Chapter 2

Factors affecting virus dynamics and microbial host-virus interactions in marine environments

Kristina D. A. Mojica[1*], and Corina P. D. Brussaard[1,2]

[1]Department of Biological Oceanography, Royal Netherlands Institute for Sea Research (NIOZ), P.O. Box 59, 1790 AB Den Burg, Texel, The Netherlands
[2]Department of Aquatic Microbiology, Institute for Biodiversity and Ecosystem Dynamics (IBED), University of Amsterdam, P.O. Box 94248, 1090 GE Amsterdam, The Netherlands

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Abstract

Marine microorganisms constitute the largest percentage of living biomass and serve as the major driving force behind nutrient and energy cycles. While viruses only comprise a small percentage of this biomass (i.e., 5%), they dominate in numerical abundance and genetic diversity. Through host infection and mortality, viruses affect microbial population dynamics, community composition, genetic evolution and biogeochemical cycling. However, the field of marine viral ecology is currently limited by a lack of data regarding how different environmental factors regulate virus dynamics and host-virus interactions. The goal of the present mini-review is to contribute to the evolution of marine viral ecology, through the assimilation of available data regarding the manner and degree to which environmental factors affect viral decay and infectivity as well as influence latent period and production. Considering the ecological importance of viruses in the marine ecosystem and the increasing pressure from anthropogenic activity and global climate change on marine systems, a synthesis of existing information provides a timely framework for future research initiatives in viral ecology.

Introduction

Since the discovery of high viral abundance in marine environments, the ecological importance of viruses to aquatic systems has become increasingly evident. Most of these viruses infect the numerically dominant microorganisms, which constitute over 90% of the ocean's biomass and serve as the major driving force behind nutrient and energy cycles (Cotner and Biddanda 2002; Suttle 2007; Sorensen 2009). Aside from driving host population dynamics and horizontal gene transfer, viruses influence microbial community structure and function through the conversion of biomass to dissolved and particulate organic matter via host cell lysis (Suttle 2007). Viral activity thus effectively regulates biodiversity and food web efficiency. The extent and efficiency to which viruses are able to drive microbial processes can be regulated by both abiotic and biotic aspects of the environment in which they occur.

At any spatio-temporal point in the ocean, viral abundance reflects the balance between rates of removal and production through host lysis. When viral progeny are released from their hosts, they are present in the environment as free virus particles and are directly exposed to environmental factors which may reduce infectivity, degrade or remove virus particles, and adversely affect adsorption to host, thereby reducing the chance of a successful host encounter and infection (Fig. 1A). Moreover, as obligate parasites, viruses are reliant upon their host to provide not only the cellular machinery but also the necessary energy and resources required for viral replication and assembly. Consequently, the factors regulating the physiology of the host, as well as its production and removal are also important in governing virus dynamics (Fig. 1B).

In the face of continued anthropogenic activity (marine utilization, eutrophication, urbanization, tourism, and global climate change), it will become increasingly important to unravel how environmental factors regulate virus dynamics and virus-host interactions and thus influence the role that viruses have in the marine environment. Reviews on aquatic viruses have thus far only limitedly conversed the influence of 'the environment' in viral ecology. It is therefore an opportune time to synthesize the current available knowledge on factors affecting host-virus interactions in the marine pelagic environment and identify any remaining gaps. The present mini-review focuses on microbe-viruses, both in culture and in the field (with emphasis on the pelagic).

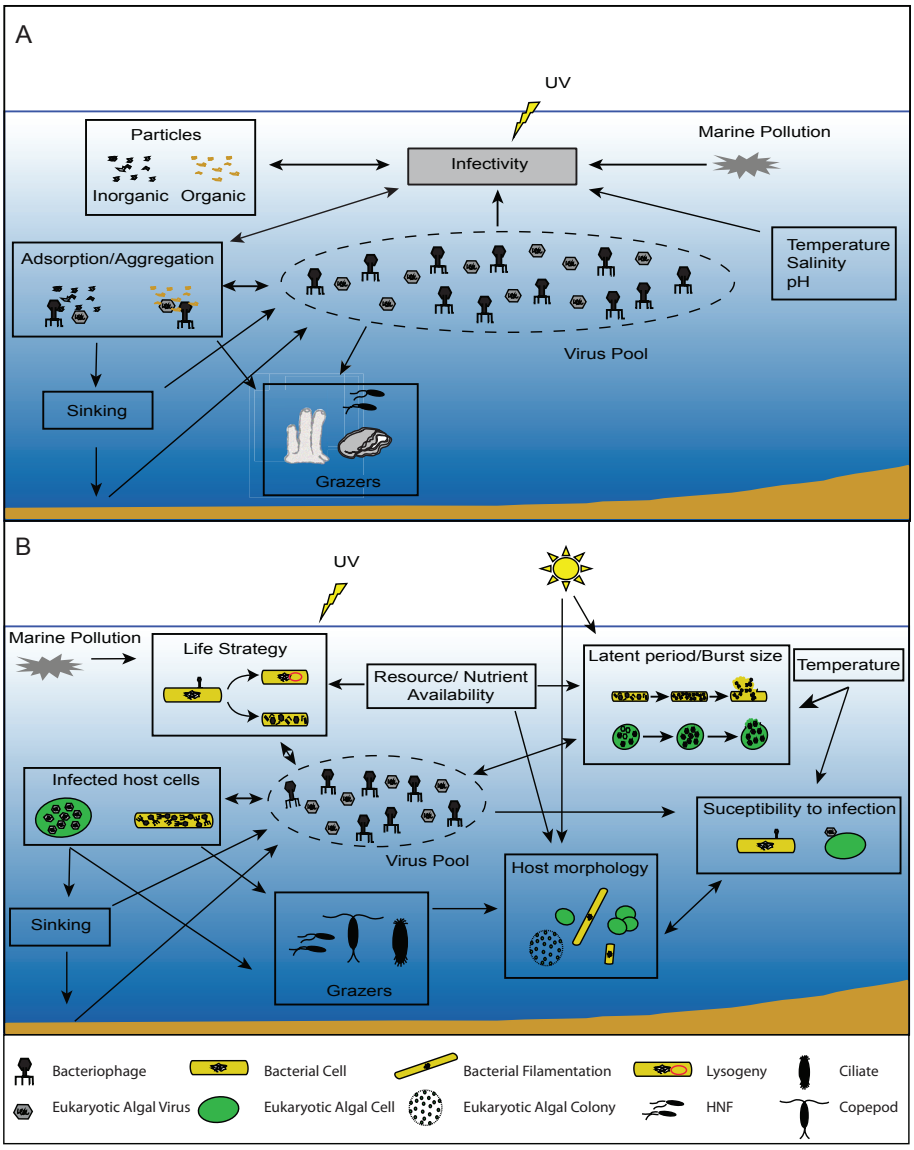

Figure 1. Schematic overview of environmental factors and processes in the marine environment that have been found thus far to affect virus dynamics and virus host interactions. (a). A synopsis of environmental factors that can lead to the removal or inactivation of virus particles reducing the chance of a successful host encounter and infection. (b) Overview of aspects that may influence the viral pool by altering host dynamics and deceasing susceptibility to infection or by modifying characteristics of viral proliferation. Heterotrophic nanoflagellates are abbreviated as NHF.

Temperature

Due to the dependency of viruses on a host for replication, the actual distribution of viruses can be expected to be constrained either by their own sensitivity to an environmental factor or by that of their hosts. Viruses may be more resistant to thermal stress than their host systems, indicating that the temperature distribution of the virus-host system is set by the host. Based on the available data from culture studies, the inactivation temperatures of most marine viruses fall outside of those at which host growth can be maintained (Table 1). Interestingly, the inactivation temperature of the psychrophilic filamentous phage SW1 infecting *Shewanella piezotolerans* showed the largest divergence from the host's optimum growth temperature. To our knowledge, this is the only marine filamentous phage tested thus far and it would be interesting to uncover if this is a general feature of this virus morphotype (Table S1). Apart from the filamentous phage, phages BVW1 and GVE1 of the hydrothermal field bacteria *Bacillus* and *Geobacillus*, respectively, were inactivated at temperatures comparable to that of their thermophilic host's optimum. The fact that non-hydrothermal field viruses have lower absolute inactivation temperatures suggests an ongoing adaption to the lower optimum growth temperature of their host's. One may then speculate that marine viruses have retained (from evolutionary origin) the genetic blueprint that is neutral in the current environment and may be useful for adaptation to (future) environmental increases in temperature.

Even though marine viruses are typically more stable to temperature than their host, it does not necessarily mean that virus-host interactions within the host's growth temperature range will lead to successful viral proliferation. For example, *Pseudomonas putrefaciens* (P19X) can grow well up to 27°C but phage-27 was unable to form plaques above 20°C (Delisle and Levin 1972a) Furthermore, the lower temperature stability of a marine sediment phage (0-23°C for phage versus 0-33°C for the host *Aeromonas* sp.) was due to an apparent inability of irreversible adsorption to host cells, as phage titers only demonstrated limited reduction when exposed to 30°C for 24h in the presence of their host bacterium (Wiebe and Liston 1968). However, whether the inactivation was a consequence of thermal alterations to phage structure or host receptors remains unknown.

Table 1. Temperature (°C) range and optimum for host growth and the range tested and values for inactivation, successful host lysis and maximum plaque forming unit (PFU) of associated viruses. When optimum temperature of the host was not reported, the culture temperature of host employed was assumed to be optimum. Parenthesis indicate that range provided is not strain-specific but obtained from the literature.

Host	Range	Optimum	Virus	Genome Type	Tested	Inactivation	Host lysis	Max PFU	References
Phaeocystis globosa Pg-I	8 to 20	15	Group I PgV	dsDNA	20 to 75	35	15		(Baudoux & Brussaard, 2005), Brussaard unpubl data (host growth)
P. globosa Pg-I	8 to 20	15	Group II PgV	dsDNA	20 to 75	25	15		(Baudoux & Brussaard, 2005)
M. pusilla LAC38	4 to 22	15	MpRNAV-01B	dsRNA	20 to 95	40	4 to 15		(Brussaard et al., 2004), Brussaard unpubl data (host lysis)
M. pusilla LAC38	4 to 22	15	MpV-03T, 06T, 08-12T, 14T, R3-4, B4-5	dsDNA	4 to 45	40	4 to 15		(Martinez-Martinez & Brussaard)
M. pusilla CCMP1545	8 to 24	20	MpV-02T, 04-05T, 07T, 13T, R1-R2, SP1	dsDNA	4 to 45	40	4 to 15		(Martinez-Martinez & Brussaard)
Chaetoceros debilis Ch48	(9 to 30)*	15	CdebDNAV18	ssDNA	4 to 20	>20	15		(Tomaru et al., 2008), (Tomaru et al., 2011a)
C. lorenzianus IT-Dia51	(9 to 27)*	15	ClorDNAV	ssDNA	4 to 20	>20	15		(Tomaru et al., 2011b)
C. setoensis IT07-C11	(7 to 28)*	15	CsetDNAV	ssDNA	4 to 20	>20	15		(Tomaru et al., 2013)
C. socialis	(7 to 28)*	15	CsfrRNAV	ssRNA	4 to 20	>20	15		(Tomaru et al., 2009b)
C. tenuissimus 2-10	(9 to 30)*	15	CtenRNAV01	ssRNA	4 to 20	>20	15		(Shirai et al., 2008) (Tomaru et al., 2011a)
Heterosigma akashiwo H93616	(5 to 30)	20	HaV01	dsDNA	4 to 20	>20	15 to 30		(Tomaru et al., 2005), (Nagasaki & Yamaguchi, 1998), (Graneli & Turner, 2007) (host growth
H. akashiwo NM96	(5 to 30)	20	HaV01	dsDNA	4 to 20	>20	15 to 25		(Nagasaki & Yamaguchi, 1998)
H. akashiwo H93616	(5 to 30)	20	HaV08	dsDNA	4 to 20	>20	20 to 30		(Nagasaki & Yamaguchi, 1998)
H. akashiwo NM96	(5 to 30)	20	HaV08	dsDNA	4 to 20	>20	20 to 25		(Nagasaki & Yamaguchi, 1998)
H. akashiwo H93616	(5 to 30)	20	HaV53	dsDNA	4 to 20	>20	20		(Tomaru et al., 2005)
H. akashiwo H93616	(5 to 30)	20	HaRNAV	ssRNA	4 to 20	>20	20		Tomaru et al., 2005)
Heterocapsa circularisquama HU9433-P	(15 to 30)	20	HcV03	dsDNA	4 to 20	>20	20		(Tomaru et al., 2005), (Yamaguchi et al., 1997) (host growth)
H. circularisquama HU9433-P	(15 to 30)	20	HcV05	dsDNA	4 to 20	>20	20		(Tomaru et al., 2005)
H. circularisquama HU9433-P	(15 to 30)	20	HcV08	dsDNA	4 to 20	>20	20		(Tomaru et al., 2005)
H. circularisquama HU9433-P	(15 to 30)	20	HcV10	dsDNA	4 to 20	>20	20		(Tomaru et al., 2005)
H. circularisquama HU9433-P	(15 to 30)	20	HcRNAV34	ssRNA	4 to 20	>20	20		(Tomaru et al., 2005)

Table 1. Continued.

Host	Range	Optimum	Virus	Genome Type	Tested	Inactivation	Host lysis	Max PFU	References
H. circularisquama HCLG-1	(15 to 30)	20	HcRNAV109	ssRNA	4 to 20	>20	20		(Tomaru et al., 2005
Pseudomonas putrefaciens P19X	2 to 27	2	Phage 27	dsDNA	-5 to 26, 55	55	-5 to 13	-5 to 2	(Delisle & Levin, 1972a), (Delisle & Levin, 1972b)
P. putrefaciens P10	2 to 27	2	Phage 27	dsDNA			2 to 20	2	(Delisle & Levin, 1972a)
P. putrefaciens P13	2 to 27	20	Phage 23	dsDNA	2 to 26, 55	55	2 to 26	20 to 26	(Delisle & Levin, 1972a)
P. putrefaciens P2	2 to 27	2	Phage 25F	dsDNA	2 to 26, 55	55	2 to 26	=	(Delisle & Levin, 1972a)
Pseudoalteromonas marina KCTC 12242†	(2 to 25)	25	φRIO-1	dsDNA	20 to 50	40	10 to 25	20 to 25	(Hardies et al., 2013)
Vibrio sp. ATCC19648	6 to 30	18	unknown	dsDNA	6 to 30, 50	50	6 to 25	=	(Johnson, 1968)
Vibrio (Beneckea) natriegens ATTC 14048	(4 to 40)	27	nt-1	dsDNA	5 to 60	50	27		(Zachary, 1976), (Farmer III & Janda, 2005) (host growth)
V. natriegens ATTC 14048	(4 to 40)	27	nt-6	dsDNA	5 to 60	37	27		(Zachary, 1976)
V. fischeri MJ-1	(5 to 30)	15	rp-1	dsDNA	23 to >45	45	25		(Levisohn et al., 1987), (Waters & Lloyd, 1985) (host growth)
Pseudomonas sp.	25 to 37	25 to 28	06N-58P	ssRNA	5, 45 to 50	45	25		(Hidaka & Ichida, 1976)
Bacillus sp. w13	45 to 85	68	BVW1	dsDNA	60 to 80	70	> 60	60	(Liu et al., 2006)
Geobacillus sp. E26323	45 to 85	65	GVE1	dsDNA	60 to 80	70	> 60	60	(Liu et al., 2006)
Colwellia psychrerythraea 34H	-18 to 18	10 to 18	Phage 9A	dsDNA	-12 to 55	25	-6 to 4		(Wells & Deming, 2006b), (Wells & Deming, 2006a), (Bowman et al., 1998)
C. demingiae ACAM 459^{T}	-10 to 18	10 to 18	Phage 9A	dsDNA			-6 to 8		(Bowman et al., 1998), (Wells & Deming, 2006b)
21C (*C. psychrerythraea)*[b]	0 to 15	4	21c	dsDNA			0 to 5		(Borriss et al., 2003)
Aeromonas sp.	0 to 33	12	unknown	dsDNA	45 to 60	45	0 to 23	5 to 12	(Wiebe & Liston, 1968)
1A *(Shewanella frigidimarina* LMG 19867)[b]	0 to 21	4	1a	dsDNA			0 to 14		(Borriss et al., 2003)
S. piezotolerans WP2	0 to 28	15 to 20	SW1	ssDNA	4-25, 60, 70,	70	4 to 15	4	(Wang et al., 2004), (Wang et al., 2007)

* Values are the reported natural temperature range where strains are found
† highest identities based on 16S analysis
'=, equal efficiency

Temperature affects the structural conformation of proteins and the elasticity of biomolecules such as proteins and membrane lipids, therefore variability in the response of different viruses to modifications in temperature will most likely arise from molecular or structural differences that regulate the sensitivity of viral lipid membranes or capsid proteins to thermal deformation or thermal fracture (Selinger et al. 1991; Evilevitch et al. 2008). Group I PgVs infecting *Phaeocystis globosa* were inactivated above 35°C while infectivity of Group II PgVs could only be maintained below 25°C (Baudoux and Brussaard 2005). These viruses differ in their phylogenetic origin, genome size, and in the size and composition of capsid proteins, most likely underlying the observed variation (Table S1) (Baudoux and Brussaard 2005; Santini et al. 2013). In contrast, the larger dsDNA viruses infecting *Heterocapsa circularisquama* were more sensitive to losses to infectivity at the different temperatures tested compared to the smaller ssRNA virus infecting the same species (Tomaru et al. 2004; Nagasaki et al. 2005; Tomaru et al. 2005). Although very different virus types, it would be interesting to test if the smaller size of the putative major capsid protein of the HcV03 (591 nt) as compared to HcRNAV109 (678 nt) may explain the discrepancy in the expected viral stability (Table S1) (Hickey and Singer 2004; Tomaru et al. 2009a). Although the underlying mechanisms remain unknown, variation in temperature sensitivity provides a driving force for virus and host population dynamics, and can be expected to affect the outcome of adaptation to changing environments (Bolnick et al. 2011). It is important to note that in general unfiltered or 0.2 μm pore-size filtered water which is commonly used for investigating the stability of viruses may include components such as extracellular enzymes that can contribute to the inactivation of viruses in a temperature dependent manner.

Temperature can also regulate infection dynamics and can vary amongst viruses infecting the same host as demonstrated for viruses infecting *Heterosigma akashiwo*. The dsDNA virus HaV01 only infects *H. akashiwo* strain H93616 between 15-30°C, while the comparable virus strain HaV08 is infective between 20-30°C (Nagasaki and Yamaguchi 1998). In addition, phenotypic variability can also be dependent on the host stain being infected. *H. akashiwo* strain H93616 was infected by HaV01 and HaV08 up to 30°C, whereas strain NM96 (with same growth optimum temperature) was not sensitive to infection above 25°C (Nagasaki and Yamaguchi 1998). Similar results have also been found in a bacterium-phage system, wherein Phage 27 could successfully form plaques between 2-20°C on host *P. putrefaciens* P10, but was restricted to 2-13°C on host P19X (Delisle and Levin 1972a). However,

in this case, it was not due to an inability of the virus to adsorb to host cells, as temperature (0 and 26°C) had no effect on the absorption of Phage 27 to P19X. Such virus-host co-occurring variability in temperature sensitivity, within the optimum range host growth, will enhance the temporal intraspecies diversity index.

Temperature is a major regulatory factor for microbial growth (through the regulation of enzyme kinetics, molecular diffusion, and membrane transport) and therefore can be expected to affect viral life strategy and viral production (White et al. 1991; Wiebe et al. 1992). Indeed, seasonal variations in temperature (from 15 to 30°C) correlated to prophage (φHSIC) induction in a eutrophic estuarine environment were found to be a consequence of the 2-fold higher growth rate of the host (*Listonella pelagia)* at 28°C compared to 18°C (Cochran and Paul 1998; Williamson et al. 2002; Williamson and Paul 2006). Temperature-induced difference in growth rate is also the most probable cause for the delay in the onset of viral lysis of infected *H. akashiwo*, i.e., 2 to 3-fold delay in lysis under suboptimal temperature conditions of host (Nagasaki and Yamaguchi 1998). Unfortunately, the authors did not sample for virus abundance so no conclusions can be made on alterations to adsorption, latent period or burst size. Nevertheless, this study illustrates the importance of studying different host strain and virus strain models in order to accurately extrapolate to natural virus-host dynamics (e.g. red-tide bloom dynamics in the case of *H. akaswhiwo*). In contrast, production of a filamentous phage (SW1) in the deep-sea bacterium *S. piezotolerans* WP3 only occurred at temperatures below the optimum of the host, producing 2 to 9-fold more plaques at 4°C compared to 10°C and 15°C (Wang et al. 2007). Moreover, SW1 was shown to have a negative effect on the swarming ability of the host at low temperatures, which may provide energy for SW1 proliferation under suboptimal growth conditions of the host and therefore may play a role in adjusting the fitness of the host cells to the cold deep-sea environments (Jian et al. 2013).

Salinity

Osmotic shock assays demonstrate that viral capsids have differing permeabilities to water and salt ions, which can lead to inactivation or virus particle destruction when exposed to rapid changes in ionic strength (Cordova et al. 2003). There is also evidence which suggests that phage morphology may play a role in resistance. Tailed viruses appear to be the most resistant to changes in ionic strength. In

addition, membrane viruses are more sensitive compared to non-lipid containing, and of the membrane containing viruses, enveloped viruses are more sensitive than those with internal membranes (Kukkaro and Bamford 2009). Similar to temperature, the realized niche of viruses can be constrained either by their own sensitivity to variations in ionic strength or by that of their hosts. Bacteriophages and archaeoviruses isolated from a wide range of ionic strength environments, have been found to be more resistant to variations in ionic strength than their host (Table 2). Moreover, marine bacteriophages appear to have specific ionic requirements to maintain structural stability and remain infective indicating an adaptation to the marine environment. Both sodium and magnesium ions were necessary for retention of viability for bacteriophages NCMB384 and 385 infecting the marine *Cytophaga* sp. NCMB397 (Chen et al. 1966). Keynan and colleagues (1974) found sodium ion concentration was the most important for the stability of h*ν*-1 phage of the marine luminous bacterium *Beneckea harveyi* (maximum stability in seawater, followed by 3% NaCl; Keynan et al. 1974). Stability, infectivity, plating efficiency and uniformity of plaque formation, but not adsorption to host, were improved by divalent ions such as Mg^{2+} and Ca^{2+}, indicating that the requirement was related to infection of phage DNA. Thus salt stress may affect the survival and successful infection of bacteriophages, potentially through decreases in capsid pressure and consequent reductions to DNA injection efficiency or release of phage DNA though cracks in the capsid, which has been shown for the coliphage λ. Wherein the injection of DNA into host is driven by energy stored in the DNA due to its confinement, therefore any changes in ion concentrations can interact with the DNA and change the state of stress and hence the ejection force (Cordova et al. 2003; Evilevitch et al. 2008).

There is high variability in the effect of salt concentration on the adsorption of virus to host, even up to four orders of magnitude, suggesting different mechanisms for host binding (Zachary 1976; Torsvik and Dundas 1980; Kukkaro and Bamford 2009; Wigginton et al. 2012). Marine bacteriophages appear to have maximum host cell binding at salt concentrations similar to seawater, suggesting that viruses adapt to the ionic strength of their native environment. However, this could also be due to the host, as cationic imbalance, particularly deficiencies of various cations are believed to affect the permeability and other properties of cells surface structures in certain marine bacteria, which would impede attachment or penetration mechanisms of the phage (Brown 1964). Slowest binding kinetics were found amongst viruses isolated from Archaea-dominated high salt environments

(Kukkaro and Bamford 2009). The slower adsorption kinetics of archaeoviruses compared to bacteriophages might be explained by dissimilarity of surface structures of bacteria and archaeal hosts (i.e., as Archaea have lower membrane permeability; Valentine 2007). Halophages may have evolved to exert minimal selective pressure on their sensitive hosts (Santos et al. 2012). Prokaryotic hosts are more sensitive to changes in ionic strength and the physiological state of cells decreases at high-salt concentrations (Kukkaro and Bamford 2009; Bettarel et al. 2011). Therefore slow absorption, in combination with slow decay rate, might be selected for as a mechanism to avoid decimating host population under higher ionic stress or could in turn be tied to the low generation times of hosts, as fast adsorption and infection dynamics could decimate host populations (Bettarel et al. 2011). Indeed, over a wide range of salinities (10 - 360) along the coast of Senegal the frequency of infected prokaryotes in was negatively correlated with salinity, whereas a high percentage lysogenic prokaryotes at the higher salinities (> 150) was found to correlate to the abundance of archaeal cells (Bettarel et al. 2011).

Although salinity has been found to trigger the marine temperate phage φHSIC to switch to a lysogenic existence when incubated at brackish salinity, it is likely that this was due to a reduction of host growth rate (Williamson and Paul 2006). However, there is also some indication that salinity can alter viral proliferation independent of host growth. A study employing a estuarine salt marsh bacterium *B. natriengens* reported phage-specific effects on production with alterations in salinity (Zachary 1976). Phage nt-1 showed longer latent periods and highly reduced burst sizes (plaque forming units) at salinities below 18, while nt-6 revealed highest phage production rates at brackish salinities (which was below the host's growth optimum). The differences between these phages exemplify the potential importance of salinity on virus-host interactions and suggest a mechanism for alterations in viral population dynamics under changing salinity, both particularly meaningful for estuarine viral ecology

Table 2. Salinity (expressed as M NaCl) ranges for host growth and the range tested and values for successful infectivity and adsorption of associated viruses. Parenthesis indicate that value was assumed based on range tested for absorbance of virus to host.

Host	Range	Virus	Ions required	Tested	Infective	Max adsorption	References
Salmonella enterica	0 to 0.75	PRD1		0 to 4.50	< 4.25	0	(Kukkaro & Bamford, 2009)
S. enterica	0 to 1.00	P22		0 to 4.50	0 to 4.50	0	(Kukkaro & Bamford, 2009)
Pseudomonas syringae	0 to 0.25	φ6		0 to 4.50	< 2.75	0.25	(Kukkaro & Bamford, 2009)
Pseudolateromonas sp.	0 to 1.75	PM2		0 to 4.50	< 3.25	0.75	(Kukkaro & Bamford, 2009)
Halorubrum sp.	2.00 to 4.50	HRTV-1		0 to 4.50	0 to 4.50	> 4.00	(Kukkaro & Bamford, 2009)
H. hispanica	2.25 to 4.50	HHTV-1		0 to 4.50	0 to 4.50	4	(Kukkaro & Bamford, 2009)
H. hispanica	2.25 to 4.50	HHPV-1		0 to 4.50	> 1.75	3.5	(Kukkaro & Bamford, 2009)
H. hispanica	2.00 to 4.50	SH1	$MgCl_2$ & NaCl*	0 to 4.50	0	> 4.00	(Kukkaro & Bamford, 2009) (Porter et al., 2005)
H. californiae	2.25 to 4.50	HCTV-1		0 to 4.50	0 to 4.50	3	(Kukkaro & Bamford, 2009)
Salicola sp.	1.00 to 3.50	SCTP-1		0 to 4.50	0 to 4.50	3.5	(Kukkaro & Bamford, 2009)
Salicola sp.	1.00 to 4.50	SCTP-2		0 to 4.50‡	0 to 4.50	3	(Kukkaro & Bamford, 2009)
Vibrio (Beneckea) natriegens	0.06 to 0.40	nt-1	Na^+, K^+ †	0 to 0.16	0 to 0.16	> 0.16	(Zachary, 1976)
V. natriegens		nt-6	ns †	0 to 0.16	≥ 0.06	=	(Zachary, 1976)
V. harveyi	0.10 to 0.60	hν-1	Na^+, Ca^{2+}, Mg^{2+}*	0 to 0.60	> 0.085		(Keynan et al., 1974)
V. fischeri MJ-1	(0 to 1.03)	rp-1		0 to 1.37	0 to 1.37	0.34 to 0.68	(Levisohn et al., 1987)
Aeromonas sp.	0.085 to 0.50	unknown	Mg^{2+}, Ca^{2+} ‡				(Wiebe & Liston, 1968)
Cytophaga sp.		NCMB 384	Mg^{2+}, Na^+ *				(Chen et al., 1966)
Cytophaga sp.		NCMB 385	Mg^{2+}, Na^+ *				(Chen et al., 1966)
Colwellia psychrerythraea 34 H	0.29 to 0.96	Phage 9A		0.29 to 0.74	0.29 to 0.74		(Wells & Deming, 2006b)
C. demingiae ACAM 459^T	0.29 to 0.96	Phage 9A		0.40 to 0.50	0.40 to 0.50		(Wells & Deming, 2006b)

* required for stability
† required for adsorption
‡ required for lysis of host
=, equal efficiency
^{T} highest identities based on 16S analysis

UV

Biologically harmful ultraviolet radiation (UV, 100-400 nm) can penetrate to depths exceeding 60 m in clear oceanic waters (Booth et al. 1997; Whitehead et al. 2000) and has been found to be a principal factor contributing to the decline of viral infectivity in bacteriophages, cyanophages and viruses infecting eukaryotic hosts, with average losses of 0.2 h^{-1} and rates up to 0.8 h^{-1} in phage isolates (Table 3). Solar radiation can directly affect free viruses by degrading proteins, altering structure, and decreasing infectivity (Suttle and Chen 1992; Wommack et al. 1996; Wilhelm et al. 1998a; Weinbauer et al. 1999). However, viral particles appear more vulnerable to inactivation than to destruction (Wommack et al. 1996; Jacquet and Bratbak 2003). While a strong link between UVA and loss of infectivity of marine viruses has not been found, UVB shows a clear correlation (Table 3) (Weinbauer et al. 1997; Wilhelm et al. 1998a; Jacquet and Bratbak 2003). The shorter wavelength (290 - 320 nm) can result in the modification of viral proteins and the formation of photoproducts such as cyclobutane pyrimidine dimers (CPD) (Kellogg and Paul 2002; Hotze et al. 2009; Wigginton et al. 2010). As common lethal photoproducts of UV are thymine dimers, DNA viruses (containing thymine) are generally more sensitive to damage by UV than RNA viruses (not containing thymine). Furthermore, double stranded DNA or RNA viruses are more resistant to UV than single stranded viruses (Lytle and Sagripanti 2005). However, these differences have yet to be demonstrated for marine viruses. Interestingly, Kellogg and Paul (2002) found a significant negative correlation between the G+C content of marine phage DNA and the degree of DNA damage induced by solar radiation. Viruses with AT-rich genomes and thus higher potential dimer (T-T) sites, had a higher potential for UV damage (Kellogg and Paul 2002). In addition, AT-rich DNA also enhances the generation of oxygen species, which cause oxidative damage (Wei et al. 1998). Repair mechanisms can reduce the lethal effect of UV, especially for viruses possessing double stranded DNA (Lytle and Sagripanti 2005). The dsDNA virus PBCV of Chlorella contains a DNA repair gene giving it access to 2 DNA repair mechanisms, i.e., photoreactivation using host encoded gene products and a virus-encoded enzyme that initiates dark repair (Furuta et al. 1997). The combined activities of these repair systems should enhance survival and maintenance of viral activity, particularly in the relatively UV-rich surface waters.

Table 3. Average decay rates (h-1) reported for losses in infectivity of virus isolates in the dark, under full sunlight, and in the absence of UVB.

		Infectivity			Sample information		
Host	**Viral isolate**	**Dark**	**Sunlight**	**no UVB**	**Location**	**Time of year**	**Reference**
LMG1	LMG1-P4		0.68[1], 0.27[2]	0.181	Gulf of Mexico	May[1], various[2]	(Suttle & Chen, 1992)[1,†], (Suttle & Chan, 1994)[2]
PWH3a	PWH3a-P1	0.00[*]	0.35[1], 0.24[2], 0.80[3]	0.081	Gulf of Mexico	May[1], various[2], June[3]	(Suttle & Chen, 1992)[1,†], (Suttle & Chan, 1994)[2], (Wilhelm et al., 1998a)[3]
Photobacterium leiognathi (LB1VL)	LB1VL-P1b	0.00[*]	0.52[1], 0.28[2]	0.151	Gulf of Mexico	May[1], various[2]	(Suttle & Chen, 1992)[1,†], (Suttle & Chan, 1994)[2]
CB 38	CB 38Φ	0.05[*]	0.11[*]		York river estuary	October	(Wommack et al., 1996)[‡]
CB 7	CB 7Φ	0.04[*]	0.06[*]		York river estuary	October	(Wommack et al., 1996)[‡]
H2	H2/1	0.02[*]	0.07		Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
H11	H11/1	0.02[*]	0.07		Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
H40	H40/1	0.01[*]	0.09	0.06	Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
H85	H85/1	0.03[*]	0.07	0.04	Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
PR1	PR1/1	0.02[*]	0.05		Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
PR2	PR2/1	0.02[*]	0.04		Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
PR3	PR3/1	0.02[*]	0.05		Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
PR4	PR4/1	0.02[*]	0.04		Santa Monica Bay	March-July	(Noble & Fuhrman, 1997)[†,§]
Synechococcus sp.	S-PWM1		0.19		Gulf of Mexico	various	(Suttle & Chan, 1994)[†]
Synechococcus sp.	Natural Community *Syn DC2 phages*		0.19		Gulf of Mexico	1 year	(Garza & Suttle, 1998)
Synechococcus sp.	Syn DC2 isolates (S-PWM1 and S-PWM3)		0.39		Gulf of Mexico	1 year	(Garza & Suttle, 1998)[b]
Micromonas pusilla	MpV SP1	0.00	0.3		Gulf of Mexico	March-April	(Cottrell & Suttle, 1995)

[*] 0.2 µm filter seawater or artificial seawater

[†] estimated from figures in referred paper.

[‡] light transmission: UVC (200-290nm) 3-23%, UVB (290-320nm) 23-26%; UVA (320-400nm) 26-32%, PAR (400-700 nm) 32-55%

[§] light transmission: UVB (290-320nm) 67%; UVA (320-400nm)

Numerical superscipts link data in table to the appropriate reference

While some large dsDNA algal viruses may encode for their own DNA repair enzymes most viruses rely on repair mechanisms of their hosts, which are achievable only after DNA has been inserted into the host cell (Furuta et al. 1997; Weinbauer et al. 1997; Shaffer et al. 1999; Orgata et al. 2011; Santini et al. 2013). In the summer, Garza and Suttle (1998) found 2-fold lower decay rates of natural cyanophage communities as compared to isolates, whereas in the winter they were equal, suggesting (seasonal) selection for viruses that encoding host-mediated repair mechanisms. Alternatively, this may be explained by the rapid inactivation and removal of more sensitive cyanophages. Either way, it is important to note that UV-impact studies employing viral isolates may not represent the natural community response, and that viruses in surface waters may be more infective than previously thought based on the literature decay values.

CPDs in marine viruses have been found to increase over a latitudinal gradient (i.e., 41°S to 4°N), from 250 in the south to 2000 Mb^{-1} DNA near the equator, consistent with longer solar days and decreased solar angle. In addition, in the Gulf of Mexico, higher rates (i.e., 0.35 h^{-1}) for the loss of infectivity were recorded in natural cyanophage communities and cyanophage isolates (S-PWM1 and S-PWM3) during the summer and early fall when solar insolation was the highest, compared to undetectable levels in winter and spring (Garza and Suttle 1998; Wilhelm et al. 2003). Variations in level of DNA damage in waters with similar transparency and optical properties but different mixing depths have been found (Wilhelm et al. 1998b). When the mixing depth was reduced by half, photoreactivation was prevented and resulted in increased levels of CPD beyond what could be repaired overnight, leading to accumulation of damage over time (Wilhelm et al. 1998b). Similarly, Wilhelm and coworkers found high residual CDP levels in the surface water viral community of the pacific coastal waters of South America at high latitudes, at the time experiencing an equatorial upwelling event (Wilhelm et al. 2003). Viruses were, therefore, constricted to the surface waters leading to higher residence times and DNA damage levels exceeding normal daily levels. There is substantial evidence that marine viruses may adapt to local conditions of solar radiation; making them less susceptible to the degradation. Phages isolated from the coastal waters of Santa Monica Bay (USA) were 50 - 75% less susceptible to decay under local solar radiation than non-native phages of the North Sea (Noble and Fuhrman 1997). In addition, phages isolated from tropical waters have higher G+C content and higher survival rates over a range of UV radiation compared to phages isolated from temperature regions (Kellogg and Paul 2002). Similarly,

the proportion of lysogens induced by sunlight was found to be lower at oceanic than at coastal stations, which may be due to higher resistance to induction in the more transparent oligotrophic open ocean, or to induction of most UV-inducible lysogens. Natural solar radiation may thus alter the viral life cycle by inducing lysogenic phage production, but it does not appear to be an important source of phage production (max. 3.5%; Wilcox and Fuhrman 1994; Jiang and Paul 1998; Weinbauer and Suttle 1999; Weinbauer and Suttle 1996).

Due to the difference in susceptibility and abilities of viruses to repair the damaging effects of UV, it is not surprising that a considerable amount of variability exists in the sensitivity of viral particles to UV radiation, which has important implications for host population dynamics and species diversity (Suttle and Chen 1992; Wommack et al. 1996; Noble and Fuhrman 1997; Kellogg and Paul 2002; Jacquet and Bratbak 2003; Lytle and Sagripanti 2005). Five large dsDNA algal viruses, showed varying sensitivities to UVB from no effect for PoV infecting *Pyramimonas orientalis* to complete inactivation for PpV infecting *Phaeocystis pouchetii* (Jacquet and Bratbak 2003). Interestingly, the same study showed that some of these algal viruses, i.e., of *P. pouchetii* and *M. pusilla*, had a protective effect on surviving host cells when exposed to UVB subsequent to infection (Jacquet and Bratbak 2003). Although the mechanisms of UVB stress and resistance to viral infection remain largely unclear it demonstrates the complexity of how environmental factors interact with host-virus systems.

Photosynthetic Active Radiation (PAR)

Light is the essential energy source for photosynthetic organisms and most often drives synchronization of phytoplankton cell division and thus DNA synthesis and mitosis. Production of the virus infecting *P. orientalis* was found to depend on the host cell cycle, with 3 to 8-fold increase in progeny viruses when infection occurred at the end of cell division cycle (around the onset of the light period; Thyrhaug et al. 2002). During a mesocosm study of *E. huxleyi* blooms, EhV abundance increased during the first part of the day (light period) suggesting that viral production was also synchronized to host cell cycle (Jacquet et al. 2002). A diel cycle-dependent cyanophage infection has been hypothesized, with maximal phage production and reinfection occurring at night, to explain the sharp decline in *Synechococcus* abundance at the onset of darkness (Suttle 2000). However, support

for this hypothesis from field observations vary (Bettarel et al. 2002; Clokie et al. 2006). Light-dependent viral infection and proliferation which triggers infection by dawn and lysis by dusk or dark, would reduce exposure of the viruses to light (UV) and allow viral replication to align with its host's reproduction cycle (Clokie and Mann 2006). Diel patterns have even been described in virally infected bacterioplankton (Winter et al. 2004), i.e., viral lysis of bacteria with high viral progeny occurring around noon or early afternoon when bacterial activity was most likely responding to photosynthetic extracellular release (in combination with increased bioavailability of dissolved organic carbon by UV radiation). In addition, the newly released phages may accumulate less DNA damage (by UV) in the afternoon (Winter et al. 2004). This concept would also explain the diel variability described for a natural microbial community of NW Mediterranean Sea (Bettarel et al. 2002). One mechanism by which viruses could synchronize infection with host cell cycle is to have light dependent absorption. The effect of light on the adsorption of 9 cyanophages to *Synechococcus* sp. (WH7803) were found to be either light-independent (S-PWM1, S-BM3, S-MM4, S-MM1, S-MM5) or light-dependent (S-BnM1, S-BP3, S-PWM3, S-PM2) (Jia et al. 2010). However, the adsorption rate and dependence on light was host strain-specific. Light-dependent adsorption may be due to light-induced charge neutralization at the cell surface or by light-induced alterations to the ionic composition of the host cell surfaces, which could vary according to the host (Cseke and Farkas 1979).

In contrast, the production of PpV infecting *P. pouchetii* was cell cycle-independent which was in agreement with earlier work showing that the duration of the lytic cycle of PpV was of similar duration in darkness as in light and therefore not dependent on photophosphorylation (Bratbak et al. 1998). Similarly, the latent period of algal viruses infecting Chlorella (PBCV-1, first algal virus characterized, although not marine) and *H. akashiwo* (1 ssRNA and 2 uncharacterized DNA viruses) were unaffected by darkness (Van Etten et al. 1983; Juneau et al. 2003; Lawrence and Suttle 2004). However, the viral burst size strongly decreased (50% for PBCV and 90% for PpV), implying that light independent processes such as exploitation of host energy via chlororespiration, ATP reserves and/or production via respiration could provide the energy needed for viral replication and host cell lysis (Juneau et al. 2003). The degree to which darkness affects viral production can also depend upon the previous light conditions experienced by the host (Baudoux and Brussaard 2008). Viral production in *P. globosa* pre-adapted to a low irradiance level (25 μmol quanta m^{-2} s^{-1}) was inhibited under darkness but resumed once

light was reinitiated. Conversely, mid and high light (100 and 250 µmol quanta m^{-2} s^{-1}) pre-adapted host cells did not show additional viral production when reintroduced to the light. The burst sizes of the low and high light-adapted *P. globosa* cells were only half of the mid light cultures indicating PgV proliferation is sensitive to shortage of energy (low light) as well as high irradiance inhibition that likely induces reactive oxygen species formation. Conversely, light level did not affect the virus growth cycle of MpV infecting *Micromonas pusilla* (Baudoux and Brussaard 2008). Yet, prolonged darkness (48 - 65h) did delay host cell lysis and consequent release of virus progeny (Brown et al. 2007; Baudoux and Brussaard 2008). It is presumed that energy and potentially reductants derived from stored metabolic intermediates were sufficient to permit viral multiplication to proceed, but at the expense of the host DNA replication (Brown et al. 2007). Late stages of infection and lysis, however, may be too energy expensive to be overcome in the dark and thus required host photosynthetic energy. We speculate that such response to viral infection under darkness is related to cell size, with the small sized picophytoplankton having insufficient reserves to complete the virus growth cycle under darkness. Darkness is an extreme condition of light limitation, in nature phytoplankton cells are exposed periodically to dark at night and prolonged darkness occurs only once cells sink out of the euphotic zone. However, within the euphotic zone, light conditions are far from static and algae can experience changes in light of several orders of magnitude throughout the day depending on mixing conditions and cloud cover.

These studies show that viral production of phytoplankton species may occur at low light levels and even below the photic zone, although the extent is species-specific and dependent on the growth conditions prior to viral infection. Typically, the dependence of viral replication on light is characterized by a gradual shut down of host photosynthesis, with a portion of photosynthetic capacity being maintained until the end of the lytic cycle (Waters and Chan 1982; Suttle and Chan 1993; Juneau et al. 2003; Brown et al. 2007; Baudoux and Brussaard 2008). In some hosts this dependence was investigated in more detail and key photosynthetic complexes such as the chloroplasts, and ratios of several key photosynthetic proteins (rubisco, PSI, PSII and ATP synthase) were maintained during the course of infection (Juneau et al. 2003; Brown et al. 2007). The importance of light in marine virus-host systems is further exemplified by the acquisition of key photosynthetic functional genes by cyanophages infecting *Prochlorococcus* and *Synechococcus* during the course of evolution (Sullivan et al. 2006). These photosynthetic genes are expressed during

viral replication and aid in maintaining host photosynthesis and ensuring the provision of energy for viral replication until the onset of lysis (Lindell et al. 2005). To our knowledge, no studies on the potential impact of the color (wavelength) of the photosynthetically active radiation (PAR, 400 - 700nm) have been reported.

Nutrients

Lysogeny often prevails in systems with a lower trophic status, independent of geographical location (Williamson et al. 2002; Weinbauer et al. 2003; Payet and Suttle 2013). In the deep-sea, microbes, experiencing a low nutrient flux and rapidly changing conditions, have high numbers of lysogenic hosts (Weinbauer et al. 2003; Williamson et al. 2008; Anderson et al. 2011). Hence, lysogeny seems to represent a survival strategy under conditions of low host productivity and abundance, and exemplifies the crucial role that host physiology plays in determining viral life strategy. Seasonal studies and nutrient addition experiments demonstrate that viral production can be enhanced through alterations in bacterial host metabolism either by increasing host growth rate or by prophage induction (Williamson et al. 2002; Motegi and Nagata 2007; Payet and Suttle 2013). Likewise, P-limitation of cyanobacterium *Synechococcus* sp. induces lysogens, while P-addition stimulated the production of temperate cyanophage from natural P-depleted *Synechococcus* spp. (Wilson et al. 1996; Wilson et al. 1998). Viral-induced lysis of host resulting from lytic infection, may then act as a switch for lytic infection of prophages of the community in uninfected host which can utilize the cellular compounds released from lysed cells. These findings illustrate the highly dynamic and responsive nature of viral life strategies to environmental factors. As the impact on host population dynamics, food web functioning and biogeochemical cycling are very different for lysogenic or lytic viral infection, there is need for more detailed studies on this topic.

In addition to altering virus life strategies, environmental conditions that affect host physiology can also regulate the characteristics of a lytic viral infection. The latent period of marine bacteriophages typically corresponds closely with host generation time (Proctor et al. 1993; Middelboe 2000). Similarly, viral production is often found to have a negative relationship to host growth phase, i.e., lowest for host cells in stationary phase in comparison to exponentially growing cultures (Moebus 1996; Middelboe 2000). Interestingly, no distinct trend has been found between

burst size (mostly determined by whole cell TEM analysis) and bacterial production across different systems (Parada et al. 2006), which may be due to high bacterial host diversity under these natural conditions or selection for lysogeny under non-favorable conditions. However, in some algal host-virus model systems, burst size has been found to be linked to host growth phase (Van Etten et al. 1983; Bratbak et al. 1998). Experiments with Chlorella virus PBCV-1 revealed that this was not due to differences in adsorption but rather enhanced viral replication in actively growing cells (Van Etten et al. 1983). Shirai and coauthors (2008) found that while host growth phase had no effect on the burst size of the ssRNA virus CtenRNAV01 infecting the diatom *Chaeotoceros tenuissimus*, viral lysis occurred earlier in the stationary-phase culture. Interestingly, Nagasaki and Yamaguchi (1998) showed that the harmful algal bloom-forming *H. akashiwo* was sensitive to infection by both dsDNA viruses HaV01 and HaV08 when growing exponentially, but became resistant to HaV01 when in stationary phase. While, the underlying mechanism is unknown, the results indicate that the functional status of the host cell is an important determinant of virus-host interactions. Despite the potential importance of host growth as a driving factor of virus-host dynamics and subsequent organic matter cycling, there is surprisingly little attention focused on this area of research. A large proportion of the seas and oceans are oligotrophic and phytoplankton growth is often limited by inorganic nutrient (P, N, Si, Fe) availability, increasing the potential importance of host growth as a regulatory factor of virus-host interactions. Only two studies have investigated the consequence of N-depletion in host cells on viral production and demonstrate either no effect or reduced virus yield (Bratbak et al. 1993; Bratbak et al. 1998). Alternatively, the few studies which have focused on the effect of P-depletion on algal host-virus interactions consistently show reductions in the production of viruses, i.e., on average 70% for EhV infecting *E. huxleyi*, 30% for PpV infecting *P. pouchetii* after correction for growth phase differences and 80% for MpV infecting *M. pusilla* (Bratbak et al. 1993; Bratbak et al. 1998; Jacquet et al. 2002; Maat et al. in press). Furthermore, the length of the latent period of *M. pusilla* virus MpV-08T was positively correlated to the degree of P-limitation (Maat et al. in press). Although in theory viral production can be depressed in P-limited cultures due to insufficient intracellular P for the production of nucleic acid-rich (and thus P-rich) viruses, it may also be caused by reduced energy availability (Clasen and Elser 2007; Maat et al. in press).

Wikner and colleagues (1993) have shown that bacterial host nucleic acids serve as a major source of nucleotides for marine bacteriophages, and suggests a mechanism

by which marine phages limit their sensitivity to P-limitation which may be common in some open ocean areas (Paytan and McLaughlin 2007). Interestingly, *Prochlorococcus* cyanophage genomes may contain the putative ribonucleotide reductase (RNR) domain, which could function as extra nucleotide-scavenging genes in P-limited environments (Sullivan et al. 2005). The highest fraction of cyanophage genomes containing host-like P-assimilation genes originate from low-P source waters (Sullivan et al. 2010; Anderson et al. 2011; Kelly et al. 2013). Moreover, host-derived *pho*-regulon genes, which regulate phosphate uptake and metabolism under low-phosphate conditions, are found specifically in marine phages (40% of marine vs 4% of non-marine phage genomes (Goldsmith et al. 2011). Recently, Zeng and Chisholm (2012) showed enhanced transcription of the *Prochlorococcus* cyanophage-encoded alkaline phosphatase gene (phoA) and the high-affinity phosphate-binding protein gene (pstS), both of which have host orthologs, in phages infecting P-starved hosts. Such adaptations suggests that manipulation of host-PO_4 uptake may be an important adaptation strategy for viral proliferation in many marine ecosystems (Monier et al. 2011). Moreover, phage-genes are controlled by the host's PhoR/PhoB system, illustrating nicely the regulation of lytic phage genes by nutrient limitation of host.

Inorganic particles

Turbidity not only affects light penetration in sea but may also passively adsorb viruses. In natural waters, viruses possess a net negative surface charge due primarily to the ionization of carboxyl groups present on the external surfaces of viral capsid proteins (Wait and Sobsey 1983). Low molecular weight peptides and amino acids have a natural binding affinity for clay minerals, with the amount being absorbed and bound dependent on the type of clay and the type of cation saturating the clay (Dashman and Stotzky 1984). The addition of functional groups, such as amino or carboxyl groups, enhances absorption suggesting that these molecules play an important role in the absorption kinetics (Dashman and Stotzky 1984). Many studies have demonstrated the capacity of viruses to adsorb and bind to sediment and clay particles in the marine system (Kapuscinski and Mitchell 1980; Kimura et al. 2008). These reveal that association with particles can enhance survival and persistence of viruses by providing protection against UV radiation (most likely due to shading) and chemical pollutants relative to free

viruses in seawater (Vettori et al. 2000; Templeton et al. 2005; Kimura et al. 2008). In addition, the ability of marine viruses to irreversibly or reversibly bind to clay particles depends on virus and clay type and can be affected by environmental factors such as temperature, mixing, changes in ionic strength, organic matter type, size and concentration that either enhance adsorption or induce desorption of viruses from some particles (Kapuscinski and Mitchell 1980; Lipson and Stotzky 1984). However, these studies only focused on the effect of particles on enteric phages. Hewson and Furhman (2003) reported that between 20 - 90% of natural marine viruses can be absorbed by mineralogically uncharacterized suspended sediments, dependent on sediment concentration, size and source. The marine (cold-active) heterotrophic bacteriophage-9A failed to be inactivated by incubation with different ecologically relevant clay types, however, this could have been due to the high concentration of organics in the medium used, as organic matter can inhibit phage adsorption to clays, presumably by outcompeting phage for binding sites (Lipson and Stotzky 1984; Wells and Deming 2006a). Alternatively, a marine bacteriophage under simple media conditions has been shown to serve as nuclei for iron adsorption and precipitation, presumably via the same binding mechanism as clays, i.e., carboxyl and amino functional group reactive sites enabling iron atoms to penetrate and bind to the protein capsid (Daughney et al. 2004). Anthropogenic pollution has also led to the introduction of non-native particles such as black carbon to the marine environment, which has also been shown to adsorb viruses (Cattaneo et al. 2010). Due to the lack of scrutiny applied to the study the effects of clay and sediment on native marine viruses, as well as other native and non-native particles, additional research is needed to understand the ecological role, either as protective or removal agents, that the association of marine viruses with these particles has in seas and oceans.

Organic particles

In the marine environment, phytoplankton and bacteria generate large amounts of extracellular polysaccharides (EPS). In addition, intracellular substances released during viral lysis, or sloppy feeding also contribute to the organic matter pool. One important type of EPS is transparent exoploymer particles (TEP). TEP particles originate from colloidal DOM precursors, which may also bind to viruses and lead to inactivation, and might explain the discrepancy of inactivation rates in the <0.2

μm fraction of seawater (Mitchell and Jannasch 1969; Suttle and Chen 1992; Noble and Fuhrman 1997; Passow 2002; Finiguerra et al. 2011). TEP form a chemically and heterogeneous group of particles and their chemical composition and physical properties are dependent on the species releasing them and the prevailing environmental conditions. TEP aggregation is non-selective implying that all categories of particles present in the water become incorporated into aggregates (including viruses). In addition, TEP is primarily composed of negatively charged polysaccharides which would have a high affinity to viruses.

TEP is abundant in all marine waters (1 - 8000 ml^{-1} for the TEP > 5 μm and 3000 - 40000 ml^{-1} for the >2 μm fraction) and is often in the same size range as phytoplankton, dramatically increasing the potential collusion frequency of particles. Suttle and Chen (1992) estimated that viruses can adsorb to microaggregates at a rate of 0.41 d^{-1} (averaged over the upper 10 m of the water column), which rivaled the loss due to solar radiation (0.38 d^{-1}), indicating that ocean wide virus association with TEP could be a significant mechanism leading to the inactivation or removal of viruses from the pelagic system. Weinbauer and coworkers (2009) reviewed that viral abundance on suspended matter ranges between 10^5 to 10^{11} viruses cm^{-3} of aggregate, although it remains largely unclear how many viruses are truly attached and how many occur in the pore water of the TEP matrix and aggregates. In *P. globosa* mesocosms, TEP production resulting from colony disintegration and viral lysis adsorbed large quantities of the viral progeny (10 - 80%, also depending on whether N or P was limiting algal growth; Brussaard et al. 2005b). This could provide a mechanism by which viruses can be rapidly removed from the pelagic system after the collapse of a bloom. Conversely, reversible virus association may prolong survival and infectivity (Weinbauer et al. 2009). In addition, TEP colonized by microorganisms may continue to produce viral progeny, as viral production measurements of TEP have been shown to rival those of surrounding seawater (Proctor and Fuhrman 1991; Wommack and Colwell 2000; Weinbauer et al. 2009). Viral-induced cell lysis of the prokaryotic hosts releases organic matter that can further stimulate aggregation. While bacterial exo-enzymes (such as aminopeptidase) and dissolved extracellular proteases and nucleases (through cell lysis) within TEP may degrade viral capsid proteins and inactivate the viruses attached to aggregates (Simon et al. 2002; Bongiorni et al. 2007) in a similar manner to that found in seawater (although not always; Finiguerra et al. 2011) and sediment (Cliver and Herrmann 1972; Noble and Fuhrman 1997; Corinaldesi et al. 2010) and contribute to the disintegration of TEP aggregates. The magnitude to which

viruses are associated with different type, quality, size, and age of aggregates thus represents the sum of passive adsorption and the active production by the microbial community living on the aggregates (Weinbauer et al. 2009).

Grazing

Removal of viruses by heterotrophic nanoflagellate (HNF) grazing seem to play only a minor role in the removal of viruses (0.1% of virus community h^{-1}; Suttle and Chen 1992). Gonzalez and co-authors (1993) demonstrated that fluorescently labeled viruses were ingested and digested by cultured and natural HNFs at clearance rates of about 4% of those for bacterial prey, with rates depending on abundance and species of grazer and virus grazed. In contrast, Hadas et al. (2006) showed a removal of viruses by a coral reef sponge at an average efficiency of 23%, which may affect the virus-to-host ratios in the surrounding waters (depending on the removal rate of bacteria by the sponge) (Hadas et al. 2006). Enteric viruses have been found to accumulate in filter-feeding shell-fish (oysters, clams and mussels), revealing the potential of these organisms to dilute ambient virus concentrations (Rao et al. 1986; Enriquez et al. 1992; Faust et al. 2009). In addition to the direct removal of virus particles, organic particles present in seawater can also be grazed (Passow 2002). As these particles may have adsorbed viruses, the rate of viral removal by grazing might actually be underestimated.

Virus-specific selective grazing has the potential (when in high enough rates) to influence the specific virus-host dynamics and affect biodiversity. This effect can be further influenced by selective grazing on the virally infected host. Grazing of infected host cells will also alter the contact rate between virus and uninfected host cell by reducing the number of progeny viruses released from infected hosts (Ruardij et al. 2005). Preferential grazing of infected cells has been observed for *E. huxleyi* (Evans and Wilson 2008), but was unconfirmed using lower, more ecologically relevant algal abundances (Martínez-Martínez and Brussaard unpubl. data). Preferential grazing has also been hypothesized as a response to the inhibited release of star-like structures from infected *P. globosa* cells (Sheik et al. 2012). These rigid chitinous filaments are thought to provide a protective benefit against grazers (Zingone et al. 1999; Dutz and Koski 2006). In addition to increasing the susceptibility to grazers, this process directly reduces the availability of newly released PgVs by the release of hydrated flocculants, i.e., the intracellular precursors of the star-like structures

which exist in a fluid state within vesicles in the cell (Chretinennot-Dinet et al. 1997), which passively adsorb a high percentage of the viral progeny (~68%; Sheik et al. 2012). Through the use of nanoSIMS technology and single cell investigations, it has been revealed that viral infection of *P. globosa* results in a leakage or excretion of ^{13}C-labeled compounds prior to lysis which elicited an immediate response by the microbial community (Sheik et al. 2012). Leakage of intracellular material prior to lysis would provide a chemical trail which could be followed by chemotaxic grazers, thereby supporting a mechanism by which preferential grazing of infected cells could occur.

Grazing may also result in the release of viral antagonists. Upon grazing of *E. huxleyi* cells by *Oxyrrhis marina*, dimethyl sulfide (DMS) and acrylic acid was released which diminished the viral titers of EhV (Evans et al. 2006; Evans et al. 2007). While viral lysis of *E. huxleyi* also led to the production of DMS and acrylic acid the rate was reduced, which has been postulated to serve as a counter strategy of the virus to protect the infectivity of progeny viruses (Evans et al. 2006). The same mechanism might explain the earlier results by Thyrhaug and colleagues (2003) who demonstrated that the viral lysate of *E. huxleyi* contained inhibitory compounds that delayed cell lysis. The finding that the DMS concentrations differ between *E. huxleyi* strains (because of diverse intracellular dimethylsulfoniopropionate (DMSP) concentrations and DMSP lyase activities; Steinke et al. 1998), clearly illustrates how quickly multiple ecologically relevant factors complicate natural virus-host dynamics and promote co-existence of host and virus. Moreover, uninfected *E. huxleyi* cells subjected to viral glycosphingolipids, normally produced by infected *E. huxleyi* to induce the release of EhV progeny, promptly executed programmed cell death (Vardi et al. 2009). It has been suggested that during blooms of *E. huxleyi*, production of viral glycosphingolipids may act as a strategy to limit viral propagation through clonal host populations.

Host morphology

Grazing on heterotrophic prokaryotes can also lead to alterations in bacterial phenotypes (Pernthaler 2005). Filamentation, and the formation of microcolonies or biofilms reduces the likelihood that a specific prokaryotic host cell encounters a phage due to partial shading (Abedon 2012). On the other hand, if successfully infected, progeny viruses might have easy access to host in such an arrangement,

decreasing encounter time (Abedon 2012). However, the extent to which these processes influence viral encounter rate and host survival in the marine environment remain unknown, as detailed studies using marine phage-bacteria model systems are largely missing. Such information is essential as many marine bacteria are found in filaments or attached to particles (Tang et al. 2012).

It has been argued that not all host cell morphotypes of *E huxleyi* are equally sensitive to viral infection, and that exposure of *E. huxleyi* diploid cells to EhV would promote transition to the more infection resistant haploid phase, thereby ensuring that that genes of dominant diploid clones are passed on to the next generation in a virus-free environment (Frada et al. 2008). However, during a natural bloom of *E. huxleyi* both the diploid coccolith-bearing C-cells and the haploid scale-bearing S-cells were found to be virally infected (Brussaard et al. 1996). Another prymnesiophyte, *Phaeocystis,* also has a polymorphic life cycle phase which consists of solitary cells and cells embedded in a colony matrix. In this case, the haploid flagellated single cells are readily infected whereas the colonial stage protects against viral infection (Brussaard et al. 2005a; Jacobsen et al. 2007; Rousseau et al. 2007). Model evidence shows that the probability of a virus coming in contact with an individual colonial cell decreased with the size of the colony (Murray and Jackson 1992; Ruardij et al. 2005). *P. globosa* colony formation requires sufficient light for excess carbon fixation necessary to form the colonial matrix. Under reduced light conditions (20 µmol quanta m^{-2} s^{-1}) only exponential growth of the flagellated single cell morphotype was maintained and consequently viruses were able to control host abundance at low abundance and prevent bloom formation (Brussaard et al. 2005a; Brussaard et al. 2007).

Outlook

The present mini-review summarizes our current understanding of how environmental factors can influence virus dynamics and regulate virus-microbe host interactions. Marine viruses affect microbial host population abundance, community structure, and biogeochemical cycling in the ocean. Identifying environmental factors which regulate these processes is therefore essential to our understanding of global geochemical cycling and ecosystem functioning. This review illustrates a variety of factors in the marine environment which can influence viruses at all stages of their life cycle (Fig. 2). Moreover, it highlights the

fact that we are currently restricted by the availability of information regarding the effect that different environmental factors have on marine viruses and by the scarcity of reported rates. Factors which have been studied in more detail, e.g. UV radiation, provide useful insights into how viruses have multiple strategies by which they can adapt to their environment emphasizing the need for more detailed studies. We therefore encourage further research aimed at unraveling the role that the environment plays in regulating virus dynamics and virus-host interactions and recommend using both prokaryotic (both bacterial and archaeal) and eukaryotic virus model systems from a variety of locations and depths. We would also like to stress the importance of reporting the physicochemical and biological characteristics during field studies which is crucial for optimal interpretation. Moreover, standardization of approaches is warranted in order to allow comparison between different studies.

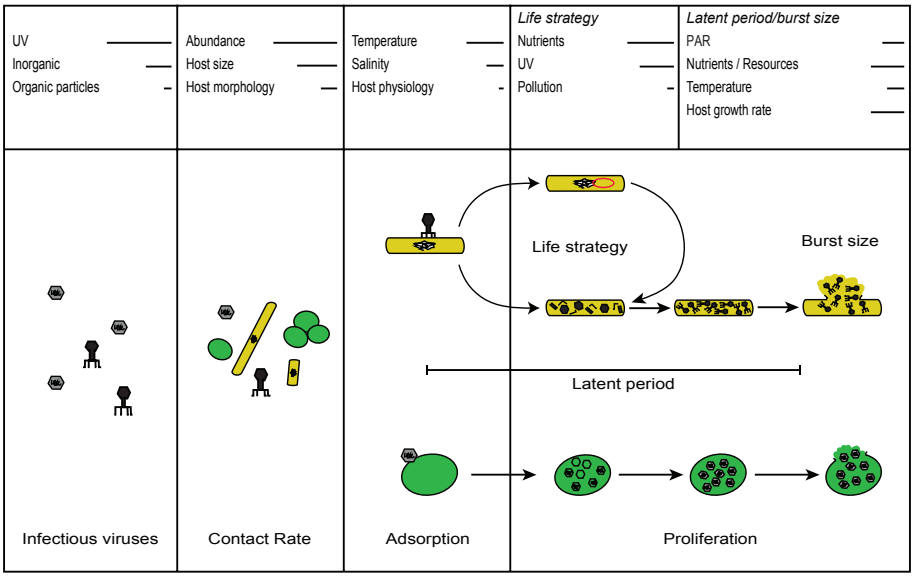

Figure 2. Conceptual diagram illustrating the influence of environmental factors on the different stages of virus life cycle. Horizontal bars indicate the amount of information known about the effect of the specific environmental variable on life cycle stage.

In order to fully understand the ecological relevance of environmental factors to viral ecology, the mechanisms behind losses of infectivity and absorption under natural conditions need to be elucidated. It may therefore be beneficial to consider extreme environments, where adaptations may be more apparent due to their

necessity for survival under such conditions, i.e., the stability of viral proteins in and around deep sea hydrothermal vents, resistance to extreme pH values near black smokes, and adaptation of viral G+C content to UV in the surface versus the deep ocean. In addition, some aspects which have not been addressed here, due to the scarcity of data, may have devastating consequences for marine ecosystems and should be considered in future endeavors. For example, lysogens have been found to be very sensitive to anthropogenic pollution. Pesticides, PCBs, trichloroethylene, PAHs, fuel oil and sunscreen, have all been reported to cause substantial prophage induction, even at low concentrations (Cochran et al. 1998; Paul et al. 1999; Danovaro et al. 2003). In addition, organic UV filters originating from sunscreen triggered lytic infection in prophages of symbiotic zooxanthellae resulting in the release of large amounts of coral mucous and complete bleaching within few days of exposure (Danovaro et al. 2008). There is also very little known about the stability of marine viruses to changes in pH, particularly for eukaryotic viruses. Although the few studies including pH sensitivity demonstrate that virus inactivation only occurred at relatively low pH values <7, pH could be an important environmental factor in some marine systems, as pH can vary both on local and seasonal scales (Hidaka and Ichida 1976; Borsheim 1993; Brussaard et al. 2004; Hofmann et al. 2011; Traving et al. 2013).

Finally, it is important to recognize that changing environmental conditions are most often multifactorial, thus shifting of one factor that influences viral infectivity, production or decay may change the sensitivity of the viruses to other factors. Therefore, it would be valuable to investigate interactions between different environmental stimuli. Especially considering that changing environmental conditions are themselves often comprised of multiple factors, i.e., alterations in sea surface temperature may be accompanied by changes in salinity, as well as UV exposure and nutrient limitation due to alterations in stratification. The lack of mechanistic understanding strongly restrains insight and predictive capacity of how, for example, global warming induced climate change (affecting multiple environmental variables) will influence viral production, activity and decay. Realizing the important ecological role viruses have for biodiversity and element fluxes, we would advocate for additional focus on this particular topic.

In summary, at this moment in time it is difficult to identify general patterns on how environmental factors regulate virus dynamics and virus-host interactions. In order to provide a broader overview which would permit viral ecologists to identify ecological functional patterns, virus-host systems need to be investigated in more

detail, across different types of environment and/or factors. While the current review is far from exhaustive, it provides a useful framework for identifying gaps in our understanding of (1) model host/virus systems (Table S1) and (2) field based testing which will likely lead to exciting new discoveries in marine viral ecology.

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Supporting Information

Table S1. Overview of marine viruses discussed in the review sections of temperature, salinity, and UV (Table 1-3).

Host	Virus	Family	genome type	genome size (kb)	Shape	Capsid size (nm)	Tail (W x L)	Size of major polypeptides (kDa	Latent period (h)	Particle accumulation site and pattern	Burst size (cell^{-1})	Strain specificity	Source
Eukaryote Algal Viruses													
Phaeocystis globosa (Pg-I)	Group I: PgV-06, -07T, -09T, -12T, -13T, -T14	*Phycodnaviridae*	dsDNA	466 ± 4	icosahedral l	153 ± 8		257, 161, 111, 52	10	cytoplasm	248 ± 120	4/12	water, North Sea [1]
P. globosa (Pg-I)	Group IIA:PgV-03T, -05T	*Phycodnaviridae*	dsDNA	177 ± 3	icosahedral	106 ± 7		119, 99, 75, 44	12	cytoplasm	344 ± 100	5/12	water, North Sea[1]
P. globosa (Pg-I)	Group IIB:PgV-04T, -10T, -11T	*Phycodnaviridae*	dsDNA	177 ± 3	icosahedral	106 ± 7		119, 99, 75, 44	16	cytoplasm	382 ± 26	5/12	water, North Sea[1]
P. globosa (Pg-I)	Group IIC:PgV-01T	*Phycodnaviridae*	dsDNA	177 ± 3	icosahedral	106 ± 7		119, 99, 75, 44	16	cytoplasm	378	12/12	water, North Sea[1]
Micromonas pusilla (LAC 38)	MpRNAV-01B	*Reoviridae*	Segmented, dsRNA	25.5	icosahedral	65-80		120, 95, 67, 53, 32	36	cytoplasm	460-520	1/6	coastal, water, Norway[2]
M. pusilla (LAC 38)	MpV-03T, 06T, 08-12T, 14T, R3-4, B4-5[3]	*Phycodnaviridae*	dsDNA		icosahedral								water, North Sea
M. pusilla (CCMP 1545)	MpV-02T, 04-05T, 07T, 13T, R1-R2, SP1[3]	*Phycodnaviridae*	dsDNA		icosahedral								water, North Sea
M. pusilla (27)	SP1	*Phycodnaviridae*	dsDNA										coastal water, Scripps pier [4]
Chaetoceros debilis (Ch48)	CdebDNAV18		ssDNA	unknown	icosahedral	30 ± 2		41, 37.5	12-24	cytoplasm, random	55 [a]	3/4	water and sediment, Japan[5]

Table S1. Continued.

Host	Virus	Family	genome type	genome size (kb)	Shape	Capsid size (nm)	Tail (W x L)	Size of major polypeptides (kDa	Latent period (h)	Particle accumulation site and pattern	Burst size (cell^{-1})	Strain specificity	Source
C. lorenzianus (IT-Dia51)	ClorDNAV	*Bacilladnavirus*	ssDNA, circular and dsDNA linear	5.9, 0.9	icosahedral	32 ± 2			<48	nucleus, random	22,000 [a]		coastal, water, Hiroshima Bay[6]
C. socialis	CsfrRNAV	*Bacilladnavirus*	ssRNA	9.5	icosahedral	22		32, 28, 25	< 48	cytoplasm, random	66 [a]		coastal, water, Hiroshima Bay [8]
C. tenuissimus	CtenRNAV01		ssRNA, circular?	9.4	icosahedral	31 ±2		33.5, 31.5, 30.0	<24	cytoplasm, crystalline arrays	12,000 [a]		water, Ariake Sound
Heterosigma. akashiwo (93616)	HaV01		dsDNA	294 [9]	icosahedral [10]	202 ± 6 [10]			30-33 [11]		770 [11]	13/18 [12]	coastal, water, Nomi Bay[10]
H. akashiwo (H93616)	HaV08		dsDNA										
H. akashiwo (H93616)	HaV53		dsDNA										
H. akashiwo (H93616)	HaRNAV		ssRNA	9100 nt [13]	icosahedral [13]	25 [13]		33.9, 29.0, 26.1, 24.6, 24.0 [13]	29 [14]	cytoplasm, crystalline arrays [13]	21,000 [14]		river plume, Strait of Georgia [13]
Heterocapsa circularisquama (HU9433-P)	HcV03		dsDNA		icosahedral	197 ± 8			48-72	cytoplasm, viroplasm	~1300	18/18	coastal, water, Japan [15]
H. circularisquama (HU9433-P)	HcV05		dsDNA										coastal, water, Japan [16]
H. circularisquama (HU9433-P)	HcV08		dsDNA									12/14	coastal, water, Japan [16]

Table S1. Continued.

Host	Virus	Family	genome type	genome size (kb)	Shape	Capsid size (nm)	Tail (W x L)	Size of major polypeptides (kDa	Latent period (h)	Particle accumulation site and pattern	Burst size (cell^{-1})	Strain specificity	Source
H. circularisquama (HU9433-P)	HcV10		dsDNA										coastal, water, Japan [16]
H. circularisquama (HU9433-P)	HcRNAV34		ssRNA	4.4 [17]	icosahedral	30 ± 2		38 [17]	24-48	cytoplasm, crystalline arrays or random	21000 [a]	2/4 [b]	coastal, water, Japan [17]
H. circularisquama (HCLG-1)	HcRNAV109		ssRNA	4.4 [17]	icosahedral	30 ± 2		38 [17]	24-48	cytoplasm, crystalline arrays or random	3400 [a]	2/4 [b]	coastal, water, Japan [17]
Bacteriophages													
Pseudomonas putrefaciens P19X	Phage 27		dsDNA		icosahedral	77	14 x 176						coastal, water [18]
P. putrefaciens P13	Phage 23		dsDNA		icosahedral	76	?						fish [18]
P. putrefaciens P2	Phage 25F		dsDNA		icosahedral	55	14 x 176						coastal, water [18]
Pseudoalteromonas marina	φRIO-1	*Podoviridae*	dsDNA		icosahedral	51	12 x 15		1		118	1/2	water, East Sea [19]
Vibrio sp.	unknown		dsDNA		hexagonal	60	?						Deep, water, mud, Indian Ocean [20]
Vibrio (Beneckea) harveyi	h*v*-1		dsDNA	43.9	icosahedral	70	12 x 220		0.75		100		squid/mud, Atlantic [21]
V. natriegens	nt-1	*Myoviridae* [22]	dsDNA		prolate	120	? X 110						estuary water, Chesapeake Bay [23]
V. natriegens	nt-6		dsDNA		icosahedral	60	? X 40						estuary water, Chesapeake Bay [23]

Table S1. Continued.

Host	Virus	Family	genome type	genome size (kb)	Shape	Capsid size (nm)	Tail (W x L)	Size of major polypeptides (kDa	Latent period (h)	Particle accumulation site and pattern	Burst size (cell^{-1})	Strain specificity	Source
V. fischeri MJ-1	rp-1		dsDNA		icosahedral	83	16 x 83		0.3833		100	21/27	coastal water, Mexico [24]
PWH3a	PWH3a-P1		dsDNA			50	? x ?						coastal water, Gulf of Mexico [25]
Pseudomonas sp.	06N-58P		ssRNA		icosahedral	60			0.5833		170		seawater, 5 miles offshore, Japan [26]
Bacillus sp. w13	BVW1		dsDNA	18	icosahedral	70	15 x 300	32, 45, 50, 57, 60, 70				1/5	deep, water and sediment, hydrothermal fields, Pacific Ocean [27]
Geobacillus sp. E26323	GVE1	*Siphoviridae*	dsDNA	41	icosahedral	130	30 x 180	34, 37, 43, 60, 66, 100				1/4	deep, water and sediment, hydrothermal fields, Pacific Ocean [27]
Colwellia psychrerythraea 34 H	Phage 9A	*Siphoviridae* [c 28]	dsDNA	104 [28]	icosahedrall	90	? X 200		4-5		55	2/8	water, nepheloid layer, Arctic [29]
21C (C. *psychrerythraea)* [d]	21c	*Siphoviridae*	dsDNA	40-50	icosahedral	46-48	9-11 x 151-188						sea ice, Artic [30]
Aeromonas sp.	unknown				icosahedral	53	15 x 160						sediment, Pacific Ocean [31]

Table S1. Continued.

Host	Virus	Family	genome type	genome size (kb)	Shape	Capsid size (nm)	Tail (W x L)	Size of major polypeptides (kDa	Latent period (h)	Particle accumulation site and pattern	Burst size (cell^{-1})	Strain specificity	Source
Listonella pelagia	φHSIC	*Siphoviridae*	dsDNA	36	icosahedral	47±3.7	? x 146±3.7		1.5		47		coastal water, Hawaii [32]
Shewanella piezotolerans	SW1		ssDNA	7,718 nt [33]	filamen-tous[33]								prophage, host was isolated from deep, sediment, Pacific [34]
1A (*S. frigidimarina* LMG 19867)[d]	1a	*Myoviridae*	dsDNA	70	icosahedral	94-103	11-15 x 94-103						sea ice, Artic [30]
Salmonella enterica	PRD1	*Tectiviridae* [22]	dsDNA	15 [22]	icosahedral	66 [22]							sewage
S. enterica	P22		dsDNA		icosahedral								
Pseudomonas syringae	φ6		dsRNA										
Pseudolateromonas sp.	PM2	*Corticoviridae* [35]	circular, dsDNA [35]	10.1 [35]	icosahedral	60 [36]			1 [36]	Cytoplasm [36]	50-600 [36]		coastal, water, Pacific [35]
Salicola sp.	SCTP-1				Icosahedral	55	? x 95						water, solar salterns, Italy [37]
Salicola sp.	SCTP-2				Icosahedral	125	? x 145						water, solar salterns, Italy [37]
Cytophaga sp.	NCMB 384		dsDNA						2.5		28		coastal, water, North Sea [38]
Cytophaga sp.	NCMB 385		dsDNA			78	? x 97		3.0		20		coastal, water, North Sea [38]
Photobacterium leiognathi	LB1VL-P1b		dsDNA										

Table S1. Continued.

Host	Virus	Family	genome type	genome size (kb)	Shape	Capsid size (nm)	Tail (W x L)	Size of major polypeptides (kDa	Latent period (h)	Particle accumulation site and pattern	Burst size (cell^{-1})	Strain specificity	Source
Unknown bacteria	LMG1-P4		dsDNA			83	? x 104						water, hypersaline lagoon, Gulf of Mexico [25]
Unknown CB 38	CB 38Φ												
Unknown CB 7	CB 7Φ												
Unknown H2	H2/1		dsDNA		Icosahedral	64	? x 71						water, North Sea [39]
Unknown H11	H11/1		dsDNA		Icosahedral	62	? x 75						water, North Sea [39]
Unknown H40	H40/1		dsDNA		Icosahedral	62	? x 117						water, North Sea [39]
Unknown H85	H85/1		dsDNA		Icosahedral	57	? x 120						water, North Sea [39]
Unknown PR1	PR1/1		dsDNA		Icosahedral	41							Coastal waters, Santa Monica Bay [39]
Unknown PR2	PR2/1		dsDNA		Icosahedral	86	? x 142						Coastal waters, Santa Monica Bay [39]
Unknown PR3	PR3/1		dsDNA		Icosahedral	42	? x ?						Coastal waters, Santa Monica Bay [39]

Table S1. Continued.

Host	Virus	Family	genome type	genome size (kb)	Shape	Capsid size (nm)	Tail (W x L)	Size of major polypeptides (kDa	Latent period (h)	Particle accumulation site and pattern	Burst size (cell^{-1})	Strain specificity	Source
Unknown PR4	PR4/1		dsDNA		Icosahedral	52	? x ?						Coastal waters, Santa Monica Bay [39]
Archaeal Viruses													
Halorubrum sp.	HRTV-1				Icosahedral	55	? x 85						water, solar salterns, Italy [37]
Haloarcula hispanica	HHTV-1				Icosahedral	55	? x 110						water, solar salterns, Italy [37]
H. hispanica	HHPV-1		dsDNA		pleomorphic								water, solar salterns, Italy [37]
H. hispanica	SH1		dsDNA	30.9±1.0 [40]	Icosahedral	70 [40]			5-6 [40]		200 [40]		water, hypersaline lake, Australia
H. californiae	HCTV-1				Icosahedral	70	? x 80						water, solar salterns, Italy [37]
Cyanophages													
Synechococcus sp. DC2	S-PWM1	*Myoviridae*	dsDNA	65	Icosahedral							1/8	coastal water, Gulf of Mexico [41]
Synechococcus sp. DC2	S-PWM3	*Myoviridae*	dsDNA		Icosahedral							4/8	coastal water, Gulf of Mexico [41]

[a] infectious units, [b]viruses infectious against different hosts, [c]"*unclassified*", [d] highest identities based on 16S analysis

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