophages also included in the present study. Further, the host pool comprises of more than 30 unicellular eukaryotic species; including chlorophytes, diatoms, haptophytes, raphidophytes, dinoflagellates, ~35 strains of cyanobacteria and 6 archaeal species (Table 3-13).

The genome size of the eukaryote viruses (collected in this study) ranges from 4.4-560 kb, where size range for dsDNA viruses are 77-560 kb, for ssDNA 5.5-7 kb and for ssRNA viruses 4.4-11.2 kb (Table 1). All the size ranges for other traits are summarized in the Table.1. According to the obtained results through literature, there is a more than one order of magnitude variation in virion head (capsid) size across the virus families while the variation is more than two orders of magnitude when it comes to genome sizes of the different virus families. There are four orders of magnitude variation in burst sizes and latent periods (Table 1). There is a clear distinction between dsDNA virus and ssDNA/RNA eukaryote virus in relation to virion size, where the dsDNA viruses are larger in one order of magnitude than the single strand viruses. Such clear differentiation is not apparent among three phage families. (Table 1). In his review, Weinbauer, (2004) reported an average head diameter of 55-64 nm for prokaryotic viruses in marine environments. In addition, a virion head size variation with different layers of the freshwater lake system, where phages in oxic surface layer shows the smallest average head size while it increases with the depth.

Table 1. Range of reported latent periods, burst sizes, genome sizes and virion sizes for different virus and phage categories. Summarized data from Table 3-13 in the appendix.

	Latent period	Burst size	Genome size (kb)	Virion size (nm)	Host
dsDNA	2-72 Hrs	25-4100	77-560	113-310	Pry, HNF,Ch,,Pra,Rh,Di
ssDNA	12-96 Hrs	29-22000	5.5-7	32-38	Baci
ssRNA	<8- < 80 Hrs	66-93400	4.4-11.2	22-38	Baci, Thr
Cyanophages	2-96 Hrs	15-400	28-196	50-97	cyanobacteria
Siphoviridae	10-10800 min	5-794	13.4-180	40.92-140	B, Ar
Myoviridae	8-240 min	5-750	8-250.4	10-164	B, Ar
Podoviridae	10-300 min	10-9000	18-90	41-153	B
Archaeal viruses	2-21 Hrs	20-470	14.4-35.8	44-900	Ar

Bold and blue text- viruses of eukaryotes, B-Bacteria, Ar-Archaea, Pry-Prymnesiophyceae, Chl-Chlorophyceae (green algae), Pra-Prasinophyceae, HNF-heterotrophic nanoflagellate, Rh-Rhaphidophyceae, Di-Dinophyceae, Baci-Bacillariophyceae, Thr-Thraustochytriaceae

The ICTV 9th report (King et al., 2011) also has indicated size ranges for *Myoviridae* members as; head 60-145 nm, elongated heads 80-110 nm, tail 16-20 X 40 455nm , genome size 31-317 kb. According to above reference, size ranges for *Podoviridae* members are; head 60-70 nm, tail 10-20 nm, genome size 16-78 kb. For *Siphoviridae*; head 40-80 nm, tail 5-10 X 100- 210 nm, genome size 21-134 kb. Approximately 100 new bacterial and archaeal viruses described per year (Ackermann and Prangishvili, 2012). Since the ICTV 9th report, there are possibly 800 new viruses that have been described, and the variation between Table 1 and King et al., (2011) may be due to these new discoveries. I took into consideration only the references which indicated burst size and latent period data, and therefore I gathered only a fraction of references available on these families.

3.1 Viruses of eukaryotes

3.1.1 dsDNA viruses

3.1.1.a Relationship between burst size and latent period

There is a weak positive correlation between burst size and latent period (hrs) for dsDNA viruses, $R^2 = 0.3118$ (Fig.6 B).

Longest latent period for dsDNA viruses in eukaryotes

The maximum latent period observed is 60 (48-72) hrs in virus (HcV03)- infecting *Heterocapsa circularisquama* HU9433-P (Tarutani et al., 2001) (Fig. 6A and 6B). *Heterocapsa circularisquama* is a dinoflagellate which can cause mortality in shellfish. HcV03 virus belongs to the family- - *Phycodnaviridae*. (Nagasaki et al., 2003) has also reported lengthy latent periods for *H. circularisquama*, 40 hrs at 25º C and 56 hrs at 20 º C. HcV03 demonstrate species specificity but is able to infect all 18 strains tested. The authors have suggested that the reason for this longer latent period and larger burst size (>1300) compared to other algal viruses is large host cell size (approximate dimensions -length 24.5x 17μm width).

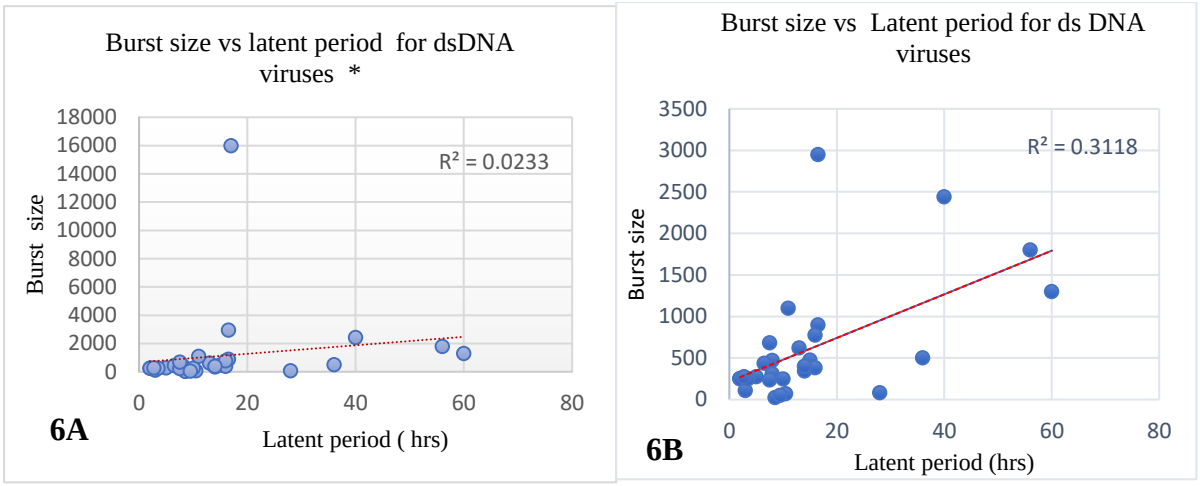

Fig. 6 Relationship between burst size and latent period (hrs) for dsDNA viruses infecting eukaryotes. **(A)** *including the extreme data point (burst size=16000). **(B)** *Excluding the extreme data point (burst size=16000). Data are given in (Table 3.1-3.3). All the data are derived from the literature cited in (Table 3.1-3.3).

Shortest latent period for dsDNA viruses in eukaryotes

The shortest latent period of approximately 2 hrs belongs to the chloro-viruses; OSy-NE5 and PBCV-1, which infect two *Chlorella variabilis* strains (Fig. 6A and 6B). The chlorovirus host, *C. variabilis* (synonym -zoochlorellae) is an ex-endosymbiont of *Paramecium bursaria* (Protozoan) (Quispe et al., 2017). Even if the authors have not stated the latent period clearly, it was calculated from the given one step growth curve graph in the reference. There are some other literature, which also show similar shorter latent periods, 2.5 to 3 hrs and 3 hours in PBCV-1 (Paramecium bursaria chlorella virus-1) grown in light and dark conditions,

respectively (Van Etten et al., 1983). It has observed that continuous dark conditions have no influence on the latent period while reduce the burst size by 50%. Compared to its large size (190 nm), large genome size (327 kb for OSy-NE5 and 334 kb for PBCV-1) and complexity as an eukaryote infecting virus, such a short latent period and growth cycle is noteworthy (Van Etten et al., 1983).

Highest burst size for dsDNA viruses in eukaryotes

Highest burst size among dsDNA virus is, 16000 for virus OIs1- (small morphotype) in *Heterosigma akashiwo* (Lawrence et al., 2006). *Heterosigma akashiwo* is a bloom forming toxic algal species (*Rhaphidophyceae*), which is harmful to fish. It is distributed in temperate coastal habitats. There are huge differences in burst size between two morphotypes of the virus OIs1. The large morphotype has burst size 1100 and latent period 11 hrs, while the smaller morphotype has burst size (BS) 16000 and latent period 17 hrs. It is sensible that the high burst size belongs to the smaller morphotype of the virus (particle size-30 nm and genome size-20 kb) compared to the larger morphotype (particle size-80 nm and genome size-130 kb). There is more room inside the host cell for high number of smaller size virus particles. Besides that, when allocating the limited host resources to make viral progeny, there is a **trade-off** of high numbers of small morphotype against low number of larger morphotype. The same study has also characterized another ssRNA virus (HaRNAV) infecting *H. akashiwo*. Its infection characteristics are totally differed from previously described dsDNA virus, OIs1. HaRNAV shows latent period 29 hrs and much higher BS of 21000. This is a clear example that the infection characteristics are rather virus depended than host dependent. When the same host is infected by different viruses, distinguishable infection characteristics are observable. The authors of this study (Lawrence et al., 2006) have proposed that the intrinsic dynamics and biological variability of rapidly fluctuating phytoplankton blooms, allow two virus types to coexist in the same host, here in this example. The dsDNA virus OIs, which have the shorter LP and the shorter lytic cycle (rapid progeny release thus swift propagation), have the competitive advantage over RNA virus during the high host density. Anyhow, during bloom termination and thus low host density, HaRNAV with its longer latent period and higher BS, have the upper hand. However, in some other cases heterogeneity can better explained the paradox of the viruses (Lawrence et al., 2006).

Note that Fig.6A and 6B are both derived from the same data set. The extremely high data point of BS= 16000 has been removed from the graph in Fig.6B, which enables us to get a closer look on the other data points.

The second largest burst size of dsDNA viruses is 3000 (1800-4100) for CeV- 01B infecting *Chrysochromulina ericina* (Sandaa et al., 2001). It has a genome size of 510kb and a virion size of 160nm.

Lowest burst size for dsDNA viruses in eukaryotes

Virus OtV5 (host-*Ostreococcus tauri*- OTH95), has the lowest reported burst size (=25) for eukaryote infecting dsDNA viruses (Derelle et al., 2008b). The second lowest burst size (=51) is observed in OlV7 (more virulent virus type and under limited light condition) infecting *Ostreococcus lucimarinus* (Zimmerman et al., 2019) (Fig 6 B). *Ostreococcus* is a widely distributed pico-prasinophyte alga, smaller than *Micromonas spp.* OlV7 has capsid diameter of 140nm and genome size approximately 200kb. However results should be interpreted carefully for OlV7, since the experimental setup adopted in this study differs from standard one-step growth curve type which has followed by majority of the other literature. Nevertheless, under higher irradiance, burst size of OlV7 increased approximately 50 times. Even though *Ostreococcus* are the smallest eukaryotes known to date, measuring ≈1-µm cell diameter, it is difficult to interpret the host cell volume as the limiting factor for low burst size. Since it has demonstrated that, under high irradiance (=higher host growth rate) *Ostreococcus lucimarinus* can harbour more than 1000 virions. Instead, the limiting factor here could be the host physiology (growth rate).

3.1.1. b Burst size and latent period variation compared to genome size of the dsDNA viruses of eukaryote

Genome size for dsDNA viruses of eukaryotes varies from **77-560 kb** (Fig.7). There is no prominent correlation between burst size and genome size (kb). Neither between latent period and genome size (Fig.7A and 7B). Largest genome size belongs to the virus, PoV-01B (host-*Pyramimonas orientalis*) which is 560 kb, 14-19 hours latent period , burst size of 800- 1000 and a capsid size of 220x180 nm (Sandaa et al., 2001). Next largest genome size is 530 kb, H. ericina virus RF02 (HeV RF02), which has a broad host range (hosts- *Haptolina ericina* strains and *Prymnesium kappa* UIO033). It has a latent period of 14-18 hrs, burst size of 775 and 190±20×160±10 in size (Johannessen et al., 2015). The former is in the family, *Phycodnaviridae* and the latter is in the *Mimiviridae* family. The *Mimiviridae* family belongs

to the Nucleocytoplasmic large DNA viruses (NCLDV), together with certain prymnesioviruses and prasinoviruses and chloroviruses. (Cottrell and Suttle, 1991) report the dsDNA virus with the smallest genome 77-110 kb, MpV (host- *Micromonas pusilla*). It also has the smallest (113 nm) virion size. The host, *Micromonas pusilla* is a 1.5-2 µm large photosynthetic marine flagellate (*Prasinophyceae*).

This data point (with smallest genome and virion size), is not included in the fig 7A and 7B, since the study does not provide latent period or burst size. The next smallest genome 177 kb belongs to the PgV Group II A and PgV Group II B (host-*Phaeocystis globosa*). It has a latent period of 12 and 16 and a mean burst size of 345 and respectively (Baudoux and Brussaard, 2005). The host is a member of harmful bloom forming marine *Prymnesiophyceae.*

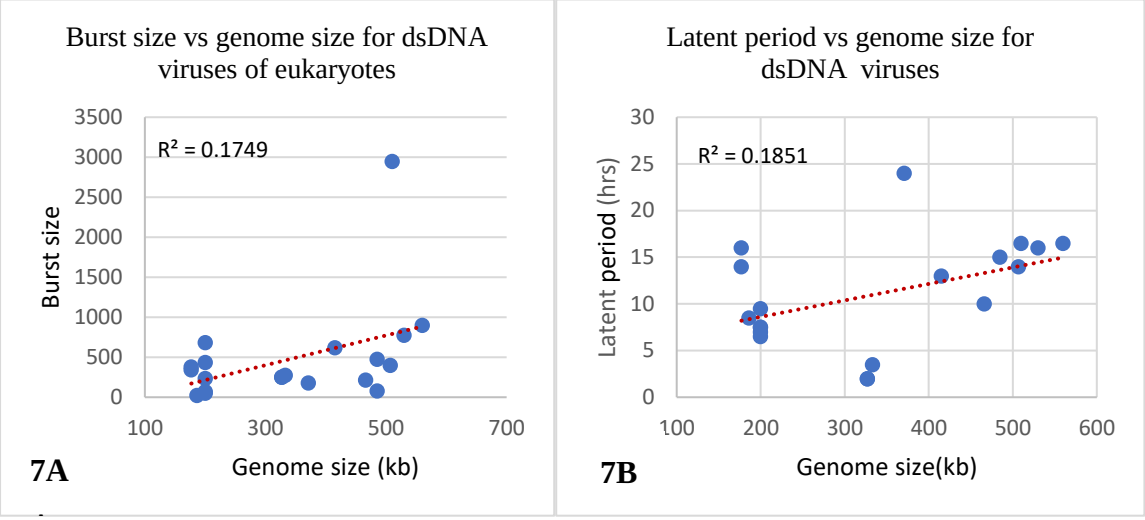

Fig. 7 (A) Relationship between burst size and genome size (kb) (B) Relationship between latent period (hrs) and genome size (kb) for dsDNA viruses of eukaryotes. Data are given in (Table 3.1-3.3). All the data are derived from the literature cited in (Table 3.1-3.3).

Since several studies with large burst size values has not given the genome size or virion size, those were excluded from the plots above.

3.1.1.c Burst size and latent period vs virion size

Virion (head) size for the dsDNA viruses of eukaryotes varies from 113-310 nm (Fig.8). The largest virion is Prymnesium kappa virus RF01 (PkV RF01) (hosts-*H. ericina* strains and *P. kappa* UIO033) This largest virion does not contain the largest genome for dsDNA virus group, instead it has a much smaller genome of ~ 310 kb. It is also include in the *Mimiviridae*

family (Johannessen et al., 2015). There is no correlation between burst size and virion size (nm). Neither between latent period and virion size of dsDNA viruses (Fig 8A and 8 B).

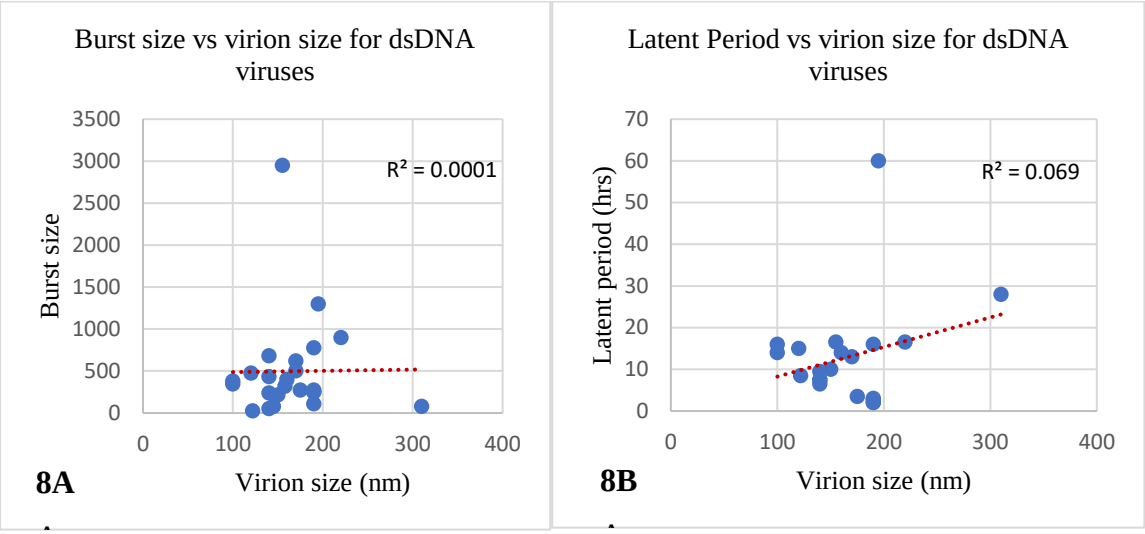

Fig. 8 (A) Relationship between burst size and virion size (nm) **(B)** latent period and virion size (nm) for dsDNA viruses of eukaryotes. Data are given in (Table 3.1-3.3). All the data are derived from the literature cited in (Table 3.1-3.3).

3.1.1.d Virion size and genome size

When we compare the 77-110 kb genome and 560 kb largest genome, genome size increases approximately x5 times while virion size only has doubled (Fig.9). However, we can observe a general trend that, with increasing genome size, virion size increases as well (R^2= 0.387) in dsDNA viruses (Fig.9).

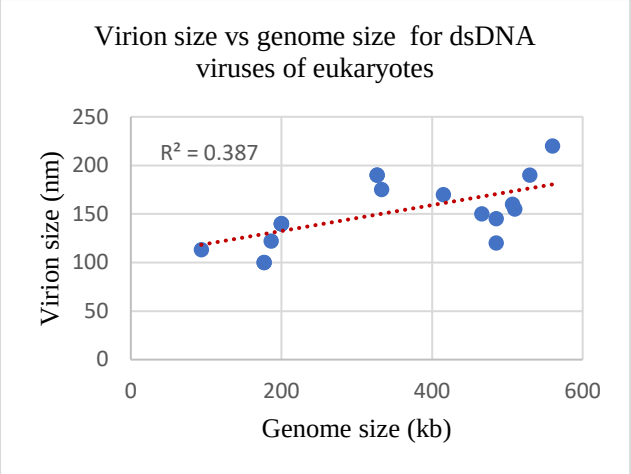

Fig. 9 Relationship between virion size (nm) vs genome size (kb) for dsDNA viruses of eukaryotes. Data are given in (Table 3.1-3.3). All the data are derived from the literature cited in Table (3.1-3.3).

3.1.2 ssDNA viruses

3.1.2. a Burst size vs latent period

According to the R^2 value (0.0653), it can be concluded that there is no apparent correlation between burst size and latent period (hrs) for ssDNA viruses (Fig.10). All the ssDNA viruses found in the literature have diatom hosts (Table.4). **The longest latent period** (96 hours) is displayed by CtenDNAV (host-*C. tenuissimus*) (Tomaru et al., 2011a). Then there are several diatom infecting viruses, which show more or less similar latent periods, approximately 48 hours, for e.g. ClorDNAV (host-*Chaetoceros lorenzianus* Grunow (Tomaru et al., 2011b) and CsetDNAV (host -*Chaetoceros setoensis* IT07-C11) (Tomaru et al., 2013). It seems like latent period around 48 h, gives the highest burst size, while both shorter (< 24 hrs) and longer latent periods do not yield high burst sizes.

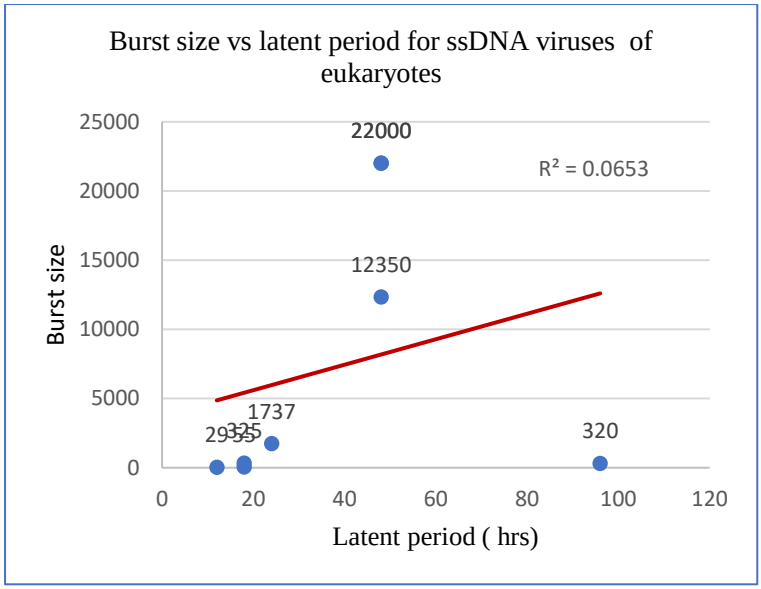

Fig. 10 Relationship between burst size and latent period (hrs) for ssDNA viruses of eukaryotes. Data are given in Table 4. All the data are derived from the literature cited in Table 4.

Shortest latent period for ssDNA viruses of eukaryotes

Bacilladnavirus- Csp07DNAV, (host-*Chaetoceros* sp. Strain SS628-11) has the shortest reported latent period (<12 hours) among ssDNA viruses (Kimura and Tomaru, 2013) (Fig.10). Next, there are two viruses that have approximately the same mean latent period of 18 hours (12-24 hours), CsalDNAV (CsNIV) host -*Chaetoceros salsugineum* ((Nagasaki et al., 2005) and CdebDNAV (host-*Chaetoceros debilis*) (Tomaru et al., 2008) (Fig.10). All these three viruses have smaller burst sizes, 29, 59 and 325 respectively.

Highest burst size for ssDNA viruses of eukaryotes

Among the ssDNA viruses, ClorDNAV (Bacilladnavirus), (host-*Chaetoceros lorenzianus* Grunow, bloom forming diatom belong to *Bacillariophyceae*) has the highest burst size of 22000 (Tomaru et al., 2011b) (Fig.10). Chaetoceros setoensis ssDNA virus (CsetDNAV) comes next, with mean burst size of 12350 (4700-20000).

Lowest burst size for ssDNA viruses of eukaryotes

Csp07DNAV- (host-*Chaetoceros* sp. Strain SS628-11) shows the lowest burst size of 29 (Kimura and Tomaru, 2013), followed by CdebDNAV (host-*Chaetoceros debilis*) with a burst size of 55 (Tomaru et al., 2008) (Fig.10).

3.1.2. b Burst size and latent period vs genome size

Unlike dsDNA viruses (77-560 kb), ssDNA viruses show notably smaller and limited genome size range from 5.5-7 kb (Fig.11A). There is no relationship between burst size and genome size (kb). Neither between latent period and genome size (Fig.11 A and 11B).

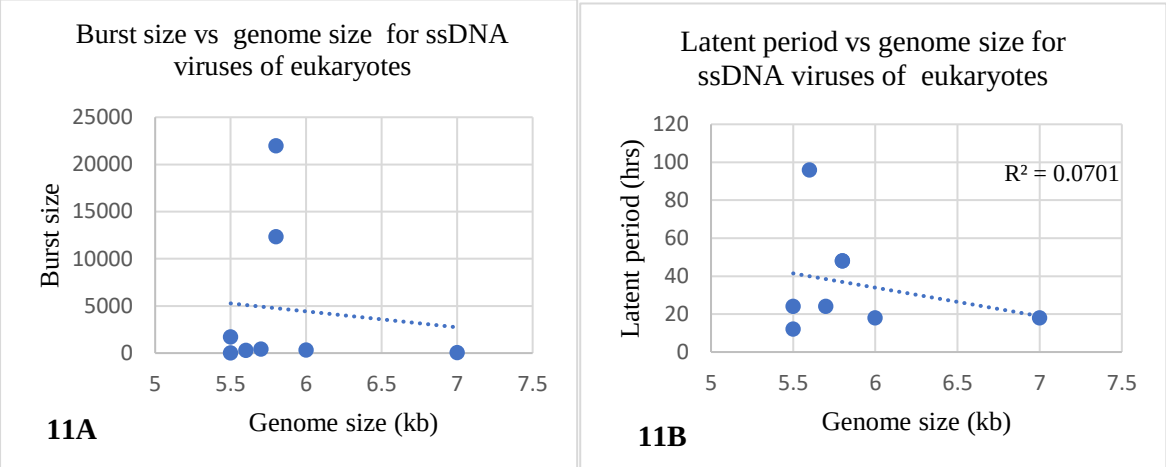

Fig. 11 (A) Relationship between burst size and genome size (kb) (B) Relationship between latent period (hrs) and genome size (kb) for ssDNA viruses of eukaryotes. Data are given in Table 4. All the data are derived from the literature cited in Table 4.

3.1.2. c Burst size and latent period vs virion size

There is no relationship between burst size and virion size (nm). Neither between latent period and virion size for ssDNA viruses of eukaryotes (Fig. 12 A and 12 B).

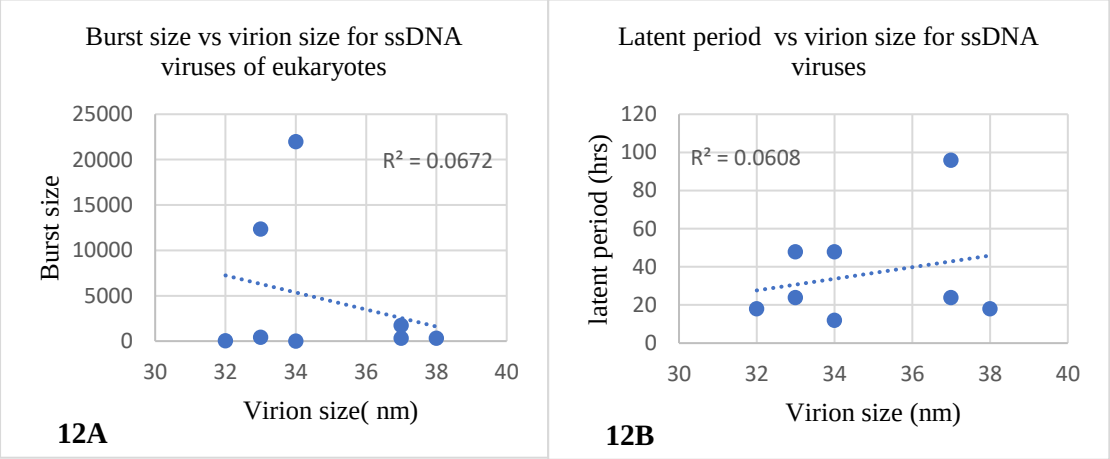

Fig. 12 (A) Relationship between burst size and virion size (nm) (B) Relationship between latent period (hrs) and virion size (nm) for ssDNA viruses of eukaryotes. Data are given in Table 4. All the data are derived from the literature cited in Table 4.

When comparing the virion size range, dsDNA viruses have a virion size range 113-310 nm, while ssDNA viruses display a much smaller and limited range of 32-38 nm. The reason might be that there is a larger host variety from where the data has been collected on dsDNA virus. On the contrary, all the reported data on ssDNA viruses came from two host species, *Thalassiosira nitzschioides* and various C*haetoceros* sp. However, within this very narrow virion size range and genome size range, there is a huge disperse of burst sizes (29-22000) as well as latent period values (12-96 hrs) (Fig. 12 A and 12 B).

3.1.3 ssRNA viruses

3.1.3. a Burst size vs Latent period

We can observe a weak negative correlation (R^2= 0.41) between burst size and latent period for ssRNA viruses of eukaryotes (Fig.13).

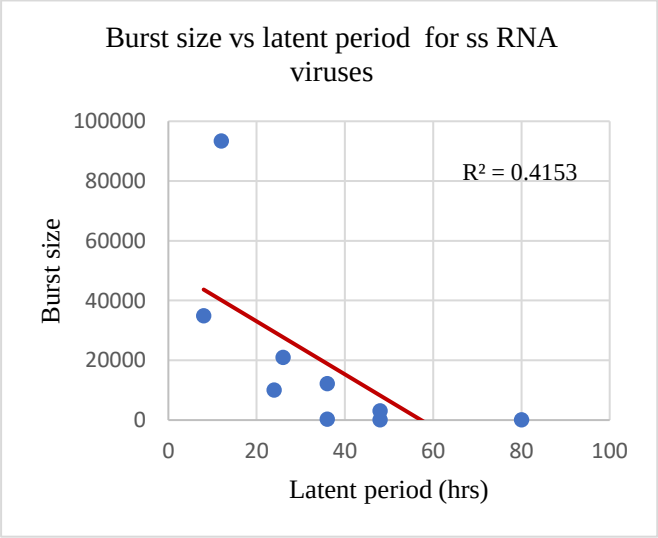

Fig. 13 Relationship between burst size and latent period (hrs) for ssRNA viruses infecting eukaryotes. Data are given in Table 5. All the data are derived from the literature cited in Table 5.

Longest latent period for ssRNA viruses in eukaryotes

RsRNAV, (host- bloom forming diatom, *Rhizosolenia setigera*) has the longest latent period of 48 hrs (Nagasaki et al., 2004). CsfrRNAV (host- *Chaetoceros socialis* f. Radians) also has similar latent period of <48 hrs (Tomaru et al., 2009a).

However, SpalV (diatom host-*Stephanopyxis palmeriana*) has a latent period of < 80 hrs (Kim et al., 2015b). The reference has not characterized the genome type and size of the virus, SpalV. They report the particle size of the virus as ~ 20 nm. The authors suggested, a ssRNA genome for SpalV, based on the fact that phytoplankton virus which has diameter less than <40 nm usually contain ssRNA genomes (Culley et al., 2003). All the diatom infecting viruses described so far in the literature has either ssDNA genomes or ssRNA genomes.

Shortest latent period for ssRNA viruses in eukaryotes

SssRNAV, infecting *Schizochytrium* sp. has the shortest latent period (<8 hours) for ssRNA viruses in eukaryotes (Takao et al., 2005). Guinardia delicatula RNA virus (GdelRNAV) has a latent period of <12 hours (Arsenieff et al., 2019). The host, *Guinardia delicatula* is a bloom forming cosmopolitan marine diatom.

Highest burst size for ssRNA viruses in eukaryotes

Guinardia delicatula RNA virus (GdelRNAV) has the highest burst size of 93400 virions per host cell (Arsenieff et al., 2019). It's small size (35-38 nm) and small genome ~9 kb, might be the reason for this highest burst size.

SssRNAV , infecting *Schizochytrium* sp. has the next highest mean burst size 34900 (5800-64000) (Takao et al., 2005). *Schizochytrium* is a cosmopolitan marine fungoid protist (class-labyrinthulea, kindom-chromista) pathogenic to molluscs. They are important decomposers in coastal ecosystems and can be found in sediment, water, algae and plants. SssRNAV has a small genome of 10.2 kb and small capsid of 25nm. The size of the host, *Schizochytrium* sp. can be 7–15 µm diameter (Honda et al., 1998). It is assumed based on geometric analysis, that the host can contain > 600000 virus particles and the burst size given above might be an underestimation (Takao et al., 2005). The second largest burst size (=21000) reported in ssRNA viruses HaRNA belongs to the family- *Marnaviridae*, (host- toxic bloom forming phytoflagellate, *Heterosigma akashiwo*) ((Tai et al., 2003). HcRNAV infecting bivalve killing dinoflagellate, *Heterocapsa circularisquama* comes next with mean burst size 12200 (3400-16000) (Tomaru et al., 2004).

Lowest burst size for ssRNA viruses in eukaryotes

According to gathered data in this study, CsfrRNAV (diatom host-*Chaetoceros socialis* f. radians) has the lowest burst size of 66 (Tomaru et al., 2009a). CtenRNAV type II infecting C. *tenuissimus* Meunier shows also low burst size of 287 (Kimura and Tomaru, 2015). It is

interesting to point out that CtenRNAV01 with the same host (*C. tenuissimus* Meunier) has much higher burst size of 10000 (Shirai et al., 2008).

3.1.3. b Burst size and latent period vs genome size

Within the members of the ssRNA viruses, there is no correlation between burst size and genome size. Neither between latent period and genome size (Fig.14A and 14B). Like ssDNA viruses, genome size variation within the ssRNA virus group is narrow (4.4-11.2kb). The same applies to the virion size variation, which occurs within a narrow limit of (22-38 nm) (Fig.15A). Similar again to the ssDNA viruses, there is a massive burst size variation (66-93400) and a latent period variation (<8- < 80Hrs) (Fig.15 A and B).

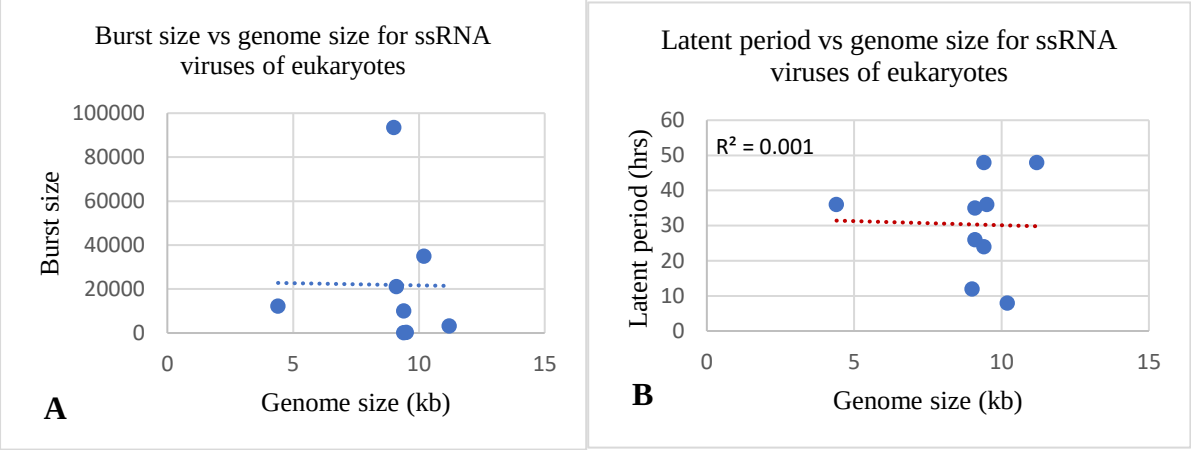

Fig. 14 (A) Relationship between burst size and genome size (kb) (B) Relationship between latent period (hrs) and genome size (kb) for ssRNA viruses of eukaryotes. Data are given in Table 5. All the data are derived from the literature cited in Table 5.

3.1.3 .c Burst size and latent period vs virion size

Within the members of the ssRNA viruses, there is no correlation between burst size and virion size (nm). Neither between latent period and virion size (Fig.15A and 15B).

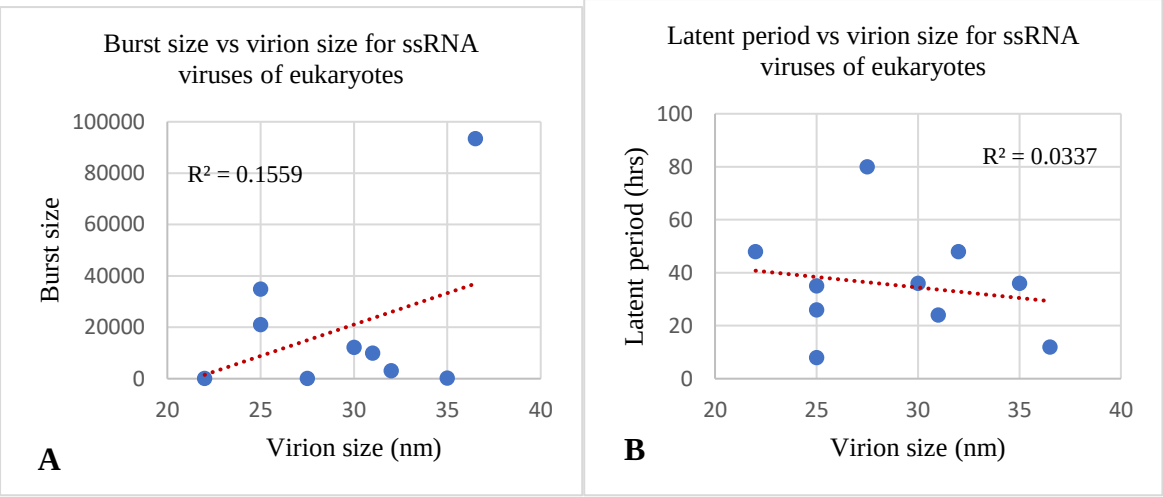

Fig. 15 Relationship between **(A)** burst size and virion size (nm) (B) latent period (hrs) and virion size (nm) for ssRNA viruses of eukaryotes. Data are given in Table 5. All the data are derived from the literature cited in Table 5.

3.2 Viruses of prokaryotes- Bacteriophages

3.2.1 Family *Siphoviridae*

3.2.1. a Burst size vs Latent period

There is no apparent relationship between burst size and latent period for dsDNA bacteriophages- Family-*Siphoviridae* (Fig.16).

There are several viruses of psychrophilic bacterial hosts that show extra-long latent periods compared to other members of the family- *Siphoviridae*. The mean latent period values range from 95, 165,240,270 360 and up to 10800 minutes. Sipho phage 9A, (host- psychrophilic bacterium, *Colwellia psychrerythraea* Strain 34H) has the **longest latent period** of them all (7200-14400 min or 5-10 d, mean- 10800 min) (Wells and Deming, 2006). However, the one step growth experiment has carried out at –10 and –12°C. This latent period is 10-20% of the generation time of its host at this temperature. The phage 9A has isolated from seawater from an Arctic nepheloid layer (a particle-rich region of the water column). The authors use the same virus host system at three different temperature, (–10 and –12), -1 and 8°C, which expressed the latent periods, 7200-14400, 240-300 and 150-180 minutes respectively.

Apart from the sipho-phages infect psychrophilic bacterial hosts, phage, FnpΦ02 (host- *Fusobacterium nucleatum*, a periodontal pathogen) has a latent period of 900min (15h) at

37°C (Machuca et al., 2010). Here, the temperature alone is not the decisive factor ruling latent period. All the above data points were not included in Fig.16.

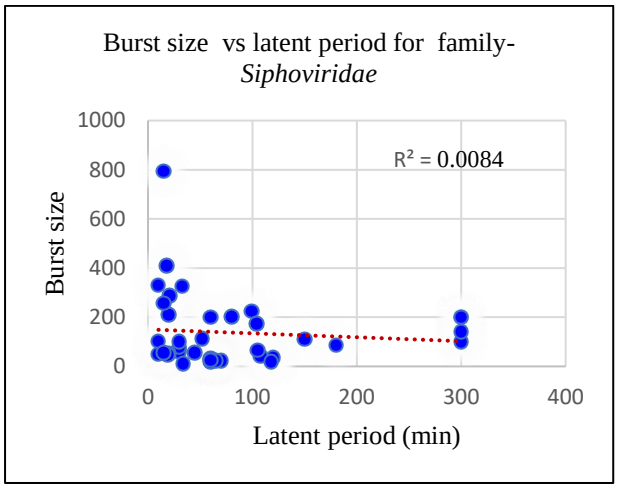

Fig. 16 Relationship between burst size and latent period (min) for dsDNA bacteriophages-Family-*Siphoviridae*. Data are given in (Table 6.1-6.5). All the data are derived from the literature cited in Table 6.1-6.5.

The next longest latent periods (300min) belongs to ΦRP1, ΦRP2 and ΦRP3 (host-*Robinia pseudoacacia rhizobia*, legume microsymbiont). Virus were isolated from soil, rhizosphere of legumes (Małek et al., 2009). Sipho phage SR2, (host-*Bradyrhizobium japonicum* CB1809, endosymbiont of soy bean) has the same 300 min latent period (Fig.16) (Appunu and Dhar, 2008) , and both are rhizobiophages.

The shortest observed latent period for Siphoviridae is 10 min. Several phages have the same value, namely; LPST10 (host- *Salmonella Typhimurium*) (Huang et al., 2018), P-16 (O111) (host- Non-O157 Shiga toxigenic *Escherichia coli* (STEC) (Litt et al., 2018), Av-05 (host- *Escherichia coli* O157:H7 (ATCC 4076)) (López-Cuevas et al., 2011), P9C, (host-*Vibrio* strain B8D, closely related to *Vibrio owensii* (Yu et al., 2013a). All of them share a common feature, they have enteropathogenic hosts and grow at 37°C. However, the explanation for this shortest latent period may not be the high experimental temperature alone, since siphoviridae phage TSP4 (host- *Thermus* strain TC4, a thermophilic bacteria), which is adapted to high temperature as 65°C , have much longer latent period of 60 m (Lin et al., 2010a) (table 6.1-6.5).

The highest burst size of siphoviridae is 794 per cell, coming from the phage, P-11 (O26) (host- Non-O157 Shiga toxigenic *Escherichia coli* (STEC) (Fig.16) (Litt et al., 2018). The next

highest number is 409 from phage AB1, (host-*Acinetobacter baumannii* KD311) (Yang et al., 2010).

Phage 9A (host- *Colwellia psychrerythraea* Strain 34H, psychrophilic bacteria) has the **lowest burst sizes** of 5 (Wells and Deming, 2006), followed by the burst size of 7, also belonging to a psychrophilic bacteria infecting phage FpV-7 (host- *Flavobacterium psychrophilum* 950106-1/1 (Stenholm et al., 2008). If we set aside the phages of psychrophilic bacteria, based on the temperature factor, phage φC6, (host-*Clostridium difficile*) has a low burst size of 19 (Goh et al., 2005a) and the phage Cp1 (host- *Xanthomonas axonopodis* pv. citri (syn., *Xanthomonas campestris* pv. citri or *Xanthomonas citri*) has more or less similar low burst size of 20 (Ahmad et al., 2014).

3.2.1. b Burst size and latent period vs genome size -*Siphoviridae*

Within the members of *Siphoviridae* phages, there is no correlation between burst size and genome size. There is only a weak positive correlation between genome size and latent period (Fig.17A and 17B). Genome size variation within siphoviridae members is 13.4-180 kb.

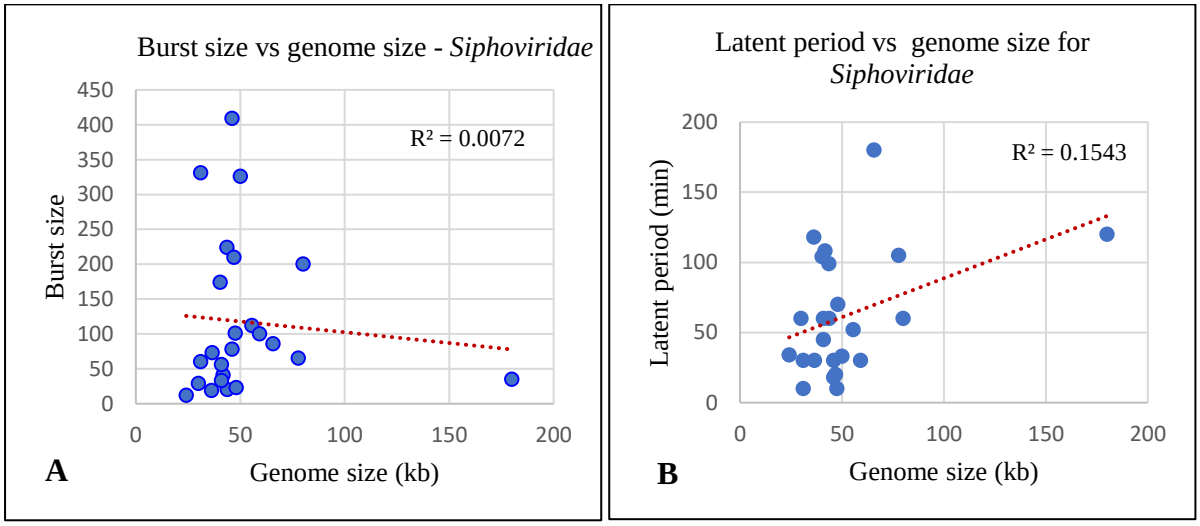

Fig. 17 (A) Relationship between burst size and genome size (kb) (B) latent period (min) and genome size (kb)for dsDNA bacteriophages- Family-*Siphoviridae*. Data are given in Table (6.1-6.5). All the data are derived from the literature cited in Table (6.1-6.5).

The largest genome within *Siphoviridae* members is 180 kb, belonging to phage XTP1 (host-*Xanthomonas campestris* pv. campestris (Weiss et al., 1994) . It has a burst size of 35 and a latent period of 120 min) (Fig.17A and 17B). The next largest genome is 80-90 kb, phage 9A (host-cold active bacterium, *Colwellia psychrerythraea* Strain 34H- from Arctic). This

particular phage also has the longest latent period among siphoviridae members. This phage host system demonstrates negative correlation between decreasing temperature and increasing latent period, but no such effect is clear with temperature and burst size. With decreasing experimental temperatures, 8, -1 and (-10 to -12), the latent period increases from (150-180), (240-300) to (7200-14400) min, while burst size varies from 5, 55 and 5 respectively (Wells and Deming, 2006). However, this data point is not included in Fig. 17 A and 17B, since phages of psychrophilic /cold active bacterial host were excluded from the graph. Phage Φps05 (host- *Pediococcus* sp. LA0281, a gram positive lactic acid bacterium) contains **the smallest genome** of 24.1 kb (Yoon et al., 2007). It also has a latent period of 34 min and a low burst size of 12. It is normally the fact that smaller genome size phages may produce higher burst size. Because normally host's nucleotide pool and break down of host genome is used to make new viral progeny rather than producing *de novo* nucleotides.

According to the gathered data from the literature, phages with the largest genome did not always produce lesser progeny and vice versa. Here we can observe that phages with smallest genomes have much low burst sizes even than the largest genome group.

There are several phages which have smaller genome sizes next to Φps05 such as, 30, 31,31 and 32 kbs (Fig.17 A and 17B). Phage P8D, P3K and P9C (host- *Vibrio* strain B8D (closely related to) *Vibrio owensii*) (Yu et al., 2013b). Phage EV3 (host- *Lactobacillus sanfranciscensis* H2A) has the 32 kb genome (Foschino et al., 2005). However, phage CG33 (host- *Corynebacterium glutumicum*) could be the member with the smallest genome of 13.4 kb (burst size 16 and latent period 18 min) among siphoviridae (Trautwetter et al., 1987). The authors have classified the phage only according to Bradley's classification (Bradley, 1967) , as group B, but according to (Ackermann and Prangishvili, 2012) Bradley's group B correspond to *Siphoviridae* family. If we try to make inferences among genome size and corresponding burst sizes of the above mentioned smallest phages (13.4 kb-16 burst), (24 kb-12 burst), (30 kb-29 burst), (31 kb-60 burst), (31 kb-331 burst) and (32kb-30 burst) still we cannot see any pattern. Because phages with nearly similar genome size of ~30 kb, shows BS variation of one order magnitude; 29-331.

3.2.1. c Burst size and latent period vs virion size - *Siphoviridae*

Within the members of phages, there is no correlation between burst size and virion size, neither between latent period and virion size (Fig. 18A and18 B). Virion size variation within siphoviridae members is 40.92-140 nm. The largest virion is ΦRP1,virion size of 60x140 nm,

(host-*Robinia pseudoacacia rhizobia*) , it has a quite long latent period (300min) and a large burst size of 200 (Małek et al., 2009). It is difficult to relate the larger virion size with host or with environment type of factors. Since some of the largest sipho-phages infect vibrio stains, while some infect marine roseobacter clade or phytopathogenic bacteria. The smallest siphoviridae phage is 39.97x40.92nm size ΦCP6-2 (host- *Serratia liquefaciens* CP7) with a latent period of 104.8 min and a burst size of 174. However it does not have one of the smallest genomes, or a higher burst size either (Ashelford et al., 1999). Both the largest and the smallest siphoviridae phages are isolated from phytosphere.

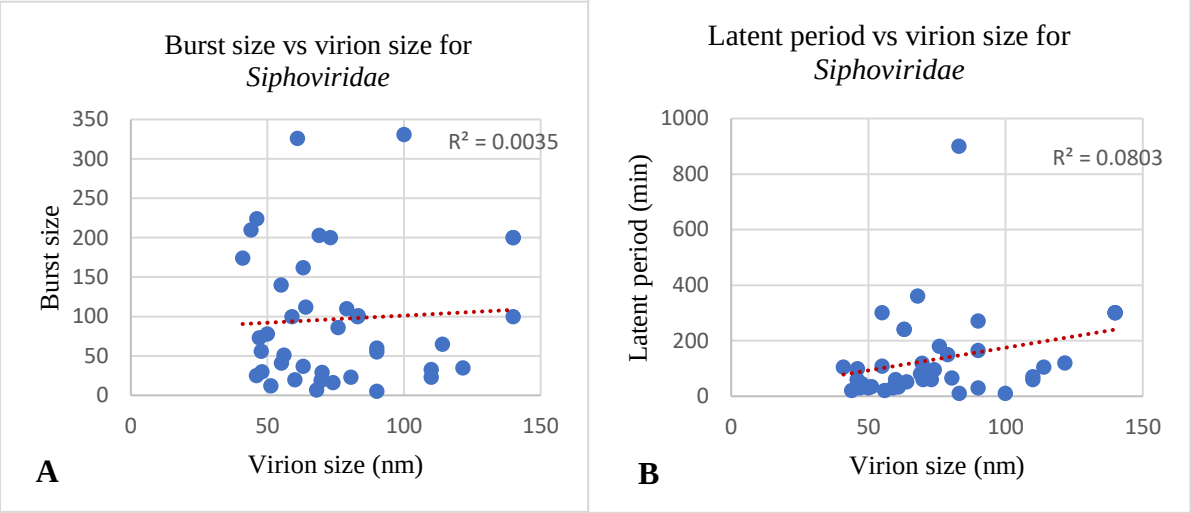

Fig. 18 (A) Relationship between burst size and virion size (nm) (B) Relationship between latent period (hrs) and virion size (nm) for family *Siphoviridae*. Data are given in (Table 6.1-6.5). All the data are derived from the literature cited in (Table 6.1-6.5).

3.2.2 Family *Myoviridae*

3.2.2. a Burst size vs latent period

It is outstanding that as a general observation, all the phages in the *Myoviridae* family show a latent period =< 100 minutes, except a phage with psychrophilic host and halophilic host (Fig.19). In contrast, *Siphoviridae* phages display much longer latent periods up to 10800 min (Table 6.1-6.5). If we remove the psychrophilic hosts from the *Siphoviridae* group, still latent periods extend up to 300 min, which is much higher than that of *Myoviridae* phages.

The longest latent period for *Myoviridae* is 240 min for phage FpV-19 (host- *Flavobacterium psychrophilum* 950106-1/1, a psychrophilic bacterium) (Stenholm et al., 2008) (Fig.19)..

Outside the psychrophilic group, phage ΦCP6-6 (host-phytosphere bacterium, *Serratia liquefaciens* CP11) has the longest latent period of ~99.5 min (Ashelford et al., 1999). Even though (Daniels and Wais, 1990) describe a phage S5100, with halophilic host, *Halobacterium cutirubrum* shows a latent period of 660 and 540 minutes at 38 ^{o}C, under different NaCl levels 4.5 M and 3.5 M respectively. They only classify it according to Bradley Group A, which corresponds to present myoviridae family. Authors have not indicated its nucleic acid type or genome size, so those data were excluded from Fig.19.

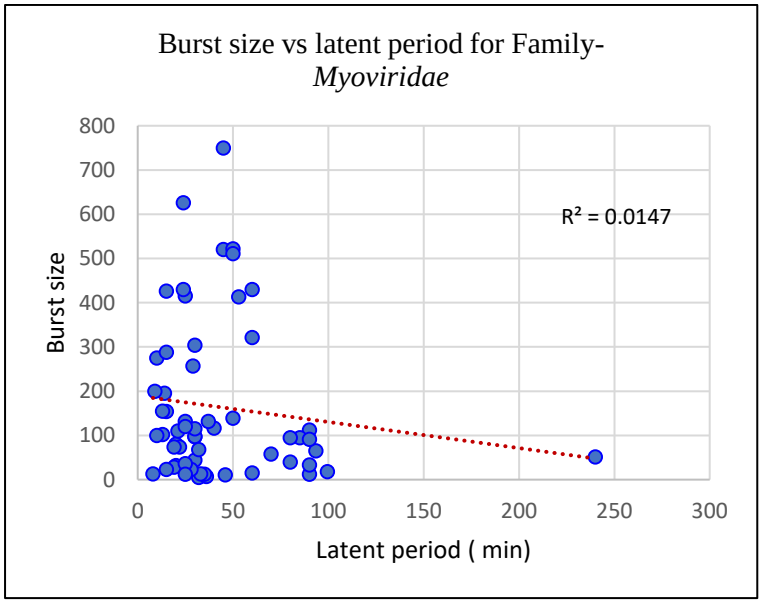

Fig. 19 Relationship between burst size and latent period (min) for dsDNA bacteriophages-Family-*Myoviridae*. Data are given in (Table 7.1-7.5). All the data are derived from the literature cited in Table (Table 7.1-7.5).

Shortest latent period for *Myoviridae*

There are several phages of Non-O157 Shiga toxigenic *Escherichia coli* (STEC) that have nearly equally short latent periods of 8 , 10, 13 minutes (Litt et al., 2018). Besides them, phage ZZ1 (host-*Acinetobacter baumannii*) has a 9 minutes short latent period (Jin et al., 2012) (Fig. 19). Above phages replicate at 37 ^{o}C.

Highest burst size for *Myoviridae*

The highest burst size of 750 belongs to T-even-like , type A2 myovirus , nt-1, (host- *Beneckea natriegens*, a halophilic species) under the experimental conditions of 0.16 M NaCl +4SN media (Zachary, 1976) (Fig. 19). Here in that study we can observe a range of burst sizes (12 - 750) for the same phage-host system under the various NaCl concentrations. Phage IHQ1(

host- *Aeromonas punctata*) also shows a high burst size of 626 (Haq et al., 2012). **The lowest burst size** of 5 and 7 belongs to phages φC2 and φC5, respectively (Fig. 19) (host- *Clostridium difficile*) (Goh et al., 2005a). Phage Y5, (host-*Lactobacillus plantarum* LA 280) has also a low burst size of 11 (Yoon et al., 2002), and phages vB_KpnM_KP15 and vB_KpnM_KP27 (host- *Klebsiella pneumoniae*) has a value 10-15 (Kasik-Szeloch et al., 2013). There are no apparent common traits among the 3 host or their phages with respect to habitat or latent periods.

3.2.2. b Burst size and latent period vs genome size- *Myoviridae*

Within the members of *Myoviridae* phages, there is no statistically significant correlation between burst size and genome size, neither between latent period and genome size (Fig 20 A and B). Genome size variation within *Myoviridae* members is (8-250.4) kb.

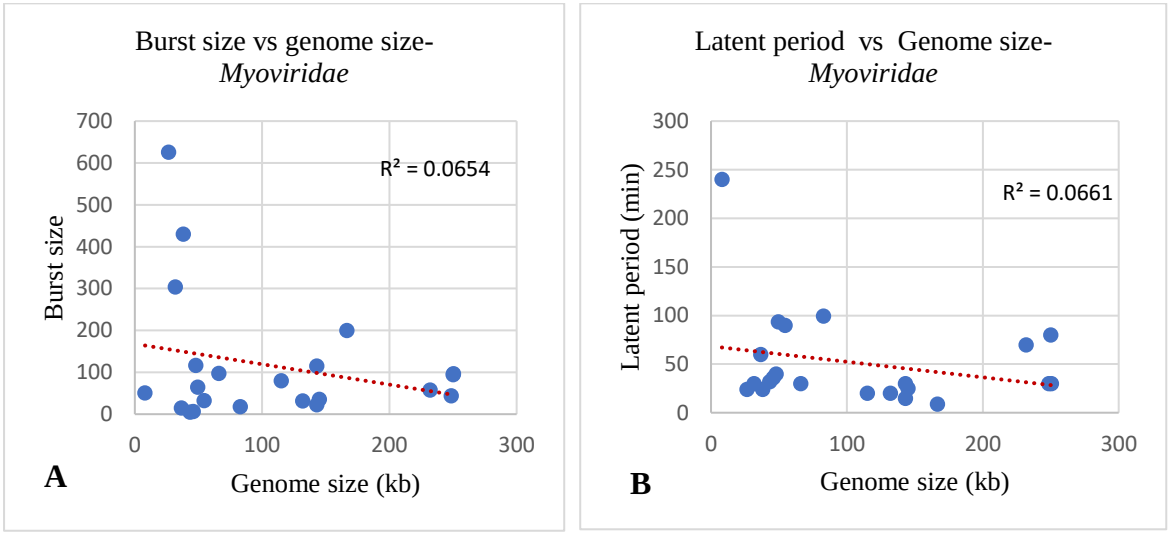

Fig. 20 (A) Relationship between burst size and genome size (kb) (B) latent period (min) and genome size (kb) for dsDNA bacteriophages- Family-*Myoviridae*. Data are given in (Table 7.1-7.5). All the data are derived from the literature cited in (Table 7.1-7.5).

Largest genomes of *Myoviridae*

Phage φSMA5 (host- *Stenotrophomonas maltophilia* T39 syn. *Xanthomonas maltophilia* and *Pseudomonas maltophilia*) has one of the largest (250 kb) genomes within the *Myoviridae* family (Chang et al., 2005) (Fig.20A and 20B). Its host is a widespread opportunistic bacterium causing nosocomial infections. The host has a wide habitat range too for e.g. water, sediment, sewage, soil, rhizospheres of plants, and frozen foods. Though it is not documented, we can assume that the phage also must have a wide habitat range to infect its host. This large genome

size (means more genes) could facilitate its survival. Despite of this large genome size, phage φSMA5 has a burst size of 95. Phage φSt2 (host- *Vibrio alginolyticus* V1) also has a large genome of 250 .4 kb (Kalatzis et al., 2016). The same study describes another phage, φGrn1 with the same host and nearly equal genome size of 248.6 kb. Then there is a third phage with 231.9 kb genome named pVa-21with same host but different strain (Kim et al., 2019) Nonetheless, there is no visible pattern either in burst sizes (95, 97, 44 and 58) or in latent periods (80,30,30 and 70) of these phages φSMA5, φSt2, φGrn1 and pVa-21 respectively (table 7.1 and 7.5) All these phages share one trait, they display broad host range, not only multiple strain but also different sub species level. This observation raises the question; is there a relationship between genome size and host range?

Smallest genomes of *Myoviridae*

Phage FpV-19 (host- *Flavobacterium psychrophilum* 950106-1/1) has the smallest genome of 8 kb (Stenholm et al., 2008) . Next comes phage IHQ1 (host- *Aeromonas punctata*) with a genome of 25–28 kb (Haq et al., 2012). (Fig.20A and 20B). FpV-19 is a fish pathogen and IHQ1is an opportunistic pathogen of human with burst sizes 51 and 626 respectively. None of the literature on phages with smallest burst size (5, 7, 11 and 10-15) have mentioned their genome sizes, so it is difficult to make a connection between the two traits. However, all the three phages with highest burst sizes have a genome size < 50 kb.

We can't make any conclusions on the relationship between latent period and genome size of myoviridae members either. For e.g., if we consider latent periods less than 50 min, there is a huge genome size variation from 24- 250 kb within that group (Fig. 20B).

3.2.2 .c Burst size and latent period vs virion size - *Myoviridae*

Within the members of *Myoviridae phages*, there is no correlation between burst size and virion size, neither between latent period and virion size (Fig. 21A and 21B). Virion size variation within myoviridae phages is 10-164 nm. Burst size, latent period data of myoviridae phages with the halophilic host *Beneckea natriegens* were excluded from the graph since the variation of the parameters were related to the changes in NaCl concentrations in the experimental setup.

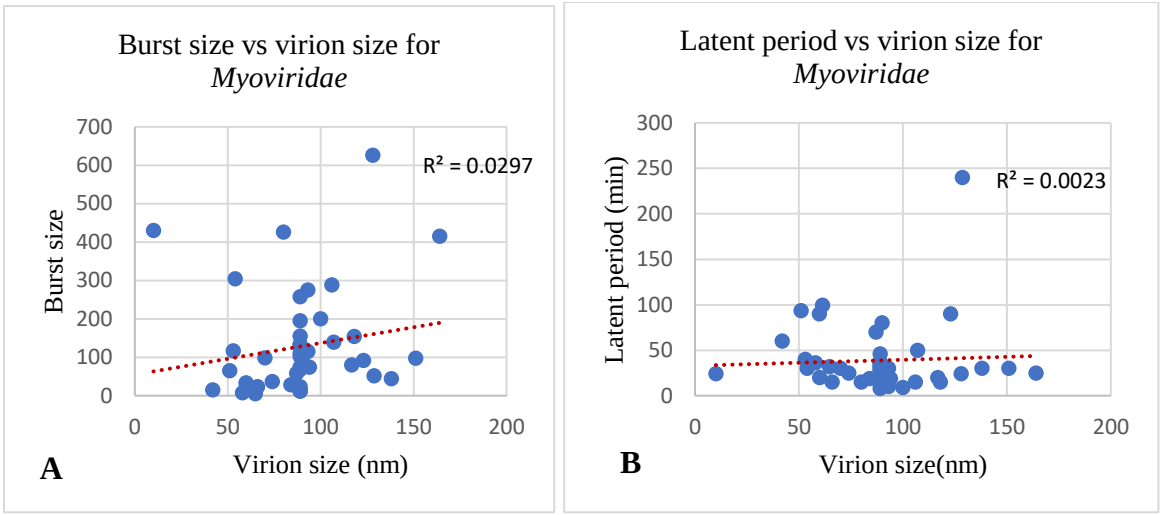

Fig. 21(A) Relationship between burst size and virion size (nm) (B) latent period (hrs) and virion size (nm) for family *Myoviridae*. Data are given in (Table 7.1-7.5). All the data are derived from the literature cited in (Table 7.1-7.5).

The largest virion size 164 nm, belongs to the VP01, (host- *Vibrio alginolyticus*) with a shorter latent period of 25 min and a quite large burst size of 415 (Sasikala and Srinivasan, 2016). The smallest virion (10 nm capsid) is WZ1, infecting *Shigella dysenteriae* with a latent period of 24 min and a burst size of 430 (Fig. 21A and 21B). Compared to its smaller size, it has a considerably large genome of 38kb (Jamal et al., 2015).

3.2.3 Family *Podoviridae*

3.2.3. a Burst size vs Latent period

Within the members of *Podoviridae phages*, there is no robust correlation between burst size and latent period (Fig. 22).

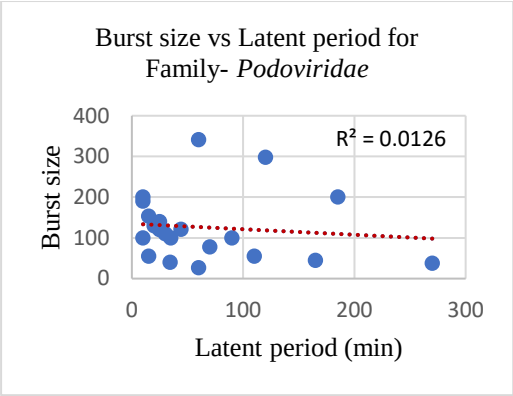

Fig. 22 Relationship between burst size and latent period (min) for dsDNA bacteriophages-Family-*Podoviridae*. Data are given in (Table 8.1-8.4). All the data are derived from the literature cited in (Table 8.1-8.4).

The highest burst size of 9000 infectious particles per cell was reported in phiAxp-3, an N4-like bacteriophage (host-*Achromobacter xylosoxidans* (Yanyan et al., 2016). This data point was excluded from the graph, allowing more visibility to inspect the other data points. *A. xylosoxidans* is a medically significant opportunistic bacterium regularly linked with nosocomial infections. It has quite large genome of 72,8 kb compared to the genome size range found within other members of the family- *Podoviridae* (18-90 kb). Virion size of the phage phiAxp-3 is 67 nm (head diameter), while the size of the other members of the podoviridae family varies between 41-153nm (Table 8.1-8.4). Therefore, neither genome size nor the virion size can explain the extremely high burst size compared to that of other members of the family which is (27-341). However, this high burst size together with its species specificity of phiAxp-3, make it very attractive biocontrol agent of its host, *Achromobacter xylosoxidans* (Yanyan et al., 2016). The second highest burst size(>1000) was found in roseophage EE36ϕ1 (host-*Sulfitobacter* sp. EE-36) (Zhao et al., 2009). This data point also has not included into the graph above (Fig.22).

The third highest burst size of 341, belongs to the podophage (RD-1410Ws-07) (host-*Roseobacter denitrificans* OCh114) (Li et al., 2016a) (Fig.22). Roseobacter lineage comprises

a group of marine α-proteobacteria. It is outstanding that significantly high (341) and low (27) burst size within podoviridae, comes from the same host - *Roseobacter denitrificans* OCh114 but two different phages, RD-1410Ws-07 and RD-1410W1-01 respectively (Li et al., 2016). The two phages have similar G+C content, approximately similar genome sizes (76.3 and 72.7 kb), genome structures, capsid diameters (70.8 and 63.2 nm), latent periods (< 60 min and 60 min), and similar host range. Moreover, one step growth curve experiment performed with similar conditions (m.o.i = 0.1, incubated at 28^{o} C). But there is low nucleotide sequence similarity between them (~ 40%) (Li et al., 2016).

The lowest burst size of 10 also belongs to a Roseophage, (vB_RsvN_RPP1) (RPP1, Roseovarius Plymouth Podovirus) (host- *Roseovarius nubinhibens*) (Chan et al., 2014) (Table 8.3).

The same virus above, (vB_RsvN_RPP1) with lowest burst size, also display the **longest latent period for podovirus**, which is 300 minutes. The same study describes another virus (vB_Rsv217_RLP1) (RLP1, Roseovarius Langstone Podovirus) with similar long latent period of 300 min. (Table 8.3). However, note that this particular study has used modified one step growth curve experiment (Chan et al., 2014). (Sinha et al., 2018) has discussed in detail that phage-host interactions in low viscosity (for e.g. liquid broth) and high viscosity (agar) media can be quite variable. Therefore, it is not included into the graph above (Fig.22) to avoid variation.

Then comes next, FpV-2 (host-*Flavobacterium psychrophilum* 950106-1/1) with a latent period of 270 min (Stenholm et al., 2008). The host is a psychrophilic bacterium and the study was performed at 15^{o}C, which explain the longer latent period associated with low temperature. Podophage SR3, isolated from rhizosphere of (host-*Bradyrhizobium japonicum*-endosymbiont of soy bean) also has a long latent period of 185 min (Appunu and Dhar, 2008) (Fig.22) .

There are 3 podophages, all displaying shortest latent period of 10 min (Fig.22). 1) ΦSPB (host-*Salmonella enterica* serovar Paratyphi B) (Ahiwale et al. 2013), 2) ϕAB2 (host- *Acinetobacter baumannii* ATCC17978) <10 min (Lin et al., 2010b) and 3) vB_PmuP_PHB01 (host-*Pasteurella multocida*) (Chen et al. 2019). *S. enterica* serovar Paratyphi B is an enteric pathogen while *A. baumannii* and *P. multocida* are opportunistic nosocomial pathogens. The explanation for short latent periods could be, all three experiments were performed at 37^{o}C.

Other than the similar experimental temperature, the viruses were isolated from fresh water (1) and sewage water (2 and 3), they have different genome sizes 59, 40 and 37.2 kb respectively. Despite the short latent period, they have quite high burst sizes 100, 200 and 190 (burst size range for podo phages is 10- 341 except the extreme point of 9000 (data from table 8.1-8.4).

3.2.3. b Burst size and latent period vs genome size- *Podoviridae*

Within the members of *Podoviridae phages*, there is no correlation between burst size and genome size (Fig. 23A). Anyhow, we can predict for very weak positive correlation between genome size and latent period for Podoviridae members $R^2 = 0.2921$. (Fig.23B). Shortest latent periods correspond with medium size genomes (Fig.23B).

Compared to Sipho- and *Myoviridae phages*, *Podoviridae* phages display a smaller range of genome size, from 18 kb only up to 90 kb. Where, sipho and myoviridae phages have a range of 13.4-180 and 8-250.4 respectively (Fig.23 A and Table 2).

The largest genome of *Podoviridae* (90 kb) belongs to the phage, FpV-2 (host- *Flavobacterium psychrophilum* 950106-1/1 (Stenholm et al., 2008). It has a burst size of 38 ± 6 and the longest latent period of 270 minutes too (Fig.23 A and 23B). Next in the line of largest genome sizes, there are several phages of marine Roseobacter clade that have genome sizes ranging between 72.7-76.5 kb. Their burst sizes range from 10-1000< and latent period varies from 60-360 min. Some of them show broad host range, while others have narrow host range (Zhao et al., 2009 , Li et al., 2016a , Chan et al., 2014). One of the **smallest genome of** is 18.7 kb, belongs to the phage asccφ28 (host- *Lactococcus lactis*, a lactic acid producing bacterium) (Kotsonis et al., 2008). The other is phage VMY22 with 18 - 20 kb genome (host- cold-active *Bacillus cereus* MYB41-22) (Ji et al., 2015). Their burst sizes are 121, 78 and latent periods are 44 and 70 minutes respectively (Fig.23 A and 23B).

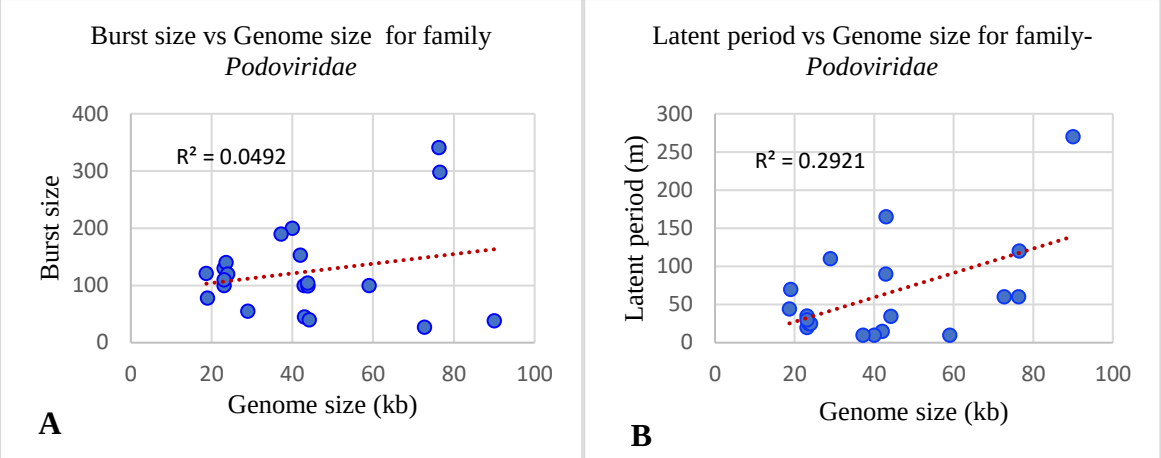

Fig. 23 (A) Relationship between burst size and genome size (kb) (B) latent period (min) and genome size (kb) for dsDNA bacteriophages- Family-*Podoviridae*. Data are given in (Table 8.1-8.4). All the data are derived from the literature cited in (Table 8.1-8.4).

3.2.3 .c Burst size and latent period vs virion size

There is no relationship between burst size and virion size of podophages (Fig. 24A).

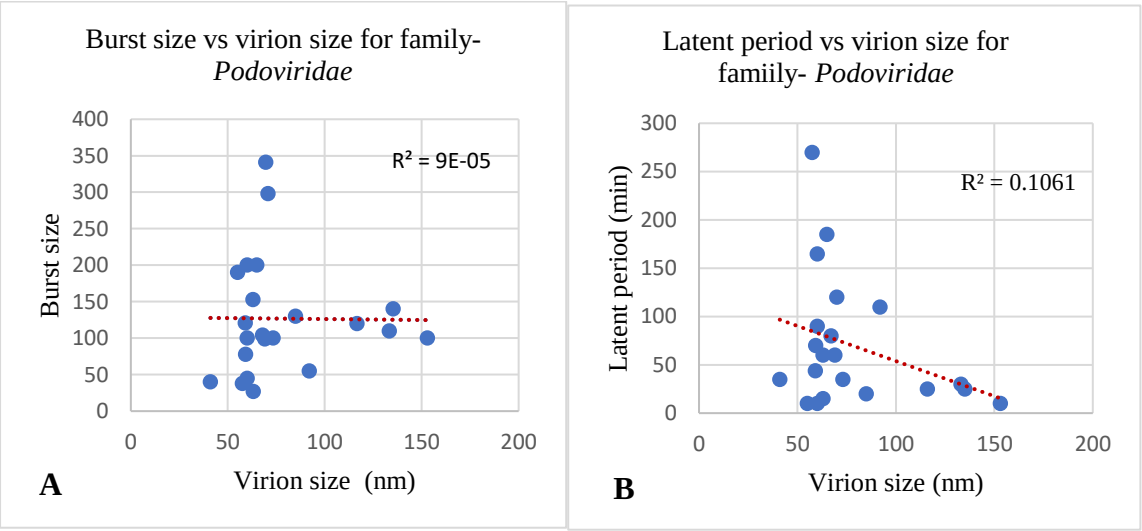

Fig. 24 Relationship between **(A)**burst size and virion size (nm) **(B)** latent period (min) and virion size (nm) for dsDNA bacteriophages- Family-*Podoviridae*. Data are given in (Table 8.1-8.4). All the data are derived from the literature cited in (Table 8.1-8.4).

The largest virion (head/ capsid) size is 153 × 57nm belongs to the phage φSPB, (host- *Salmonella enterica* serovar Paratyphi B). It has a latent period of 10 min and a burst size of 100. This phage has a 59 kb genome and a very long, cigar-shaped head (Ahiwale et al., 2013). The next largest virion is 135.2 nm head diameter, Kpn12 (host- *Klebsiella pneumoniae* B5055)

(Kumari et al., 2010). It has a much smaller genome of 23.6 kb, latent period of 25 min and burst size of 140 (Fig. 24A and 24B).

The smallest virion size is 41.03 nm ΦCP6-4, with a latent period of 35.4 and burst size of 40 (host- *Serratia liquefaciens* CP9) (Ashelford et al., 1999). Most of the podophages have a head diameter size range of 55-75 nm, but their burst sizes vary to a great extent within the limit, 27-350 virions per cell (Fig. 24A).

We cannot see a pattern between changes in latent period and virion size (Fig. 24B). Anyhow, largest virions have shorter latent periods than that of medium size virions. It should also be pointed out that, between 55-75 nm virion size, there is a huge variation in latent period from 10-270 min (Fig. 24B).

3.2.3. d Virion size vs genome size

There is no strong relationship between the virion size (nm) and genome size (kb) either (Fig.25 A, B and C). Though, for *Siphoviridae* and *Myoviridae* members, the R^2 value is 0.2 (Fig.25 A and C), thus we can predict a weak positive relationship.

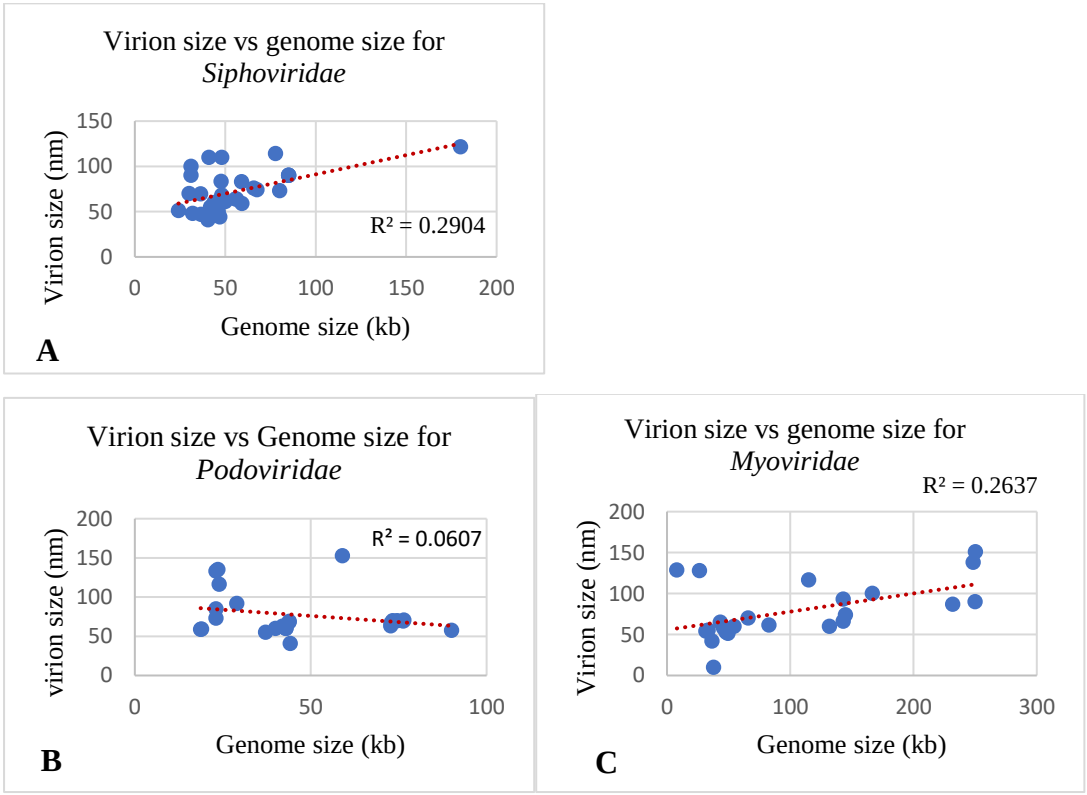

Fig. 25 Relationship between virion size (nm) and genome size (kb) for dsDNA bacteriophages (A) Family- *Siphoviridae* (B) Family-*Myoviridae*. (C) Family-P*odoviridae*.

3.2.4 Cyanophages

The reasons for analysing cyanophages separately from other prokaryote phages and using host traits into the comparison and graphs are discussed in section 4.5.7. Several virus traits, host traits and combination of both virus-host traits were used to discover any trends within cyanophages. Weak trends were observed between BS vs LP, BS vs host; virus genome ratio and LP vs host growth rate (R^2=0.3) (Fig.26B, 26C and 27D respectively). Virus capsid size and virus genome size showed moderate positive relationship with R^2= 0.56 (Fig.27E). No trends were observed between other trait pair combinations (Fig. 26A, 26D, 26E, 26F, 27A, 27B and 27C).

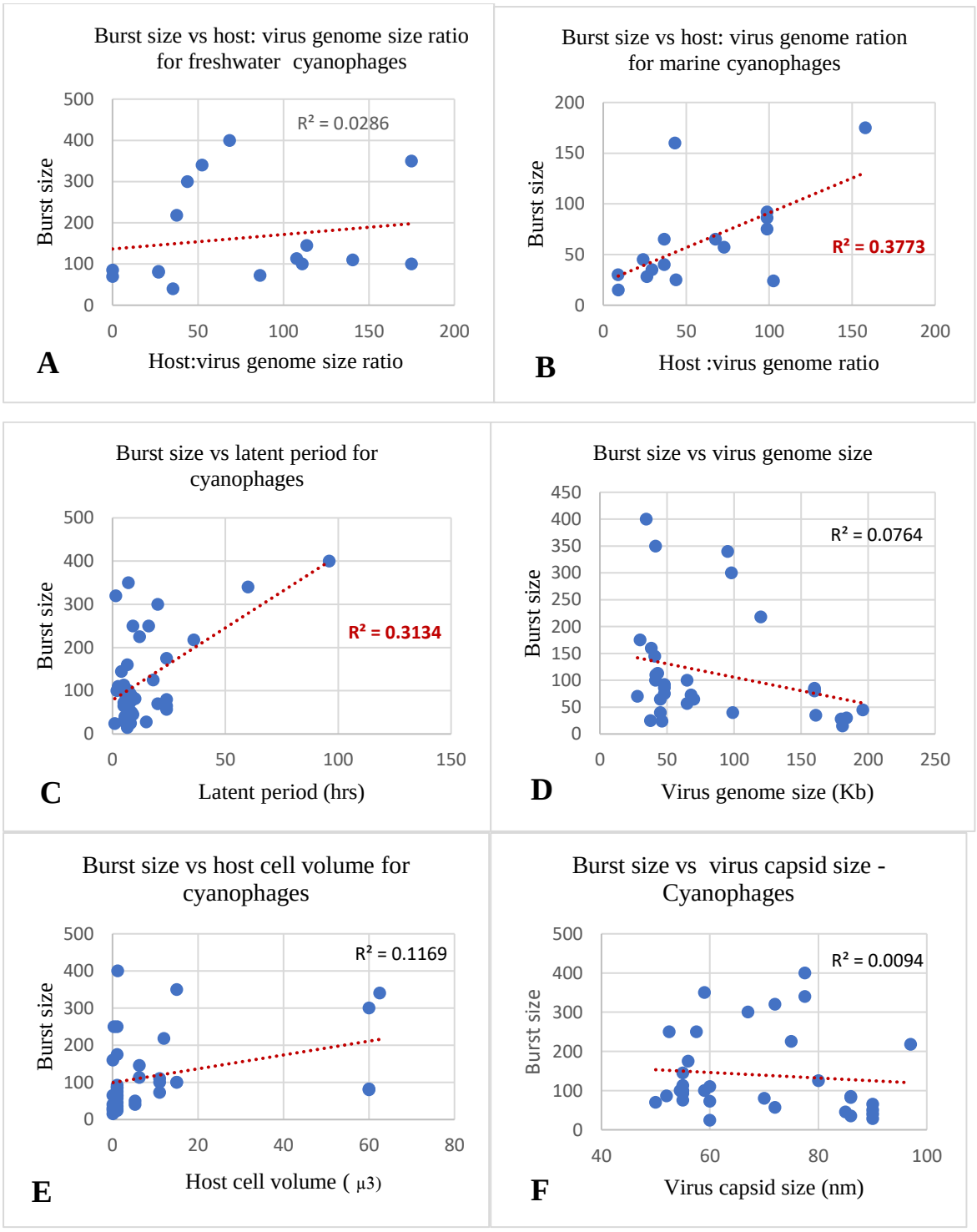

Fig.26. Relationship between (A) burst size and host: virus genome size ratio for freshwater cyanophages (B) burst size and host: virus genome size ratio for marine cyanophages (C) burst size and latent period for cyanophages (D) burst size and viral genome size (E) burst size and host cell volume (μ^3) (F) burst size and virus capsid size (nm) for cyanophages. In graphs C, D, E and F both marine and freshwater species taken together in the analysis

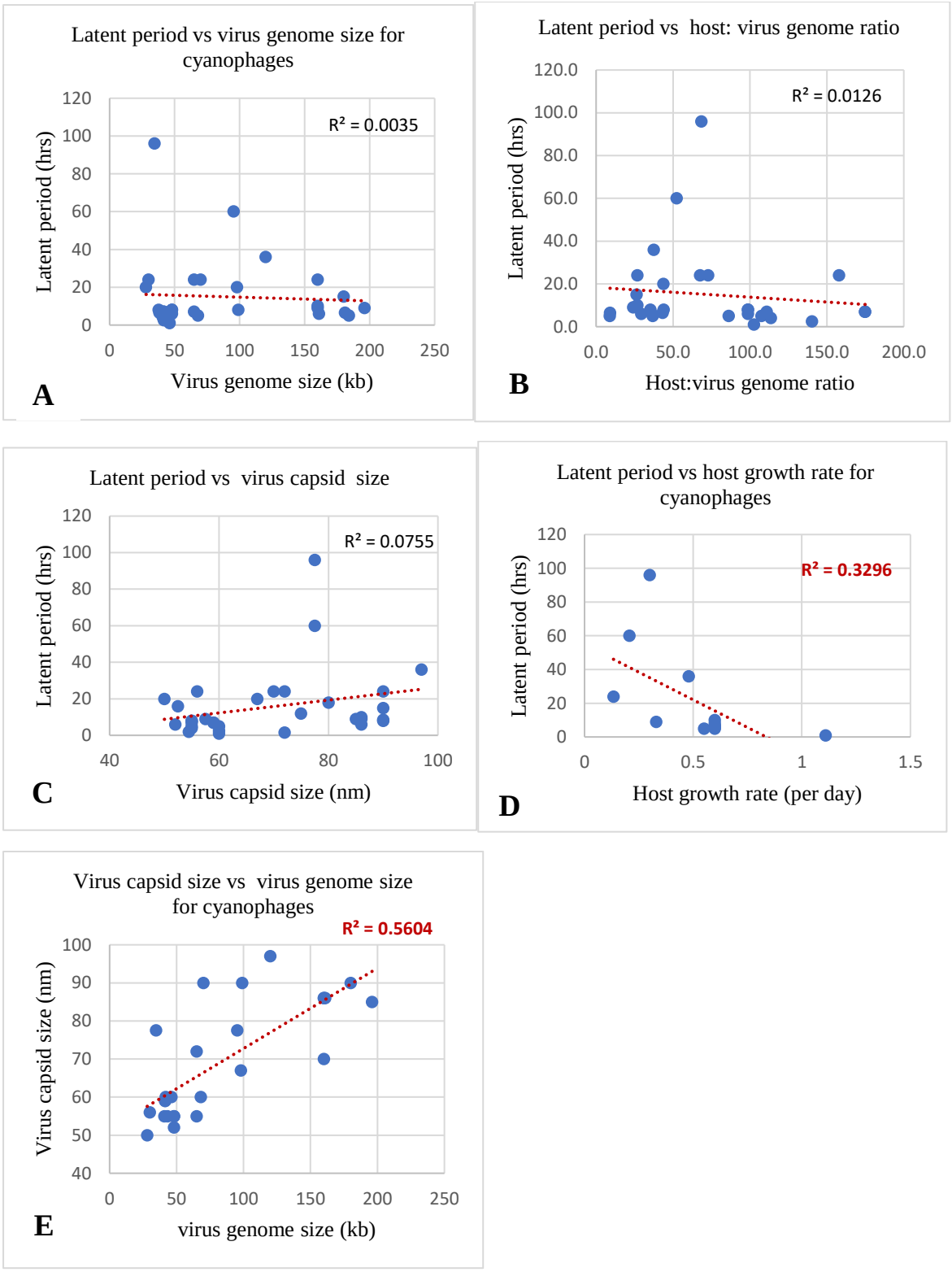

Fig.27. Relationship between (A) latent period and virus genome size (B) latent period and host: virus genome size ratio (C) latent period (hrs) and virus capsid size (nm) (D) latent period (hrs) and host growth rate (day $^{-1}$) (E) virus capsid size (nm) and virus genome size (kb) for cyanophages.

3.2.5 Archaeal viruses

There is a weak trend (R^2= 0.3-0.4) between BS and LP, VS and GS, LP and GS for archaeal viruses, while the relationship is moderately strong for LP and VS (R^2= 0.53). However, no relationship observed for BS vs GS and BS vs VS (Fig. 28 A-F).

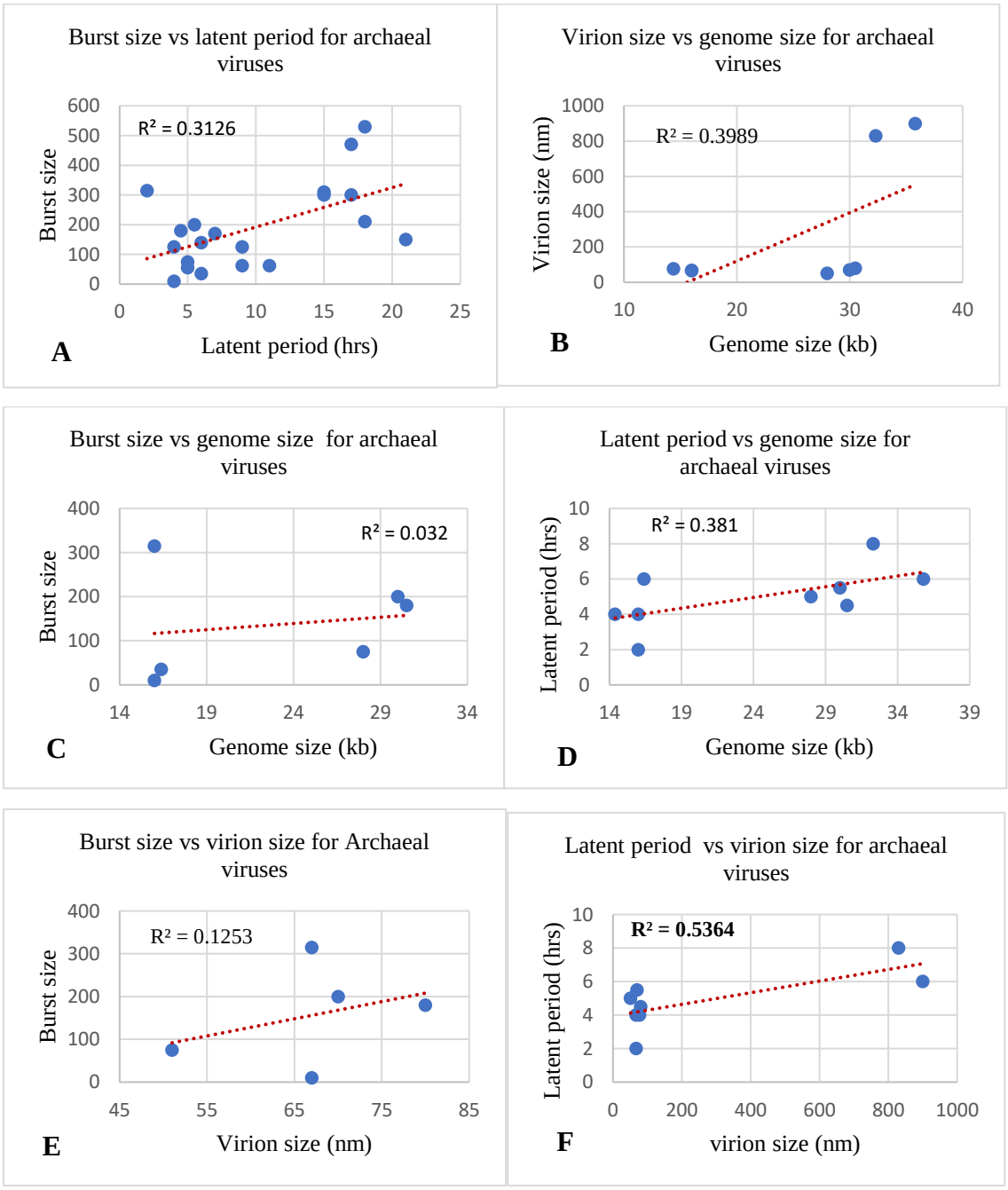

Fig.28 Relationship between (A) burst size and latent period (hrs) (B) virion size (nm) and virus genome size (kb) (C) burst size and genome size (nm) (D) latent period (hrs) and genome size (kb) (E) burst size and virion size (nm) (F) latent period (hrs) and virion size (nm) for archaeal viruses.

4. General discussion

4.1 Limitations, reasons for caution, methodological errors

The majority of burst size and latent period data have been obtained using isolated phage-host systems, under experimental conditions using one step growth curves. But in natural field conditions both viruses and hosts face different challenges, than in experimental setup. The host's metabolic and nutritional status has an impact on virus growth parameters and in nature, we can thus expect nutrient and other growth limitation factors for host, as well as environmental stress factors for viruses. It has been reported that in natural environments (*in situ* conditions), the burst sizes tend to be smaller and the latent periods tend to be longer (Zhang and Jiao, 2009, Børsheim, 1993). Some of the trait variation found within members of a group can arise due to physiological plasticity (Brown et al., 2006).

(Zimmerman et al., 2019) has highlighted that comparison between studies does not always give an accurate picture of the viral traits because of the variances in other experimental parameters, which have a direct control over the virus host dynamics. Such parameters, which are identified and studied are; host physiological state, virus-host contact rates (Murray and Jackson, 1992), temporal resolution, multiplicity of infection (m.o.i.) (Brown and Bidle, 2014) and percentage of infectious virions (Zimmerman et al., 2019 and references therein), temperature, level of irradiance and host density (Mojica and Brussaard, 2014, Murray and Jackson, 1992). In addition, differences could arrive due to the method used to estimate the BS. For example, indirect BS estimation by "dilution to extinction assay of infectivity" can underestimate the virus numbers compared to direct counts using TEM or flow cytometry (Van Etten et al., 1983, Cottrell and Suttle, 1995). There might be a connection between burst size variation within a virus group (for e.g. ssDNA diatom viruses) and the ecological strategies adapted by the individual viruses (Toyoda et al., 2012). Further, the studies, which reported strong or moderate correlations between traits have used relatively fewer data points than I have used in the present study (sometimes only 4 data points) (Parada et al., 2006).

Further, studies which compare viral traits have been restricted to use aquatic viral types. However, in the present study I used viruses isolated from various habitats other than freshwater or marine environments, for e.g. rhizospheres of plants, soil, cheese, sausages and other fermented food, curd, vegetables, compost, gut, wounds of animals, straw, seeds and human body. Sinha et al., (2018) has described the effect of the viscosity of the media on virus host interactions. Where, low viscosity liquid media such as natural aquatic or liquid broth in

an experimental setup allows for well mixed homogenous viral, host populations while in high viscosity solid media (agar, soil etc) interactions between viral and host populations are constrained spatially. Once again it was not wise to group together aquatic viruses with other types.

Constraints in latent period determination

Most of the studies on diatom infecting viruses and cyanophages give the latent period as a time range or indicated as "less than 12 hours" (Kimura and Tomaru, 2013), <24 hrs (Bettarel et al., 2005), < 48 hrs (Kim et al., 2015a). Sometimes the given time range is so wide as; 24-48 hrs (Tarutani et al., 2001), 48-72 hrs (Kimura and Tomaru, 2015, Gao et al., 2009) (24 hours difference) (table. 4,11). There is huge uncertainty in such readings and difficult to incorporate such data into graphs for comparative tasks.

4.2 Pool or not to pool the data

There are both advantages and disadvantages with pooling viruses into larger groups, intending to search for traits and trade-offs. Pooling of data allows to find universal or common trends, which can be incorporated into models to better understand the role of virus in processes of global significance.

But at the same time, it is easy to miss trends because of too much noise from high level of case specific exceptions. When we pool data from a very wide group of viruses, into broader categories (such as siphovirus, podovirus and myovirus) it could mask the relationships within smaller groups. Within each of the above groups, we can see that there are smaller groups adapted for specific environments or with distinct physiological properties differentiated from the rest. For e.g. bacteriophages of halophilic, thermophilic, psychrophilic, phytopathogenic, enteric, lactic acid, nosocomial/ opportunistic, roseobacter clade, etc. Bacteriophages and their hosts within those niche segregated groups apparently have specific adaptive, survival, ecological and evolutionary strategies to cope with the explicit challenges offered by their surroundings.

It was not wise to pool marine, fresh water and other viruses from different environments, since those fundamentally exclusive environments force their inhabitants (including viruses) to choose unique trade-offs independent of their phytogenic or systematic assemblage. It is

reasonable to conclude that "the search for trade-offs should concentrate on species that co-occur" (Litchman and Klausmeier, 2008).

4.3 Certain virus groups are understudied

Characterizing the viruses is an essential part of exploring and understanding their enormous diversity. There are at least several folds more literature published on bacteriophages than algal viruses. There are only a handful of reports on characterization of dsRNA viruses infecting algae or bacterial host. I could find only one study on eukaryote dsRNA virus characterization (Brussaard et al., 2004), two on dsRNA phage, φ6 (host- *Pseudomonas phaseolicola*) (Vidaver et al., 1973) and phiYY (host-*Pseudomonas aeruginosa*) (Yang et al., 2016). Out of the algal viruses, there is an abundance of literature on dsDNA viruses, but few on ssDNA/RNA viruses. There are nearly 1900 genome sequences available in NCBI database (NCBI, 2019) on bacteriophages, anyhow, out of them there are only 6 dsRNA sequences (10 isolations by 2016) and 12 ssRNA sequences of phages available (Yang et al., 2016).

An interesting question is, does this unevenness represent the actual viral species abundance in nature or is this a consequence of "great plate count anomaly" and lack of suitable methods to isolate other virus-host systems than dsDNA types? Because recent advances in metagenomic studies has revealed the existence of previously unknown, diverse and genetically distinct viruses in environments worldwide (Labont and Suttle, 2013, Angly et al., 2006, Breitbart and Rohwer, 2005, Breitbart et al., 2002). When we investigate bacteriophages, there is an obvious bias towards, phages of medically (pathogenic enteric hosts) and industrially (lactic acid producing, pathogenic to aquaculture species) important hosts, compared to other types.

Here in the present study, I only focus on viruses infecting unicellular algae species, but there are ssRNA viruses which infect multicellular algae species, for e.g. virus CAV, (host-*Chara australis* -green alga) (Gibbs et al., 2011). Anyhow, it was out of the scope of the present study.

4.4 General trends

Because of their vast diversity, it was difficult to find generalized traits and trade-offs among different virus and phage groups. If we possess the knowledge of the driving forces behind the viral traits and underlying mechanisms together with their discernible patterns, this information is valuable in predicting viral-host interactions, dynamics and ecological impact of viruses at various levels (from population to ecosystems) (Gudelj et al., 2010).

4.4.1 Burst size

Life history traits are highly variable across the virus groups. Therefore, Edwards and Steward, (2018) pointed out the importance of bringing eco-evolutionary dynamics (Fussmann et al., 2007) onto the table when reasoning the viral traits, since some traits (e.g. LP and BS) might employ feedback mechanisms. Brown et al., (2006) has assumed that most of the time, larger burst size represents the greater viral success in natural environments. Burst size is a result of 1) molecular and other resources of the host and 2) the ability of the virus to utilize these resources effectively for its own reproductive success. So it is a well-accepted fact that genome size of the host, has an impact on burst size (Edwards and Steward, 2018b). Hosts with larger genomes have more resources available for virus replication, thus produce larger burst size (positive correlation) (Brown et al., 2006), while it is expected negative relation between virus genome size and burst size. Viruses with larger genome size need more resources per progeny, so the trade-off is to produce less progeny (= lower burst size) (Edwards and Steward, 2018b).

In general, virus of eukaryotes possesses higher BS than bacteriophages and BS varies over 4 orders of magnitude across virus categories (Table.1). Out of all gathered information on burst size of viruses and phages, Heterosigma akashiwo nuclear inclusion virus (HaNIV) has the largest ever burst size of 100000. However, the authors (Lawrence et al., 2001) have not determined its nucleic acid type (whether DNA or RNA or single stranded or double stranded). Therefore, it could not be incorporated into any of the graphs in the result section.

A question arising during this study was, why some viruses show extreme burst size and latent period values compared to other members in the same group? Are those extreme traits controlled by the virus itself or is there an involvement of host traits as well? To understand this further, I investigated host traits corresponding to those viruses displaying extreme BS and LP values.

Host of the highest burst size producing dsDNA virus, *H.akashiwo* has a genome size of 1945 **Mb**, a cell volume of 1103 μ^3 and a host: virus genome size ratio of 97250 (Table 2A). The host *Chrysochromulina ericina* of the second largest BS producing virus, has a genome size of 1070 **Mb**, a cell volume of 113 μ^3 and a genome ratio of 2098. The host of lowest BS producing virus, *Ostreococcus tauri*, has a genome size of 12.6 Mb, a cell volume of 0.52 μ^3 (Edwards and Steward, 2018a) and a genome ratio of 67. Sometimes cell volume becomes a limiting factor for burst size (Brussaard et al., 2004). For ssDNA viruses, highest BS producing hosts are *Chaetoceros lorenzianus and Chaetoceros setoensis* whereas lowest BS producing

host is *Chaetoceros sp.* strain SS628-11. In contrast to the lowest BS producing host, the largest BS producing host has one order of magnitude higher cell volume and a host genome (Table 2A). Their host traits are shown in Table 2A.

Table 2A Eukaryotic host traits correspondent to highest and lowest burst sizes. Host trait values were extracted from (Edwards and Steward, 2018a)

Virus Catergory	Host	Host genome Size (Mb)	Virus genome size (Kb)	Host: virus genome Ratio	Burst size	Host cell volume (μ^3)	Host growth rate (per day)
dsDNA	*H.akashiwo*	1945	20	97250	16000	1103	0.43
dsDNA	*C.ericina*	1070	510	2098	1800-4100	113	-
dsDNA	*O. tauri*	12.6	186.2	67	**25**	0.52	1.23
ssDNA	*C. lorenzianus*	1000	5.8	172413	22000	8000	1.12
ssDNA	*C. setoensis*	239	5.8	41206	4700-20000	800	1.78
ssDNA	*Chaetoceros sp. strain SS628-11*	239	5.5	43454	**29**	424	0.94
ssRNA	*H.circularisquama*	20500	4.4	4659090	3400-16000	2200	-
ssRNA	*C.socialis f. radians*	239	9.4	25425	**66**	350	1.17

Table 2B Eukaryotic host traits correspondent to highest and lowest latent periods. Host trait values were extracted from (Edwards and Steward, 2018a)

Host	Host genome size (Mbp)	Virus genome size (Kb)	Host: virus genome Ratio	Latent period (hrs)	Host cell volume (µ3)	Host growth rate (per day)
Chlorella variabilis NC64A	46	327	140	2	40	1.39
Rhizosolenia setigera	100000	11.2		48	63000	1.02
Chaetoceros sp. strain SS628-11	239	5.5	43454	<12	424	0.94
Heterocapsa circularisquama	20500	356	57584	56	2200	0.265

Edwards and Steward, (2018b), have used genome size ratio to compare with viral traits BS and LP. They found a strong positive relation between genome ratio and BS, but BS and host growth rate are not related (Table. 2A). Half of the variation in BS can describe by genome ratio (Edwards and Steward, 2018b). There higher BS correlated with higher genome ratio. In the present study, this trend is clear with dsDNA and ssRNA viruses but not so obvious for ssDNA viruses (Table 2). The study aforesaid has found the similar results. In addition, they have demonstrated that the total viral nucleotide output upon cell lysis is alike to the number of nucleotides in the host genome.

Primary traits of the host such as; growth rate, genome size, reasons for burst size and latent period variation up to 40-50% (Edwards and Steward, 2018b). Therefore, it is a necessity to include host traits when searching for trade-offs. Host: virus genome size ratio (hereafter abbreviated as "genome ratio") demonstrate a saturating correlation with both BS and LP. At low "genome ratio" there is a positive correlation between BS, LP and "genome ratio" whereas, at high "genome ratio" there is no relationship observable (Edwards and Steward, 2018). Further, there is an inverse relationship between host growth rate and latent period (Edwards and Steward, 2018). This highlight the need of information on host traits, when investigating trends in viral traits. However, most of the literature studied for this study, on viral traits, have not indicate the corresponding host traits, other than merely indicating the growth rate as "exponentially growing culture". So, lack of quantitative information on host growth rate and genome size is an inadequacy in this study. To find genome sizes of the host, I had to look elsewhere. Some data were obtained from Edwards and Steward, (2018a). Lack of published data on host genome size (specially diatom hosts) is also pointed out by Edwards and Steward, (2018). Their model identified the host's genetic resources allocated to viral replication as the decisive factor for BS setting. Further, LP is a progressive trait (regulated by the host metabolic rate) facilitating this "prescribed" BS. Therefore, viral traits alone are not adequate to explain the trade-offs and I should have incorporated host traits into all virus categories of the present study (for *Siphoviridae*, *Myoviridae* and *Podoviridae* families) as well, then I would be able to present more accurate interpretations.

(Parada et al., 2006) pooled and summarized burst sizes of phages infecting heterotrophic prokaryotes in various environments such as coastal shelf, offshore, deep sea, fresh water oligotrophic, oligo- mesotrophic, mesotrophic, meso-eutrophic, eutrophic, solar saltern. Further the reported BS range for freshwater phage category is 4-140, while for marine systems, upper range is twice larger than freshwater habitats, 6-300 (Parada et al., 2006). The

reported mean burst sizes are, marine -oligotrophic ~20, marine eutrophic ~25, fresh water oligotrophic ~28 and freshwater eutrophic ~ 40. According to that, viruses from marine - oligotrophic environments show lowest values of burst size, while viruses in freshwater eutrophic environments have the highest burst size values. These results support the intuitive theory of host nutrient status and BS relationship (Wilson et al., 1996). However, this BS correlation with trophic level of the habitat not always adhere. Because some phages isolated from eutrophic habitats show much lower BS e.g. Roseophages (Li et al., 2016a, Chan et al., 2014). Besides two Roseophages isolated from the same host, *Dinoroseobacter shibae*, grew in similar nutrient conditions display one order difference in BS (Li et al., 2016a, Yang et al., 2017). Since I did not classify my data according to the trophic status of the habitat or as freshwater and marine, it is difficult to compare my results with Parada et al., (2006). In addition, bacteriophage group in the present study does not limit to the freshwater, marine aquatic division, but consists of much extensive categories.

Van Etten et al., (1983) reported that burst size is altered by m.o.i. With increasing m.o.i. from 0.1 to 50, there is a consistent decrease in burst size. They assumed that the underlying cause might be the "lysis from without" phenomenon or some sort of interference. The same article also mentioned that the growth of host in continuous dark conditions reduce the burst size by 50% compared to continuous light conditions. However, the burst size is not depending on photosynthesis (Van Etten et al., 1983). There is wide variation in m.o.i., among the studies included in this literature survey. For viruses of eukaryotes it varies from 0.1-20, while for diatom viruses, higher m.o.i. values such as; 66 and 270 have been used. For archaeal viruses have also been used higher m.o.i. values. For prokaryotic viruses applied m.o.i. values vary from 0.001- 10, data not shown in the tables in the appendix, but reported in the pivot table. Therefore, once again it has to be noted that experimental conditions are highly variable among studies.

There are several hypotheses that have been presented in the literature to explain the BS variation, namely; size and morphology of the host and the virus, host physiological control, habitat control, latent period control, superinfection and lysis inhibition, phage control, control by different environments (Parada et al., 2006 and references therein). Out of them I have discussed most of them in the present study under different sections.

4.4.2 Latent period

LP is correlated with both, host growth rate and host: virus genome size ratio (Edwards and Steward, 2018b). Those two host traits combined, can explain 38% of LP variation in eukaryote viruses and the correlation is much stronger in dsDNA viruses, explaining 57% of the variation (Edwards and Steward, 2018b). LP increases with genome size ratio, but the correlation is not strong as for BS. (Edwards and Steward, 2018b). Their study reports negative relationship between LP and host growth rate, but no relationship between BS and host growth rate for dsDNA viruses. In addition, LP is approximately similar to demi- host doubling time (Edwards and Steward, 2018b). According to Fig.7B, we can see a weak trend that LP increase with the virus genome size for dsDNA viruses. Even though host traits were not focused on the present study, I gathered some data on host traits for the viruses showing latent period extremes (Table 2B). At least for the examples in Table 2B, we can see a pattern; when host growth rate increases, latent period tend to decrease. I found weak positive relation between LP and BS for dsDNA viruses (Fig.6B), weak positive trend between LP and GS (Fig.7B) and no relation between LP and VS (Fig. 8B) for dsDNA viruses. For ssDNA viruses I could not find any relation between LP and any of the viral traits compared (Fig.10, 11B,12B). For ssRNA viruses there is a moderate negative correlation between LP and BS (Fig.13), but no relation with GS and VS (Fig. 14B and 15B). For prokaryote viruses I could not find any relation between LP and other traits, except a weak positive trend between LP and GS for family *Podoviridae* (Fig 23B). However, several studies has been claimed a correlation between LP and BS, where prolonged LP provide the time needed to assemble more virions, so identified as a stratagem for optimizing BS (Abedon et al., 2001, Wang et al., 2004). However, this trade-off is only useful in slow growing host populations, where it is advantageous to wait for more favourable circumstances to arrive before lysis. This guarantees virus spread and next generation continuance (Wommack and Colwell, 2000). In additions, (Lawrence et al., 2006) have demonstrated that latent period and BS variation or "biological variability" among different virus types, which infect the same host population at the same time (e.g. harmful algal bloom of *H. akashiwo*, "red tide") makes room for coexistence of viral types during fluctuating host densities.

Further, longer latent periods are observed when the genome ratio is high, since much time is needed to exploit all host resources (Table 2B). However, latent period evolution is a combined result of many factors. When latent period is determined using one step growth curve under experimental conditions, the host density is high. It is documented that under very high host

density, latent period can shift by genetic adaptation. Such as short LP mutants in the population can out-compete the longer LP and higher BS types. Since under high host density, host encounter time become less, compared to the lytic cycle, which is an advantage (Abedon et al., 2003). Similar prediction has also done by models; high host density leads the virus population to select short LP and short BS (Mann, 2003).

4.4.2.a Latent period, burst size and host physiology

Effect of host metabolic rate (physiological status) on burst size and latent period

Some studies have extensively discussed the influence of factors connected to host growth rate on viral traits (You et al., 2002). Physiological state of the host, (whether if it is actively growing or in stationary phase) has clear influence on both latent period and burst size. RsRNAV, (host- bloom forming diatom, *Rhizosolenia setigera*) shows two distinct burst size values of 1010 and 3100, when they infect their host growing at stationary phase and exponential phase respectively (Nagasaki et al., 2004). This can be taken as an example demonstrating the influence of host metabolic rate (physiological status) on burst size (Van Etten et al., 1983, Bratbak et al., 1998). Host's growth phase (physiological status) not only affects the burst size , but it also affects the host's susceptibility to the virus infection (Nagasaki et al., 2003). They have observed that during the stationary phase host, *H. circularisquama*, is less susceptible to the virus, HcV 03. While during the log phage growth, the host is more sensitive to viral infection. However, a study by (Bratbak et al., 1998) showed otherwise, that all growth stages of *Phaeocystis pouchetii* is susceptible to viral infection. Host's increased biosynthesis activity, which is favourable for viral replication is an eminent fact (Nagasaki et al., 2004). Actively growing cells may supply more resources for virus production, so it can increase the BS and shorten the LP (Proctor et al., 1993, Middelboe, 2000, Young et al., 2000).

Effect of nutrient and light depletion of algal host on burst size and latent period

Some studies have concluded that, in comparison to nutrient replete conditions, nutrient (such as phosphorus) depletion cause prolongation of latent period (Wilson et al., 1996). While others found no prolongation of latent period due to nutrient limitation (Bratbak et al., 1998). When discussing light limitation and burst size, there are studies to support both theories. Some

conclude that there is no effect of light on burst size (Bratbak et al.,1998), while others observed burst size reduction of 50% due to light limitation (Van Etten et al., 1983, Piedade et al., 2018).

Effect of temperature on burst size and latent period

When some algal hosts grow at high temperature, the latent period of the infected virus gets shortened and the burst size increases. In contrast, when some algal hosts grow at low temperature, the latent period of the infected virus get longer and the burst size decreases (Nagasaki et al., 2003, Demory et al., 2017).

4.4.3 Virus genome size

Upon infection, when virus take control over the host metabolism, genome size of the virus is the trait mainly influencing this control. Since it decides how many proteins encoded by the virus and thus the complexity of the infection process. Besides that this trait contribute to the outcome of other traits such as, metabolic cost of virion production process and virion size as well (Edwards and Steward, 2018b). Genome of progeny virions are made from host nucleotide pool and disintegration of the host genome. However, *de novo* nucleotide synthesis has also been discussed in some literature (Wikner et al., 1993, Van Etten et al., 1984, Brown et al., 2007). It is suggested that larger viral genome contribute to better control over the host metabolism, which leads to higher viral production rate (=BS divided by LP) and de novo nucleotide synthesis (Hurwitz et al., 2013 , Edwards and Steward, 2018b and references therein).

Edwards and Steward, (2018) have used host: virus genome size ratio (instead I use virus genome size alone, in the present study) to compare with BS and LP. However, Edwards and Steward, (2018b), concluded that "virus genome size alone is a poor predictor of viral traits". I found similar conclusion in this study for ssDNA, ssRNA, *Siphoviridae* and *Myoviridae* members since virus genome size alone did not correlate with other tested traits. Yet, there are weak trends observable for GS vs BS, GS vs LP and VS vs GS for dsDNA viruses (Fig. 7A, 7B, 9), VS vs GS for *Siphoviridae and Myoviridae* members (Fig. 25 A and 25 C), LP vs GS for *Podoviridae* (fig.23 B). Edwards and Steward, (2018b), suggests that there is a tendency for larger viruses having smaller BS, thus my results do not support such tendency. The same study states that "latent period is unrelated to viral genome size", but I found weak correlations between those two traits for some virus groups; namely within dsDNA and *Podoviridae*. There is no correlation between eukaryote host genome size and viral genome size, but as pointed out by Edwards and Steward, (2018b), smallest viruses (both having smaller genomes and capsids),

which are single stranded DNA and RNA viruses, only have been isolated from larger eukaryotes such as diatoms dinoflagellates.

4.4.4 Virion size

Edwards and Steward, (2018) have found strong correlation between genome size and virion size in eukaryote viruses. However, I only found moderate or weak relationship between these two traits for dsDNA (Fig.9), *Siphoviridae* and *Myoviridae* (Fig. 25 A and 25C), while no relationship for other groups. Similar trend has also been reported by Ashelford et al., (1999), in phages of *Serratia liquefaciens* , isolated from the sugar beet phytosphere. Nevertheless, virion head size can increase separately from genome size (Kumari et al., 2010). This can happen as a result of errors in DNA replication followed by erroneous synthesis of viral capsid proteins (Kumari et al., 2010 and references therein). I have not included tail length of the phages into comparison of traits, yet some studies have detected a robust correlation between tail length and LP (Ashelford et al., 1999). These authors also raise the question; are these size differences (even for the members of a same family), reflect an explicit evolutionary change? or a random consequence of mutation?

dsDNA virus of eukaryotes, which were included in this study, possess wide size range (113-310 nm), but ssDNA and ssRNA viruses show limited variation, 32-38nm and 22-38nm respectively. The reason for this great size variation observed in dsDNA viruses and prokaryote viruses is yet to be answered (Edwards and Steward, (2018). Larger virions need longer LP to produce the progeny and need more resources. Usually under limited host resources, we can assume that low BS and longer LP for larger virions. But my results do not support this theory, as none of the traits (neither BS nor LP) had a relationship with virion size for any of the virus groups. (Fig. 8A.8B, 12A,12B, 15A,15B,18A,18B,21 A, 21B, 24A and 24B). when calculating the energetic cost for building a virus particle (Mahmoudabadi et al., 2017),virion size is a critical parameter. it has been calculated that for viruses with head size over 80 nm, genome replication cost overweighs the translation cost of energy (Mahmoudabadi et al., 2017).

4.5 Specific traits for different groups

4.5.1 DNA versus RNA viruses

When comparing the double strand and single strand viruses, ssDNA and ssRNA viruses have the smallest genome sizes and comparably smaller capsid sizes than dsDNA viruses and phages (Table.1). Since single stranded viruses have one nucleic acid strand compared to the double

strand, a smaller capsid is sufficient to accommodate the smaller single strand genome. To date it has not been reported any RNA viruses infecting cyanobacteria or archaea (Prangishvili, 2013, Pietila et al., 2014). If we could find out the reason why those two groups are resistance for RNA viruses it would be an enlightening addition to our understanding of the virosphere. (Prangishvili et al., 2006) has suggested the idea that since archaea flourish in hyperthermal environment and RNA is less stable to high temperatures might partially explain the lack of RNA viruses infecting archaea, or could it be due to methodological problem? DNA viruses have less complex replication cycles than a RNA viruses (Sinha et al., 2018). Further, the energetic cost of building a DNA virus is different than that of a RNA virus (Mahmoudabadi et al., 2017), however the overall cost of infection depend on the BS. Could it be that the more advance and complicate eukaryote species support that complex replication cycle of RNA viruses, but comparatively simpler prokaryotes could not support it?

4.5.2 Viruses of eukaryotes vs phages of prokaryotes

The burst size for viruses infecting eukaryotes ranges from 25- 93400 while for phages it varies between 5-9000 (Table. 1). Though, (Børsheim, 1993) has reported the BS variation of phages in aquatic system as 5-300. This huge BS variation is mainly due to larger host cell volume and host genome size in eukaryotes compared to that of prokaryotes. Average bacterial cell volume in lake aquatic environment estimated to be $<0.15\mu m^3$ with a range of (0.01- 0.4 <) (Weinbauer and Hoefle, 1998 and references therein) while eukaryote host cell volume can be as high as 5 orders of magnitude. With the reported positive relationship between BS and host cell volume (Weimbauer and Peduzzi, 1994, Weinbauer and Hoefle, 1998), the differences in BS between eukaryote versus prokaryote viruses can be explained. Further, there is a huge latent period difference between the two groups as, LP of eukaryote viruses is measured in hours, while for phages it is measured with minutes (except for cyanophages, who have very long LP values). This is directly coinciding with differences in host generation times. In addition, there is a virion size difference between eukaryote and prokaryote dsDNA viruses and phages. Prokaryote phages show virion size variation 10-164nm, while eukaryote viruses are much larger 113-310nm.

4.5.2.a Viruses infecting eukaryotes

So far, around 20 diatom viruses have been documented (Arsenieff et al., 2019). All the diatom infecting viruses described so far in the literature has either ssDNA genomes or ssRNA genomes. It would be interesting to find a reason for such trait. Do diatom hosts show any

resistance to dsDNA virus infection? Perhaps the silica in the diatom wall has some kind of resistance mechanism against dsDNA viruses.

4.5.2.b ssDNA viruses

It is noteworthy to mention that, all the diatom- infecting ssDNA viruses characterized so far, share a common trait. ssDNA virus particles aggregate in the nucleus of the host while dsDNA and ssRNA viruses accumulate in the cytoplasm of the host cell. Most of the ssDNA viruses belong to the genus- basilladnavirus. In contrast, most of the dsDNA viruses belong to the family-- *Phycodnaviridae* and some also in *Mimiviridae* family.

There are 6 single stranded RNA viruses with marine microalgal hosts that have been included in the present study, for e.g. HaRNA virus (host- toxic bloom forming phytoflagellate, *Heterosigma akashiwo*) (Tai et al., 2003), HcRNAV (host-bivalve killing dinoflagellate, *Heterocapsa circularisquama*) (Tomaru et al., 2004). In contrast to dsDNA viruses, lack of correlations between traits of ssDNA and ssRNA viruses leads to the explanation, due to their smaller capsid size together with smaller genome size make them vulnerable for different types of selective pressures and specific viral-host interaction mechanisms than the dsDNA viruses (Edwards and Steward, 2018b.)

When assessing host range, diatom infecting viruses are highly species specific and some are confined as strain specific (narrow host range) (Tomaru et al., 2011b , Arsenieff et al., 2019 and references therein). At the same time, they possess the smallest genomes among all the other groups, even smaller than bacteriophages genomes. This might be a specificity and generality trade-off (host range). As described in section 1.10.2, to infect several hosts, a virus needs to have a larger genome encoding more genes to defeat various host defence tactics and enhance their adsorption mechanisms. A tiny genome may not support a strategy like this. All those single stranded diatom infecting viruses have longer LPs, which lower the reproductive rate. However, the trade-off here is not between reproduction rate and host range, instead between genome size and host range. All the reported diatom infecting ssDNA viruses have smaller virion size, approximately 35nm and smaller genome size around 5.5-6 kb. But there is a huge variation in burst sizes (29-22000) and latent periods (<12-96 hors) see table 1. Therefore, virion size or genome size of the virus cannot explain the reported huge variation of the traits. The host diatom species were collected from surface water.

Despite the fact that usually phages are exhibiting narrow host ranges, even specific to serovars/biovars/ subspecies (Grose and Casjens, 2014), there are phages infecting *Vibrio*

alginolyticus, which show broader host range. Interestingly they have the largest genomes within *Myoviridae* members too (Kim, et al. 2019, Kalatzis et al., 2016).

4.5.3 Cyanophages

Since cyanophages are a distinct group among phages, they were analysed separately. Searching for trade-offs, considering only viral traits was unsuccessful. Therefore, here I combined host and viral traits together to reveal any trade-offs among cyanophages, even though, host traits were not used in analysing eukaryote viruses or other bacteriophages in the previous sections 3.1 and 3.2. Since viruses must always depend on their host for resources, it is natural that host resources may become limiting factors for virus replication. Amino acids, nucleotides, cell volume, energy, ribosomes, enzymes and co factors can be named as such resources need for virus replication and among them nucleotide content (basically represent by the host genome size) has been chose in many studies and models as a predictor to maximum burst size (Brown et al., 2006, Edwards and Steward, 2018b). It has been reported a robust correlation between cyanophage burst size and their marine host's genome size (Paul et al., 2002). Guided by Edwards and Steward, (2018b), I used, host: virus genome ratio to compare with burst size. To check whether there is a difference between freshwater isolated and marine isolates, the two categories were separately plotted (Fig. 26A and 26B). However, for the rest of the plots both freshwater and marine data were grouped together since pooling did not substantially change the R^2 value for the trend line in the graphs. Edwards and Steward, (2018b) have found correlation with R^2=0.49, (half of the observed burst size variation is explained by the genome size ratio). In addition, they have found positive correlation between LP and genome size ratio (R^2=0.30). They have not specified cyanophages but included all ds/ss/DNA and RNA viruses together into the graph. However, in my analysis I could not find such robust correlation between BS and genome size ratio for freshwater species (Fig. 26A). But there is a weak relationship between two traits for the marine species (Fig.26 B). There is a weak positive relationship between BS and LP (R^2 = 0,3134), but there is no relationship between BS and host cell volume (R^2 = 0,1169) (fig. 26 E) or BS and virus capsid size (Fig. 26F). However, virus capsid size and virus genome size have a correlation of R^2 = 0,5604 (Fig.27E).

One obvious trait among **cyanophages** in contrast to other bacteriophages, is their extremely long latent periods (Table.12). If we look into the latent period and other traits; latent period and host: virus genome ratio shows no correlation (Fig. 27B), but, Edwards and Steward,(

2018b) reported a weak (R^2= 0.3) correlation for those two traits for viruses infecting phytoplankton (including cyanophages). All the other traits compared with LP showed no relationship except there is a negative correlation between LP and host growth rate (Fig. 27D). Correlation values and compared traits are summarized in the Table 2C.

Table 2C. Summarization of compared trait pairs and correlation values (R^2) for cyanophages.

Compared trait pairs	R^2 values (correlation)	Figure
Burst size and host: virus genome size ratio -Fresh water	0.02	26A
Burst size and host: virus genome size ratio -marine	**0.37**	26B
BS and LP	**0.31**	26C
Burst size and virus genome size	0.07	26D
Burst size and host cell volume	0.11	26E
Burst size and virus capsid size	0.00	26F
Latent period and virus genome size	0.00	27A
Latent period and host: virus genome size ratio	0.01	27B
Latent period and virus capsid size	0.07	27C
Latent period and host growth rate	**0.33**	27D
Virus capsid size and virus genome size	**0.56**	27E

Brown et al., (2006), converted host and virus genomes into nucleotides and then compared with BS. In that study also they have not restricted to cyanophages but used other viruses of phytoplankton as well. They have reported strong correlation between Log Burst and Log host genome nucleotides. This trade-off in burst size with host's genome size is also reported earlier for cyanophages (Sullivan et al., 2003). Because of cyanobacteria are carrying multiple genome copies, Brown et al., (2006) has highlighted the need of incorporating this when dealing with host genome size.

4.5.4 Phages of halophilic bacterial hosts

Overall, viruses of hypersaline habitats are dominated by archaeal viruses, very few eukaryote viruses and some bacteriophages have also been reported (Atanasova et al., 2015). When investigating trade-offs and fitness costs, bacteriophages infecting halophilic bacteria is useful,

because their environment provides them the selective pressure, which is the starting point of natural selection. Further viruses are the sole predators in this unique habitat (Jaakkola et al., 2012). One of the hypotheses presented by Parada et al., (2006), on burst size variation is that, hosts inhabiting in favourable environment conditions may produce higher BS than that of challenging environments. In parallel, unfavourable environment conditions lead to low host quality, reduced BS and prolonged LP (Zachary, 1976, Proctor et al., 1993, Middelboe, 2000, Young et al., 2000, Kokjohn et al., 1991).

Phage UTAK, infecting moderate halophilic host, *Vibrio* B1 shows range of LP and burst size values under varying NaCl concentration (Goel et al., 1996). At 1M NaCl concentration (which is the most favourable and least stress) the phage has the optimum values for BS and LP, which is shortest LP and highest BS (higher reproductive success). Towards the lower and higher NaCl concentrations, host growth rate decrease and doubling time increases. It is clear in this example; latent period evolution occurs parallel to host doubling time (Table. 9). This fitness trade-off can be observed in all the other phages infecting halophilic bacterial hosts in the present study as well (Zachary, 1976, Torsvik and Dundas, 1980, Daniels and Wais, 1990). In addition to the impact on LP, adsorption and BS, salinity level of the hypersaline environments involve in determining the lifecycle mode of the viruses (lytic to carrier state switching) (Torsvik and Dundas, 1980). Further, phages nt-1 and nt-6 of *Beneckea natriegens*, show narrow host range, with only one host (Zachary, 1976). Will it be a trade-off between narrow host range and stability over wide salinity range? It is safe to state that both halophilic and psychrophilic phages possess considerably larger genomes above average for other members. It is interesting to investigate whether phages isolated from extreme habitats have larger genomes. Since larger genomes can encode for plethora of functional traits which is necessarily for the survival in those stressful habitats.

Siphoviridae phage of cold-active host -*Colwellia psychrerythraea* shows a similar fitness trade-off with temperature as changing factor. It was reported that, latent period shortened together with increasing temperature (Wells and Deming, 2006) and both low and high temperature yields low BS, while at optimum temperature , maximum BS value can be observed.

4.5.5 Archaeal viruses

Most of the archaeal viruses documented so far has hyperthermophile or hyperhalophile hosts. In the present study also, I found mostly viruses infecting hyperhalophile hosts and some

methanogen hosts. Genome size range for archaeal **specific** viruses is 5.3- 143.8 kb with an average of 23.9 kb. Normally it has been calculated that the average genome size for other cosmopolitan archaeal viruses to be 67 kb (Krupovic et al., 2018 and references therein). However, I could only find handful of literature, which reported burst size values of the archaeal viruses. Instead of cell lysis, they continuously release the viral progeny hence, traditional one step growth curve method can't apply. It has been suggested that this viral release mode seems to optimize the viral production as well as transmission to new hosts. Natural hypersaline environments in spite of their harsh conditions, harbour dense microbial populations dominated by haloarchaea (Porter et al., 2007). So, once released to the outside, finding a host is not a problem to archaeal viruses. However, host growth rates are slower in hypersaline habitats, which influence BS. In addition, once release to the environment, there is a higher probability of reduced survival of virions due to specifics laid by the surrounding. So, collectively it is a fitness trade-off to gradual release of viruses keeping the host live for an extended period (Porter et al., 2013).

The small collection of archaeal viruses in the present study has a genome size range of 14-35 kb (Table. 13) however, genome size range for the ~117 reported archaeal viruses to date is 5.2-230 kb (Dellas et al., 2014). This indicates that the sample in the present study only represents a very small portion of the current knowledge on archaeal viruses. These halophilic archaeal viruses often show remarkably broad host ranges infecting various species as well as genera (Atanasova et al., 2015). Most of the trait pairs plotted in archaea virus displayed weak to moderate trends between them. This might be due to less sample size of archaeal viruses in the present study (Table 13.1and 13.2). Some studies, which characterized archaeal viruses have not provided data on GS and VS. So, the data set became even smaller for some of the plots (Fig. 28 E and F).

Multiplicity of infection

According to the observed data in the present study, it seems like there is a characteristic m.o.i. range for each virus group, where, diatom viruses need the highest m.o.i. values compared to other groups. Summarized ranges are mentioned in the Table. 2D. I could not find any specific explanation in the literature reasoning that bacteriophages can infect bacteria successfully with such a low m.o.i. values while archaeal and diatom viruses need to have up to seven orders of magnitude higher m.o.i. levels. It might relate to host, virus morphological differences, percentage of defective virus particles, attachment and entry mechanisms, adsorption efficiency etc. Generally, reported adsorption rates for bacteriophages are faster (30% of viral

inoculum adsorb within 15 seconds) compared to that of some archaeal viruses (needs 60 sec-3 hours for 30% adsorption) (Dellas et al., 2014), which explains part of the differences in m.o.i. Nevertheless, fast adsorption rates were reported for hyperthermophillic crenarchaeal hosts, which assumed to be an evolutional strategy to decrease the time spend outside in high temperature, acidic environment (Dellas et al., 2014 and references therein). It was found that some viruses are less tolerant to environmental fluctuations than their hosts, while the opposite has been reported as well (Mei et al., 2015). Anyhow, optimum parameters for virus production do not necessarily parallel to those for optimum host growth (Wells and Deming, 2006).

Table 2D. Multiplicity of infection ranges observed for each virus group in the present study (data not shown).

Virus group	m.o.i. range
Algal virus	1-20
Diatom virus	66-270
Bacteriophages	10^{-1} - 10^{-4}
Cyanophages	1> mostly 10^{-1}
Archaeal virus	3-50

It is proposed that low initial m.o.i. could leads to overestimation of BS, since uninfected hosts can proliferate continuously during the experiment (Bratbak et al., 1998). However, several other studies reported contradictory observations on impact of m.o.i. on other viral traits (Brussaard et al., 2004, Yoshida et al., 2006). Therefore, it is safe to assume that influence of m.o.i. on process of viral infection is variable across different virus-host pair (Šulčius et al., 2015).

4.6 Fitness trade-offs

Identification of possible trade-offs and testing them is a main step when parameterizing models in ecology (Zimmerman et al., 2019). It is difficult to generalize the trade-offs for broad virus families or for larger groups, because there is a huge variation and a diversity within these larger groups in relation to traits as, genome size, virion size, burst size and latent period. Not to mention that they are adapted to specialized and unique habitats which presented them unique challenges. Consequently, each smaller group (e.g. halophilic, thermophilic or psychrophilic) must evolve according to their own environments, which needs specialize trade-offs which differ among those smaller groups. Pooling those different exclusive groups into one large group might mask away those individual trade-offs. Such has also been proposed by (Goldhill and Turner, 2014), where trade-offs are not generalizable to whole family.

Zimmerman et al., (2019) proposed a fitness trade-off for two phycodnaviruses; OlV I and OlV7, which infect *Ostreococcus lucimarinus.* Increased infection efficacy (= burst size) against reduced plasticity (resilience to changes in host physiology (=growth rate)) has been observed in this virus-host system. Ostreococcus lucimarinus virus 7 (OlV7) shows higher virulence (swift host cell lysis, early interference of cell cycle) than OlV1, however virion production (virus replication) in OlV1 is more resilient (robust) to diminished host growth rate than that of OlV7 (Zimmerman et al., 2019). Further, OlV7 bear the cost for higher burst size under favourable host growth condition by reducing its ability to withstand fluctuating host growth. While less virulent OlV1's lower burst size under favourable host growth condition traded off by increased ability to withstand changes in host physiology (Zimmerman et al., 2019) . Under limited irradiance, latent time has increase compared to that of higher irradiance. Under limited irradiance, host growth rate reduced. In parallel, burst size reduction observed from 435 ± 251 to 237 ± 47 in OlV1, while in the same limited irradiation, there is a drastic reduction in burst size observed, from 682 ± 408 to 51 ± 20. This is a good example showing the functional diversity of closely related marine viruses.

All phages (both *Podoviridae* and *Siphoviridae* members) of **Roceobacter clade** listed in this study has similar genome sizes (72-77 kb), comparatively longer latent periods1-6 hours (mean 146 minutes) thus their burst sizes are highly variable (10-1000<) (Table.6.5 and 8.2). If we investigate the reason for these prolonged latent periods in phages of Roceobacter clade compared to other *Podoviridae* members, we can use the two phages infecting *Silicibacter pomeroyi* DSS-3 and *Sulfitobacter* sp. EE-36 as examples (Zhao et al., 2009). These two Roceobacter bacteria have four times slower growth rates compared to *E. coli*. So, the two N4 like phages, which have Roceobacter hosts show longer latent periods parallel to their host in contrast to other N4 phages infecting *E. coli* (Zhao et al., 2009). Therefore, in this example viral trait directly depend on host trait rather than following its own taxonomic features.

4.7 Hypotheses- Accept, reject, inconclusive, need more information or methodological errors?

Here I repeat the hypotheses made earlier followed by answering it according to the results obtained.

a) There is a positive trend between **LP and BS**-

Inconclusive, weak positive relation for dsDNA viruses (Fig. 6B), cyanophages (Fig.26C) and archaeal viruses (fig.28A), weak negative relation for ssRNA viruses of eukaryotes (Fig.13). For other categories, no trend observed. Edwards and Steward, (2018) have the same conclusion on dsDNA viruses.

b) There is a positive trend between **LP and GS**-

Rejected according to the present study results for all groups (Fig.7B,11B,14B,17B,20B,23B). But weak trend observed for archaeal viruses (R^2=0.38) (fig.28D).

c) There is a positive trend between **LP and VS**-

Rejected according to the present study results (Fig. 8B,12B,15B,18B,21B and 24B). However, observed moderate trend for archaeal viruses (R^2=0.53) (fig. 28F).

d) There is a positive trend between **VS and GS**-

Only weak relation for dsDNA viruses (Fig.9) and for archaeal viruses (fig.28B). Accepted, moderate for cyanophages (R^2=0.56). rejected for other categories.

e) There is a negative trend between **BS and GS**-

need to incorporate more information as; host: virus genome ratio, instead of merely virus GS (Fig. 7A,11A, 14A, 17A,20A and 23A).

f) There is a negative trend between **BS and VS**-

Rejected, need more information (Fig.8A,12A,15A,18A, 21A and 24A).

g) There is a negative relationship between **LP and host growth rate** –

Accepted, but it is weak (R^2=0.3) and tested only for cyanophages.

h) There is a positive relationship between **BS and host: virus genome size ratio**-

Tested only for cyanophages. Accepted only for marine cyanophages but weak (R^2=0.37).

i) There is a positive relationship between **BS and host cell volume**-

Tested only for cyanophages. Rejected.

j) There is a relationship between **latent period and host: virus genome size ratio**-

Tested only for cyanophages. Rejected.

I could not find any distinct trends between compared trait pairs, however there is a weak positive trend between (BS vs LP), (VS vs GS) for dsDNA viruses of eukaryotes and weak negative relation between (BS vs LP) for ssRNA viruses of eukaryotes. There are mainly two environment categories observed for viruses of eukaryotes; marine and freshwater. However, bacteriophages mentioned in this study have been isolated from much larger environmental

range; for e.g. human or animal body, soil, aquatic, food etc. This could be the main reason for disappearance of any existing trends among the wide background variation (explained in detail in section 4.2.

4.8 Concluding remarks

When we describe variations observed in viral traits, it should be kept in mind that these expressed viral trait variances are a combined product of both viral and host genotypes (Edwards and Steward, 2018 and references therein). The reason for this is, viruses are using the molecular machinery of the host for their replication. This highlight the necessity of including information on host traits, when investigating trends in viral traits and searching for trade-offs. Overall, I could not find evidence for any strong correlations, but moderate or weak correlations suggesting that while one trait in the compared pair might in certain cases limit the other trait, however other variables are probably more important. Most of the time, traits were uncorrelated.

To adequately explain the observed variations in viral traits, we need to consider both viral and host traits together. It is interesting to find out why the same trait pair (for e.g. BS and LP) shows a correlation for one virus group but shows no correlation for another virus group. There are viral trait evolution models in the literature to understand the patterns of trait covariation (Edwards and Steward, 2018b). It explains that when a small genome size virus infects a large genome size host (for e.g. ssDNA virus and its diatom host), there is a potential for producing very large burst size, but there is a fitness cost for having a long latent period, which is necessary to produce such a large burst size. In theory, it is assumed that there is a positive correlation between BS and LP, anyhow there is a huge variation observed in the data. The reason might be, latent period is highly influenced by the host growth rate, but it does not affect the burst size (Edwards and Steward, 2018). Nevertheless, as also assumed by Parada et al., (2006), most of the time the trends between traits are shaped by the domination and diversity of virus-host systems.

Here in the present study I use pairwise comparison of traits. However, it is a well-known fact that many factors are interconnected and involved in determination of trait variations and trade-offs. Therefore, instead of a pairwise approach, I would in the future involve more traits when identifying trade-offs, even though it is complicated.

4.9 Knowledge gaps and suggestions for future work;

Record et al., (2016) have pointed out the utility of trait base approach in virus ecology, as in many other fields in ecology. Litchman and Klausmeier, (2008) has suggested the "*need for a global database of phytoplankton traits, both marine and freshwater, with guidelines for standardized measurements of various traits*" (Carroll et al., 2018) has discussed "*Building a global atlas of zoonotic viruses*" in connection to Global Virome Project. Such data bases exist for medically important viruses of humans, and for genomic information of other viruses (NCBI, 2019), but I could not find any such database for functional and morphological traits of algal viruses or ecologically important virus. Following that idea, I would like to suggest;

- There is a need for a global database of viral traits, both marine, freshwater and other environments including medically important viruses, "with guidelines for standardized measurements of various traits" (idea extracted from Litchman and Klausmeier, 2008).
- Viral "trade-offs need to be characterized, including the shapes and interaction of multiple traits" (idea extracted from Litchman and Klausmeier, 2008).
- Need more attempts and approaches to isolate and characterized dsRNA, ssRNA and ssDNA viruses of unicellular eukaryote species.
- Expand the knowledge of viruses infecting freshwater algae.
- In the recent years more and more studies have focused on genetic characterization of viral genomes using molecular biological tools. If there is growth characterization at the same time the knowledge will be more complete.
- Search for novel approaches such as phage FISH and gene ELISA (Dang et al., 2015) to characterize virus's growth parameters and traits outside the conventional methods, especially when the virus -host system unculturable in the laboratory.
- Search for novel laboratory approaches or model base methods to estimate the BS values for archaeal viruses (for e.g. using the proposed model for calculate for energetic cost of building a virus) (Mahmoudabadi et al., 2017), Since traditional one-step growth curve method cannot utilize for most of the archaeal species.
- Recommend to use the recently proposed nomenclature system (Kropinski et al., 2009) when naming the new virus isolates for better consistency.
- Submit the assembled data in the present study to the **DRYAD research database** (DRYAD, 2019) for citable easy access, discoverability, and reuse in future research.

6. Appendix

6.1 Viruses of eukaryotes

Table 3.1 (continued) Eukaryotic host, latent period, burst size, genome size, dimensions of the virion for dsDNA viruses. Family- *Phycodnaviridae*

	Name of the virus	Eukaryotic host	Latent period (hours)	Burst size	Genome size (kb)	Virion size (Head diameter*) (nm)	Host Genome size (KB)	Reference
1	AaV(NCLDVs)#	*Aureococcus anophagefferens*	24	164 - 191	371			(Brown and Bidle, 2014)
2	LVLPs /E. huxleyi virus	*Emiliania huxleyi*	NG	350-700		180 & 140**		(Bratbak et al., 1996)
3	EhV	*Emiliania huxleyi*	12-14	400-1000	415	160-180		(Castberg et al., 2002)
4	ChlV	*Chlorella* sp.	3-4	200-350	330	190		(Van Etten et al., 1991)
5	PBCV-1(dark cond.)	*Chlorella*-like alga, NC64A	3	70- 150 (50 % less)				(Van Etten et al., 1983)
6	PBCV-1(light cond.)	*Chlorella*-like alga, NC64A	2,5-3	200-350				(Van Etten et al., 1983)
7	prototype chlorovirus PBCV-1	*Chlorella variabilis* strains, NC64A and Syngen 2–3	~2	250	327	190		(Quispe et al., 2017)
8	OSy-NE5 -Only Syngen (OSy) viruses	*Chlorella variabilis* Syngen 2–3	~2	250	327	190		(Quispe et al. 2017)
9	MpV	*Micromonas pusilla*	NG	NG	**77-110**	113		(Cottrell and Suttle, 1991)
10	M. pusilla virus (MPV)	*Micromonas pusilla*	7	72	190-210			(Waters and Chan, 1982)

* Head-diameter X length

** to size groups described in the literature

Nucleocytoplasmic large DNA viruses (NCLDV). A group of highly complex ds DNA viruses, infecting variety of eukaryotes (Gallot-Lavallée and Blanc, 2017)

Table 3.2 (continued) Eukaryotic host, latent period, burst size, genome size, dimensions of the virion for dsDNA viruses. Family- *Phycodnaviridae*

	Name of the virus	Eukaryotic host	Latent period (hours)	Burst size	Genome size (kb)	Virion size Head diameter* (nm)	Reference
11	Cafeteria roenbergensis virus (CroV) +	*Cafeteria roenbergensis* moi=1	5.5 ± 0.5 – 10.5 ± 1.5	470 ± 100			(Taylor et al., 2018)
12	Cafeteria roenbergensis virus (CroV) +	*Cafeteria roenbergensis* moi=10	5.5 ± 0.5 – 10.5 ± 1.5	310 ± 70			(Taylor et al. 2018)
13	PpV01	*Phaeocystis pouchetii*	12-18	350-600	485	120	(Jacobsen et al., 1996)
14	PgV Group I	*Phaeocystis globosa*	10	248 (77-356)	466	150	(Baudoux and Brussaard, 2005)
15	PgV Group II A	*Phaeocystis globosa*	12 or 16	345 (274-415)	177	100	(Baudoux and Brussaard 2005)
16	PgV Group II B	*Phaeocystis globosa*	16	381	177	100	(Baudoux and Brussaard 2005)
17	CeV- 01B	*Chrysochromulina ericina*	14-19	1800-4100	510	150-160	(Sandaa et al., 2001)
18	PoV-01B	*Pyramimonas orientalis*	14-19	800- 1000	**560**	220x180	(Sandaa et al. 2001)
19	CbV-PW1	*Chrysochromulina brevifilum*	NG	>320	NG	145-170	(Suttle and Chan, 1995)
20	CpV-BQ1 **Ж**	*Chrysochromulina parva*		63-96	485	145	(Mirza et al., 2015)
21	OIs1- large morphotypes	*Heterosigma akashiwo*	11	1100			(Lawrence et al., 2006)
22	OIs1- small morphotypes	*Heterosigma akashiwo*	17	16000			(Lawrence et al., 2006)

* Head-diameter X length , NG- not given in the reference, + giant viruses , **Ж** - *Phycodnaviridae* or *Mimiviridae*

Table 3.3 (continued) Eukaryotic host, latent period, burst size, genome size, dimensions of the virion for dsDNA viruses. Family- *Phycodnaviridae*

	Name of the virus	Eukaryotic host	Latent period (hours)	Burst size	Genome size (kb)	Virion size Head diameter* (nm)	Reference
23	HcV03	*Heterocapsa circularisquama* HU9433-P	48-72	1300<		180-210	(Tarutani et al., 2001)
24	(HcV)	*Heterocapsa circularisquama* at 20° C	56	1800			(Nagasaki et al., 2003)
25	(HcV)	*Heterocapsa circularisquama* at 25 ° C	40	2440			(Nagasaki et al., 2003)
26	OtV5	*Ostreococcus tauri*- OTH95	8-9	25	186.2	122	(Derelle et al., 2008a)
27	OlV1- standard	*Ostreococcus lucimarinus*	4.5*–8.5	435 ± 251	200	140	(Zimmerman et al., 2019)
28	OlV1- under limited light	*Ostreococcus lucimarinus*	6.5–8.5	237 ± 47	200	140	(Zimmerman et al., 2019)
29	OlV7-more virulent -standard	*Ostreococcus lucimarinus*	6.5–8.5	682 ± 408	200	140	(Zimmerman et al., 2019)
30	OlV7-more virulent -- under limited light	*Ostreococcus lucimarinus*	8.5–10.5	51 ± 20	200	140	(Zimmerman et al., 2019)
31	H. ericina virus RF02 (HeV RF02) #	*Haptolina ericina* (all tested strains), *P. kappa* UIO033	14-18	775 (683–933)	530	190±20×160±10	(Johannessen et al., 2015)
32	Prymnesium kappa virus RF01 (PkV RF01)#	*H. ericina* (all tested strains), *P. kappa* UIO033	24-32	80 (34–253)	NG	310±30	(Johannessen et al., 2015)
33	P. kappa virus RF02 (PkV RF02)#	*Prymnesium kappa* UIO034 and UIO032	12-16	400 (305–471)	507	160±30	(Johannessen et al., 2015)
		Range	2-72 Hrs	25-4100	77-560 kb	113-310 nm	

*Mimiviridae* family- NCLDV, Extreme values for each trait within the group, are marked in red

Table 4. **Eukaryotic host, latent period, burst size, genome size, dimensions of the virion for ssDNA viruses.**

Genus - Bacilladnavirus- tailless, rod shape

	Name of the virus	Eukaryotic host	Latent period (hours)	Burst size	Genome size (kb)	Virion size/ Head diameter (nm)	Reference
1	TnitDNAV	*Thalassiosira nitzschioides*	NG	NG	5.5	35	(Tomaru et al., 2012)
2	Csp05DNAV	C. sp. strain TG07- C28	<24	430	5.7	33	(Toyoda et al., 2012)
3	Csp07DNAV-surface water	*Chaetoceros* sp. Strain SS628-11	**<12**	**29**	5.5	34	(Kimura and Tomaru, 2013)
4	ClorDNAV	*Chaetoceros lorenzianus* Grunow	<48	**22000**	5.8	~34	(Tomaru et al., 2011b)
5	CtenDNAV	*C. tenuissimus*	**96**	320	5.6	37	(Tomaru et al., 2011a)
6	CsetDNAV	*Chaetoceros setoensis* IT07-C11	48	4700-20000	5.8	33	(Tomaru et al., 2013)
7	CtenDNAV type II	*C. tenuissimus* Meunier	<24	1737	5.5	37	(Kimura and Tomaru, 2015)
8	CsalDNAV (CsNIV)	*Chaetoceros salsugineum*	12-24	325	6	**38**	(Nagasaki et al., 2005)
9	CdebDNAV	*Chaetoceros debilis*	12-24	55	**7**	**32**	(Tomaru et al., 2008)
		Range	**12-96**	**29-22000**	**5.5-7**	**32-38**	

Extreme values for each trait within the group, are marked in red

Table 5. **Eukaryotic host, latent period, burst size, genome size, dimensions of the virion for ssRNA viruses.**

	Name of the virus	Eukaryotic host	Latent period (hours)	Burst size	Genome size (kb)	Virion size * (nm)	Virus Family	Reference
1	SssRNAV	*Schizochytrium* sp.	**<8**	5800-64000	10.2	25	superfamily-picornavirus like	(Takao et al., 2005)
2	HaRNAV	*Heterosigma akashiwo*	23-29	21000	9.1	25	*Marnaviridae*	(Lawrence et al., 2006)
3	HaRNAV	*Heterosigma akashiwo* (Hada) Hada ex Hada et Chihara	35	NG	9.1	25		(Tai et al., 2003)
4	HcRNAV	*Heterocapsa circularisquama*	24-48	3400-21000	**4.4**	30	NG	(Tomaru et al., 2004)
5	SpalV **	*Stephanopyxis palmeriana*	**< 80**	92	-	25-30		(Kim et al., 2015b)
6	CsfrRNAV	*Chaetoceros socialis* f. radians	<48	**66**	9.4	**22**	*Bacillariornaviridae*	(Tomaru et al., 2009a)
7	CtenRNAV01	*C. tenuissimus* Meunier	< 24	10000	9.4	31		(Shirai et al., 2008)
8	CtenRNAV type II	*C. tenuissimus* Meunier	24-48	287	9.5	35	Bacillarnavirus	(Kimura and Tomaru, 2015)
9	RsetRNAV (RsRNAV)	*Rhizosolenia setigera*	48	3100-exponential and 1010-stationary growth phase	**11.2** +smaller RNA molecules	32		(Nagasaki et al., 2004)
10	GdelRNAV	*Guinardia delicatula*	<12	**93400**	9	35-38	Bacillarnavirus	(Arsenieff et al., 2019)
			<8- < 80	66-93400	4.4-11.2	22-38		

** round shape virus while all the other viruses listed below, have icosahedral shape, tailless
Extreme values for each trait within the group, are marked in red.

6.2 Viruses of prokaryotes

6.2.1 Family- *Siphoviridae*

Table 6.1 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Siphoviridae.* Extreme values for each trait are marked in red.

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
1	QHHSV-1 (halovirus)	*Halomonas ventosae* -Halophilic	30	73	NG		(Fu et al., 2016)
2	TSP4	*Thermus* strain TC4 - Thermophilic	60	200	80	H- 73, T -785 x 10	(Lin et al., 2010b)
		psychrophilic bacteria					
3	9A- cold-active ¤	*Colwellia psychrerythraea* Strain 34H- from Arctic	270	55	80-90	H- 90 , T-200	(Wells and Deming, 2006)
	9A- cold-active ¤	*Colwellia psychrerythraea* Strain 34H	165	**5**	80-90	H- 90, T-201	(Wells et al., 2006)
	9A- cold-active ¤	*Colwellia psychrerythraea* Strain 34H	**10800**	**5**	80-90	H- 90, T-202	(Wells et al., 2006)
4	MYSP06	*Janthinobacterium* sp.	95	16	65–70	H-74, T-210x 10	(Li et al., 2016b)
5	FpV-7 ¤	*Flavobacterium psychrophilum* 950106-1/1	360	7	48	H- 67.2 x 68, T -239.6 x13.1	(Stenholm et al., 2008)
	FpV-9 ¤	*Flavobacterium psychrophilum* 950106-1/1	240	37	48	H- 60.1 x 63.1, T-172.6x 10.8	(Stenholm et al., 2008)
	FpV-9 ¤	*Flavobacterium psychrophilum* 010418-2/3	240	162	48	H- 60.1 x 63.1, T-172.6x 10.8	(Stenholm et al., 2008)
		phytopathogenic bacteria					
6	Cp1	*Xanthomonas axonopodis* pv. citri (syn., *Xanthomonas campestris* pv. citri or *Xanthomonas citri*)	60	20	43.8	H-60, T- 135 x 12	(Ahmad et al., 2014)

***** Head-diameter X length, Tail- Length X width, Extreme values for each trait within the group, are marked in red,¤ different experimental temperature

Table 6.2 (continued.) **Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Siphoviridae***

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
7	XTP1	*Xanthomonas campestris* pv. campestris	120	35	**180**	H- 102.4 x 121.6	(Weiss et al., 1994)
	rhizobiophages						
8	ΦRP1	*Robinia pseudoacacia rhizobia*	300	200	NG	H- 60 × 140, T- 260	(Małek et al., 2009)
9	ΦRP2	*Robinia pseudoacacia rhizobia*	300	200	NG	H- 60 × 140, T- 260	(Małek et al. 2009)
10	ΦRP3	*Robinia pseudoacacia rhizobia*	300	100	NG	H- 60 × 140, T 260 L	(Małek et al. 2009)
11	SR1 -	*Bradyrhizobium japonicum* USDA123	150	110	NG	H-79, T-115	(Appunu and Dhar, 2008)
12	SR2	*Bradyrhizobium japonicum CB1809*	300	140	NG	H-55, T-165	(Appunu and Dhar 2008)
13	ΦCP6-1 (temperate phage)	*Serratia liquefaciens* CP6	99	224	43.6	H -46.11x42.21, T-129.7	(Ashelford et al., 1999)
14	ΦCP6-2	*Serratia liquefaciens* CP7	104.8	174	40.4	H- 39.97x40.92, T- 135.04	(Ashelford et al. 1999)
15	ΦCP6-5	*Serratia liquefaciens* CP10	108.5	41	41.8	H-55,13x50.99, T- 138.62	(Ashelford et al. 1999)
							(Ashelford et al. 1999)
	ϕC6	*Clostridium difficile*	118	19	36.3	H -69.6, T -337.4	(Goh et al., 2005b)
16	P3K	*Vibrio* strain B8D (closely related to) *Vibrio owensii*	30	60	31	H -90, T- 150	(Yu et al., 2013a)
17	P4A	*Vibrio* strain B8D (closely related to) *Vibrio owensii*	70	23	48	H- 110, T -180	(Yu et al. 2013)
18	P7A	*Vibrio* strain B8D (closely related to) *Vibrio owensii*	60	33	41	H -110, T- 180	(Yu et al. 2013)
19	P8D	*Vibrio* strain B8D (closely related to) *Vibrio owensii*	60	29	30	H- 70, T- 160	(Yu et al. 2013)
20	P9C	*Vibrio* strain B8D (closely related to) *Vibrio owensii*	**10**	331	31	H- 100, T -160	(Yu et al. 2013)

* Head-diameter X length, Tail- Length X width

Table 6.3 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Siphoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
21	SSP002	*Vibrio vulnificus*	65	23	NG	H - 80.5, T- 161	(Lee et al., 2014)
22	DCEIV-9	*Exiguobacterium indicum*	20	51	NG	H- 56 , T -163	(Zhang et al., 2019)
23	AZ 1	*Pseudomonas aeruginosa*-2995	33	326	50	H- 61x 49, T- 128x 10-15	(Jamal et al., 2017)
24	vB_EliS-R6L	*Erythrobacter litoralis* DSM 8509	180	86	65.7	H- 75.9, T- 165.6	(Lu et al., 2017)
25	Sano #	*Xylella fastidiosa* strain Temecula 1	NG	100	56.1	H- 64, T- 204	(Ahern et al., 2014)
26	Salvo#	*Xanthomonas* strain EC-12	52	112	55.6	H- 64, T-207	(Ahern et al. 2014)
27	phiC119	*E. coli* O157 EC-48	20	210	47	H- 44, T-168–172 x 8	(Amarillas et al., 2016)
28	P-11 (O26)	Non-O157 Shiga toxigenic *Escherichia coli* (STEC)	15	**794**	NG		(Litt et al., 2018)
29	P-12 (O26)	non-O157 STEC O26	19	48	NG		(Litt et al. 2018)
30	P-14 (O111)	non-O157 STEC O111	21	288	NG		(Litt et al. 2018)
31	P-16 (O111)	non-O157 STEC O111	**10**	49	NG		(Litt et al. 2018)
32	P-17 (O111)	non-O157 STEC O111	15	257	NG		(Litt et al. 2018)
33	NG	*Corynebacterium (Propionibacterium) acnes*	60	25	NG	H -42 x 46, T- 130	(Zierdt, 1974)
34	FnpΦ02	*Fusobacterium nucleatum*	900	100	59	H- 83, T- 211	(Machuca et al., 2010)
35	iEPS5 - flagellatropic phage	*Salmonella enterica* serovar Typhimurium	30	100	59.2	H- 59,T- 217	(Choi et al., 2013)
36	LPST10	*Salmonella Typhimurium*	**10**	101	47.6	H- 83.2, T- 144.8 x10.9	(Huang et al., 2018)
37	FGCSSa2	*Salmonella typhimurium* PT 160 strains	NG	NG	NG	H 66, T -112x 9	(Carey-Smith et al., 2006)

* Head-diameter X length, Tail- Length X width, Extreme values for each trait within the group, are marked in red, # sano and Salvo >80% nucleotide identity

Table 6.4 (continued.) **Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Siphoviridae***

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
38	PW2	*Vibrio harveyi*	30	78	46	H- 50, T-136 x 11	(Phumkhachorn and Rattanachaikunsopon, 2010)
39	CG33	*Corynebacterium glutumicum*	18	16	**13.4**	H -40 , T-78	(Trautwetter et al., 1987)
		Range values	**10-10800**	**5-794**	**13.4-180**	**40.92-140**	

Extreme values for each trait within the group, are marked in red

Table 6.5 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Siphoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
	Lactic acid bacteria						
40	Φps05	*Pediococcus* sp. LA0281	34	12	24.1	H -51.2 , T- 129.6 x 11.6	(Yoon et al., 2007)
41	phiLdb	*Lactobacillus delbrueckii* subsp. *bulgaricus* ATCC11842	45	56	41	H-47.7, T- 129.8	(Wang et al., 2010)
42	Φ iLp84	*Lactobacillus paracasei*	85		38		(Mercanti et al., 2015)
43	FRC 1	Streptococcus cremoris-Lactic streptococci	40	70- 90	NG	H- 50-53 x 34-47, T- (74-100) x 7-10	(Bhimani and Freitas, 1991)
44	FRC 2	Streptococcus cremoris-Lactic streptococci	40	90-110	NG	H- 56-63, T- (100-120) x 7-10	(Bhimani and Freitas 1991)
45	FRC 3	Streptococcus cremoris-Lactic streptococci	40	80-100	NG	H- 66x 47-50, T- (100-110) x 6-10	(Bhimani and Freitas 1991)
46	FRC 4	Streptococcus lactis ssp. diacetylactis	45	100-150	NG	H- 56-70, T- (92- 100) x 7-10	(Bhimani and Freitas 1991)
47	EV3	*Lactobacillus sanfranciscensis* H2A	NG	30	32	H -48, T -180x 8.4	(Foschino et al., 2005)
	Nosocomial / Oppurtunistic				NG		
48	vB_KpnS_KP16 and vB_KpnS_KP36	*Klebsiella pneumoniae*	15	55	NG		(Kasik-Szeloch et al., 2013)
49	AB1	*Acinetobacter baumannii* KD311	18	409	45.2-46.9		(Yang et al. 2010)
50	RDJLΦ1	*Roseobacter denitrificans* OCh114	80	203	NG	H -69, T- 170	(Zhang and Jiao, 2009)
51	vB_DshS-R5C	*Dinoroseobacter shibae* named DFL12T	105	65	77.8	H -114 x70, T-142	(Yang et al., 2017)
		Range values	**10-10800**	**5-794**	**13.4-180**	**40.92-140**	

* Head-diameter X length, Tail- Length X width

6.2.2 Family- *Myoviridae*

Table 7.1 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Myoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
1	MMP17 - (Meiothermus Myoviridae phage 17)-	*Meiothermus* TG17- **Thermophilic**	60	15	33.5–39.5	H- 42 , T -120 x 17	(Lin et al., 2011)
2	PAS-1	*Aeromonas salmonicida* subsp. salmonicida	40	116.7	48	H- 53, T- 123x16	(Kim et al., 2012)
3	IHQ1	*Aeromonas punctata*	24	626	25–28	H- 128, T-108	(Haq et al., 2012)
4	Aeh1	*Aeromonas hydrophila* strain A3	39	17	NG	H-134.4x 89, T- 122.8 x21.7	(Chow and Rouf, 1983)
5	Aeh2	*Aeromonas hydrophila* ATCC 7966	52	92	NG		(Chow and Rouf 1983)
	Psychrophilic						
6	VNPH-1 (cold-active)	*Aeromonas sobria* NPH-1	20	80	110 - 120	H-116.7, T - 166.7x10	(Ji et al. 2015)
7	FpV-19	*Flavobacterium psychrophilum* 950106-1/1	240	51 ± 7	**8**	H- 127.2 x 128.7, T - 102.9 x28.7	(Stenholm et al. 2008)
	phytopathogenic						
8	ΦCP6-3	*Serratia liquefaciens* CP8	93.5	65	49.5	H- 51.08 x 45.18, T- 67.82	(Ashelford et al. 1999)
9	ΦCP6-6	*Serratia liquefaciens* CP11	99.5	18	82.8 ± 4.72	H- 61.3x52.73 ,T- 121.69	(Ashelford et al. 1999)
10	ϕC2	*Clostridium difficile*	32	5	43.3 ± 3.6	H- 64.8 ± 3.4, T- 147.7 ± 46.9	(Goh et al., 2005b)
11	ϕC5	*Clostridium difficile*	36	7	45.9 ± 3.8	H- 57.9 ± 6.9, T- 118.3 ± 9.6	(Goh et al., 2005b)
12	ϕC8	*Clostridium difficile*	90	33	54.5 ± 3.8	H- 59.8 ± 3.7, T- 139.6 ± 22.3	(Goh et al., 2005b)
13	VPUSM 4, 7, 8	*Vibrio cholerae* O1 El Tor Inaba	NG	NG	33.5	H 50– 60 , T 90–100	(Al-Fendi et al., 2014)
14	VP01	*Vibrio alginolyticus*	25	415		**H-164x85** , T -121 x13	(Sasikala and Srinivasan, 2016)

* Head-diameter X length, Tail- Length X width, Extreme values for each trait within the group, are marked in red

Table 7.2 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Myoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
15	pVa-21	*Vibrio alginolyticus* rm-8402	70	58	231.9	H- 87 ± 3, T- 240 ± 9	(Kim et al. 2019)
16	ϕSt2	*Vibrio alginolyticus* V1	30	97	**250.4**	H-81x151, T- 132 x20	(Kalatzis et al., 2016)
17	ϕGrn1	*Vibrio alginolyticus* V 1	30	44	248.6	H- 74x138, T- 134x20	(Kalatzis et al., 2016)
18	øZCW1	*Pseudoalteromonas* sp. strain SP48	90	91	NG	H- 123, T- 235	(Cai et al., 2011)
19	PH101	*Pseudoalteromonas marina* BH101	20	31.6	131.9	H- 60, T-40	(Wang et al., 2015)
20	vB_PaeM_LS1	*Pseudomonas aeruginosa*	30	98	66	H- 70, T-120	(Yuan et al., 2019)
	Enteric						
21	WZ1	*Shigella dysenteriae*	24	430	38	H-10x10, T-128 ± 25 x 21	(Jamal et al. 2015)
22	Bo-21	*Escherichia coli* O157:H7 (ATCC 4076)	15	426 [1] & 195 [2]		H- 80, T-100x18, neck 18	(López-Cuevas et al. 2011)
23	Av-05	*Escherichia coli* O157:H7 (ATCC 4076)	10	275 [1] & 112 [2]		H- 62x93, T- 100 x 12	(López-Cuevas et al. 2011)
24	Av-06	*Escherichia coli* O157:H7 (ATCC 4076)	15	288 [1] & 173 [2]		H -75x 106, T- 93x18	(López-Cuevas et al. 2011)
25	Av-08	*Escherichia coli* O157:H7 (ATCC 4076)	15	154 [1] & 147 [2]		H- 93 x 118, T-106 x 18	(López-Cuevas et al. 2011)
26	P-13 (O26)	non-O157 STEC O26	35	12		H -89, T- 115	(Litt et al. 2018)
27	P-19 (O103)	non-O157 STEC 0103	13	102		H -89, T- 115	(Litt et al. 2018)
28	P-21 (O103)	non-O157 STEC 0103	32	68		H -89, T- 115	(Litt et al. 2018)
29	P-22 (O103)	non-O157 STEC 0103	8	13		H -89, T- 115	(Litt et al. 2018)

* Head-diameter X length, Tail- Length X width, 1= 1st burst, 2= 2nd burst, Extreme values for each trait within the group, are marked in red

Table 7.3 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Myoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
30	P-8 (O121)	non-O157 STEC 0121	21	110		H -89, T- 115	(Litt et al. 2018)
31	J-4 (O121)	non-O157 STEC 0121	29	257		H -89, T- 115	(Litt et al. 2018)
32	J-7 (O121)	non-O157 STEC 0121	37	132		H -89, T- 115	(Litt et al. 2018)
33	J-14 (O45)	non-O157 STEC 045	33	13		H -89, T- 115	(Litt et al. 2018)
34	J-15 (O45)	non-O157 STEC 045	13	155		H -89, T- 115	(Litt et al. 2018)
35	J-18 (O145)	non-O157 STEC 145	22	74		H -89, T- 115	(Litt et al. 2018)
36	J-19 (O145)	non-O157 STEC 145	14	195		H -89, T- 115	(Litt et al. 2018)
37	J-25 (O145)	non-O157 STEC 145	28	23		H -89, T- 115	(Litt et al. 2018)
38	J-30 (O145)	non-O157 STEC 145	25	132		H -89, T- 115	(Litt et al. 2018)
39	Salmonella phage phi PVP-SE1 (multivalent)	*Escherichia coli* Bl21	19	28		H-84, T- 120x18	(Santos et al., 2010)
40	MSA6- Twort like phages	*Staphylococcus aureu*s strains	15	23	143	H- 66, T-173	(Kwiatek et al., 2012)
41	ϕ4D	*Enterococcus faecalis*	25	36	145	H-74, T-164	(Kwiatek et al., 2012)
42	ϕEF24C	*Enterococcus faecalis*	30	110-120	143	H- 93, T -204	(Uchiyama et al., 2008)
43	FGCSSa1	*Salmonella Typhimurium* PT 160 strains	50	139 ±13		H- 107, T- 123x20	(Carey-Smith et al. 2006)

* Head-diameter X length, Tail- Length X width, Extreme values for each trait within the group, are marked in red

Table 7.4 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Myoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
	Lactic acid bacteria						
44	Y 4	*Leuconostoc mesenteroides* LA112	NG	200<			(Yoon et al. 2002)
45	Y 5	*Lactobacillus plantarum* LA 280	46	11		H- 89, T- 166x20	(Yoon et al. 2002)
46	Y 20	*Lactobacillus* sp. LA296	19	74 ±10		H- 94, T- 118x28	(Yoon et al. 2002)
	Nosocomial / Oppurtunistic						
47	ZZ1	*Acinetobacter baumannii*	9	200	166.6	H-100, T- 120	(Jin et al., 2012)
48	vB_KpnM_KP15 and vB_KpnM_KP27	*Klebsiella pneumoniae*	25	10–15			(Kasik-Szeloch et al. 2013)
49	ϕSMA5	*Stenotrophomonas maltophilia* T39 (=*Xanthomonas maltophilia* & *Pseudomonas maltophilia)*	80	95	250	H-90 , T-90	(Chang et al. 2005)
50	Phage ST79	*Burkholderia pseudomallei*	30	304	31.7	H-54, T-148x17	(Yordpratum et al., 2011)

Table 7.5 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Myoviridae*- Halophilic Host

	Name of the virus	Bacterial host	NaCl (mol)	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
51	nt-1	*Beneckea natriegens*	0.06	90	12	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.08	90	112	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.16	60	321	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.20	53	413	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.25	45	520	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.33	50	522	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.41	50	511	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.16 M +4SN media	45	750	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.08 M	80	40	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.08 M+ mannose	85	95	NG	H- 120x70, T- 110	(Zachary, 1976)
	nt-1	*Beneckea natriegens*	0.08 M + KCl	60	430	NG	H- 120x70, T- 110	(Zachary, 1976)
52	S5100 ✶	*Halobacterium cutirubrum*	3 M	540	125	NG	NG	(Daniels and Wais, 1990)
	S5100	*Halobacterium cutirubrum*	3.5 M	540	60-65	NG	NG	Daniels and Wais, 1990)
	S5100	*Halobacterium cutirubrum*	4.5 M	660	60-65	NG	NG	Daniels and Wais, 1990)
		Range		**8-240**	**5-750**	**(8-250.4)**	**10-164**	

6.2.3 Family- *Podoviridae*

Table 8.1 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Podoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
		Psychrophilic					
1	VMY22- cold-active	*Bacillus cereus* MYB41-22	70	78	18 - 20	H- 31.9x 59.2 , T -43.2	(Ji et al. 2015)
2	FpV-2	*Flavobacterium psychrophilum* 950106-1/1	270	38	**90**	H- 50.1 x57.5	(Stenholm et al. 2008)
		Phytopathogenic					
3	Prado and Paz (phiKMV-like)	*Xylella fastidiosa* strain Temecula 1		99	43.9	H- 69	(Ahern et al. 2014)
4	Paz	*Xylella fastidiosa* strain Temecula 2		104	43.8	H-68	(Ahern et al. 2014)
5	Φ RSB1-(T7-Like)	*Ralstonia solanacearum*	165	30-60	43	H-60, T-20	(Kawasaki et al., 2009)
6	Cp2	*Xanthomonas axonopodis* pv. citri (syn., *Xanthomonas campestris* pv. citri or *Xanthomonas citri*)	90	100	42.9	H- 60 , T 15	(Ahmad et al. 2014)
7	ΦCP6-4	*Serratia liquefaciens* CP9	35.4	40	44.2	H- 40.**91x41.03**, T-12.35	(Ashelford et al. 1999)
8	φIBB-PF7A	*Pseudomonas fluorescens*	15	153	42	H- 63, T- 13x8	(Neubauer et al., 2008)
9	phiAxp-3 (N4-like)	*Achromobacter xylosoxidans*	80	9000	72,8	H 67, T-20	(Yanyan et al. 2016)
10	ϕSPB	*Salmonella enterica* serovar Paratyphi B	10	100	59	H- 153 × 57, T-12 × 7	(Ahiwale et al. 2013)
		Lactic acid producing					
11	asccϕ28 (P034 phage species)	*Lactococcus lactis*	44	121	18.7	H- 59 x42, T 21x9	(Kotsonis et al., 2008)
12	Φ 22	*Weissella cibaria* N 22	110	55	29	H- 92x50, T-27	(Pringsulaka et al., 2011)

* Head-diameter X length, Tail- Length X width, Extreme values for each trait within the group, are marked in red

Table 8.2 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Podoviridae*

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
		Nosocomial / Oppurtunistic					
13	φAB2	*Acinetobacter baumannii* ATCC17978	**<10**	200	40	H -60, T- 11x9	(Lin et al., 2010b)
14	vB_KpnP_KP32 and vB_KpnP_KP34	*Klebsiella pneumoniae*	15	~50-60			(Kasik-Szeloch et al. 2013)
15	Kpn5	*Klebsiella pneumoniae* B5055	20 ± 10.0	130	23.1	H- 85 , T- 17.2 x 18.3	(Kumari et al. 2010)
16	Kpn12	*Klebsiella pneumoniae* B5055	25 ± 5.0	140	23.6	H-135.2, T- 40.1x 28.4	(Kumari et al. 2010)
17	Kpn13	*Klebsiella pneumoniae* B5055	25 ± 8.7	120	24	H- 116.6, T- 17.4 x15.8	(Kumari et al. 2010)
18	Kpn17	*Klebsiella pneumoniae* B5055	35 ± 10.0	100	23.1	H -73.3, T- 18 x 16.1	(Kumari et al. 2010)
19	Kpn22	*Klebsiella pneumoniae* B5055	30 ± 5.0	110	23.1	H- 133.3, T- 26.6 x 33.3	(Kumari et al. 2010)
20	vB_PmuP_PHB01	*Pasteurella multocida*	**10**	190	37.2	H-55, t- 13	(Chen et al., 2019)
21	SR3	*Bradyrhizobium japonicum*	185	200		H 65 , T-25	(Appunu and Dhar 2008)
		Roseobacter clade					
22	RD-1410W1-01	*Roseobacter denitrificans* OCh114	60	27	72.7	H- 63.2, T-40	(Li et al., 2016a)
23	RD-1410Ws-07	*Roseobacter denitrificans* OCh115	<60	341	76.3	H-69.6, T-41.4	(Li et al., 2016a)
24	DS-1410Ws-06	*Dinoroseobacter shibae* DFL12	120	298	76.5	H-70.8, T-41.6	(Li et al., 2016a)
25	vB_Rsv217_RLP1	*Roseovarius* (Rsv.) 217	**240-360**	100	74.6	H-72.4	(Chan et al., 2014)
26	vB_RsvN_RPP1	*Roseovarius nubinhibens*	**240-360**	10	74.7	H-77.4	(Chan et al., 2014)
		Range values	**(10-300)**	**10-9000**	**18-90**	**H-41-153**	

* Head-diameter X length, Tail- Length X width

Table 8.3 (continued.) Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages similar to coliphage N4, (a large, short-tailed phage infecting Escherichia coli K12), in terms of genomic structure and morphology -podovirus-like roseophages

	Name of the virus	Bacterial host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
		Roseobacter clade					
27	DSS3φ2	*Silicibacter pomeroyi DSS-3*	180		74.6	H- 70 , T -26	(Zhao et al., 2009)
28	EE36φ1	*Sulfitobacter* sp. EE-36	120	>1000	73.3	H -70, T- 27	(Zhao et al., 2009)
		Range values	**(10-300)**	**10-9000**	**18-90**	**H-41-153**	

* Head-diameter X length, Tail- Length X width

Table 8.4 (continued.) Halophilic Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *Podoviridae* (Type C1)

	Name of the virus	Bacterial host	NaCl (mol) in experiment	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
		Halophilic						
29	nt-6	*Beneckea natriegens*	0.06	90	300	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.08	67-75	605	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.13	60	609	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.16	55	610	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.20	57	552	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.25	60	499	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.33	60	311	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.41	60	311	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.16 M +4SN media	60	1655	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.06 M	90-95	520	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.06 M + mannose	70	712	NG	H- 60, T -40	(Zachary, 1976)
	nt-6	*Beneckea natriegens*	0.06 M + KCl	60	1000	NG	H- 60, T -40	(Zachary, 1976)

* Head-diameter X length, Tail- Length X width

Table 9 Halophilic Bacterial host, latent period, burst size, genome size, dimensions of the virion for dsDNA -Bacteriophages - Family- *unknown*

	Name of the virus	Bacterial host	NaCl (M) in experiment	Latent period (min) (HDT)	Burst size	Genome size (kb)	Virion size * (nm)	Reference
		Moderately Halophilic						
1	UTAK	*Vibrio* B1	0.25	60 (68)	1.52	80	H- 86x 44, T-100	(Goel et al., 1996)
	UTAK	*Vibrio* B1	0.5	30 (40)	12.5	80	H- 86x 44, T-101	(Goel et al., 1996)
	UTAK	*Vibrio* B1	1	28 (37)	105	80	H- 86x 44, T-102	(Goel et al., 1996)
	UTAK	*Vibrio* B1	2	64 (62)	80.3	80	H- 86x 44, T-103	(Goel et al., 1996)
	UTAK	*Vibrio* B1	2.5	88 (131)	3.83	80	H- 86x 44, T-104	(Goel et al., 1996)

* Head-diameter X length, Tail- Length X width, (HDT) host doubling time

Table 10. Eukaryotic Host, latent period, burst size, genome size, dimensions of the virion for dsRNA viruses of eukaryotic algae and diverse other unclassified virus types

Nucleic acid type	Name of the virus	Host	Latent period (hours)	Burst size	Genome size (kb)	Virion size * (nm)	Virus Family	Morphology	Reference
dsRNA	MpRNAV-01B	*Micromonas pusilla* LAC 38	36 h	460–520	25.5 kb	65–80	*Reoviridae*	nearly spherical	(Brussaard et al., 2004)
NG	Heterosigma akashiwo Virus clone 01 (HaV01)	*Heterosigma akashiwo*	30-33h	770	NG	200			(Nagasaki et al., 1999)
NG	HaNIV	*Heterosigma akashiwo*	42h	100000	NG	30			Lawrence et al. 2001
NG	Chaetoceros nuclear inclusion virus: CspNIV	*Chaetoceros cf. gracilis*	<24		NG	25		icosahedral	(Bettarel et al., 2005)
NG	ScosV	*Skeletonema costatum*	<48 h	90-250	NG	45-50		polyhedral	(Kim et al., 2015a)

Table 11 Prokaryotic Host, latent period, burst size, genome size, dimensions of the phage for diverse groups and other unclassified phage types

Nucleic acid type	Name of the virus	Host	Latent period (min)	Burst size	Genome size (kb)	Virion size * (nm)	Virus Family	Morphology	Reference
dsDNA	P-9 (O45)	non-O157 STEC 045	30	302			*Tectiviridae*- Tailless phages		(Litt et al. 2018)
ssDNA	vB_Cib_ssDNA_P1	*Citromicrobium bathyomarinum* RCC1878		180	4.3	26	*Microviridae* - Amoyvirinae	polyhedral	(Zheng et al., 2018)
ds RNA	ϕ6	*Pseudomonas phaseolicola*	80-115	250-400	2.9 (S), 4 (M), 6.3(L) 3 segments	60	*Cystoviridae*	polyhedral head surrounded by a membranous, compressible lipid envelope	(Vidaver et al., 1973)
ds RNA	ϕ6	*Pseudomonas phaseolicola*	120-160	125-150			*Cystoviridae*		(Vidaver et al. 1973)
dsRNA	phiYY	*Pseudomonas aeruginosa* ¤	10	10-14	3 (S), 3,8 (M), 6,6 (L) 3 segments	50	*Cystoviridae*	Spherical, tailless	(Yang et al., 2016)
NG	ϕμ-4	*Bacillus stearothermophilus* NU strain 10	35	175		10		spherical	(Shafia and Thompson, 1964)

¤ opportunistic pathogen

6.2.4 Cyanophages

Table 12.1 (continued) **Cyanobacterial Host, latent period, burst size, genome size, dimensions of the virion for cyanophages**

Name of the virus	Cyanobacterial Host	Latent period (hours)	Burst size	Genome size (kb)	Host Genome size (Mbp)	Host cell Volume (cubic microns)	Growth rate (per day)	Virion size (nm)	Virus Family	Morphology	Reference
AS-1	*Anacystis nidulans & Synechococcus cedrorum*	8.5	50	-		5.3	-	H-90, T-243 x22	CM?	polyhedral, contractile tail	(Safferman et al., 1972)
PaV-LD	*Planktothrix agardhii*	48 - 72	340	95.3	5	62.5	0.207	70 - 85		icosahedral, no tail	(Gao, Yuan et al. 2009)
No name given	*Plectonema boryanum*	**2**	100	-	7.26	15	-	H- 54.5 T -116x16	CM?	polyhedral	(Singh, 1974)
N(S)1-temperate	*Anabaena* 77S15 = *Nostoc muscorum* sp. (previous)	20	70	**28**				H- 50, T- 10	CP	hexagonal, rarely visible tail	(Franche, 1987)
Ma-LMM01	*Microcystis aeruginosa*	6-12	50-120	160				H- 86, T- 209 x24	CM	Anisometric, tail helical symmetry	(Yoshida et al., 2006)
Ma-LMM02	*Microcystis aeruginosa*	8-12	82	160	4.3	60	0.6	H- 86, T- 209 x25	CM	Anisometric, tail helical symmetry	(Yoshida et al. 2006)
Vb-AphaS-CL131	*Aphanizomenon* flos-aquae Ralfs ex Bornet et Flahault	36	218	120	4.5	12	0.48	**H -97, T - 361 x 11**	CS	isometric	(Šulčius et al., 2015)
Cynophage clones S-BBS1 S-BBP1 S-PWP 1 S-PWP 2 S-PWP 3 S-PWP 4	*Synechococcus* spp. strains (DC2, SYN 48	9	250	-	2.4	1.15	-	H- 50-65, T-230	CM, CP, CS		(Suttle and Chan, 1993)
SN1	*Sphaerotilus natans*	1.5	320					H- 68x72, T-145	bradley Gr b,CS	hexagonal +plate and appendages	(Winston and Thompson, 1979)

Table 12.2 Cyanobacterial Host, latent period, burst size, genome size, dimensions of the virion for cyanophages

Name of the virus	Cyanobacterial Host	Latent period (hours)	Burst size	Genome size (kb)	Host Genome size (Mbp)	Host cell Volume (cubic microns)	Growth rate (per day)	Virion size (nm)	Virus Family	Reference
S-LBS1	*Synechococcus* st.TCC793	**96**	**400**	34.6*	2.37	1.2	0.3 max	H-75-80	CS-FW	(Zhong et al., 2018)

*for Synechococcus , genome size should multiply by two, since it contains usually 2 genome copies per cell (Binder and Chisholm, 1995)
Bold letters- Fresh water isolates
CM-Cyanomyovirus, , CP-Cyanopodoviris, CS- Cyanosiphovirus

Table 12.3 Cyanobacterial Host, latent period, burst size, genome size, dimensions of the virion for cyanophages (Freshwater). Modified after Obtained from (Edwards and Steward, 2018a)

Name of the virus	Cyanobacterial Host	Latent period (hours)	Burst size	Genome size (kb)	Host Genome size (Mbp)	Host cell Volume (cubic microns)	Growth rate (per day)	Virion size * (nm)	Reference	
N-1	*Nostoc muscorum-Anabaena* PCC 7120	7	100	65	7.21	11		55	(Adolph and Haselkorn, 1972)	
LPP-1	*Plectonema boryanum-Leptolyngbya boryana*	7	350	41.5	7.26	15		59	(Padan et al., 1970)	
LPP-1	*P.boryanum-L.boryana*	7	100	41.5	7.26	15		59	(Goldstein et al., 1967)	
AS-1M	*Synechococcus cedrorum UI 1911*	8	40	99	3.5	5.3		90	(Sherman et al., 1976)	
S-5(L)	*S.elongatus CALU 698*	18	125					80	**	
SM-2	*S.elongatum UTEX 563*	16	250		2.7	0.32		52.5	**	
S-4(L)	*S.elongatus CALU 698*	12	225					75	**	
MaMV-DC	*Microcystis aeruginosa FACHB-524*	24	80	160	4.3	60	0.133	70	**	
SM-1	*M. aeruginosa NRC-1, PCC 7941*	20	300	98	4.3	60		67	**	
A-1(L)	*Anabaena variabilis Myers strain, PCC 7118*	5	72.5	68	5.87	11		60	**	
A-4(L)	*A. variabilis Myers strain, PCC 7118*	2.5	110	41.8	5.87	11		60	**	
Pf-WMP4	*Phormidium foveolarum*	4	145	40.9	4.65	6.3		55	**	
Pf-WMP3	*Phormidium foveolarum*	5	113	43.2	4.65	6.3		55	**	

** (Edwards and Steward, 2018a and references therein)

Table 12.4 Cyanobacterial Host, latent period, burst size, genome size, dimensions of the virion for cyanophages (Marine). Modified after Obtained from (Edwards and Steward, 2018a)

Name of the virus	Cyanobacterial Host	Latent period (hours)	Burst size	Genome size (kb)	Host Genome size (Mbp)	Host cell Volume (cubic microns)	Growth rate (per day)	Virion size * (nm)	Reference
S-PM2	*Synechococcus* WH7803	9	45	**196**	2.37*	1.1	0.33	85	(Wilson et al., 1996)
S-RIM1	*Synechococcus WH7803*	7	63		2.37	1.1			**
S-RIM8	*Synechococcus WH7803*	7	86		2.37	1.1			**
S-PM2	*Synechococcus WH7803*								**
S-CBM2	*Synechococcus CB0101*	15	28	180	2.37	1.1		90	**
S-CBP1	*Synechococcus CB0102*	6	86	48	2.37	1.1		52	**
S-CBP2	*Synechococcus CB0208*	8	92	48	2.37	1.1		55	**
S-CBP3	*Synechococcus CB0101*	8	75	48	2.37	1.1		55	**
S-CBS2	*Synechococcus CB0204*	24	65	70	2.37	1.1		90	**
S-CBS3	*Synechococcus CB0202*	24	175	30	2.37	1.1		56	**
S-CBS4	*Synechococcus CB0101*	24	57	65	2.37	1.1		72	**
P60	*Synechococcus* WH7803		81	47.8	2.37	1.8			Brown et al., 2006 and ref. therein
Syn5	*Synechococcus WH8109*	1	24	46.2	2.37	1.1	1.11	60	**
S-TIM5	*Synechococcus WH8102*	6	35	161	2.37	1.1		86	**
P-SSP7	*Prochlorococcus MED4*	5.5	40	45	1.66	0.113	0.6		**
P-GSP1	*Prochlorococcus MED4*	5	65	45	1.66	0.113	0.55		**
P-HM1	*Prochlorococcus MED4*	6.5	15	181	1.66	0.113	0.6		**
P-HM2	*Prochlorococcus MED4*	5	30	184	1.66	0.113	0.6		**
P-HS2	*Prochlorococcus MED4*	6.5	160	38.3	1.66	0.113	0.6		**
P-HS3	*Prochlorococcus MED4*	8	25	37.8	1.66	0.113	0.6		**
	Range	**2-96**	**15-400**	**28-196**				**50-97**	

Blue-Cyanomyovirus, red-cyanopodo, ** (Edwards and Steward, 2018a and references therein)

6.3 Viruses of archaea

Table 13.1 Halophilic archaeal host, latent period, burst size, genome size, dimensions of the virion for dsDNA -archaeal viruses -

	Name of the virus	Archaeal host	NaCl (M) in experiment	Latent period (hours)	Burst size	Genome size (kb)	Taxonomy	Reference
1	S5100	*Halobacterium cutirubrum*	3	9	125	-	*Myoviridae*	(Daniels and Wais, 1990)
	S5100	*Halobacterium cutirubrum*	3.5	9	60-65	-	*Myoviridae*	(Daniels and Wais, 1990)
	S5100	*Halobacterium cutirubrum*	4.5	11	60-65	-	*Myoviridae*	(Daniels and Wais, 1990)
2	Halophage Ja.l	*Halobacterium cutirubrum*		6	140			(Wais et al., 1975)
		extremely halophilic						
3	Hs1	*Halobacterium salinarium* str. 1	17.5%	17	300	NG		(Torsvik and Dundas, 1980))
	Hs1	*Halobacterium salinarium* str. 1	17.5%	**21**	150	NG		(Torsvik and Dundas, 1980)
	Hs1	*Halobacterium salinarium* str. 1	20% w/v	15	310	NG		(Torsvik and Dundas, 1980)
	Hs1	*Halobacterium salinarium* str. 1	20% w/v	17	**470**	NG		(Torsvik and Dundas, 1980)
	Hs1	*Halobacterium salinarium* str. 1	25 % w/v	15	300	NG		(Torsvik and Dundas, 1980)
	Hs1	*Halobacterium salinarium* str. 1	25 % w/v	18	210	NG		(Torsvik and Dundas, 1980)
	Hs1	*Halobacterium salinarium* str. 1	25 % w/v	18	530	NG		(Torsvik and Dundas, 1980)

Table 13.2 Halophilic archaeal host, latent period, burst size, genome size, dimensions of the virion for dsDNA -archaeal viruses

	Name of the virus	Archaeal host	NaCl % experiment	Latent period (hours)	Burst size	Genome size (kb)	Virion size * (nm)	Taxonomy	Morphology	Reference
4	SNJ1	*Natrinema* sp.J7-2	18	6	**20**-50	16.4	-		spherical	(Mei et al., 2015)
	SNJ1	*Natrinema* sp.J7-2	25	4	100-150					(Mei et al., 2015)
	SNJ1	*Natrinema* sp.J7-2	30	5	40-70					(Mei et al., 2015)
5	øH	*Halobacterium halobium*		7	170					Schnabel et al., 1982)
6	SH1	*Haloarcula hispanica*		5-6	200	30	70	halosphaerovirus-proposed	spherical	(Porter et al., 2005)
7	PH1	*Haloarcula hispanica*		4-6	50–100	28	51	halosphaerovirus-proposed	round	(Porter et al., 2013)
8	His1	*Haloarcula hispanica*		4	continuous	**14.4**	**44** × 77	Salterprovirus-*Fuselloviridae*	spindle	(Bath and Dyall-Smith, 1998)
9	His2	*Haloarcula hispanica*		**2**	315	16	44 × 67	Salterprovirus	spindle	(Bath et al., 2006)
10	His2	*Haloarcula hispanica*		4	10	16	45 × 67	Salterprovirus	spindle	(Bath et al., 2006)
11	HHIV-2	*Haloarcula hispanica*		4-5	180	30.5	80		icosahedral tailless	(Jaakkola et al., 2012)
12	SIRV1	*Sulfolobus islandicus*		8	continuous	32.3	830	*Rudiviridae*-crenarchaeotal viruses	unenveloped rod	(Prangishvili et al., 1999)
13	SIRV2	*Sulfolobus islandicus*		6	continuous	**35.8**	900	*Rudiviridae*	unenveloped rod	(Prangishvili et al., 1999)
		Range		**2-21**	**20-470**	**14.4-35.8**	**44-900**			

6. References

2011. *ICTV 9th report [WWW Document]. International Committee on Taxonomy of Viruses (ICTV).* [Online]. Available: https://talk.ictvonline.org/ictv-reports/ictv_9th_report/ [Accessed 07.11.19].

ABEDON, S., T & MURRAY, K., L. 2013. Archaeal Viruses, Not Archaeal Phages: An Archaeological Dig. *Archaea,* 2013.

ABEDON, S. T. 1999. Bacteriophage T4 resistance to lysis-inhibition collapse. *Genet. Res.,* 74**,** 1-11.

ABEDON, S. T., HERSCHLER, T. D. & STOPAR, D. 2001. Bacteriophage Latent-Period Evolution as a Response to Resource Availability. *Applied and Environmental Microbiology,* 67**,** 4233.

ABEDON, S. T., HYMAN, P. & THOMAS, C. 2003. Experimental Examination of Bacteriophage Latent-Period Evolution as a Response to Bacterial Availability. *Applied and Environmental Microbiology,* 69**,** 7499.

ACKERMANN, H. & PRANGISHVILI, D. 2012. Prokaryote viruses studied by electron microscopy. *Arch. Virol.,* 157**,** 1843-1849.

ADOLPH, K. W. & HASELKORN, R. 1972. Photosynthesis and the development of blue-green algal virus N-1. *Virology,* 47**,** 370-374.

AHERN, S. J., DAS, M., BHOWMICK, T. S., RY, Y. & GONZALEZ, C. F. 2014. Characterization of novel virulent broad-host-range phages of *Xylella fastidiosa* and *Xanthomonas*.(Report). *Journal of Bacteriology,* 196**,** 459.

AHIWALE, S. S., BANKAR, A. V., TAGUNDE, S. N., ZINJARDE, S., ACKERMANN, H. W. & KAPADNIS, B. P. 2013. Isolation and characterization of a rare waterborne lytic phage of *Salmonella enterica* serovar Paratyphi B. *Canadian Journal of Microbiology,* 59**,** 318-323.

AHMAD, A. A., OGAWA, M., KAWASAKI, T., FUJIE, M. & YAMADA, T. 2014. Characterization of Bacteriophages Cp1 and Cp2, the Strain-Typing Agents for *Xanthomonas axonopodis* pv. *citri*. *Applied and Environmental Microbiology,* 80**,** 77-85.

AL-FENDI, A., SHUEB, R. H., RAVICHANDRAN, M. & YEAN, C. Y. 2014. Isolation and characterization of lytic vibriophage against Vibrio cholerae O1 from environmental water samples in Kelantan, Malaysia. *Journal of Basic Microbiology,* 54**,** 1036-1043.

AMARILLAS, L., CHAIDEZ, C., GONZALEZ-ROBLES, A., LUGO-MELCHOR, Y. & LEON-FELIX, J. 2016. Characterization of novel bacteriophage phiC119 capable of lysing multidrug-resistant Shiga toxin-producing *Escherichia coli* O157:H7. *PeerJ,* 4**,** e2423.

ANGLY, F. E., FELTS, B., BRIETBART, M., SALAMON, P., EDWARDS, R. A., CARLSON, C., CHAN, A. M., HAYNES, M., KELLEY, S., LIU, H., MAHAFFY, J. M., MUELLER, J. E., NULTON, J., OLSON, R., PARSONS, R., RAYHAWK, S., SUTTLE, C. A. & ROHWER, F. 2006. The marine viromes of four oceanic regions. *Pl o S Biology,* 4**,** 2121.

APPUNU, C. & DHAR, B. 2008. Morphology and General Characteristics of Lytic Phages Infective on Strains of *Bradyrhizobium japonicum*. *Current Microbiology,* 56**,** 21-27.

ARSENIEFF, L., SIMON, N., RIGAUT-JALABERT, F., LE GALL, F., CHAFFRON, S., CORRE, E., COM, E., BIGEARD, E. & BAUDOUX, A.-C. 2019. First Viruses Infecting the Marine Diatom *Guinardia delicatula*. *Frontiers in microbiology,* 9**,** 3235.

ASHELFORD, K. E., FRY, J. C., BAILEY, M. J., JEFFRIES, A. R. & DAY, M. J. 1999. Characterization of Six Bacteriophages of *Serratia liquefaciens* CP6 Isolated from the Sugar Beet Phytosphere. *Applied and Environmental Microbiology,* 65**,** 1959.

ATANASOVA, N. S., OKSANEN, H. M. & BAMFORD, D. H. 2015. Haloviruses of archaea, bacteria, and eukaryotes. *Current Opinion in Microbiology,* 25**,** 40.

AZAM, F., FENCHEL, T., FIELD, J. G., GRAY, J. S., MEYER-REIL, L. A. & THINGSTAD, F. 1983. The Ecological Role of Water-Column Microbes in the Sea. *Marine Ecology Progress Series,* 10**,** 257-263.

BACHY, C., CHARLESWORTH, C. J., CHAN, A. M., FINKE, J. F., WONG, C.-H., WEI, C.-L., SUDEK, S., COLEMAN, M. L., SUTTLE, C. A. & WORDEN, A. Z. 2018. Transcriptional responses of the marine green alga *Micromonas pusilla* and an infecting prasinovirus under different phosphate conditions. *Environmental Microbiology,* 20**,** 2898-2912.

BARR, J. J. 2017. A bacteriophages journey through the human body.

BATH, C., CUKALAC, T., PORTER, K. & DYALL-SMITH, M. L. 2006. His1 and His2 are distantly related, spindle-shaped haloviruses belonging to the novel virus group, Salterprovirus. *Virology,* 350**,** 228-239.

BATH, C. & DYALL-SMITH, M. L. 1998. His1, an Archaeal Virus of the *Fuselloviridae* Family That Infects *Haloarcula hispanica*. *The Journal of Virology,* 72**,** 9392.

BAUDOUX, A. C. & BRUSSAARD, C. P. D. 2005. Characterization of different viruses infecting the marine harmful algal bloom species *Phaeocystis globosa*. *Virology,* 341**,** 80-90.

BAUDOUX, A. C. & BRUSSAARD, C. P. D. 2008. Influence of irradiance on algal-host interactions 1. *Journal of Phycology,* 44**,** 902-908.

BEKLIZ, M., COLSON, P. & SCOLA, B. 2016. The Expanding Family of Virophages. *Viruses,* 8**,** 317.

BERGH, I., KNUT YNGVE, B., GUNNAR, B. & MIKAL, H. 1989. High abundance of viruses found in aquatic environments. *Nature,* 340**,** 467.

BERNARD LA, S., CHRISTELLE, D., ISABELLE, P., CATHERINE, R., LINA, B., GHISLAIN, F., MICHÈLE, M., MARIE, S.-M., PATRICK, F., EUGENE, K. & DIDIER, R. 2008. The virophage as a unique parasite of the giant mimivirus. *Nature,* 455**,** 100.

BETTAREL, Y., KAN, J., WANG, K., WILLIAMSON, K., COONEY, S., RIBBLETT, S., CHEN, F., WOMMACK, K. & COATS, D. 2005. Isolation and preliminary characterisation of a small nuclear inclusion virus infecting the diatom *Chaetoceros cf. gracilis*. *Aquat. Microb. Ecol.,* 40**,** 103-114.

BHIMANI, R. S. & FREITAS, Y. M. 1991. Isolation and Characterization of the Bacteriophages of Lactic *Streptococci*. *Journal of Dairy Science,* 74**,** 1461-1471.

BIZE, A., KARLSSON, E. A., EKEFJÄRD, K., QUAX, T. E. F., PINA, M., PREVOST, M.-C., FORTERRE, P., TENAILLON, O., BERNANDER, R. & PRANGISHVILI, D. 2009. A unique virus release mechanism in the Archaea. *Proceedings of the National Academy of Sciences,* 106**,** 11306.

BØRSHEIM, K. Y. 1993. Native marine bacteriophages. *FEMS Microbiology Letters,* 102**,** 141-159.

BOUVIER, T. & DEL GIORGIO, P. A. 2007. Key role of selective viral-induced mortality in determining marine bacterial community composition. *Environ. Microbiol.,* 9**,** 287-297.

BRADLEY, D. E. 1967. Ultrastructure of bacteriophage and bacteriocins. *Bacteriological reviews,* 31**,** 230.

BRATBAK, G., EGGE, J. K. & HELDAL, M. 1993. Viral mortality of the marine alga *Emiliania huxleyi* (Haptophyceae) and termination of algal blooms. *Marine Ecology Progress Series,* 93**,** 39-48.

BRATBAK, G., JACOBSEN, A., HELDAL, M., NAGASAKI, K. & THINGSTAD, F. 1998. Virus production in *Phaeocystis pouchetii* and its relation to host cell growth and nutrition. *Aquat. Microb. Ecol.,* 16**,** 1-9.

BRATBAK, G., THINGSTAD, F. & HELDAL, M. 1994. Viruses and the microbial loop. *An International Journal,* 28**,** 209-221.

BRATBAK, G., WILSON, W. & HELDAL, M. 1996. Viral control of *Emiliania huxleyi* blooms? *Journal of Marine Systems,* 9**,** 75-81.

BREITBART, M. & ROHWER, F. 2005. Here a virus, there a virus, everywhere the same virus? *Trends in Microbiology,* 13**,** 278-284.

BREITBART, M., SALAMON, P., ANDRESEN, B. & MAHAFFY, J. 2002. Genomic analysis of uncultured marine viral communities. *Proceedings of the National Academy of Sciences of the United States of America***,** 14250-14255.

BREITBART, M., THOMPSON, L., SUTTLE, C. & SULLIVAN, M. B. 2007. Exploring the Vast Diversity of Marine Viruses. *Oceanography,* 20**,** 135-139.

BROWN-JAQUE, M., CALERO-CÁCERES, W. & MUNIESA, M. 2015. Transfer of antibiotic-resistance genes via phage-related mobile elements. *Plasmid,* 79**,** 1-7.

BROWN, C. M. & BIDLE, K. D. 2014. Attenuation of virus production at high multiplicities of infection in *Aureococcus anophagefferens*. *Virology,* 466-467**,** 71-81.

BROWN, C. M., CAMPBELL, D. & LAWRENCE, J. 2007. Resource dynamics during infection of *Micromonas pusilla* by virus MpV-Sp1. *Environ. Microbiol.*

BROWN, C. M., LAWRENCE, J. & CAMPBELL, D. 2006. Are phytoplankton population density maxima predictable through analysis of host and viral genomic DNA content? *J. Mar. Biol. Assoc. U.K.,* 86**,** 491-498.

BRUSSAARD, C. P., WILHELM, S., THINGSTAD, F., WEINBAUER, M., BRATBAK, G., HELDAL, M., KIMMANCE, S., MIDDELBOE, M., NAGASAKI, K., PAUL, J., SCHROEDER, D., SUTTLE, C., VAQU**?**, D. & WOMMACK, K. 2008. Global-scale processes with a nanoscale drive: the role of marine viruses. *The ISME Journal,* 2**,** 575-8.

BRUSSAARD, C. P. D., NOORDELOOS, A. A. M., SANDAA, R. A., HELDAL, M. & BRATBAK, G. 2004. Discovery of a dsRNA virus infecting the marine photosynthetic protist *Micromonas pusilla*. *Virology,* 319**,** 280-291.

CAI, W., WANG, H., TIAN, Y., CHEN, F. & ZHENG, T. 2011. Influence of a Bacteriophage on the Population Dynamics of Toxic Dinoflagellates by Lysis of Algicidal Bacteria. *Applied and Environmental Microbiology,* 77**,** 7837.

CANCHAYA, C., FOURNOUS, G., CHIBANI-CHENNOUFI, S., DILLMANN, M.-L. & BRÜSSOW, H. 2003. Phage as agents of lateral gene transfer. *Current Opinion in Microbiology,* 6**,** 417-424.

CAREY-SMITH, G. V., BILLINGTON, C., CORNELIUS, A. J., HUDSON, J. A. & HEINEMANN, J. A. 2006. Isolation and characterization of bacteriophages infecting *Salmonella* spp. *FEMS Microbiology Letters,* 258**,** 182-186.

CARROLL, D., WATSON, B., TOGAMI, E., DASZAK, P., MAZET, J. A., CHRISMAN, C. J., RUBIN, E. M., WOLFE, N., MOREL, C. M., GAO, G. F., BURCI, G. L., FUKUDA, K., AUEWARAKUL, P. & TOMORI, O. 2018. Building a global atlas of zoonotic viruses. *Bulletin of the World Health Organization,* 96**,** 292-294.

CASTBERG, T., THYRHAUG, R., LARSEN, A., SANDAA, R.-A., HELDAL, M., VAN ETTEN, J. & BRATBAK, G. 2002. Isolation and characterization of a virus that infects *Emiliania huxleyi* (Haptophyta). 38**,** 767-774.

CHAN, J. Z.-M., MILLARD, A. D., MANN, N. H. & SCHÄFER, H. 2014. Comparative genomics defines the core genome of the growing N4-like phage genus and identifies N4-like Roseophage specific genes. *Frontiers in Microbiology,* 5.

CHANG, H.-C., CHEN, C.-R., LIN, J.-W., SHEN, G.-H., CHANG, K.-M., TSENG, Y.-H. & WENG, S.-F. 2005. Isolation and Characterization of Novel Giant *Stenotrophomonas maltophilia* Phage {phi}SMA5. *Applied and Environmental Microbiology,* 71**,** 1387.

CHEN, Y., GUANGHAO, G., ERCHAO, S., JIAOYANG, S., LAN, Y., LILI, Z., WAN, L., LIN, H., ZHONG, P., XIBIAO, T., HUANCHUN, C. & BIN, W. 2019. Isolation of a T7-Like Lytic *Pasteurella* Bacteriophage vB_PmuP_PHB01 and Its Potential Use in Therapy against *Pasteurella multocida* Infections. *Viruses,* 11**,** 86.

CHIBANI-CHENNOUFI, S., SIDOTI, J., BRUTTIN, A., DILLMANN, M. L., KUTTER, E., QADRI, F., SARKER, S. A. & BRUSSOW, H. 2004. Isolation of *Escherichia coli* bacteriophages from the stool of pediatric diarrhea patients in Bangladesh. *J Bacteriol,* 186.

CHOI, Y., SHIN, H., LEE, J.-H. & RYU, S. 2013. Identification and characterization of a novel flagellum-dependent *Salmonella*-infecting bacteriophage, iEPS5. *Applied and environmental microbiology,* 79**,** 4829.

CHOW, M. S. & ROUF, M. A. 1983. Isolation and Partial Characterization of Two *Aeromonas hydrophila* Bacteriophages. *Applied and environmental microbiology,* 45**,** 1670.

COLSON, P., DE LAMBALLERIE, X., FOURNOUS, G. & RAOULT, D. 2012. Reclassification of Giant Viruses Composing a Fourth Domain of Life in the New Order Megavirales. *Intervirology,* 55**,** 321-332.

CORREA, A. M., WELSH, R. & VEGA THURBER, R. 2013. Unique nucleocytoplasmic dsDNA and +ssRNA viruses are associated with the dinoflagellate endosymbionts of corals. *The ISME Journal,* 7**,** 13-27.

COTTRELL, M. & SUTTLE, C. 1995. Dynamics of a lytic virus infecting the photosynthetic marine picoflagellate *micromonas pusilla*. *Limnol. Oceanogr.,* 40**,** 730-739.

COTTRELL, M. T. & SUTTLE, C. A. 1991. Wide-spread occurrence and clonal variation in viruses which cause lysis of a cosmopolitan, eukaryotic marine phytoplankter, *Micromonas pusilla*. *Marine Ecology Progress Series,* 78**,** 1-9.

CULLEY, A. I., ANDREW, S. L. & CURTIS, A. S. 2003. High diversity of unknown picorna-like viruses in the sea. *Nature,* 424**,** 1054.

DANG, V. T., HOWARD-VARONA, C., SCHWENCK, S. & SULLIVAN, M. B. 2015. Variably lytic infection dynamics of large Bacteroidetes podovirus phi38:1 against two *Cellulophaga baltica* host strains. *Environmental Microbiology,* 17**,** 4659-4671.

DANIELS, L. L. & WAIS, A. C. 1990. Ecophysiology of Bacteriophage S5100 Infecting *Halobacterium cutirubrum*. *Applied and Environmental Microbiology,* 56**,** 3605.

DANOVARO, R., DELL**?**ANNO, A., CORINALDESI, C., RASTELLI, E., CAVICCHIOLI, R., KRUPOVIC, M., NOBLE, R. T., NUNOURA, T. & PRANGISHVILI, D. 2016. Virus-mediated archaeal hecatomb in the deep seafloor. *Science Advances,* 2.

DANOVARO, R., RASTELLI, E., CORINALDESI, C., TANGHERLINI, M. & DELL'ANNO, A. 2017. Marine archaea and archaeal viruses under global change. *F1000Research,* 6**,** 1241-1241.

DE PAEPE, M., TADDEI, F. O. & KIRKWOOD, T. 2006. Viruses' Life History: Towards a Mechanistic Basis of a Trade-Off between Survival and Reproduction among Phages (Decay/Reproduction Tradeoff among Phages). *PLoS Biology,* 4**,** e193.

DELLAS, N., SNYDER, J. C., BOLDUC, B. & YOUNG, M. J. 2014. Archaeal Viruses: Diversity, Replication, and Structure. *Annual Review of Virology,* 1**,** 399-426.

DEMORY, D., LAURE, A., NATHALIE, S., CHRISTOPHE, S., FABIENNE, R.-J., DOMINIQUE, M., PEI, G., ESTELLE, B., STEPHAN, J., ANTOINE, S., OLIVIER,

B., SOPHIE, R. & ANNE-CLAIRE, B. 2017. Temperature is a key factor in *Micromonas*-virus interactions. *The ISME Journal*, 11.
DENNEHY, J. J. & WANG, I.-N. 2011. Factors influencing lysis time stochasticity in bacteriophage λ. *BMC Microbiology*, 11**,** 174.
DERELLE, E., FERRAZ, C., ESCANDE, M.-L., EYCHENI, S., COOKE, R., PIGANEAU, G. L., DESDEVISES, Y., BELLEC, L., MOREAU, H., GRIMSLEY, N. & WARD, N. 2008a. Life-Cycle and Genome of OtV5, a Large DNA Virus of the Pelagic Marine Unicellular Green Alga *Ostreococcus tauri* (Marine Picoplankton Virus). *PLoS ONE*, 3**,** e2250.
DERELLE, E., FERRAZ, C., ESCANDE, M.-L., EYCHENI, S., COOKE, R., PIGANEAU, G. L., DESDEVISES, Y., BELLEC, L., MOREAU, H., GRIMSLEY, N. & WARD, N. 2008b. Life-Cycle and Genome of OtV5, a Large DNA Virus of the Pelagic Marine Unicellular Green Alga Ostreococcus tauri (Marine Picoplankton Virus). *PLoS ONE*, 3**,** e2250.
DESSAU, M., GOLDHILL, D., MCBRIDE, R. L., TURNER, P. E., MODIS, Y. & MALIK, H. S. 2012. Selective Pressure Causes an RNA Virus to Trade Reproductive Fitness for Increased Structural and Thermal Stability of a Viral Enzyme (Structural Basis of a Viral Evolutionary Tradeoff). 8**,** e1003102.
DRYAD. 2019. *The Dryad Digital Repository* [Online]. Available: (https://datadryad.org/stash/) [Accessed 11.11.2019].
EDWARDS, K. F. & STEWARD, G. F. 2018a. *Data from: Host Traits Drive Viral Life Histories across Phytoplankton Viruses, Dryad Digital Repository*, [Online]. Available: https://datadryad.org/stash/dataset/doi:10.5061/dryad.31sm2 [Accessed 17.11.2019].
EDWARDS, K. F. & STEWARD, G. F. 2018b. Host Traits Drive Viral Life Histories across Phytoplankton Viruses. *The American Naturalist*, 191**,** 566-581.
ELLIS, E. L. & DELBRÜCK, M. 1939. The growth of bacteriophage. *The Journal of general physiology*, 22**,** 365.
FANCELLO, L., TRAPE, S., ROBERT, C., BOYER, M., POPGEORGIEV, N., RAOULT, D. & DESNUES, C. 2013. Viruses in the desert: a metagenomic survey of viral communities in four perennial ponds of the Mauritanian Sahara. *The ISME Journal*, 7**,** 359-69.
FLORES, C. O., MEYER, J. R., VALVERDE, S., FARR, L. & WEITZ, J. S. 2011. Statistical structure of host–phage interactions. *Proceedings of the National Academy of Sciences*, 108**,** E288.
FORTERRE, P. 2010. Defining Life: The Virus Viewpoint. *Origins of Life and Evolution of Biospheres*, 40**,** 151-160.
FOSCHINO, R., VENTURELLI, E. & PICOZZI, C. 2005. Isolation and Characterization of a Virulent Lactobacillus sanfranciscensis Bacteriophage and Its Impact on Microbial Population in Sourdough. *An International Journal*, 51**,** 413-418.
FRANCHE, C. 1987. Isolation and characterization of a temperate cyanophage for a tropical Anabaena strain. *Archives of Microbiology*, 148**,** 172-177.
FROST, L., S. , RAPHAEL, L., ANNE, O. S. & ARIANE, T. 2005. Mobile genetic elements: the agents of open source evolution. *Nature Reviews Microbiology*, 3**,** 722.
FU, C.-Q., ZHAO, Q., LI, Z.-Y., WANG, Y.-X., ZHANG, S.-Y., LAI, Y.-H., XIAO, W. & CUI, X.-L. 2016. A novel Halomonas ventosae-specific virulent halovirus isolated from the Qiaohou salt mine in Yunnan, Southwest China.(Report). 20**,** 101.
FUHRMA, J. A. & SUTTLE, C. A. 1993. Viruses in Marine Planktonic Systems. *Oceanography*, 6**,** 51-63.

FUHRMAN, J. A. 1999. Marine viruses and their biogeochemical and ecological effects. *Nature,* 399**,** 541-548.
FUSSMANN, G. F., LOREAU, M. & ABRAMS, P. A. 2007. Eco-evolutionary dynamics of communities and ecosystems. *Functional Ecology,* 21**,** 465-477.
GALLOT-LAVALLÉE, L. & BLANC, G. 2017. A Glimpse of Nucleo-Cytoplasmic Large DNA Virus Biodiversity through the Eukaryotic Genomics Window. *Viruses,* 9**,** 17.
GAO, E., YUAN, X., LI, R. & ZHANG, Q. 2009. Isolation of a novel cyanophage infectious to the filamentous cyanobacterium Planktothrix agardhii (Cyanophyceae) from Lake Donghu, China. *Aquatic Microbial Ecology,* 54**,** 163-170.
GARCÍA-VILLADA, L. & DRAKE, J. W. 2013. Experimental selection reveals a trade-off between fecundity and lifespan in the coliphage Qß. *Open biology,* 3**,** 130043.
GIBBS, A. J., TORRONEN, M., MACKENZIE, A. M., WOOD, J. T., ARMSTRONG, J. S., KONDO, H., TAMADA, T. & KEESE, P. L. 2011. The enigmatic genome of Chara australis virus. *Journal of General Virology,* 92**,** 2679-2690.
GOBLER, C. J., HUTCHINS, D., FISHER, N., COSPER, E. & SANUDO-WILHELMY, S. A. 1997. Release and bioavailability of C, N, P, Se, and Fe following viral lysis of a marine chrysophyte. *Limnol. Oceanogr.,* 42**,** 1492-1504.
GOEL, U., KAURI, T., KUSHNER, D. J. & ACKERMANN, H.-W. 1996. A moderately halophilic Vibrio from a Spanish saltern and its lytic bacteriophage. *Canadian Journal of Microbiology,* 42**,** 1015-1023.
GOH, S., RILEY, T. V. & CHANG, B. J. 2005a. Isolation and Characterization of Temperate Bacteriophages of <em>Clostridium difficile</em&gt. *Applied and Environmental Microbiology,* 71**,** 1079.
GOH, S., RILEY, T. V. & CHANG, B. J. 2005b. Isolation and Characterization of Temperate Bacteriophages of Clostridium difficile. *Applied and Environmental Microbiology,* 71**,** 1079-1083.
GOLDHILL, D. H. & TURNER, P. E. 2014. The evolution of life history trade-offs in viruses. *Current Opinion in Virology,* 8**,** 79-84.
GOLDSTEIN, D. A., BENDET, I. J., LAUFFER, M. A. & SMITH, K. M. 1967. Some biological and physicochemical properties of blue-green algal virus LPP-1. *Virology,* 32**,** 601-613.
GROSE, J. H. & CASJENS, S. R. 2014. Understanding the enormous diversity of bacteriophages: The tailed phages that infect the bacterial family Enterobacteriaceae. *Virology,* 468-470**,** 421-443.
GUDELJ, I., WEITZ, J. S., FERENCI, T., CLAIRE HORNER-DEVINE, M., MEYER, J. R., FORDE, S. E. & MARX, C. J. 2010. An Integrative Approach to Understanding Microbial Diversity: From Intracellular Mechanisms to Community Structure. *Ecology Letters,* 13.
GUIXA-BOIXAREU, N., CALDERON-PAZ, J., HELDAL, M. & BRATBAK, G. 1996. Viral lysis and bacterivory as prokaryotic loss factors along a salinity gradient. *Aquatic Microbial Ecology,* 11**,** 215-227.
HAMDI, S., ROUSSEAU, G. M., LABRIE, S. J., TREMBLAY, D. M., KOURDA, R. S., BEN SLAMA, K. & MOINEAU, S. 2017. Characterization of two polyvalent phages infecting Enterobacteriaceae. *Scientific Reports,* 7**,** 40349.
HAQ, I. U., CHAUDHRY, W. N., ANDLEEB, S. & QADRI, I. 2012. Isolation and Partial Characterization of a Virulent Bacteriophage IHQ1 Specific for Aeromonas punctata from Stream Water. *Microbial Ecology,* 63**,** 954-963.
HEINEMAN, R. H., BROWN, S. P. & BROCKHURST, M. A. 2012. Experimental Evolution of a Bacteriophage Virus Reveals the Trajectory of Adaptation across a Fecundity/Longevity Trade-Off (Evolution across a Trade-Off). 7**,** e46322.

HELDAL, M. & BRATBAK, G. 1991. Production and decay of viruses in aquatic environments. *Marine Ecology Progress Series,* 72**,** 205-212.
HENNES, K. P. & SIMON, M. 1995. Significance of Bacteriophages for Controlling Bacterioplankton Growth in a Mesotrophic Lake. *Applied and Environmental Microbiology,* 61**,** 333.
HONDA, D., YOKOCHI, T., NAKAHARA, T., ERATA, M. & HIGASHIHARA, T. 1998. Schizochytrium limacinum sp. nov., a new thraustochytrid from a mangrove area in the west Pacific Ocean. *Mycological Research,* 102**,** 439-448.
HUANG, C., SHI, J., MA, W., LI, Z., WANG, J., LI, J. & WANG, X. 2018. Isolation, characterization, and application of a novel specific Salmonella bacteriophage in different food matrices. *Food Research International,* 111**,** 631-641.
HURWITZ, B. L., HALLAM, S. J. & SULLIVAN, M. B. 2013. Metabolic reprogramming by viruses in the sunlit and dark ocean. *Genome biology,* 14**,** R123.
HYMAN, P. & ABEDON, S. T. 2009. Practical methods for determining phage growth parameters. *Methods Mol Biol,* 501**,** 175-202.
HYMAN, P. & ABEDON, S. T. 2010. Chapter 7 - Bacteriophage Host Range and Bacterial Resistance. *Advances in Applied Microbiology.* Academic Press.
ICTV. 2019a. *International Committee on Taxonomy of Viruses* [Online]. Available: (https://talk.ictvonline.org) [Accessed 30.10.19 2019].
ICTV. 2019b. *Introduction to the ICTV Online Report, Virus diagrams* [Online]. International Committee on Taxonomy of Viruses (ICTV). Available: https://talk.ictvonline.org/ictv-reports/ictv_online_report/introduction/w/introduction-to-the-ictv-online-report/422/hosts-bacteria-and-archaea and https://talk.ictvonline.org/ictv-reports/ictv_online_report/introduction/w/introduction-to-the-ictv-online-report/421/hosts-algae-fungi-and-protozoa [Accessed 12.11.2019].
JAAKKOLA, S. T., PENTTINEN, R. K., VIL?N, S. T., JALASVUORI, M., R?NNHOLM, G., BAMFORD, J. K. H., BAMFORD, D. H. & OKSANEN, H. M. 2012. Closely related archaeal Haloarcula hispanica icosahedral viruses HHIV-2 and SH1 have nonhomologous genes encoding host recognition functions. *Journal of virology,* 86**,** 4734.
JACOB, H. M.-M., JAMIE, C. S. & MARK, J. Y. 2018. Archaeal Viruses from High-Temperature Environments. *Genes,* 9**,** 128.
JACOBSEN, A., BRATBAK, G. & HELDAL, M. 1996. ISOLATION AND CHARACTERIZATION OF A VIRUS INFECTING PHAEOCYSTIS POUCHETII (PRYMNESIOPHYCEAE) 1. *Journal of Phycology,* 32**,** 923-927.
JAMAL, M., ANDLEEB, S., JALIL, F., IMRAN, M., NAWAZ, M. A., HUSSAIN, T., ALI, M. & DAS, C. R. 2017. Isolation and characterization of a bacteriophage and its utilization against multi-drug resistant Pseudomonas aeruginosa-2995. *Life Sciences,* 190**,** 21-28.
JAMAL, M., CHAUDHRY, W. N., HUSSAIN, T., DAS, C. R. & ANDLEEB, S. 2015. Characterization of new Myoviridae bacteriophage WZ1 against multi-drug resistant (MDR) Shigella dysenteriae.(Report).
JI, X., ZHANG, C., FANG, Y., ZHANG, Q., LIN, L., TANG, B. & WEI, Y. 2015. Isolation and characterization of glacier VMY22, a novel lytic cold-active bacteriophage of Bacillus cereus. *Virologica Sinica,* 30**,** 52-58.
JIAO, N., GERHARD, J. H., DENNIS, A. H., RONALD, B., GERHARD, K., STEVEN, W. W., DAVID, L. K., MARKUS, G. W., TINGWEI, L., FENG, C. & FAROOQ, A. 2010. Microbial production of recalcitrant dissolved organic matter: long-term carbon storage in the global ocean. *Nature Reviews Microbiology,* 8**,** 593.

JIN, J., LI, Z.-J., WANG, S.-W., WANG, S.-M., HUANG, D.-H., LI, Y.-H., MA, Y.-Y., WANG, J., LIU, F., CHEN, X.-D., LI, G.-X., WANG, X.-T., WANG, Z.-Q. & ZHAO, G.-Q. 2012. Isolation and characterization of ZZ1, a novel lytic phage that infects Acinetobacter baumannii clinical isolates. *BMC Microbiology,* 12**,** 156.

JOHANNESSEN, T. V., BRATBAK, G., LARSEN, A., OGATA, H., EGGE, E. S., EDVARDSEN, B., EIKREM, W. & SANDAA, R.-A. 2015. Characterisation of three novel giant viruses reveals huge diversity among viruses infecting Prymnesiales (Haptophyta). *Virology,* 476**,** 180-188.

JOVER, L., F., EFFLER, T. C., ALISON, B., STEVEN, W. W. & JOSHUA, S. W. 2014. The elemental composition of virus particles: implications for marine biogeochemical cycles. *Nature Reviews Microbiology,* 12**,** 519.

JUN-HUA, T., XIAN-DAN, L., YAN-JUN, K., LIANG-JUN, C., XIN-CHENG, Q., XU, J., HOLMES, E. & YONG-ZHEN, Z. 2015. Unprecedented genomic diversity of RNA viruses in arthropods reveals the ancestry of negative-sense RNA viruses. *eLife,* 4.

KALATZIS, P. G., BASTIAS, R., KOKKARI, C. & KATHARIOS, P. 2016. Isolation and Characterization of Two Lytic Bacteriophages, [phi]St2 and [phi]Grn1; Phage Therapy Application for Biological Control of Vibrio alginolyticus in Aquaculture Live Feeds.(Report). 11.

KARIN, M., FELIX, B. & CHRISTIAN, W. 2018. A Wake-Up Call: We Need Phage Therapy Now. *Viruses,* 10**,** 688.

KASIK-SZELOCH, A., DRULIS-KAWA, Z., WEBER-DABROWSKA, B., KASSNER, J., MAJKOWSKA-SKROBEK, G., AUGUSTYNIAK, D., AUSIAK-SZELACHOWSKA, M., A'ACZEK, M., GORSKI, A. & KROPINSKI, A. M. 2013. Characterising the biology of novel lytic bacteriophages infecting multidrug resistant Klebsiella pneumoniae.(Report). *Virology Journal,* 10.

KAWASAKI, T., SHIMIZU, M., SATSUMA, H., FUJIWARA, A., FUJIE, M., USAMI, S. & YAMADA, T. 2009. Genomic Characterization of Ralstonia solanacearum Phage {phi}RSB1, a T7-Like Wide-Host-Range Phage. *The Journal of Bacteriology,* 191**,** 422.

KEEN, E. C. 2014. Tradeoffs in bacteriophage life histories. *Bacteriophage,* 4.

KIM, J., KIM, C.-H., YOUN, S.-H. & CHOI, T.-J. 2015a. Isolation and Physiological Characterization of a Novel Algicidal Virus Infecting the Marine Diatom Skeletonema costatum. *The Plant Pathology Journal,* 31**,** 186-191.

KIM, J., YOON, S.-H. & CHOI, T.-J. 2015b. Isolation and physiological characterization of a novel virus infecting Stephanopyxis palmeriana (Bacillariophyta). *Algae,* 30**,** 81-87.

KIM, J. H., SON, J. S., CHOI, Y. J., CHORESCA, C. H., SHIN, S. P., HAN, J. E., JUN, J. W., KANG, D. H., OH, C., HEO, S. J. & PARK, S. C. 2012. Isolation and Characterization of a Lytic Myoviridae Bacteriophage PAS-1 with Broad Infectivity in Aeromonas salmonicida. *Current Microbiology,* 64**,** 418-426.

KIM, S., GIRI, S., KIM, H. & JEONG, D. 2019. Isolation and characterisation of pVa-21, a giant bacteriophage with anti-biofilm potential against Vibrio alginolyticus. *Scientific Reports (Nature Publisher Group),* 9**,** 1-10.

KIMURA, K. & TOMARU, Y. 2013. Isolation and Characterization of a Single-Stranded DNA Virus Infecting the Marine Diatom Chaetoceros sp. Strain SS628-11 Isolated from Western JAPAN.(Research Article). *PLoS ONE,* 8**,** e82013.

KIMURA, K. & TOMARU, Y. 2015. Discovery of two novel viruses expands the diversity of single-stranded DNA and single-stranded RNA viruses infecting a cosmopolitan marine diatom.(Report). 81**,** 1120-1131.

KING, A. M. Q., LEFKOWITZ, E., ADAMS, M. J. & CARSTENS, E. B. 2011. *Virus Taxonomy: Ninth Report of the International Committee on Taxonomy of Viruses*, Elsevier Science.

KNOWLES, B. 2016. Piggyback-the-Winner: lytic to temperate switching of viral communities.

KOKJOHN, T. A., SAYLER, G. S. & MILLER, R. V. 1991. Attachment and replication of Pseudomonas aeruginosa bacteriophages under conditions simulating aquatic environments. *Journal of Genetic Microbiology,* 137**,** 661-666.

KOTSONIS, S. E., POWELL, I. B., PILLIDGE, C. J., LIMSOWTIN, G. K. Y., HILLIER, A. J. & DAVIDSON, B. E. 2008. Characterization and Genomic Analysis of Phage ascc{phi}28, a Phage of the Family Podoviridae Infecting Lactococcus lactis. *Applied and Environmental Microbiology,* 74**,** 3453.

KRISHNAMURTHY, S. R., JANOWSKI, A. B., ZHAO, G., BAROUCH, D. & WANG, D. 2016. Hyperexpansion of RNA Bacteriophage Diversity. *PLoS Biology,* 14.

KROPINSKI, A. M., PRANGISHVILI, D. & LAVIGNE, R. 2009. Position paper: The creation of a rational scheme for the nomenclature of viruses of Bacteria and Archaea. *Environmental Microbiology,* 11**,** 2775-2777.

KRUPOVIC, M., CVIRKAITE-KRUPOVIC, V., IRANZO, J., PRANGISHVILI, D. & KOONIN, E. V. 2018. Viruses of archaea: Structural, functional, environmental and evolutionary genomics. *Virus Research,* 244**,** 181-193.

KUMARI, S., HARJAI, K. & CHHIBBER, S. 2010. Isolation and characterization of Klebsiella pneumoniae specific bacteriophages from sewage samples. *Folia Microbiologica,* 55**,** 221-7.

KUZYAKOV, Y. & MASON-JONES, K. 2018. Viruses in soil: Nano-scale undead drivers of microbial life, biogeochemical turnover and ecosystem functions. *Soil Biology and Biochemistry,* 127**,** 305-317.

KWIATEK, M., PARASION, S., MIZAK, L., GRYKO, R., BARTOSZCZE, M. & KOCIK, J. 2012. Characterization of a bacteriophage, isolated from a cow with mastitis, that is lytic against Staphylococcus aureus strains. *Official Journal of the Virology Division of the International Union of Microbiological Societies,* 157**,** 225-234.

LABONT, J., M. & SUTTLE, C., A. 2013. Previously unknown and highly divergent ssDNA viruses populate the oceans. *The ISME Journal,* 7**,** 2169.

LAWRENCE, J. E., BRUSSAARD, C. P. D. & SUTTLE, C. A. 2006. Virus-Specific Responses of Heterosigma akashiwo to Infection. *Applied and Environmental Microbiology,* 72**,** 7829.

LAWRENCE, J. E., CHAN, A. M. & SUTTLE, C. A. 2001. A NOVEL VIRUS (HaNIV) CAUSES LYSIS OF THE TOXIC BLOOM-FORMING ALGA HETEROSIGMA AKASHIWO (RAPHIDOPHYCEAE. *Journal of Phycology,* 37**,** 216-222.

LE ROMANCER, M., GAILLARD, M., GESLIN, C. & PRIEUR, D. 2007. Viruses in extreme environments. *Reviews in Environmental Science and Bio/Technology,* 6**,** 17-31.

LEE, H. S., CHOI, S., SHIN, H., LEE, J.-H. & CHOI, S. H. 2014. Vibrio vulnificus Bacteriophage SSP002 as a Possible Biocontrol Agent. *Applied and Environmental Microbiology,* 80**,** 515.

LI, B., ZHANG, S., LONG, L. & HUANG, S. 2016a. Characterization and Complete Genome Sequences of Three N4-Like Roseobacter Phages Isolated from the South China Sea. *Current Microbiology,* 73**,** 409-418.

LI, M., WANG, J., ZHANG, Q., LIN, L., KUANG, A., MATERON, L., JI, X. & WEI, Y. 2016b. Isolation and Characterization of the Lytic Cold-Active Bacteriophage MYSP06 from the Mingyong Glacier in China. *Current Microbiology,* 72**,** 120-127.

LIN, L., HAN, J., JI, X., HONG, W., HUANG, L. & WEI, Y. 2011. Isolation and characterization of a new bacteriophage MMP17 from Meiothermus. *Extremophiles,* 15**,** 253-258.
LIN, L., HONG, W., JI, X., HAN, J., HUANG, L. & WEI, Y. 2010a. Isolation and characterization of an extremely long tail Thermus bacteriophage from Tengchong hot springs in China. Berlin :.
LIN, N.-T., CHIOU, P.-Y., CHANG, K.-C., CHEN, L.-K. & LAI, M.-J. 2010b. Isolation and characterization of phi AB2: a novel bacteriophage of Acinetobacter baumannii. *Research in microbiology,* 161**,** 308.
LITCHMAN, E. & KLAUSMEIER, C. 2008. Trait-Based Community Ecology of Phytoplankton. *Annual Review of Ecology, Evolution, and Systematics,* 39**,** 615.
LITT, P., SAHA, J. & JARONI, D. 2018. Characterization of Bacteriophages Targeting Non-O157 Shiga Toxigenic Escherichia coli. *Journal of Food Protection,* 81**,** 785-794.
LONG, A., LAUREN, D. M., JENNIFER, M. & JOHN, H. P. 2007. Comparison of lysogeny (prophage induction) in heterotrophic bacterial and Synechococcus populations in the Gulf of Mexico and Mississippi river plume. *The ISME Journal,* 2**,** 132.
LÓPEZ-CUEVAS, O., CASTRO-DEL CAMPO, N., LEÓN-FÉLIX, J., GONZÁLEZ-ROBLES, A. & CHAIDEZ, C. 2011. Characterization of bacteriophages with a lytic effect on various Salmonella serotypes and Escherichia coli O157:H7. *Canadian Journal of Microbiology,* 57**,** 1042-1051.
LU, L., CAI, L., JIAO, N. & ZHANG, R. 2017. Isolation and characterization of the first phage infecting ecologically important marine bacteria Erythrobacter. *Virology Journal,* 14.
MAAT, D. & BRUSSAARD, C. 2016. Both phosphorus- and nitrogen limitation constrain viral proliferation in marine phytoplankton. *Aquatic Microbial Ecology,* 77**,** 87-97.
MACHUCA, P., DAILLE, L., VINES, E., BERROCAL, L. & BITTNER, M. 2010. Isolation of a Novel Bacteriophage Specific for the Periodontal Pathogen Fusobacterium nucleatum. *Applied and Environmental Microbiology,* 76**,** 7243.
MAHMOUDABADI, G., MILO, R. & PHILLIPS, R. 2017. Energetic cost of building a virus. *Proceedings of the National Academy of Sciences,* 114**,** E4324-E4333.
MAŁEK, W., WDOWIAK-WRÓBEL, S., BARTOSIK, M., KONOPA, G. & NARAJCZYK, M. 2009. Characterization of Phages Virulent for Robinia pseudoacacia Rhizobia. *Current Microbiology,* 59**,** 187-192.
MANN, N. H. 2003. Phages of the marine cyanobacterial picophytoplankton. Oxford, UK.
MANN, N. H. 2005. The Third Age of Phage (Primer). *PLoS Biology,* 3**,** e182.
MANRIQUE, P., BOLDUC, B., WALK, S. T., VAN DER OOST, J., DE VOS, W. M. & YOUNG, M. J. 2016. Healthy human gut phageome. *Proceedings of the National Academy of Sciences,* 113**,** 10400.
MANRIQUE, P., DILLS, M. & YOUNG, M. J. 2017. The Human Gut Phage Community and Its Implications for Health and Disease. *Viruses,* 9.
MATHIAS, C. B., KIRSCHNER, A. K. T. & VELIMIROV, B. 1995. Seasonal Variations of Virus Abundance and Viral Control of the Bacterial Production in a Backwater System of the Danube River. *Applied and Environmental Microbiology,* 61**,** 3734.
MCGEE, L. W., AITCHISON, E. W., CAUDLE, S. B., MORRISON, A. J., ZHENG, L., YANG, W., ROKYTA, D. R. & WOROBEG, M. 2014. Payoffs, Not Tradeoffs, in the Adaptation of a Virus to Ostensibly Conflicting Selective Pressures (Payoffs in the Adaptation to Conflicting Selective Pressures). 10**,** e1004611", PLoS Genetics, vol. 10, no. 10.
MEI, Y., HE, C., HUANG, Y., LIU, Y., ZHANG, Z., CHEN, X. & SHEN, P. 2015. Salinity regulation of the interaction of halovirus SNJ1 with its host and alteration of the

halovirus replication strategy to adapt to the variable ecosystem. *PloS one,* 10**,** e0123874-e0123874.
MERCANTI, D., ACKERMANN, H.-W. & QUIBERONI, A. 2015. Characterization of Two Temperate Lactobacillus paracasei Bacteriophages: Morphology, Kinetics and Adsorption. *Intervirology,* 58**,** 49-56.
MIDDELBOE, M. 2000. Bacterial Growth Rate and Marine Virus–Host Dynamics. *Microbial Ecology,* 40**,** 114-124.
MIDDELBOE, M., CHAN, A. & BERTELSEN, S. (eds.) 2010. *Isolation and life cycle characterization of lytic viruses infecting heterotrophic bacteria and cyanobacteria. in SW Wilhelm, MG Weinbauer & CA Suttle (eds), Manual of aquatic viral ecology . American Society of Limnology and Oceanography, Inc., Waco, Tex, pp. 118-33.*

.
MIRZA, S. F., STANIEWSKI, M. A., SHORT, C. M., LONG, A. M., CHABAN, Y. V. & SHORT, S. M. 2015. Isolation and characterization of a virus infecting the freshwater algae *Chrysochromulina parva*. *Virology,* 486**,** 105-115.
MOJICA, K. D. A. & BRUSSAARD, C. P. D. 2014. Factors affecting virus dynamics and microbial host–virus interactions in marine environments. *FEMS Microbiology Ecology,* 89**,** 495-515.
MURRAY, A. G. & JACKSON, G. A. 1992. Viral dynamics: a model of the effects of size, shape, motion and abundance of single-celled planktonic organisms and other particles. *Marine Ecology Progress Series,* 89**,** 103-116.
MYA, B., PETER, S., BJARNE, A., JOSEPH, M. M., ANCA, M. S., DAVID, M., FAROOQ, A. & FOREST, R. 2002. Genomic analysis of uncultured marine viral communities. *Proceedings of the National Academy of Sciences of the United States of America,* 99**,** 14250.
NAGASAKI, K., TARUTANI, K. & YAMAGUCHI, M. 1999. Growth Characteristics of *Heterosigma akashiwo* Virus and Its Possible Use as a Microbiological Agent for Red Tide Control. *Applied and Environmental Microbiology,* 65**,** 898.
NAGASAKI, K., TOMARU, Y., KATANOZAKA, N., SHIRAI, Y., NISHIDA, K., ITAKURA, S. & YAMAGUCHI, M. 2004. Isolation and Characterization of a Novel Single-Stranded RNA Virus Infecting the Bloom-Forming Diatom *Rhizosolenia setigera*. *Applied and Environmental Microbiology,* 70**,** 704-711.
NAGASAKI, K., TOMARU, Y., TAKAO, Y., NISHIDA, K., SHIRAI, Y., SUZUKI, H. & NAGUMO, T. 2005. Previously Unknown Virus Infects Marine Diatom. *Applied and Environmental Microbiology,* 71**,** 3528.
NAGASAKI, K., TOMARU, Y., TARUTANI, K., KATANOZAKA, N., YAMANAKA, S., TANABE, H. & YAMAGUCHI, M. 2003. Growth Characteristics and Intraspecies Host Specificity of a Large Virus Infecting the Dinoflagellate *Heterocapsa circularisquama*. *Applied and Environmental Microbiology,* 69**,** 2580.
NCBI. 2019. *The National Center for Biotechnology Information NCBI database* [Online]. Available: (https://www.ncbi.nlm.nih.gov/) [Accessed 11.11.2019].
NEUBAUER, P., SILLANKORVA, S. & AZEREDO, J. 2008. Isolation and characterization of a T7-like lytic phage for *Pseudomonas fluorescens*. *BMC Biotechnology,* 8**,** 80.
ORTMANN, A. C. & SUTTLE, C. A. 2005. High abundances of viruses in a deep-sea hydrothermal vent system indicates viral mediated microbial mortality. *Deep Sea Research Part I: Oceanographic Research Papers,* 52**,** 1515-1527.

PADAN, E., GINZBURG, D. & SHILO, M. 1970. The reproductive cycle of cyanophage LPP1-G in *Plectonema boryanum* and its dependence on photosynthetic and respiratory systems. *Virology,* 40**,** 514-521.
PARADA, V., HERNDL, G. J. & WEINBAUER, M. G. 2006. Viral burst size of heterotrophic prokaryotes in aquatic systems. *Journal of the Marine Biological Association of the United Kingdom,* 86**,** 613-621.
PARADA, V., SINTES, E., VAN AKEN, H. M., WEINBAUER, M. G. & HERNDL, G. J. 2007. Viral Abundance, Decay, and Diversity in the Meso- and Bathypelagic Waters of the North Atlantic. *Applied and Environmental Microbiology,* 73**,** 4429.
PAUL, J. H. & SULLIVAN, M. B. 2005. Marine phage genomics: what have we learned? *Current Opinion in Biotechnology,* 16**,** 299-307.
PAUL, J. H., SULLIVAN, M. B., SEGALL, A. M. & ROHWER, F. 2002. Marine phage genomics. *Comparative Biochemistry and Physiology, Part B,* 133 (4)**,** 463-476.
PENADÉS, J. R., CHEN, J., QUILES-PUCHALT, N., CARPENA, N. & NOVICK, R. P. 2015. Bacteriophage-mediated spread of bacterial virulence genes. *Current Opinion in Microbiology,* 23**,** 171-178.
PHUMKHACHORN, P. & RATTANACHAIKUNSOPON, P. 2010. Isolation and partial characterization of a bacteriophage infecting the shrimp pathogen *Vibrio harveyi*. *African Journal of Microbiology Research,* 4**,** 1794-1800.
PIEDADE, G. J., WESDORP, E. M., MONTENEGRO-BORBOLLA, E., MAAT, D. S., BRUSSAARD, C. P. D., FRESHWATER & MARINE, E. 2018. Influence of irradiance and temperature on the virus MpoV-45T infecting the Arctic picophytoplankter *Micromonas polaris*. *Viruses,* 10**,** urn:issn:1999-4915.
PIETILA, M. K., DEMINA, T. A., ATANASOVA, N. S., OKSANEN, H. M. & BAMFORD, D. H. 2014. Archaeal viruses and bacteriophages: comparisons and contrasts. *Trends in Microbiology,* 22**,** 334.
PORTER, K., KUKKARO, P., BAMFORD, J. K. H., BATH, C., KIVELÄ, H. M., DYALL-SMITH, M. L. & BAMFORD, D. H. 2005. SH1: A novel, spherical halovirus isolated from an Australian hypersaline lake. *Virology,* 335**,** 22.
PORTER, K., RUSS, B. E. & DYALL-SMITH, M. L. 2007. Virus-host interactions in salt lakes. *Current Opinion in Microbiology,* 10**,** 418-424.
PORTER, K., TANG, S.-L., CHEN, C.-P., CHIANG, P.-W., HONG, M.-J. & DYALL-SMITH, M. 2013. PH1: An Archaeovirus of *Haloarcula hispanica* Related to SH1 and HHIV-2. *Archaea,* 2013**,** 17.
PRANGISHVILI, D. 2013. The Wonderful World of Archaeal Viruses. *Annual Review of Microbiology,* 67**,** 565-585.
PRANGISHVILI, D., ARNOLD, H. P., G?TZ, D., ZIESE, U., HOLZ, I., KRISTJANSSON, J. K. & ZILLIG, W. 1999. A novel virus family, the *Rudiviridae:* Structure, virus-host interactions and genome variability of the *Sulfolobus* viruses SIRV1 and SIRV2. *Genetics,* 152**,** 1387.
PRANGISHVILI, D. & GARRETT, R. A. 2005. Viruses of hyperthermophilic Crenarchaea. *Trends in Microbiology,* 13**,** 535-542.
PRANGISHVILI, D., PATRICK, F. & ROGER, A. G. 2006. Viruses of the Archaea: a unifying view. *Nature Reviews Microbiology,* 4**,** 837.
PRINGSULAKA, O., PATARASINPAIBOON, N., SUWANNASAI, N., ATTHAKOR, W. & RANGSIRUJI, A. 2011. Isolation and characterisation of a novel Podoviridae-phage infecting *Weissella cibaria* N 22 from Nham, a Thai fermented pork sausage. *Food Microbiology,* 28**,** 518-525.
PROCTOR, L., OKUBO, A. & FUHRMAN, J. 1993. Calibrating estimates of phage-induced mortality in marine bacteria: Ultrastructural studies of marine bacteriophage

development from one-step growth experiments. *An International Journal,* 25**,** 161-182.

PROCTOR, L. M. & FUHRMAN, J. A. 1990. Viral mortality of marine bacteria and cyanobacteria. *Nature,* 343**,** 60.

QUISPE, C. F., ESMAEL, A., SONDERMAN, O., MCQUINN, M., AGARKOVA, I., BATTAH, M., DUNCAN, G. A., DUNIGAN, D. D., SMITH, T. P. L., DE CASTRO, C., SPECIALE, I., MA, F. & VAN ETTEN, J. L. 2017. Characterization of a new chlorovirus type with permissive and non-permissive features on phylogenetically related algal strains. *Virology,* 500**,** 103-113.

RACHEL, R., BETTSTETTER, M., HEDLUND, B. P., HÄRING, M., KESSLER, A., STETTER, K. O. & PRANGISHVILI, D. 2002. Remarkable morphological diversity of viruses and virus-like particles in hot terrestrial environments. *Archives of Virology,* 147**,** 2419-2429.

RECORD, N., TALMY, D. & VÅGE, S. 2016. Quantifying Tradeoffs for Marine Viruses. *Frontiers in Marine Science,* 3.

ROBBINS, P. D. & GHIVIZZANI, S. C. 1998. Viral Vectors for Gene Therapy. *Pharmacology & Therapeutics,* 80**,** 35-47.

SAEKI, Y., TUDA, M. & CROWLEY, P. H. 2014. Allocation tradeoffs and life histories: a conceptual and graphical framework. *Oikos,* 123**,** 786-793.

SAFFERMAN, R. S., DIENER, T. O., DESJARDINS, P. R. & MORRIS, M. E. 1972. Isolation and characterization of AS-1, a phycovirus infecting the blue-green algae, *Anacystis nidulans* and *Synechococcus cedrorum*. *Virology,* 47**,** 105-113.

SANDAA, R.-A., HELDAL, M., CASTBERG, T., THYRHAUG, R. & BRATBAK, G. 2001. Isolation and Characterization of Two Viruses with Large Genome Size Infecting *Chrysochromulina ericina* (Prymnesiophyceae) and *Pyramimonas orientalis* (Prasinophyceae). *Virology,* 290**,** 272-280.

SANTOS, S. B., FERNANDES, E., CARVALHO, C. M., SILLANKORVA, S., KRYLOV, V. N., PLETENEVA, E. A., SHABUROVA, O. V., NICOLAU, A., FERREIRA, E. C. & AZEREDO, J. 2010. Selection and Characterization of a Multivalent *Salmonella* Phage and Its Production in a Nonpathogenic *Escherichia coli* Strain. *Applied and Environmental Microbiology,* 76**,** 7338.

SASIKALA, D. & SRINIVASAN, P. 2016. Characterization of potential lytic bacteriophage against *Vibrio alginolyticus* and its therapeutic implications on biofilm dispersal. *Microbial Pathogenesis,* 101**,** 24-35.

SHAFIA, F. & THOMPSON, T. L. 1964. Isolation and preliminary characterization of bacteriophage φμ-4. *Journal of Bacteriology,* 87**,** 999-1002.

SHERMAN, L. A., CONNELLY, M. & SHERMAN, D. M. 1976. Infection of *Synechococcus cedrorum* by the cyanophage AS-1M : I. Ultrastructure of infection and phage assembly. *Virology,* 71**,** 1-16.

SHIRAI, Y., TOMARU, Y., TAKAO, Y., SUZUKI, H., NAGUMO, T. & NAGASAKI, K. 2008. Isolation and Characterization of a Single-Stranded RNA Virus Infecting the Marine Planktonic Diatom *Chaetoceros tenuissimus* Meunier. *Applied and Environmental Microbiology,* 74**,** 4022.

SHORT, S. M. 2012. The ecology of viruses that infect eukaryotic algae. *Environmental Microbiology,* 14**,** 2253-2271.

SINGH, P. K. 1974. Isolation and characterization of a new virus infecting the blue-green alga *Plectonema boryanum*. *Virology,* 58**,** 586-588.

SINHA, S., GREWAL, R. K. & ROY, S. 2018. Chapter Three - Modeling Bacteria–Phage Interactions and Its Implications for Phage Therapy. *In:* SARIASLANI, S. & GADD, G. M. (eds.) *Advances in Applied Microbiology.* Academic Press.

SPENCER, R. 1955. A Marine Bacteriophage. *Nature,* 175**,** 690.
STAHL, D. A., BUCKLEY, D. H., BENDER, K. S., BROCK, T. D., MARTINKO, J. M. & MADIGAN, M. T. 2015. *Brock biology of microorganisms,* Harlow, Pearson.
STENHOLM, A. R., DALSGAARD, I. & MIDDELBOE, M. 2008. Isolation and Characterization of Bacteriophages Infecting the Fish Pathogen *Flavobacterium psychrophilum*. *Applied and Environmental Microbiology,* 74**,** 4070.
ŠULČIUS, S., ŠIMOLIŪNAS, E., STANIULIS, J., KOREIVIENĖ, J., BALTRUŠIS, P., MEŠKYS, R., PAŠKAUSKAS, R. & LAANBROE, R. 2015. Characterization of a lytic cyanophage that infects the bloom-forming cyanobacterium *Aphanizomenon flos-aquae*. *FEMS Microbiology Ecology,* 91**,** 1-7.
SULLIVAN, M. B., JOHN, B. W. & SALLIE, W. C. 2003. Cyanophages infecting the oceanic cyanobacterium *Prochlorococcus*. *Nature,* 424**,** 1047.
SUTTLE, C. A. 1994. The significance of viruses to mortality in aquatic microbial communities. *Microbial Ecology,* 28**,** 237-243.
SUTTLE, C. A. 2005. Viruses in the sea. *Nature,* 437**,** 356.
SUTTLE, C. A. 2007. Marine viruses major players in the global ecosystem. *Nature Reviews Microbiology,* 5**,** 801.
SUTTLE, C. A. & CHAN, A. M. 1993. Marine cyanophages infecting oceanic and coastal strains of *Synechococcus*: abundance, morphology, cross-infectivity and growth characteristics. *Marine Ecology Progress Series,* 92**,** 99-109.
SUTTLE, C. A. & CHAN, A. M. 1995. Viruses infecting the marine Prymnesiophyte *Chrysochromulina* spp.: isolation, preliminary characterization and natural abundance. *Marine Ecology Progress Series,* 118**,** 275-282.
TAI, V., LAWRENCE, J. E., LANG, A. S., CHAN, A. M., CULLEY, A. I. & SUTTLE, C. A. 2003. Characterization of HaRNAV, a single-stranded RNA virus causing lysis of *Heterosigma akashiwo* (raphidophyceae) 1. *Journal of Phycology,* 39**,** 343-352.
TAKAO, Y., NAGASAKI, K., MISE, K., OKUNO, T. & HONDA, D. 2005. Isolation and Characterization of a Novel Single-Stranded RNA Virus Infectious to a Marine Fungoid Protist, *Schizochytrium* sp. (Thraustochytriaceae, Labyrinthulea). *Applied and Environmental Microbiology,* 71**,** 4516.
TARUTANI, K., NAGASAKI, K., ITAKURA, S., YAMAGUCHI, M. & NAGASAKI, K. 2001. Isolation of a virus infecting the novel shellfish-killing dinoflagellate *Heterocapsa circularisquama*. *Aquatic Microbial Ecology,* 23**,** 103-111.
TAYLOR, B., WEITZ, J., BRUSSAARD, C. & FISCHER, M. 2018. Quantitative Infection Dynamics of *Cafeteria Roenbergensis* Virus. *Viruses,* 10.
THINGSTAD, T. & LIGNELL, R. 1997. Theoretical models for the control of bacterial growth rate, abundance, diversity and carbon demand. *Aquatic Microbial Ecology,* 13**,** 19-27.
THINGSTAD, T. F. 2000. Elements of a theory for the mechanisms controlling abundance, diversity, and biogeochemical role of lytic bacterial viruses in aquatic systems. *Limnology and Oceanography,* 45**,** 1320-1328.
THINGSTAD, T. F., HELDAL, M., BRATBAK, G. & DUNDAS, I. 1993. Are viruses important partners in pelagic fend webs? *Trends in Ecology & Evolution,* 8**,** 209-213.
THINGSTAD, T. F., VÅGE, S., STORESUND, J. E., SANDAA, R.-A. & GISKE, J. 2014. A theoretical analysis of how strain-specific viruses can control microbial species diversity. *Proceedings of the National Academy of Sciences,* 111**,** 7813-7818.
TOMARU, Y., KATANOZAKA, N., NISHIDA, K., SHIRAI, Y., TARUTANI, K., YAMAGUCHI, M. & NAGASAKI, K. 2004. Isolation and characterization of two distinct types of HcRNAV, a single-stranded RNA virus infecting the bivalve-killing microalga *Heterocapsa circularisquama*. *Aquat. Microb. Ecol.,* 34**,** 207-218.

TOMARU, Y., KENSUKE, T., KEI, K., NAOTSUGU, H., MIKIHIDE, Y. & KEIZO, N. 2012. First evidence for the existence of pennate diatom viruses. *The ISME Journal,* 6**,** 1445.

TOMARU, Y., SHIRAI, Y., SUZUKI, H., NAGUMO, T., NAGASAKI, K. & TOMARU, Y. 2008. Isolation and characterization of a new single-stranded DNA virus infecting the cosmopolitan marine diatom *Chaetoceros debilis*. *Aquatic Microbial Ecology,* 50**,** 103-112.

TOMARU, Y., SHIRAI, Y., TOYODA, K., NAGASAKI, K. & TOMARU, Y. 2011a. Isolation and characterisation of a single-stranded DNA virus infecting the marine planktonic diatom *Chaetoceros tenuissimus*. *Aquatic Microbial Ecology,* 64**,** 175-184.

TOMARU, Y., TAKAO, Y., SUZUKI, H., NAGUMO, T., KOIKE, K. & NAGASAKI, K. 2011b. Isolation and Characterization of a Single-Stranded DNA Virus Infecting *Chaetoceros lorenzianus* Grunow.

TOMARU, Y., TAKAO, Y., SUZUKI, H., NAGUMO, T. & NAGASAKI, K. 2009a. Isolation and Characterization of a Single-Stranded RNA Virus Infecting the Bloom-Forming Diatom Chaetoceros socialis. *Applied and Environmental Microbiology,* 75**,** 2375-2381.

TOMARU, Y., TAKAO, Y., SUZUKI, H., NAGUMO, T. & NAGASAKI, K. 2009b. Isolation and Characterization of a Single-Stranded RNA Virus Infecting the Bloom-Forming Diatom *Chaetoceros socialis*. *Applied and Environmental Microbiology,* 75**,** 2375.

TOMARU, Y., TOYODA, K., SUZUKI, H., NAGUMO, T., KIMURA, K. & TAKAO, Y. 2013. New single-stranded DNA virus with a unique genomic structure that infects marine diatom *Chaetoceros setoensis*. *Scientific Reports,* 3.

TORSVIK, T. & DUNDAS, I. D. 1980. Persisting Phage Infection in *Halobacterium salinarium* str. 1. *Journal of General Virology,* 47**,** 29-36.

TOUCHON, M., MOURA DE SOUSA, J. A. & ROCHA, E. P. C. 2017. Embracing the enemy: the diversification of microbial gene repertoires by phage-mediated horizontal gene transfer. *Current Opinion in Microbiology,* 38**,** 66-73.

TOYODA, K., KIMURA, K., HATA, N., NAKAYAMA, N., NAGASAKI, K. & TOMARU, Y. 2012. Isolation and characterization of a single-stranded DNA virus infecting the marine planktonic diatom *Chaetoceros* sp. (strain TG07-C28). *Plankton and Benthos Research,* 7**,** 20-28.

TOYODA, K., KIMURA, K., OSADA, K., WILLIAMS, D. M., ADACHI, T., YAMADA, K. & TOMARU, Y. 2019. Novel marine diatom ssRNA virus NitRevRNAV infecting *Nitzschia reversa*. *Plant Ecology and Evolution,* 152**,** 178-187.

TRAUTWETTER, A., BLANCO, C. & BONNASSIE, S. 1987. Characterization of the Corynebacteriophage CG33. *Microbiology,* 133**,** 2945-2952.

UCHIYAMA, J., RASHEL, M., MAEDA, Y., TAKEMURA, I., SUGIHARA, S., AKECHI, K., MURAOKA, A., WAKIGUCHI, H. & MATSUZAKI, S. 2008. Isolation and characterization of a novel *Enterococcus faecalis* bacteriophage φEF24C as a therapeutic candidate. *FEMS Microbiology Letters,* 278**,** 200-206.

VAN ETTEN, J. L., BURBANK, D. E., JOSHI, J. & MEINTS, R. H. 1984. DNA synthesis in a *Chlorella*-like alga following infection with the virus PBCV-1. *Virology,* 134**,** 443-449.

VAN ETTEN, J. L., BURBANK, D. E., XIA, Y. & MEINTS, R. H. 1983. Growth cycle of a virus, PBCV-1, that infects *Chlorella*-like algae. *Virology,* 126**,** 117-125.

VAN ETTEN, J. L., GRAVES, M. V., M**?**LLER, D. G., BOLAND, W. & DELAROQUE, N. 2002. Phycodnaviridae ? large DNA algal viruses. *Archives of Virology,* 147**,** 1479-1516.

VAN ETTEN, J. L., LANE, L. C. & DUNIGAN, D. D. 2010. DNA Viruses: The Really Big Ones (Giruses). *Annu. Rev. Microbiol.,* 64**,** 83-99.

VAN ETTEN, J. L., LANE, L. C. & MEINTS, R. H. 1991. Viruses and viruslike particles of eukaryotic algae. *Microbiol Rev,* 55**,** 586-620.
VIDAVER, A. K., KOSKI, R. K. & VAN ETTEN, J. L. 1973. Bacteriophage phi6: a Lipid-Containing Virus of *Pseudomonas phaseolicola*. *Journal of virology,* 11**,** 799.
WANG, D.-B., SUN, M.-Q., SHAO, H.-B., LI, Y., MENG, X., LIU, Z.-Y. & WANG, M. 2015. Characterization and Genome Sequencing of a Novel Bacteriophage PH101 Infecting *Pseudoalteromonas marina* BH101 from the Yellow Sea of China. *Current Microbiology,* 71**,** 594-600.
WANG, I.-N., DYKHUIZEN, D. & SLOBODKIN, L. 1996. The evolution of phage lysis timing. *Evolutionary Ecology,* 10**,** 545-558.
WANG, I. 2006. Lysis timing and bacteriophage fitness. *Genetics,* 172**,** 17-26.
WANG, K., CHEN, F. & WANG, K. 2004. Genetic diversity and population dynamics of cyanophage communities in the Chesapeake Bay. *Aquatic Microbial Ecology,* 34**,** 105-116.
WANG, S., KONG, J., GAO, C., GUO, T. & LIU, X. 2010. Isolation and characterization of a novel virulent phage (phiLdb) of *Lactobacillus delbrueckii*. *International Journal of Food Microbiology,* 137**,** 22-27.
WATERS, R. E. & CHAN, A. T. 1982. Micromonas pusilla Virus: the Virus Growth Cycle and Associated Physiological Events Within the Host Cells; Host Range Mutation. *Journal of General Virology,* 63**,** 199-206.
WEI, W., WANG, N., CAI, L., ZHANG, C., JIAO, N. & ZHANG, R. 2019. Impacts of Freshwater and Seawater Mixing on the Production and Decay of Virioplankton in a Subtropical Estuary. *Microbial ecology,* 78.
WEIMBAUER, M. G. & PEDUZZI, P. 1994. Frequency, size and distribution of bacteriophages in different marine bacterial morphotypes. *Marine Ecology Progress Series,* 108**,** 11-20.
WEINBAUER, M. & HOEFLE, M. 1998. Size-specific mortality of lake bacterioplankton by natural virus communities. *Aquatic Microbial Ecology,* 15**,** 103-113.
WEINBAUER, M. G. 2004. Ecology of prokaryotic viruses. *FEMS Microbiology Reviews,* 28**,** 127-181.
WEINBAUER, M. G. & RASSOULZADEGAN, F. 2004. Are viruses driving microbial diversification and diversity? *Environmental microbiology,* 6**,** 1.
WEISS, B. D., CAPAGE, M. A., KESSEL, M. & BENSON, S. A. 1994. Isolation and characterization of a generalized transducing phage for *Xanthomonas campestris* pv. *campestris*. *The Journal of Bacteriology,* 176**,** 3354.
WEITZ, J. S., POISOT, T., MEYER, J. R., FLORES, C. O., VALVERDE, S., SULLIVAN, M. B. & HOCHBERG, M. E. 2013. Phage–bacteria infection networks. *Trends in Microbiology,* 21**,** 82-91.
WEITZ, J. S., STOCK, C. A., WILHELM, S. W., BOUROUIBA, L., COLEMAN, M. L., BUCHAN, A., FOLLOWS, M. J., FUHRMAN, J. A., JOVER, L. F., LENNON, J. T., MIDDELBOE, M., SONDEREGGER, D. L., SUTTLE, C. A., TAYLOR, B. P., FREDE THINGSTAD, T., WILSON, W. H. & ERIC WOMMACK, K. 2015. A multitrophic model to quantify the effects of marine viruses on microbial food webs and ecosystem processes. *The ISME Journal,* 9**,** 1352-1364.
WEITZ, J. S. & WILHELM, S. W. 2012. Ocean viruses and their effects on microbial communities and biogeochemical cycles. *F1000 Biology Reports,* 4**,** 17.
WELLS, L. & DEMING, J. 2006. Characterization of a cold-active bacteriophage on two psychrophilic marine hosts. *Aquatic Microbial Ecology,* 45**,** 15-29.

WIKNER, J., VALLINO, J. J., STEWARD, G. F., SMITH, D. C. & AZAM, F. 1993. Nucleic acids from the host bacterium as a major source of nucleotides for three marine bacteriophages. *FEMS Microbiology Ecology,* 12**,** 237-248.

WILHELM, S. & SUTTLE, C. 1999. Viruses and nutrient cycles in the sea. *Bioscience,* 49**,** 781-788.

WILLIAMSON, K., WOMMACK, K. E. & RADOSEVICH, M. 2007. Ecological aspects of viruses in soils. ProQuest Dissertations Publishing.

WILSON, W. H., ALLEN, M. J. & ETTEN, J. L. 2009. *The Phycodnaviridae: The Story of How Tiny Giants Rule the World,* Berlin, Heidelberg, Berlin, Heidelberg: Springer Berlin Heidelberg.

WILSON, W. H., CARR, N. G. & MANN, N. H. 1996. The effect of phosphate status on the kinetics of cyanophage infection in the oceanic cyanobacterium *Synechococcus* sp. wh7803 1. *Journal of Phycology,* 32**,** 506-516.

WINSTON, V. & THOMPSON, T. L. 1979. Isolation and characterization of a bacteriophage specific for *Sphaerotilus natans* which contain an unusual base in its deoxyribonucleic acid. *Applied and Environmental Microbiology,* 37**,** 1025.

WOMMACK, K. E. & COLWELL, R. R. 2000. Virioplankton: Viruses in Aquatic Ecosystems. *Microbiology and Molecular Biology Reviews,* 64**,** 69.

XIA, H., LI, T., DENG, F. & HU, Z. 2013. Freshwater cyanophages. *Virologica Sinica,* 28**,** 253-259.

XUE, C. & GOLDENFELD, N. 2017. Coevolution Maintains Diversity in the Stochastic "Kill the Winner" Model. *Physical review letters,* 119**,** 268101.

YANG, H., LIANG, L., LIN, S. & JIA, S. 2010. Isolation and Characterization of a Virulent Bacteriophage AB1 of *Acinetobacter baumannii*. *BMC Microbiology,* 10**,** 131.

YANG, Y., CAI, L., MA, R., XU, Y., TONG, Y., HUANG, Y., JIAO, N. & ZHANG, R. 2017. A Novel Roseosiphophage Isolated from the Oligotrophic South China Sea. *Viruses,* 9.

YANG, Y., LU, S., SHEN, W., ZHAO, X., SHEN, M., TAN, Y., LI, G., LI, M., WANG, J., HU, F. & LE, S. 2016. Characterization of the first double-stranded RNA bacteriophage infecting *Pseudomonas aeruginosa*. *Scientific Reports,* 6**,** 38795.

YANYAN, M., ERNA, L., ZHIZHEN, Q., HUAN, L., XIAO, W., WEISHI, L., RUIXIANG, Z., AIMIN, J., HUIYING, Y., ZHE, Y., JING, Y. & XIANGNA, Z. 2016. Isolation and molecular characterisation of *Achromobacter* phage phiAxp-3, an N4-like bacteriophage. *Scientific Reports,* 6.

YAU, S. & SETH-PASRICHA, M. 2019. Viruses of Polar Aquatic Environments. *Viruses,* 11.

YOON, S.-S., BARRANGOU-POUEYS, R., BREIDT, F. & FLEMING, H. P. 2007. Detection and characterization of a lytic *Pediococcus* bacteriophage from the fermenting cucumber brine. *Journal of microbiology and biotechnology,* 17**,** 262.

YOON, S. S., BARRANGOU-POUEYS, R., BREIDT, F., JR., KLAENHAMMER, T. R. & FLEMING, H. P. 2002. Isolation and Characterization of Bacteriophages from Fermenting Sauerkraut. *Applied and Environmental Microbiology,* 68**,** 973.

YORDPRATUM, U., TATTAWASART, U., WONGRATANACHEEWIN, S. & SERMSWAN, R. W. 2011. Novel lytic bacteriophages from soil that lyse *Burkholderia pseudomallei*. *FEMS Microbiology Letters,* 314**,** 81-88.

YOSHIDA, T., TAKASHIMA, Y., TOMARU, Y., SHIRAI, Y., TAKAO, Y., HIROISHI, S. & NAGASAKI, K. 2006. Isolation and Characterization of a Cyanophage Infecting the Toxic Cyanobacterium *Microcystis aeruginosa*. *Applied and Environmental Microbiology,* 72**,** 1239.

YOU, L., SUTHERS, P. F. & YIN, J. 2002. Effects of *Escherichia coli* Physiology on Growth of Phage T7 *In Vivo* and *In Silico*. *Journal of Bacteriology,* 184**,** 1888.

YOULE, M., HAYNES, M. & ROHWER, F. 2012. Scratching the Surface of Biology's Dark Matter. *In:* WITZANY, G. (ed.) *Viruses: Essential Agents of Life.* Dordrecht: Springer Netherlands.

YOUNG, R. 1992. Bacteriophage lysis - mechanism and regulation. *Microbiol. Rev.*, 56**,** 430-481.

YOUNG, R., WANG, I.-N. & ROOF, W. D. 2000. Phages will out: strategies of host cell lysis. *Trends in Microbiology, ,* 8 (3)**,** 120-128.

YU, Y.-P., GONG, T., JOST, G. N., LIU, W.-H., YE, D.-Z. & LUO, Z.-H. 2013a. Isolation and characterization of five lytic bacteriophages infecting a *Vibrio* strain closely related to *Vibrio owensii*. *FEMS Microbiology Letters,* 348**,** 112-119.

YU, Y. P., GONG, T., JOST, G., LIU, W. H., YE, D. Z. & LUO, Z. H. 2013b. Isolation and characterization of five lytic bacteriophages infecting a V ibrio strain closely related to V ibrio owensii. *FEMS Microbiology Letters,* 348**,** 112-119.

YUAN, Y., QU, K., TAN, D., LI, X., WANG, L., CONG, C., XIU, Z. & XU, Y. 2019. Isolation and characterization of a bacteriophage and its potential to disrupt multi-drug resistant *Pseudomonas aeruginosa* biofilms.

ZABLOCKI, O., VAN ZYL, L., ADRIAENSSENS, E. M., RUBAGOTTI, E., TUFFIN, M., CARY, S. C. & COWAN, D. 2014. High-level diversity of tailed phages, eukaryote-associated viruses, and virophage-like elements in the metaviromes of antarctic soils. *Applied and environmental microbiology,* 80**,** 6888.

ZABLOCKI, O., VAN ZYL, L. & TRINDADE, M. 2018. Biogeography and taxonomic overview of terrestrial hot spring thermophilic phages. *Microbial Life Under Extreme Conditions,* 22**,** 827-837.

ZACHARY, A. 1976. Physiology and ecology of bacteriophages of the marine bacterium *Beneckea natriegens:* salinity. *Applied and environmental microbiology,* 31**,** 415.

ZHANG, S., FAN, C., XIA, Y., LI, M., WANG, Y., CUI, X. & XIAO, W. 2019. Characterization of a novel bacteriophage specific to *Exiguobacterium indicum* isolated from a plateau eutrophic lake. *Journal of basic microbiology,* 59**,** 206.

ZHANG, Y. & JIAO, N. 2009. Roseophage RDJLΦ1, Infecting the Aerobic Anoxygenic Phototrophic Bacterium *Roseobacter denitrificans* OCh114. *Applied and environmental microbiology AEM,* 75 (6)**,** 1745-1749.

ZHAO, Y., WANG, K., JIAO, N. & CHEN, F. 2009. Genome sequences of two novel phages infecting marine roseobacters. *Environmental Microbiology,* 11**,** 2055-2064.

ZHENG, Q., CHEN, Q., XU, Y., SUTTLE, C. A. & JIAO, N. 2018. A Virus Infecting Marine Photoheterotrophic Alphaproteobacteria (*Citromicrobium* spp.) Defines a New Lineage of ssDNA Viruses. *Frontiers in Microbiology,* 9.

ZHONG, K. X., SUTTLE, C. A., BAUDOUX, A.-C., DERELLE, E., COLOMBET, J., CHO, A., CALETA, J., SIX, C. & JACQUET, S. 2018. A New Freshwater Cyanosiphovirus Harboring Integrase.(Report). *Frontiers in Microbiology,* 9.

ZIERDT, C. H. 1974. Properties of *Corynebacterium acnes* Bacteriophage and Description of an Interference Phenomenon. *The Journal of Virology,* 14**,** 1268.

ZIMMERMAN, A. E., BACHY, C., MA, X., ROUX, S., JANG, H. B., SULLIVAN, M. B., WALDBAUER, J. R. & WORDEN, A. Z. 2019. Closely related viruses of the marine picoeukaryotic alga *Ostreococcus lucimarinus* exhibit different ecological strategies. *Environmental Microbiology,* 21**,** 2148-2170.