BIOLOGICAL REVIEWS
Cambridge Philosophical Society
Biol. Rev. (2016), pp. 000–000.
doi: 10.1111/brv.12271

Deciphering the virus-to-prokaryote ratio (VPR): insights into virus–host relationships in a variety of ecosystems

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ABSTRACT

The discovery of the numerical importance of viruses in a variety of (aquatic) ecosystems has changed our perception of their importance in microbial processes. Bacteria and Archaea undoubtedly represent the most abundant cellular life forms on Earth and past estimates of viral numbers (represented mainly by viruses infecting prokaryotes) have indicated abundances at least one order of magnitude higher than that of their cellular hosts. Such dominance has been reflected most often by the virus-to-prokaryote ratio (VPR), proposed as a proxy for the relationship between viral and prokaryotic communities. VPR values have been discussed in the literature to express viral numerical dominance (or absence of it) over their cellular hosts, but the ecological meaning and interpretation of this ratio has remained somewhat nebulous or contradictory. We gathered data from 210 publications (and additional unpublished data) on viral ecology with the aim of exploring VPR. The results are presented in three parts: the first consists of an overview of the minimal, maximal and calculated average VPR values in an extensive variety of different environments. Results indicate that VPR values fluctuate over six orders of magnitude, with variations observed within each ecosystem. The second part investigates the relationship between VPR and other indices, in order to assess whether VPR can provide insights into virus–host relationships. A positive relationship was found between VPR and viral abundance (VA), frequency of visibly infected cells (FVIC), burst size (BS), frequency of lysogenic cells (FLC) and chlorophyll *a* (Chl *a*) concentration. An inverse relationship was detected between VPR and prokaryotic abundance (PA) (in sediments), prokaryotic production (PP) and virus–host contact rates (VCR) as well as salinity and temperature. No significant relationship was found between VPR and viral production (VP), fraction of mortality from viral lysis (FMVL), viral decay rate (VDR), viral turnover (VT) or depth. Finally, we summarize our results by proposing two scenarios in two contrasting environments, based on current theories on viral ecology as well as the present results. We conclude that since VPR fluctuates in every habitat for different reasons, as it is linked to a multitude of factors related to virus–host dynamics, extreme caution should be used when inferring relationships between viruses and their hosts. Furthermore, we posit that the VPR is only useful in specific, controlled conditions, e.g. for the monitoring of fluctuations in viral and host abundance over time.

Key words: virus, bacteria, prokaryote, relationships, aquatic ecosystems, VBR, VPR.

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I. INTRODUCTION: THE VIRUS-TO-PROKARYOTE RATIO – DEFINITION AND USE IN VIRAL ECOLOGY

With the development of direct counting techniques (i.e. transmission electron and epifluorescence microscopy as well as flow cytometry) and the increasing number of studies on viral abundance in aquatic ecosystems (Weinbauer, 2004), it became apparent that the number of virus-like particles (VLPs) greatly exceeds those of bacterial (or prokaryotic) cells. This changed the prevailing view that the role of viruses in horizontal gene transfer was limited in aquatic ecosystems due to low viral and/or prokaryotic abundance (Wommack *et al*., 1992). The early work of Bergh *et al*. (1989) on the high incidence of viral particles in aquatic ecosystems reported abundances from 10^3 to 10^7 times higher than previously estimated (then obtained by plaque-forming unit counts). Although these authors reported both viral and bacterial abundances, the proportions between the two communities were not addressed. Subsequent publications presented the ratio between enumerated VLPs and bacteria to compare the relative viral activity of different samples (Ogunseitan, Sayler & Miller, 1990; Hara, Terauchi & Koike, 1991) and termed this the 'virus-to-bacterium ratio' (VBR) (Wommack *et al*., 1992). The VBR has been used to study the relationship between viruses and bacteria in the environment (Wommack & Colwell, 2000) and as an index to demonstrate the high/low incidence of viral particles compared to bacteria in a given ecosystem. Usually, high VBR values are attributed to high and ongoing viral dynamics. Conversely, low ratios have often been interpreted as diminished viral activity, absence of viruses or high viral decay rates. These interpretations have rested on the logical supposition that at steady state, the VBR reflects the balance of viral production (VP) and loss (Maranger & Bird, 1995; Williamson, 2011). Consequently, it has been posited that declines in VBR are due to viral loss, for example by non-specific adsorption to particles (Maranger & Bird, 1995) or degradation after adsorption to humic substances (Anesio *et al*., 2004). Inversely, high VBR values have been attributed to high viral production (Middelboe *et al*., 2006; Kellogg, 2010; Yoshida-Takashima *et al*., 2012; Pinto, Larsen & Casper, 2013; Engelhardt *et al*., 2014; Parvathi *et al*., 2014) or low viral decay (Mei & Danovaro, 2004; Danovaro *et al*., 2005; Williamson *et al*., 2007; Winter, Kerros & Weinbauer, 2009; Maurice *et al*., 2010; De Corte *et al*., 2012), which in some cases (e.g. in soil and sediments) could be an artifact of extraction procedures (Middelboe, Glud & Finster, 2003; Williamson, Radosevich & Wommack, 2005; Kimura *et al*., 2008; Williamson, 2011). Trends in VBR and links with other variables (e.g. prokaryotic abundance, PA) have led to ambiguous interpretations due to apparently contradictory results. Several studies have reported a positive correlation between viral and prokaryotic abundance (e.g. Maranger & Bird, 1995; Weinbauer *et al*., 1995; Anesio *et al*., 2007; Danovaro *et al*., 2008*b*; Helton *et al*., 2012), suggesting coupling between prokaryotic production (PP) and that of viruses (Wommack & Colwell, 2000; Weinbauer, 2004). However, positive (Hara *et al*., 1996), negative/inverse (Wommack *et al*., 1992; Bratbak & Heldal, 1995; Tuomi *et al*., 1995; Nakayama *et al*., 2007; Personnic *et al*., 2009) and no (Peduzzi & Schiemer, 2004) relation have all been reported between prokaryotic abundance and VBR. To explain a positive correlation, a direct dependence of viral production on bacterial host abundance has been proposed, imposing additionally a possible selective pressure leading to a reduced volume of host cells (Hara *et al*., 1996). The inverse relationship, on the other hand, has been linked to high viral production, coupled with increased host lysis (e.g. during blooms), leading to high VBR values. Lower values would then be a result of the emergence of host cell resistance, leading to an increase in prokaryotic production and diminished viral production (Maranger, Bird & Juniper, 1994). Moreover, small VBR values have also been interpreted as a result of specific phage adsorption to host cells when host diversity is low, thus linking prokaryotic

diversity to the ratio of microbial and viral abundances (Bratbak & Heldal, 1995; Tuomi *et al*., 1995).

The lack of a well-defined index is also reflected, *inter alia*, by the use of diverse variants of the virus-to-bacterium ratio. These include both different spellings (without dashes, using a colon or the slash between words, etc.) as well as different terms such as the 'virus-to-bacteria quotient (VBQ)' (Bettarel *et al*., 2003) or the 'phage-to-bacteria ratio (PBR)' (Ogunseitan *et al*., 1990). The use of the term 'VBR' in initial microbial abundance studies was consistent with the prevailing view that prokaryotes were almost exclusively composed of heterotrophic bacteria. Initially, archaeal communities were thought to be typical of (or limited to) extreme environments; reports on the omnipresence of archaea in other environments became available only fairly recently (Chaban, Ng & Jarrell, 2006). This led to the introduction of terms such as 'virus-to-prokaryote ratio (VPR)' (De Corte *et al*., 2012) and 'virus-to-cell ratio' (Engelhardt *et al*., 2014; Pan *et al*., 2014). Herein, we suggest adoption of the term 'virus-to-prokaryote ratio' (VPR), as the most appropriate when considering the relative importance of bacterial and archaeal communities and the related virosphere, as compared to eukaryotic communities and their viruses [although use of 'prokaryote' to designate 'non-eukaryotes' remains controversial (Pace, 2006)]. The choice of term in any particular study, however, should be made according to the habitat and organisms involved.

While several interpretations have been proposed for the different results concerning the VPR, there has not yet been, to the best of our knowledge, a study investigating clearly the relevance of the VPR to viral ecology. Despite this, the VPR is commonly used to infer the importance, or absence, of viral processes within an ecosystem. The purpose of this review is to investigate the link between the VPR and the environment, and also its relationship to other microbial and viral variables. Data from 210 articles and five unpublished studies were used in our analysis. The results are presented in three parts: the first consists of a survey of the VPR and viral abundances in different environments. The second discusses, through meta-analyses, the relationship between the VPR and other microbial parameters. Lastly, two scenarios (corresponding to two models of contrasting habitat types) are proposed to illustrate our findings.

II. METHODS

(1) Data

Data from 210 articles and five unpublished studies were used in a meta-analysis. Articles were gathered using on-line databases (ScienceDirect, Wiley Online Library, Springer Link, PubMed and Google), using the key words 'virus-to-prokaryote ratio', 'virus-to-bacterium ratio', 'VPR', 'VBR' and 'viral abundance'. Interesting reports were also found within the references of publications dealing with viral ecology. Publications were chosen according to the data they contained with a priority on articles containing VPR values (184 out of the 210 publications and four out of five unpublished studies) and viral abundance data. Articles lacking information on VPR and viral numbers were discarded (e.g. reports with only prokaryotic abundance, but lacking data on viral numbers, etc.), as the focus was on the ratio between viruses and prokaryotes. When data of interest were not available in the analysed reports, authors were contacted for more details. When VPR values were not cited within a publication, they were calculated according to the viral and prokaryotic abundances provided therein. All data have been made available through 10.15454/1.4539792655245962E12 (Jacquet & Parikka, 2016).

Data were retrieved from each publication and information was listed for the individual sites studied; information from more than one site was obtained from some studies. For each site, details were recorded of its sampling location and of physical, chemical and biological variables. Sites were classified according to their environment (e.g. pelagic, sedimentary, soil), ecosystem type (e.g. marine/freshwater, saline, hot spring, etc.), habitat type (e.g. lake, coastal, deep sea, etc.) and their trophic status (eu-/meso-/oligotrophic), when possible.

(2) Conversions

Reported units were converted when necessary to enable meta-analyses. For analysis, categories 'highly eutrophic' and 'hypereutrophic' were taken as 'eutrophic'; 'meso/eutrophic' and 'oligo/mesotrophic' were taken as 'mesotrophic' and 'ultraoligotrophic' as 'oligotrophic'. Viral and prokaryotic abundance values expressed in the original papers using different units were analysed separately when conversion was not possible: cm^{-3} and ml^{-1} were considered equivalent, but values expressed as g^{-1} were analysed separately. For bacterial (or prokaryotic) production (PP), data were expressed in the original publications in three ways ($ml^{-1}\,h^{-1}$; $pmol\,l^{-1}\,h^{-1}$; $mgC\,ml^{-1}\,h^{-1}$) and these were analysed separately in our meta-analysis. Practical Salinity Unit (PSU) was considered as equivalent to parts-per-thousand (‰), as the accuracy of cited salinity in the analysed articles was inferior to the difference between the two methods of measurement.

When reported values were given as minima or maxima in the original publications (using < and >), the mathematical signs were removed.

(3) Meta-analyses

For all analyses, data from each studied site were considered as an independent sample. When a range of values was given for a single site, the reported mean value was used in analyses or, in the absence of this, the median of the range was used.

In Table 1, original minimum and maximum VPR values and abundances of viral particles and prokaryotic cells are given, together with mean values calculated from data for all sites.

Table 1. Virus-like particle (VLP) abundance, prokaryotic abundance and virus-to-prokaryote ratio (VPR) values in different ecosystems. N = number of sites

Environment	Ecosystem type	VLP abundance (VA) Mean (min–max)	Unit	N	Prokaryotic abundance (PA) Mean (min–max)	Unit	N	VPR Mean (min–max)	N
Pelagic	Marine	2.86×10^7 (3.00×10^3–7.92×10^8)	ml^{-1}	241	2.16×10^6 (0.00–6.90×10^7)	ml^{-1}	211	26.5 (0.0075–2150)	233
	Freshwater	7.00×10^7 (1.20×10^4–2.04×10^9)	ml^{-1}	223	1.33×10^7 (7.00×10^3–8.00×10^8)	ml^{-1}	202	17.2 (0.01–267.2)	229
	Saline	4.99×10^8 (5.26×10^4–7.90×10^9)	ml^{-1}	22	6.41×10^7 (3.90×10^4–3.40×10^8)	ml^{-1}	16	28.5 (0.2–144.8)	21
	Hot spring	5.62×10^6 (1.00×10^4–6.19×10^7)	ml^{-1}	46	7.42×10^5 (1.00×10^4–4.30×10^6)	ml^{-1}	46	9.1 (0.12–82.9)	46
	Ice	2.39×10^7 (1.00×10^4–1.50×10^8)	ml^{-1}	6	2.85×10^6 (4.60×10^4–1.00×10^7)	ml^{-1}	3	27.5 (0.7–119)	6
	Groundwater	9.67×10^5 (2.85×10^4–1.00×10^7)	ml^{-1}	11	2.29×10^5 (6.35×10^3–1.92×10^6)	ml^{-1}	11	5.9 (0.08–43)	11
	Aquatic snow	1.95×10^{10} (1.00×10^5–3.00×10^{11})	ml^{-1}	19	2.27×10^8 (8.00×10^4–9.60×10^9)	ml^{-1}	15	5.6 (0.01–26.9)	24
	Macrofaunal nests	1.39×10^9 (5.80×10^7–4.50×10^9)	ml^{-1}	3	6.93×10^3 (7.40×10^8–1.70×10^{10})	ml^{-1}	2	14.3 (0.06–36.5)	5
		8.70×10^9 (7.20×10^7–1.31×10^{10})	g^{-1}	3	3.16×10^8 (2.10×10^8–3.79×10^8)	g^{-1}	3		
Benthic (sedimentary)	Marine	8.77×10^9 (1.60×10^4–3.80×10^{11})	ml^{-1}	38	5.75×10^8 (1.80×10^5–3.95×10^9)	ml^{-1}	32	12.1 (0.001–225)	58
		1.28×10^9 (7.00×10^3–1.62×10^{10})	g^{-1}	23	2.39×10^8 (3.00×10^5–1.13×10^9)	g^{-1}	20		
	Freshwater	6.87×10^9 (8.79×10^6–2.20×10^{11})	ml^{-1}	33	9.33×10^8 (1.00×10^7–7.10×10^9)	ml^{-1}	31	9.2 (0.03–67)	38
		1.06×10^{10} (4.71×10^6–4.01×10^{10})	g^{-1}	2	2.54×10^9 (1.85×10^7–1.28×10^{10})	g^{-1}	2		
	Saline/hot spring	1.07×10^8 (6.00×10^4–5.62×10^8)	ml^{-1}	6	1.10×10^7 (8.90×10^3–6.17×10^7)	ml^{-1}	6	7.6 (0.002–17.5)	13
		1.46×10^9 (1.80×10^4–6.86×10^9)	g^{-1}	7	4.45×10^8 (3.20×10^5–2.67×10^9)	g^{-1}	7		
Soil		1.13×10^9 (8.53×10^6–4.17×10^9)	g^{-1}	17	1.60×10^9 (3.50×10^4–4.50×10^9)	g^{-1}	15	704.4 (0.002–8200)	15

All data were tested for normality using the Shapiro–Wilks test. Non-parametric Spearman's rank correlation coefficients were calculated to assess relationships between VPR and other biological and environmental variables. For comparisons of biological variables among trophic levels, a non-parametric Mann–Whitney U-test was used.

III. VIRAL ABUNDANCE AND VPR VALUE DISTRIBUTIONS IN DIFFERENT ECOSYSTEMS

Estimates of viral abundances of 1.2×10^{30} particles in the open ocean, 2.6×10^{30} in soils, 3.5×10^{30} in the oceanic sub-surface and between 0.25 and 2.5×10^{31} in the terrestrial subsurface (Whitman, Coleman & Wiebe, 1998; Mokili, Rohwer & Dutilh, 2012) have been reported, giving a total of 10^{31}–10^{32} particles for the whole virosphere (Krupovic & Bamford, 2008). These estimates are based on the supposition that viruses outnumber their prokaryotic hosts by roughly an order of magnitude (Wommack & Colwell, 2000). However, our review of 210 articles clearly indicates that the numerical dominance of VLPs compared to prokaryotes is highly heterogeneous. Our analysis (Table 1) reveals a wide range of VPR values from 0.001 (Yanagawa *et al*., 2014) to 8200 (Williamson *et al*., 2007), thus varying over six orders of magnitude. Mean VPR values for a variety of ecosystems, on the other hand, vary between 5.6 and 28.5 (Table 1, Fig. 1), with the exception of the soil ecosystem, which has an exceptionally high mean VPR of 704.

(1) Pelagic ecosystems

(a) *Marine and freshwater*

Most studies on natural viral abundance have been conducted in the water column of aquatic (and more specifically pelagic) ecosystems (Table 1). Previous reports describe VLP abundances ranging from scarcely detectable ($<10^4$ VLP ml^{-1}) to over 10^8 VLP ml^{-1} (Wommack & Colwell, 2000; Jacquet *et al*., 2010), and exhibit general trends such as a decrease in marine viral abundance along a transect from coastal environments to offshore waters and through the water column from the surface to the deep sea (Paul & Kellogg, 2000; Weinbauer, 2004; Sime-Ngando & Colombet, 2009). Viral abundance in freshwater ecosystems also tends to decrease along the water column from the euphotic zone to the deeper water layers, although exceptions have been reported such as in monomictic lakes (Jacquet *et al*., 2010). These trends have been attributed to the productivity of a given ecosystem influencing viral numbers through the hosts' metabolic state (Weinbauer, Fuks & Peduzzi, 1993; Maranger & Bird, 1995; Wommack & Colwell, 2000; Wilhelm & Matteson, 2008; Jacquet *et al*., 2010). Freshwater ecosystems generally harbour higher viral abundances than marine ecosystems (Wilhelm & Matteson, 2008), although little difference has been observed in viral production between these two ecosystems (Weinbauer, 2004).

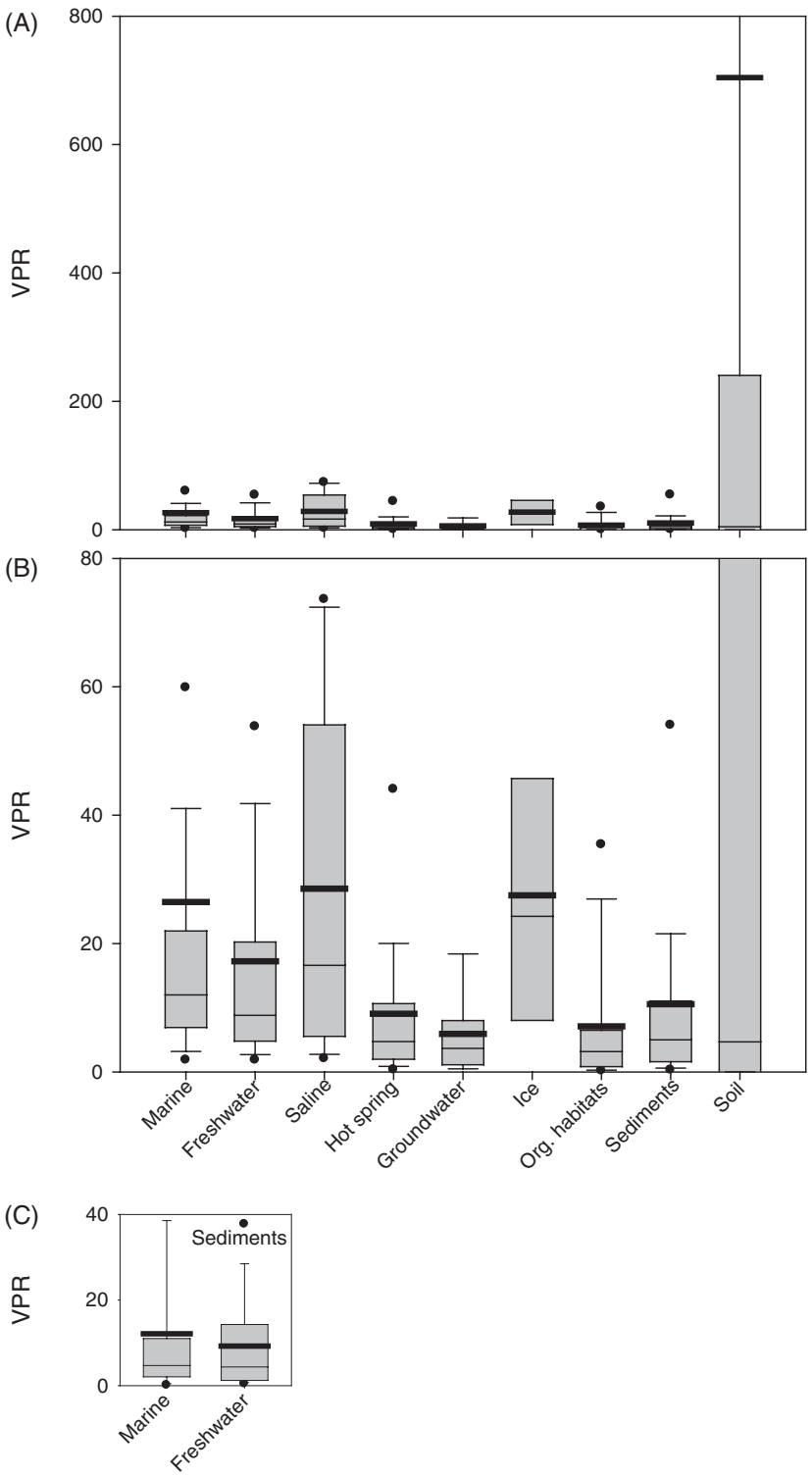

Fig. 1. Box plot representations for the virus-to-prokaryote ratio (VPR) obtained in different types of aquatic environments and soil. Panel (B) is an enlargement of (A) allowing clearer visualization of the percentiles. The boxed region shows the 25th and the 75th percentiles and the whiskers show the 5–95% percentiles; the thin horizontal line is the median and the thick horizontal line the mean. Outliers are shown as circles. Panel (C) separates data from marine and freshwater sediments. In (A) and (B), N (number of sites) = 233, 229, 20, 46, 11, 6, 29 and 96 for marine, freshwater, saline, hot spring, groundwater, ice, organic habitats (i.e. aquatic snow and macrofaunal nests) and sediments, respectively. In (C), $N = 58$ and 38 for marine and freshwater sediments, respectively. See Table 3 for results of Mann-Whitney tests between ecosystem types.

Table 2. Virus–like particle (VLP) abundance, prokaryotic abundance and virus-to-prokaryote ratio (VPR) values in aquatic habitats. N = number of sites

Environment	Habitat type	VLP abundance (ml^{-1})		Prokaryotic abundance (ml^{-1})		VPR	
		Mean	N	Mean	N	Mean	N
Marine	Coastal	3.96×10^7	97	2.67×10^6	80	20.72	98
	Estuary	6.56×10^7	21	6.88×10^6	19	11.35	19
	Open ocean/offshore	9.68×10^6	101	7.75×10^5	93	38.07	97
	Deep sea (>1000 m)	1.59×10^6	28	1.59×10^5	27	28.45	28
Freshwater	Waterflow/river	5.56×10^7	17	4.85×10^6	16	9.38	16
	Reservoir/dam	2.82×10^7	11	3.22×10^6	5	10.17	10
	Floodplain/oxbow lake	3.52×10^7	10	2.99×10^6	10	11.50	16
	Cryoconite/meltwater	4.12×10^6	4	2.63×10^5	3	16.66	4
	Lake/pond	7.46×10^7	179	1.42×10^7	166	18.84	181

Aquatic marine and freshwater ecosystems are characterized by VPR values ranging between 0.008 (Proctor & Fuhrman, 1990) and 2150 (Clasen *et al*., 2008) (Table 1), giving an overall average of 21.9. The apparent trend, based on average viral and prokaryotic abundances (Table 2), suggests higher mean VPR values in the open ocean and offshore, as well as in deep-sea waters, when compared to coastal and estuarine waters, as reported in previous reviews. This might appear surprising as higher viral numbers might be expected to give rise to higher VPR values, but is presumably due to differences in prokaryotic abundance. As coastal and estuary waters are more productive than offshore waters, high viral abundances will be accompanied by disproportionately high prokaryotic numbers, resulting in a lower VPR. Another potential explanation for the lower VPR is higher viral loss in coastal and estuarine waters compared to the open ocean. Viral loss can result, among other things, from virivory (i.e. grazing by nanoflagellates) (Gonzáles & Suttle, 1993; Bettarel *et al*., 2005), adsorption to particulate matter (Hewson & Fuhrman, 2003), temperature (Garza & Suttle, 1998; Bettarel, Bouvier & Bouvy, 2009) and degradation by heat-labile organic matter (e.g. enzymes) (Noble & Fuhrman, 1997). It is thus conceivable that in eutrophic and turbid environments viral loss is higher compared to the open ocean, resulting in lower VPR values. Viral decay is also likely to be lower in deep-sea waters due to diminished effects of temperature and sunlight (Parada *et al*., 2007).

Minimum VPR values reported for freshwater and marine habitats are similar, but the maximum reported VPR values in marine systems are one order of magnitude greater than those of freshwater systems (Table 1; Fig. 1). Although VPR values vary over six orders of magnitude within aquatic ecosystems, most reported values fall between 1 and 50. Previous authors have reported higher average mean VPR values for freshwater habitats as compared to marine habitats (20 *versus* 10, respectively) (Maranger & Bird, 1995; Weinbauer, 2004), but our literature survey suggests the opposite trend with a significantly higher mean VPR of 26.5 for marine and 17.2 for freshwater habitats ($P = 0.009$, $N = 233$ and 229, respectively) (Tables 2 and 3). These values suggest that freshwater habitats have relatively higher prokaryotic numbers compared to marine ecosystems, presumably due to higher prokaryotic production and/or higher viral loss. A higher photosynthetic biomass in freshwater than in marine habitats (Maranger & Bird, 1995; Clasen *et al*., 2008) makes it possible that prokaryotic counts are affected by autotrophic cellular abundance, causing a lower VPR. Freshwater habitats are also more impacted by human activities that introduce substances from terrestrial environments, such as chemicals and clay, which could increase the removal of viral particles (Clasen *et al*., 2008) and hence increase viral loss in freshwater systems.

(*b*) *Extreme environments*

Following the development of microbial ecology in recent decades, new possible niches have been explored, such as so-called 'extreme environments'. Although organisms from all three domains of life are found in extreme environments, bacteria and especially archaea are particularly abundant in the harshest environments. Viral numbers from undetectable levels up to 10^9 particles ml^{-1} have been documented (Le Romancer *et al*., 2007) possibly mainly comprising archaeoviruses (Prangishvili, Forterre & Garrett, 2006). Although modern technology means that increasing amounts of data are available on viruses in these environments, still little remains known about their ecology. Extreme environments comprise highly diverse habitats, from terrestrial hot springs, salterns or alkaline lakes to deep-sea hydrothermal vents, deep subsurface sediments or polar inland waters and sea-ice. Lakes and sea-ice have been studied in both Polar Regions of the earth. Although these habitats share low temperatures, highly diverse ecosystems can be found, including freshwater, brackish, saline and hypersaline lakes, as well as brine, fast-ice and sea-ice. Less-common water features are cryolakes and epishelf lakes, the latter being almost unique to Antarctica where only 2% of the continent is ice-free (Laybourn-Parry *et al*., 2013). Ice remains a poorly explored ecosystem type. Within the 'ice' habitats, the best studied are sea-ice (Maranger *et al*., 1994; Gowing *et al*., 2004), fast-ice (Paterson & Laybourn-Parry, 2012), oligotrophic glacier ice and cryoconite hole water

Table 3. Comparisons of means of virus-to-prokaryote ratio (VPR) values in different ecosystems. Significant ($P < 0.05$) values (Mann-Whitney test) are in bold. $N = 233$, 229, 20, 46, and 11 for aquatic marine, freshwater, saline, hot spring and groundwater, respectively. $N = 58$, 38, 13, 15 and 29 for sedimentary marine, freshwater, extreme (hot spring/saline) and soil, respectively. $N = 6$ and 29 for ice and organic habitats (i.e. aquatic snow and macrofaunal nests), respectively. aq., aquatic; hab., habitat; sed., sediment

VPR	Marine (aq.)	Freshwater (aq.)	Saline	Hot spring	Groundwater	Marine (sed.)	Freshwater (sed.)	Extreme (sed.)	Soil	Ice	Organic hab.	All sed.
Marine (aq.)	—	**0.0089**	0.24	**<0.001**	**0.0013**	**0.0012**	**0.037**	**0.0077**	0.13	0.32	**<0.001**	
Freshwater (aq.)		—	0.055	0.083	**0.01**	**<0.001**	**<0.001**	0.14	0.29	0.096	**0.0076**	
Saline			—	**<0.001**	**0.005**	**0.002**	**<0.001**	**0.03**	0.22	0.84	0.036	
Hot spring				—	0.77	0.77	0.77	0.77	0.77	0.77	0.77	
Groundwater					—	0.58	0.79	0.36	0.98	**0.010**	0.57	
Marine (sed.)						—	0.80	0.50	0.97	**0.021**	0.12	
Freshwater (sed.)							—	0.48	0.79	**0.013**	0.27	
Extreme (sed.)								—	0.78	**0.014**	0.082	
Soil									—	**0.33**	0.77	
Ice										—	**<0.001**	
Organic hab.											—	
All aq.												**<0.001**

(Anesio *et al*., 2007; Säwström *et al*., 2007). Recorded viral abundances in polar environments vary between 2.0×10^4 particles ml^{-1} in a freshwater lake in Antarctica (Säwström *et al*., 2008) and 1.3×10^8 particles ml^{-1} in Arctic sea ice (Maranger *et al*., 1994). Similarly high viral abundances (1.2×10^8 particles ml^{-1}) have been reported from the brackish Pendant Lake of Antarctica (Madan, Marshall & Laybourn-Parry, 2005). VPR values vary over three orders of magnitude in ice, from 0.7 to 119 (Gowing *et al*., 2004), and the mean value of 27.5 (Table 1, Fig. 1) suggests that local microbial communities are active and probably trapped (e.g. in gas bubbles) in these habitats. Despite their isolation, a substantial proportion of data on the viral ecology of brackish and saline lakes seems to derive from the Polar Regions. Few studies have provided data on the abundance of viruses and their hosts in (hyper)saline lakes, although viral numbers appear to include the highest observed in aquatic systems in general (Le Romancer *et al*., 2007), reaching abundances beyond 10^9 particles ml^{-1} (Brum *et al*., 2005; Bettarel *et al*., 2011; Peduzzi *et al*., 2014).

At the other end of the temperature scale lie the extreme thermal environments populated by 'thermophiles'. These environments share a high temperature, but are otherwise characterized by highly diverse physical and chemical characteristics (e.g. acidic or alkaline pH, various pressure conditions, etc.). Natural habitats include terrestrial geothermal springs, mud volcanoes and fumaroles, but also marine habitats such as deep-sea hydrothermal vents, submarine volcanoes and hot sediments. Moderately thermophilic eukaryotes can be found in these environments, but as the observed upper temperature limit for eukaryotes is about 60°C (Brock, 1985; Rothschild & Mancinelli, 2001), most of the organisms that populate these ecosystems are (hyper)thermophilic bacteria and archaea (the temperature limits of the archaeal communities extend beyond the observed upper limit for bacteria). Although the exploration of extreme thermal environments has revealed remarkable viral diversity, mostly associated with the hyperthermophilic Crenarchaeota phylum of Archaea (Prangishvili *et al*., 2006; Ackermann & Prangishvili, 2012), there is surprisingly little understanding of the contribution of viral processes to microbial dynamics in these ecosystems. Early studies of viral abundance in terrestrial hot spring samples reported very low viral abundances; while Rice *et al*. (2001) failed to find viral particles by direct filtration of raw hot spring samples, Zillig *et al*. (1994) had to concentrate their raw samples by 10^4 times in order to detect VLPs using transmission electron microcopy (TEM) (Prangishvili, 2006). In marine hydrothermal vents, an early study reported viral abundances of $\sim 10^4$–10^5 VLPs ml^{-1} (Juniper *et al*., 1998). Although most available data indicate low viral abundances in both marine and terrestrial hot springs, abundances up to 10^7 particles ml^{-1} in terrestrial (Chiura *et al*., 2002) and 10^6 particles ml^{-1} in marine (Wommack *et al*., 2004; Ortmann & Suttle, 2005; Williamson *et al*., 2008; Yoshida-Takashima *et al*., 2012) hot springs have been documented.

In thermal ecosystems, most VPR values vary between 1 and 20 (Fig. 1). For saline ecosystems most reported VPR values are between 1 and 100. Although VPR values from more extreme aquatic ecosystems are derived from fewer sites (Table 1), some trends can be identified. The lower mean VPR in hot springs ($P < 0.001$ except for freshwater; Table 3) (Fig. 1) suggests that there are fewer viral particles per host cell than in temperate (i.e. 'mesothermal', *ca*. 10–45°C) systems. The lower number of viral particles in hot springs (Prangishvili, 2006) could be attributed to possible instability of virions at high temperatures (Rice *et al*., 2001; Ortmann *et al*., 2006; Prangishvili *et al*., 2006; Fulton, Douglas & Young, 2009). Another observation is the apparent prevalence of lysogeny and poorly productive chronic infection, typical for, but not exclusive to, thermophilic viruses of archaea (Prangishvili & Garrett, 2005; Prangishvili *et al*., 2006; Pina *et al*., 2011). The higher reported VPR values in saline could be attributed to rather high abundances of viral particles in salines – the

mean for 22 studied sites was 4.99×10^8 particles ml^{-1} – and the apparent tendency of haloviruses to form persistent infections and have lytic lifestyles rather than lysogenic ones (Porter, Russ & Dyall-Smith, 2007; Santos *et al*., 2010; Sabet, 2012). Virions of haloviruses are reported to be stable at high salinity (Witte *et al*., 1997; Pietilä *et al*., 2012, 2013), which would contribute to the persistence of viral particles in these habitats, potentially leading to high VPR values. Furthermore, our knowledge on grazing pressure in these habitats is scarce, with documentation of eukaryotic microbial diversity in salines remaining poor. This deficit is shared by high-temperature habitats, although grazing is probably non-existent at temperatures that exceed the known limit for eukaryotic life (Breitbart *et al*., 2004); diminished or absence of grazing will impact VPR values. Thus, further information on viral decay in these two types of extreme habitats could provide insights into the reasons behind the differences in their VPR values.

(*c*) *Aquifers*

Recently, interest has arisen into the microbial viruses of groundwater and aquifers. These aquatic ecosystems are characterized by more neutral pH values and mesothermal temperatures than the extreme environments discussed above, but have remained relatively unexplored, probably due to their limited accessibility. According to initial reports, these habitats harbour low viral abundances, ranging between 2.9×10^4 (Wilhartitz *et al*., 2013) and 10^7 particles ml^{-1} (Kyle *et al*., 2008) and have low VPR values (mean 5.9, Table 1). The reasons for these low microbial and viral numbers are yet to be elucidated, but may be linked to the oligotrophic nature of groundwater (Wilhartitz *et al*., 2013). Data from Australian confined and unconfined aquifers suggest, nonetheless, small-scale heterogeneity in microbial communities, both prokaryotic and viral, which in turn suggests active microbial dynamics rather than inactive homogeneous suspensions of cells (Roudnew *et al*., 2013, 2014).

(2) Benthic ecosystems and soil

(*a*) *Benthos*

In addition to pelagic aquatic environments, increasing numbers of studies have been carried out during the past 15 years on the viriobenthos, exploring the viral ecology of freshwater and marine sedimentary ecosystems. In these environments, viral abundances have previously been reported to range from 10^7 to 10^{10} VLP g^{-1} of dry sediment, being 10–1000 times higher than viral abundances in the water column (Danovaro *et al*., 2008*a*). Viral numbers in sediments, with pore water included, between 10^4 and 10^{11} VLP ml^{-1} have been reported (Duhamel & Jacquet, 2006; Pinto *et al*., 2013). Similar general trends as in water columns can also be observed in the viriobenthos. Freshwater and low-salinity coastal sediments usually harbour the highest viral numbers, which decrease with depth of the water column (Danovaro *et al*., 2008*a*) and sediment layer (Danovaro & Serresi, 2000; Bird *et al*., 2001; Hewson *et al*., 2001; Middelboe & Glud, 2006; Borrel *et al*., 2012). Evidence also suggests a decrease in viral abundance along a decreasing trophic gradient (Weinbauer *et al*., 1993; Danovaro & Serresi, 2000; Hewson *et al*., 2001; Danovaro, Manini & Dell'Anno, 2002), although not all data support this positive correlation (Danovaro *et al*., 2008*a*). Our analysis showed significantly higher viral abundances in eutrophic and mesotrophic habitats when compared to oligotrophic ecosystems ($P < 0.001$; Table 4). Viruses have been detected in deep subsurface sediments, hundreds of meters below the seafloor (Bird *et al*., 2001; Middelboe, Glud & Filippini, 2011), reaching abundances up to 10^9 particles cm^{-3} (Engelhardt *et al*., 2014).

Out of all ecosystems, the lowest reported VPR value of 0.001 was found in deep marine sediments, 200 m below the sea floor (Yanagawa *et al*., 2014). The maximum VPR value for marine sediments was 225 (Engelhardt *et al*., 2014), with a mean of 12.1 (Table 1). Freshwater sediments, by contrast, have a VPR range from 0.03 (Farnell-Jackson & Ward, 2003) to 67 (Hewson *et al*., 2001). Marine and freshwater sediments show similar distributions of VPR values (Fig. 1C). The range of recorded VPR values is larger in marine than freshwater sediments (Table 1). VPR values in sediments are generally lower than those found in aquatic ecosystems (Table 3) implying that pelagic environments harbour higher ratios than benthic habitats. This is not due to a lower abundance of viruses as there are on average 10^9–10^{10} particles ml^{-1} or g^{-1} in both freshwater and marine sediments. The lower VPR values must therefore be the result of higher abundances of prokaryotes. This may be due to high prokaryotic production (e.g. of phage-resistant strains) and/or high viral decay rates. Low VPR values may also imply a steady state (e.g. dormant communities), in which low prokaryotic activity may be related to high metabolic costs due to phage activity (Thingstad *et al*., 2014). The low metabolic activity of phage-infected hosts would then favour the growth of strains unrecognized by phages, lowering the VPR. Similar patterns in marine and freshwater sediments imply that the constraints experienced by microbial communities in these ecosystems are similar irrespective of the nature of the physicochemical parameters of the aquatic fraction above the sediments. Viral life strategy, particularly the occurrence of lysogeny, within sedimentary and soil ecosystems is also likely to have an impact on both the microbial communities and viral production (Mei & Danovaro, 2004; Williamson, 2011).

VPR values of sediments from saline and high-temperature environments do not exceed 17.5, with a mean of 7.6 (Table 1). These sediments from extreme environments represent an unusual ecological niche: it is possible that there is an absence of protists and thus of grazing pressure. This also may be the case in deep sediments, due to the high pressure and lack of oxygen (Engelhardt *et al*., 2011). Physicochemical variables prevalent in geothermal sediments are also likely to be inhospitable to eukaryotic life throughout the whole sediment column, making these vast habitats another type

Table 4. Virus-to-prokaryote ratio, viral and prokaryotic abundance and production and the concentration of chlorophyll *a* in different trophic conditions. The significant ($P < 0.05$) results of Mann-Whitney test between trophic categories are in bold. Where *P*-value is missing, the analysis was not performed due to insufficient data ($N < 5$) of the analysed trophic categories. Chl *a*, chlorophyll *a*; Eu, eutrophic; Meso, mesotrophic; Oligo, Oligotrophic; PA, prokaryotic abundance; PP, prokaryotic production; VA, viral abundance; VP, viral production; VPR, virus-to prokaryote ratio. *N* = number of sites

Aquatic	Eutrophic Mean (min–max)	Mesotrophic Mean (min–max)	Oligotrophic Mean (min–max)	Eu–Meso *P*	Meso–Oligo *P*	Eu–Oligo *P*	*N*
VPR	15.48 (0.26–74.68)	22.68 (2.4–71.05)	43.01 (0.15–897.3)	**0.034**	0.35	0.39	183
VA (ml^{-1})	19.22×10^7 (3.00×10^5–1.30×10^9)	7.34×10^7 (5.40×10^6–1.00×10^9)	2.27×10^7 (5.00×10^4–2.20×10^8)	0.55	**<0.001**	**<0.001**	129
PA (ml^{-1})	8.39×10^6 (1.61×10^5–1.34×10^8)	4.14×10^6 (6.40×10^5–2.57×10^7)	4.23×10^6 (7.00×10^3–1.10×10^8)	**0.001**	**<0.001**	**<0.001**	140
VP (ml^{-1} h^{-1})	4.38×10^5 (6.70×10^3–1.27×10^6)	4.81×10^6 (6.25×10^4–1.33×10^7)	1.66×10^5 (5.93×10^3–5.13×10^5)	—	—	0.097	23
PP (ml^{-1} h^{-1})	2.69×10^5 (3.60×10^4–1.37×10^6)	4.54×10^4 (1.26×10^4–9.70×10^4)	2.33×10^5 (5.19×10^{-5}–1.80×10^6)	—	—	**0.005**	23
[Chl *a*] ($\mu g\,l^{-1}$)	30.82 (0.30–196.35)	7.38 (1.60–24.32)	2.55 (0.06–43.83)	**0.003**	**<0.001**	**<0.001**	96
Sediment							
VPR	7.41 (0.60–51.00)	13.12 (0.22–36.98)	23.40 (0.90–98.00)	0.011	0.57	0.48	44
VA (cm^{-3})	7.75×10^9 (1.50×10^8–1.10×10^{11})	4.88×10^9 (2.10×10^8–9.50×10^9)	1.02×10^9 (2.00×10^7–2.50×10^9)	**<0.001**	—	—	41
PA (cm^{-3})	1.22×10^9 (1.96×10^7–6.79×10^9)	6.56×10^8 (2.25×10^6–3.40×10^9)	–	0.17	—	—	33

of ecosystem where grazing is absent and viruses are solely responsible for prokaryote mortality.

(*b*) *Soil*

Only a few studies have been conducted on the viral ecology of soils (Williamson *et al*., 2005; Kimura *et al*., 2008). The abundance of viruses varies within three orders of magnitude (Table 1), between *ca.* 10^6 and 10^9 particles g^{-1} dry weight (gdw), although usually varying between 10^8 and 10^9 VLP gdw^{-1}, the lowest estimates having been obtained from deserts (Williamson, 2011). As in other ecosystems, viral abundance is dependent on prokaryotic abundance (Williamson, 2011) and the lack of data on viral processes in these habitats reflects a lack of knowledge on microbial processes that sustain soil ecosystems (Srinivasiah *et al*., 2008). Physical and chemical properties of soils (e.g. soil moisture content, pH values and temperature) all influence viral abundance; high values are likely to be found in soils with high prokaryotic abundances as well as with high clay and organic matter contents (Kimura *et al*., 2008; Williamson, 2011). The reported VPR range in soil ecosystems is immense, spanning from 0.002 (Ashelford, Day & Fry, 2003) to 8200 (Williamson *et al*., 2007). This extreme range raises several questions regarding our knowledge of soil viral ecology. These include methodological issues related to viral and prokaryotic extraction efficiency (Williamson *et al*., 2005, 2007; Swanson *et al*., 2009; Williamson, 2011). If the reported viral numbers are genuine, it has been proposed that very high and very low local viral abundances could be related to an imbalance between viral production and decay (Williamson, 2011). High VPR values could be due to a low rate of decay, as in Antarctic soils (Williamson *et al*., 2007). The low VPR values could result from high prokaryotic proliferation as compared to viruses, whose decay or decline in infectivity can be influenced by chemical, physical and/or biological parameters of the soil (Swanson *et al*., 2009).

(3) Aquatic snow and nests of macrofauna

While most studies have investigated viral processes in pelagic or benthic environments, interest in viral dynamics and their involvement in nutrient cycles has led some researchers to explore viral particle distribution in suspended organic and inorganic matter. As for extreme environments, the available information on viruses in aggregates is scarce, especially in river systems (Peduzzi & Luef, 2008). High viral numbers have been recorded on aggregates, ranging from 10^5 to 3×10^{11} particles ml^{-1} (Weinbauer *et al*., 2009; Peduzzi, 2015). Viral-mediated cellular lysis leads to the accumulation of particulate organic matter (POM), dissolved organic matter (DOM) and colloids and contributes to the formation of aggregates (Wilhelm & Suttle, 1999; Weinbauer, Chen & Wilhelm, 2011). At the larger end of the size range of non-living organic matter is so-called marine, lake or river 'snow' (Suzuki & Kato, 1953; Grossart & Simon, 1993). Microscopic and macroscopic aggregates are composed of various materials, both organic and inorganic, their

composition being dependent on the specific environment and conditions. Although inorganic compounds, such as calcite and clay, are not usually colonized by prokaryotes (Simon *et al*., 2002), they could contribute to the sinking of viral particles in the water column (Brussaard, 2004; Clasen *et al*., 2008).

Aquatic aggregates (i.e. aquatic snow, inorganic and organic material such as mucilage, cobwebs, leaves, wood, etc.) generally have lower VPR values than the surrounding aquatic environment (Tables 1 and 3, Fig. 1). This might give the impression of a diluted microbial niche, when in fact individual particles are probably microbial 'hot spots' (Simon *et al*., 2002), with high viral and prokaryotic abundances. The particles are colonized by prokaryotic communities (on average 2.27×10^8 cells ml^{-1}), which will reduce VPR values. Aggregates appear to act as scavengers rather than factories of viral particles (Weinbauer *et al*., 2009), which could also partly explain their lower VPR values. Viral and microbial numbers within aggregates are influenced by water residence time (Mari, Kerros & Weinbauer, 2007) and their role in particle scavenging and distribution is dependent on the type of surrounding environment. Aggregates likely alter the microbial ecology of their surrounds by displacing viral particles, impacting local microbial communities by removing/adding pressure by viral lysis (Weinbauer *et al*., 2009, 2011). By contrast, Mari *et al*. (2007) suggested that aggregates could constitute 'hot spots' of viral infection that elevate local viral abundance, due to reduced distances between viruses and prokaryotic cells (especially in smaller aggregates). A study involving bacterial immobilization on artificial gel beads *in vitro* showed that bacterial colonies within beads released cells into the surrounding medium (Cinquin *et al*., 2004) acting as cell factories. Organic and inorganic aggregates could act as natural beads, distributing prokaryotic cells and elevating local microbial abundance. However, little is known of virus–host interactions within aggregates.

Several studies have focused on the microbial content of water related to aquatic fauna (Table 1). Whereas some studies have been conducted on seawater found in the proximity of coral reefs (Paul *et al*., 1993; Seymour *et al*., 2005; Patten, Seymour & Mitchell, 2006; Patten *et al*., 2008; Kellogg, 2010; Yoshida-Takashima *et al*., 2012), few have focused on sampling nests of macrofauna. Water within polychaete and galatheid colonies as well as associated symbiotic microbial mats was analysed by Yoshida-Takashima *et al*. (2012). VPR values were globally low (<1). VPR values from sectioned layers of living organisms (galatheid crabs) were several orders of magnitude lower than those of water derived from their nests. The nests hosted symbiotic microbial mats, which were also analysed. The attached prokaryotic communities had lower VPR values compared to those in planktonic communities surrounding the nests. Possible reasons for this included higher frequency of lysogeny, lower frequency of virus-infected cells and/or lower viral production rates in attached prokaryotes. In another report, the VPR from symbiotic microbial communities in the nests of mussels was found to be an order of magnitude higher than those found in polychaete and galatheid colonies (Kellogg, 2010).

IV. THE RELATIONSHIPS OF VPR TO MICROBIAL AND VIRAL VARIABLES

We used meta-analysis to investigate the relationship between several microbial and viral parameters with VPR (Table 5). VPR was strongly positively correlated with viral abundance (VA) in aquatic habitats and sediment-related ecosystems, and with frequency of lysogenic cells (FLC). VPR values are also positively correlated with frequency of visibly infected cells (FVIC), burst size (BS), and chlorophyll *a* (Chl *a*) concentration. By contrast, no significant relationships were detected between VPR and viral production (VP), fraction of mortality from viral lysis (FMVL), viral decay rates (VDR), viral turnover or depth. VPR was negatively correlated with prokaryotic abundance (PA) in sediments and soil (but only shows a negative trend in aquatic ecosystems), prokaryotic production (PP), virus–host contact rates (VCR), salinity and temperature. Section VIII proposes a list of abbreviations used herein, with definitions provided where necessary.

Since VPR is the ratio of VLP to prokaryotic abundance, the value that VPR takes will be related to factors controlling both viral and prokaryotic abundance: viral production and decay, and prokaryotic production, mortality and infection rates (Williamson, 2011). The relationship between VPR and environmental variables, however, is less intuitive and the interpretations proposed below are derived from current understanding of viral and microbial processes in specific habitats.

Some data on microbial parameters were too scarce ($N < 5$) to study their relationship with VPR. Grazing rate (GR) on prokaryotes will affect prokaryotic abundance, and hence the number of available hosts for viruses. *A priori*, top-down control of prokaryotic grazing by heterotrophic nanoflagellates would increase the VPR by diminishing prokaryotic abundance, although in reality the effect of grazing is probably more complex (Miki & Jacquet, 2008). Grazing might favour some prokaryotic species/strains, which in turn could favour infection by specific viral strains. By contrast, grazing on viral particles will increase viral loss, and thus diminish viral-mediated reductions in prokaryotic abundance. Other uninvestigated variables include prokaryotic turnover rates, cell size (which will affect contact rates), sediment porosity and nutrient concentrations (e.g. dissolved/particulate organic carbon, phosphate, etc.). Future studies should investigate their effects on the VPR.

(1) Factors enhancing VPR

A positive correlation was found between VPR and VA in both aquatic ($P < 0.001$) habitats and sedimentary ($P < 0.001$) ecosystems (Table 5). An increase in viral abundance will obviously increase VPR if prokaryotic abundance remains constant as it is used in VPR calculation

Table 5. Spearman's rank correlations between virus-to-prokaryote ratio (VPR) or viral abundance (VA) and other variables (across all ecosystems). Significant correlations ($P < 0.05$) are in bold. BS, burst size; Chl *a*, chlorophyll *a*; FLC, frequency of lysogenic cells; FMVL, fraction of mortality from viral lysis; FVIC, frequency of visibly infected cells; PA, prokaryotic abundance; PP, prokaryotic production; VCR, virus–host contact rates; VDR, viral decay rate; VP, viral production. N = number of sites

	VPR		
	r	*P*	*N*
VA (ml^{-1})[a]	**0.24**	**<0.001**	571
VA (g^{-1})[b]	**0.6**	**<0.001**	48
PA (ml^{-1})[a]	−0.074	0.087	526
PA (g^{-1})[b]	**−0.32**	**0.028**	48
VP (ml^{-1} h^{-1})	−0.093	0.39	86
PP (ml^{-1} h^{-1})	**−0.31**	**0.0058**	77
PP (pmol l^{-1} h^{-1})	**−0.25**	**0.0012**	171
PP (mgC ml^{-1} h^{-1})	**−0.45**	**<0.001**	59
FVIC (%)	**0.38**	**<0.001**	79
FLC (%)	**0.58**	**<0.001**	69
FMVL (%)	0.082	0.53	61
BS	**0.19**	**0.045**	116
VCR (l^{-1} day^{-1})	**−0.63**	**<0.001**	32
VDR (h^{-1})	0.39	0.21	12
Viral turnover (day)	−0.91	0.22	42
[Chl *a*]	**0.29**	**<0.001**	200
Salinity (PSU)	**−0.27**	**0.002**	136
Temperature (°C)	**−0.20**	**<0.001**	305
Depth (m)	0.034	0.44	505
	VA (ml^{-1})		
	r	*P*	*N*
PA (ml^{-1})[a]	**0.86**	**<0.001**	524
[Chl *a*][a]	**0.37**	**<0.001**	183
	VA (g^{-1})		
	r	*P*	*N*
PA (g^{-1})[b]	**0.36**	**0.013**	47

[a]Analyses performed only with data from aquatic ecosystems.
[b]Analyses performed only with data from sediments and soil.

(VPR = VA/PA). Factors contributing to elevated viral abundance are viral production (VP), burst size (BS), frequency of visibly infected cells (FVIC) and fraction of mortality from viral lysis (FMVL). In addition, elevated virus–host contact rates (VCR), short latent period (LP) and decreased viral decay rates (VDR) will enhance viral production and the persistence of free viral particles in the environment.

Whereas a positive correlation between VPR and both BS ($P < 0.05$) and FVIC ($P < 0.001$) was confirmed by our meta-analyses, it was surprising that no correlation was found between VPR and viral production or FMVL (Table 5). Several different methods are used for quantifying viral production including: radiolabelled thymidine or ^{32}P incorporation (Steward *et al*., 1992); estimation from decay rates (Heldal & Bratbak, 1991); fluorescent labelling of viral tracers (Noble & Fuhrman, 2000); estimation from virus-induced bacterial mortality [VIBM (=FMVL)], BS and prokaryotic production (Weinbauer & Höfle, 1998); the dilution method/reduction approach (Weinbauer & Suttle, 1996; Wilhelm, Brigden & Suttle, 2002); tangential flow diafiltration (Winget *et al*., 2005); direct enumeration of viral particles over time (Hewson & Fuhrman, 2003); and using Würgler bags (Hansen, Thamdrup & Jørgensen, 2000) in anoxic conditions (Glud & Middelboe, 2004). Many of these methods have drawbacks, although the reduction approach appears to be the most reliable (Wilhelm *et al*., 2002; Helton *et al*., 2005; Weinbauer, Rowe & Wilhelm, 2010). These methods either rely on inferring viral production from other variables (e.g. FMVL, BS and prokaryotic production) or on sample processing, potentially leading to inaccurate estimates. Use of mean BS and conversion factors, such as in Weinbauer, Winter & Höfle (2002), gives viral production estimates that may be subject to error. It is possible that this diversity of techniques is reflected by the lack of correlation between VPR and viral production in our meta-analysis. An alternative possibility is that increased viral production is always associated with a high net prokaryotic production generating high prokaryotic abundance and counteracting any effect of viral production on the VPR.

Another surprising finding was the absence of a correlation between VPR and FMVL. FMVL reduces prokaryotic abundance and increases viral production (and abundance), hence a positive correlation with VPR would be expected. FMVL is related to FVIC as follows (Binder, 1999):

$$FMVL = \frac{FVIC}{[\gamma \ln(2)\,(1\text{-}\varepsilon\text{-FVIC})]}$$

where γ is the ratio between the latent period and host generation time and ε is the fraction of the latent period during which intracellular viral particles are not visible.

As VPR is positively correlated to FVIC and FMVL is directly dependent on FVIC, it is likely that the conversion factors (i.e. γ and ε) in the above equation lead to inaccuracy in calculated FMVL, which is highly sensitive to these variables (Binder, 1999; Jacquet *et al*., 2005). Estimates of FMVL may therefore be subject to more uncertainty than FVIC, which is based on direct observations of virally infected cells. This may explain the absence of a significant relationship between FMVL and VPR in our analysis. This concern should extend to studies using FMVL to estimate viral production.

A positive correlation was observed between chlorophyll *a* (Chl *a*) concentration and VPR ($P < 0.001$). [Chl *a*] has been used as an index for photoautotrophic biomass and as an indicator of trophic status (Steele, 1962; Cullen, 1982; Boyer *et al*., 2009) and has been linked to phytoplankton as potential viral hosts (Cochlan *et al*., 1993). A positive correlation between VPR and [Chl *a*] may suggest that high viral abundance is linked directly or indirectly to phytoplankton biomass; indeed a positive correlation was observed between viral abundance and [Chl *a*] (Table 5).

This implies that at least a fraction of the measured viral abundance could be linked to the abundance of primary producers (Zhong *et al*., 2014). Prokaryotic abundance is also positively correlated with viral abundance in both pelagic habitats and benthic ones (Table 5). These correlations of viral abundance with [Chl *a*] and prokaryotic abundance confirm previous conclusions that the abundance of viruses is influenced by factors affecting host productivity and abundance (Wommack & Colwell, 2000; Weinbauer, 2004 and references therein).

Table 4 presents our meta-analysis results for different trophic conditions; there appears to be an increase in both prokaryotic and viral abundance along a trophic gradient (all $P < 0.001$ between trophic categories, with the exception of VA between eutrophic and mesotrophic conditions). Prokaryotic production is higher in eutrophic conditions compared to oligotrophic conditions ($P = 0.005$). Data on mesotrophic ecosystems were too scarce for reliable analysis ($N < 3$). Prokaryotic production does not include primary production, which will be highest in eutrophic conditions as confirmed by mean [Chl *a*] (all $P < 0.005$ between trophic categories). It is interesting that the VPR values suggest the inverse pattern along the trophic gradient (although the difference was only significant between meso- and eutrophic conditions). Viral loss may contribute to the observed decrease in VPR in eutrophic relative to mesotrophic habitats where dark decay (i.e. decay caused by factors other than light) makes an important contribution to viral loss (Liu *et al*., 2011), especially heat-sensitive substances including not only colloidal dissolved organic matter but also microscopic plankton. In productive and turbid waters, factors causing viral loss could be more concentrated than in oligotrophic habitats (Bongiorni *et al*., 2005).

A highly significant positive correlation was found between VPR and FLC ($P < 0.001$; Table 5). This was unexpected: lysogeny should lead to a reduced VPR, as the production of free viral particles is occasional compared to lytic production. It has been suggested that proviruses are common within prokaryotic communities with some results suggesting that 60% of bacteria harbour at least one prophage (Casjens, 2003; Edwards & Rohwer, 2005) and others reporting that the frequency of lysogeny in marine bacteria is 40% (Jiang & Paul, 1994, 1998; Paul, 2008), between 0.1 and 16% in temperate lakes and between 0 and 73% in Antarctic lakes (Sime-Ngando & Colombet, 2009). Recent studies suggest that lysogeny may represent a transitional state or stress response to abiotic changes experienced by hosts (Palesse *et al*., 2014). However, explanations of results on lysogeny remain ambiguous (Weinbauer, 2004). The correlation between FLC and VPR observed here may also result from a methodological bias. As temperate viruses cannot be distinguished visually from virulent viruses, studies on lysogenic viruses have been conducted by estimation of FLC from the induction of proviruses harboured by lysogenized cells (in either natural samples or isolated host strains), and such induction can fail. For example, the use of mitomycin C may not produce reliable results as its effects on prokaryotic strains may vary (or it can be ineffective) (Jiang & Paul, 1994, 1998). Further complications arise because the distinction between lysogenic and lytic viruses is somewhat artificial, as noted by Lenski (1988), since some environmental conditions induce lysogenic viruses, which then reproduce similarly to lytic viruses. The observed FVIC thus includes both lytic and lysogenic viruses (probably the majority being lytic, as they reproduce without requiring induction). Biases related to the use of FVIC as a proxy for lytic infection, and FLC as a proxy for lysogeny, have been discussed by Bettarel *et al*. (2008). Another possible explanation for the relationship between FLC and VPR may be related to the hypothesized increased frequency of non-lytic viral lifestyles in oligotrophic environments. If this is the case, a majority of viral particles in these environments would be produced by induction of lysogenic cells (spontaneous or otherwise) or chronically. It would then be logical that viral abundance (and hence VPR) would correlate with FLC. Recent advances in the study of lysogeny in the environment include analyses of metagenomes and it is possible that these will illuminate its link with other viral and microbial variables in the future.

(2) Factors decreasing VPR

Microbial variables elevating prokaryotic abundance, such as prokaryotic production, are likely to have an inverse relationship with VPR. A negative correlation between VPR and prokaryotic abundance ($P < 0.05$; Table 5) was found in our analyses, but only for data from sediments (expressed g^{-1} in Table 5). A negative relationship was also noted between VPR and prokaryotic production (all $P < 0.01$). Prokaryotic production is generally measured using radiolabelled thymidine (TdR) (Fuhrman & Azam, 1980, 1982) or leucine incorporation (Kirchman, K'Nees & Hodson, 1985; Kirchman, 2001); these methods measure net production rather than total production. As net production increases, and prokaryotic abundance increases accordingly, VPR decreases.

It is worth noting that a high prokaryotic abundance does not inevitably generate a low VPR, as high prokaryotic abundance may be accompanied by high viral abundance in a eutrophic steady state. This might, in part, explain the lack of correlation between VPR and prokaryotic abundance in aquatic ecosystems.

A negative correlation was also found between VPR and VCR ($P < 0.001$). Although intuitively a positive correlation might be expected (as high contact rates lead to viral production), the negative correlation is likely due to the low probability of virus–host encounters for individual viral particles when viral abundance increases. This could also result in crowding and/or cell saturation by viruses.

Significant negative correlations were found for salinity ($P < 0.01$) and temperature ($P < 0.001$) with VPR, but no correlation was found between VPR and depth. The negative correlation between temperature and VPR is likely to be related to low viral numbers at high temperatures. Hot springs have the second lowest mean viral abundance (5.62×10^6 particles ml^{-1}), after groundwater

(9.67×10^5 particles ml^{-1}). Hot springs also have a VPR ranging from 0.12 to 82.9 and a relatively low mean VPR (9.1) (Table 1). This harsh environment may favour lysogeny or carrier state for viruses and might be hostile to the persistence of viral particles, unlike their thermophilic hosts. Preliminary work on coastal hydrothermal vents partly supports this hypothesis, as virioplankton abundance appears to decrease along a temperature gradient (Manini *et al*., 2008; K. J. Parikka, S. Jacquet & M. Le Romancer, unpublished data). Salines have one of the highest mean abundances of viral particles (4.99×10^8 particles ml^{-1}) (all $P < 0.001$ when compared to other aquatic ecosystems) and prokaryotic cells (6.41×10^7 cells ml^{-1}) (all $P < 0.001$ when compared to other aquatic ecosystems), and mean VPR is also the highest (28.5) ($P < 0.001$ when compared to hot springs and groundwater, but no significant difference for marine and freshwater habitats; Fig. 1, Table 3) for any habitat other than soil. A negative correlation with salinity may indicate heterogeneity of VPR values, as also shown by the large range of values in Fig. 1B for salinity. Investigations on Senegalese aquatic habitats (Bettarel *et al*., 2011) and Antarctic lakes (Laybourn-Parry, Hofer & Sommaruga, 2001) suggest that viral abundances are higher in salines compared to neighbouring freshwater habitats and appear to increase with salinity. Bettarel *et al*. (2011) observed an increase in prokaryotic numbers as salinity increased from 0 to *ca*. 160‰, followed by a decrease until 310‰ and then a final peak from 310 to 360‰. A similar trend was found for viral abundance. The proposed explanation relates to a switch in microbial populations: beyond a threshold, halophilic organisms thrive. These gradients can also be observed in VPR values with increasing salinity: VPR values fluctuate in synchrony with viral abundance. The negative correlation observed here between VPR and salinity may therefore be most heavily influenced by data from salinities below the threshold of halophilic viruses. Note that (halo)viruses at high salinity are probably more stable than their thermophilic counterparts.

Surprisingly, no negative correlation was found between VPR and VDR. Viral decay is influenced by many factors (e.g. particle degradation and inactivation, virivory etc.) and it is likely that negative correlations between these parameters and VPR would be found if there were sufficient data (to the best of our knowledge, data available on both virivory and VPR for comparison are very scarce, $N < 5$). VPR fluctuations should then be monitored alongside the individual parameters to assess their influence. More data on VDR might also be needed to confirm the lack of correlation ($N = 12$ in our analysis).

(3) Seasonality and VPR

Several studies have reported seasonal variability in VPR (Weinbauer *et al*., 1995; Winget *et al*., 2011; Wilhartitz *et al*., 2013) linked to fluctuations in viral abundance (Vrede, Stensdotter & Lindstrom, 2003; Laybourn-Parry *et al*., 2013; Ma *et al*., 2013) or host abundance (Nakayama *et al*., 2007; Personnic *et al*., 2009; Maurice *et al*., 2010), while other studies have failed to find seasonality (Weinbauer *et al*., 1993; Helton *et al*., 2012). Seasonal variation in viral or prokaryotic numbers will not necessarily result in consistent changes in VPR if these constituents of VPR fluctuate proportionately. Parvathi *et al*. (2014) found significant co-variation between viruses and both heterotrophic bacteria and picocyanobacterial populations in Lake Geneva, but there was no clear seasonality in the resulting VPR. The highest viral and prokaryotic abundances were observed in August and September, but VPR values peaked in July along with FVIC, the frequency of infected cells (FIC) and BS. VPR values were also high in October and November. As discussed in Sections IV.1 and IV.2, a variety of variables can influence the calculated VPR, including environmental variables that will have direct and indirect effects on both cell and free viral particle abundance. For example, seasonal temperature changes affect mixing in lakes, altering nutrient availability and turbidity. These changes will affect the biological components of these habitats and influence the food chain. Any established steady state in microbial and viral communities will be affected by such changes and virus–host populations will move towards new steady states. This may be reflected in changes in VPR that may not be predictable from the original seasonal patterns in the variable of interest.

V. VPR DYNAMICS IN TWO CONTRASTING ECOSYSTEMS

The VPR is clearly linked to a series of viral and prokaryotic variables and fluctuates in all environments. However, the relative influence of these different indices on the VPR is likely to differ among different habitats, making a mechanistic understanding of the variation in VPR values difficult. We illustrate our interpretation in light of previously published theories, by depicting two scenarios: one for a highly productive environment and another for an environment with low productivity (Fig. 2).

In a single virus–host association, viral production will follow that of its host leading eventually to a decrease in host abundance and increased abundance of free viral particles. This is usually followed by the emergence of a virus-resistant host. This type of virus–host dynamics is typically observed *in vitro* and has inspired theories on microbial ecology *in vivo*, such as the infectivity paradox or the 'Weinbauer paradox' (Weinbauer, 2004), the 'Killing the Winner (KtW)' model (Thingstad & Lignell, 1997; Thingstad, 2000), the 'King of the Mountain (KoM)' hypothesis (Giovannoni, Temperton & Zhao, 2013) and at a larger co-evolutionary scale the 'Red Queen Hypothesis (RQH)' (Van Valen, 1973; Stern & Sorek, 2011). The infectivity paradox is based on observations that during the establishment of a steady state, virus–host systems become dominated by virus-resistant host strains leading to a low VPR. However, the opposite is observed *in situ*: VPR values indicating numerical viral dominance over their hosts.

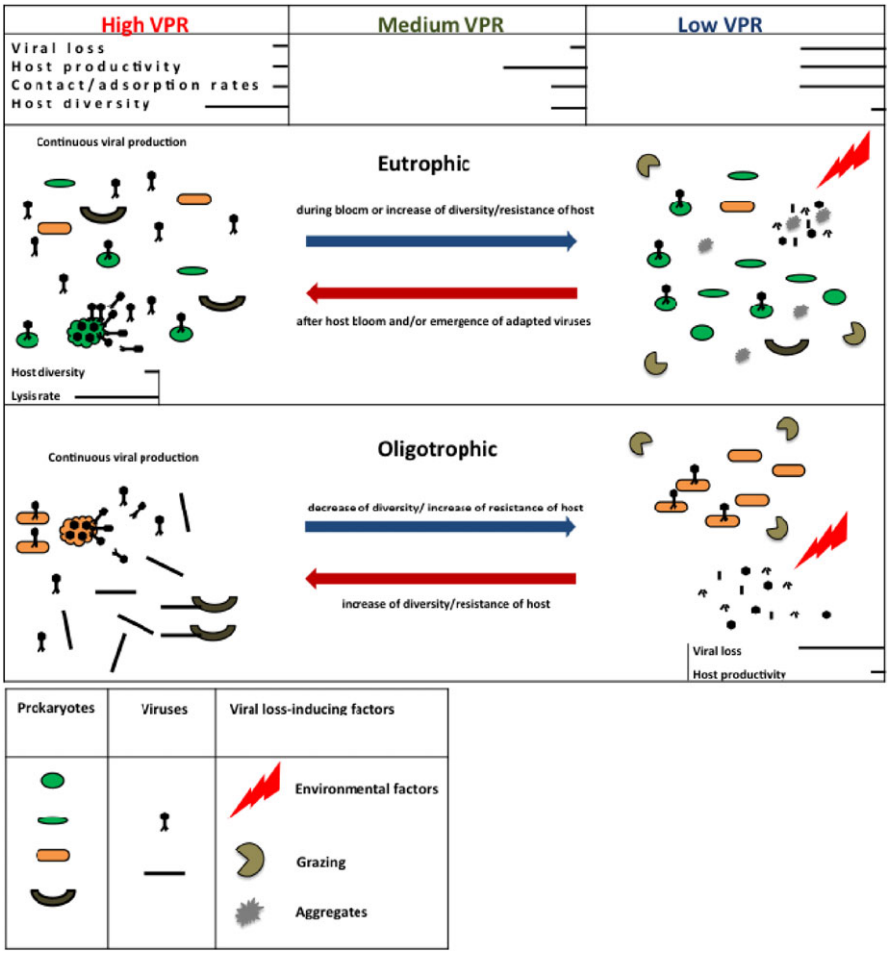

Fig. 2. Graphical representation of a conceptual scheme of factors or processes contributing to values of the virus-to-prokaryote ratio (VPR). VPR is influenced by several factors, of which host productivity and viral loss (e.g. decay, adsorption to particles, virivory, etc.) are probably the most influential. The different combinations of the characteristics on the top row generate a low, medium or high VPR, regardless of the productivity of a given ecosystem (the relative importance of each individual characteristic is illustrated by the length of its bar). However, other combinations can increase the VPR in eutrophic (separate bars on left side) or decrease in oligotrophic (separate bars on right side) ecosystems.

The KtW model predicts that fast-growing microbial strains are controlled though viral lysis enabling the co-existence of several strains/species that share similar resources, maintaining global microbial diversity. The application of the RQH to virus–host systems predicts an 'arms race', where hosts evade viral infection by genetic modification while viruses produce diversified progeny enabling recognition of resistant host cells (Hyman & Abedon, 2010; Stern & Sorek, 2011). The infectivity paradox, the KtW and RQH concepts are intuitively appealing, but can be in conflict, with attempts to reconcile them leading to a discussion (Giovannoni *et al*., 2013; Våge, Storesund & Thingstad, 2013; Zhao *et al*., 2013) over whether control over prokaryotic hosts is bottom-up or top-down, i.e. whether competitiveness over resources or resistance to viral attacks dictates the rise of dominant strains. Finally, Thingstad *et al*. (2014) recently proposed a model in which competitive traits generate strain diversity and defensive traits the abundance of individuals of each strain. Their model predicts that microbial species with the smallest trade-offs between competitiveness and resistance to viruses are likely to be the most abundant, as opposed to either highly competitive or highly defensive species.

(1) Highly productive environments

In highly productive (eutrophic) environments, the production of a virus from a single virus–host system happens at the expense of prokaryotic host production leading to a decrease in prokaryotic abundance, an increase in viral abundance and consequently an increase in VPR. As virus resistance increases in the host population, prokaryotic production will increase, elevating prokaryotic abundance and decreasing the VPR. This simplified virus–host dynamics can be observed in nature during blooms.

In an environment with diverse microbial communities, a decrease in the host abundance of the initial virus–host couple provides space and resources not only for

virus-resistant strains, but also for other prokaryotes. If the virus of the initial virus–host couple cannot infect other species of these communities, this leads to high prokaryotic production (and abundance) and therefore a reduced VPR. These propagating prokaryotic strains are either resistant due to selective pressure, as proposed by Maranger *et al*. (1994) and modelled by Thingstad *et al*. (2014), or are not recognized because they are not in the host range of the virus. On a larger scale, this scenario can be applied to primary producers releasing nutrients to secondary producers in productive environments leading to a viral shunt (i.e. virus-mediated lysis of microbial cells, which disables their consumption by higher trophic levels) and the redistribution and recycling of matter (Proctor & Fuhrman, 1990; Wilhelm & Suttle, 1999; Suttle, 2005; Weinbauer *et al*., 2011; Weitz & Wilhelm, 2012). Eventually, a modified or new virus will infect the newly dominant strains and the process will repeat, until new resistant strains occur. As this co-evolutionary arms race between resistant hosts and new viral progeny capable of recognizing them continues, fluctuations will be observed in the VPR before a steady state is reached. At steady state, one would expect to observe several host strains and related viruses. In the model of Thingstad *et al*. (2014), at steady state, the last strain of a species has only limited resources and therefore no longer represents a potential host for a new virus. As viruses replicate in proportionally greater numbers than their hosts, one can predict the VPR to reach a value above 1 at equilibrium; it is likely to take a value within the averages found for pelagic environments (i.e. the number of viral particles being an order of magnitude higher than that of its hosts).

Another hypothesis proposed by Bratbak & Heldal (1995) and Tuomi *et al*. (1995) suggested that the observed inverse relationship between prokaryotic abundance and VPR could be due to the adsorption of viruses to their host (e.g. when blooms occur). As viruses adsorb to their hosts, or viral receptors are released after lysis, the proportion of the virioplankton decreases and VPR would reflect this decrease. This would be temporary and followed by viral release and an ensuing increase in VPR. Presumably, as the co-evolutionary arms race proceeds and host diversity increases, viruses would spend more time as free particles without adsorption to specific hosts. This would lead to a steady increase in VPR until steady state is reached. A second factor influencing steady state is viral loss or decay by natural processes, which will act to decrease VPR. Factors contributing to viral loss in highly productive environments are temperature (Garza & Suttle, 1998; Bettarel *et al*., 2009), heat-sensitive substances (Noble & Fuhrman, 1997; Bongiorni *et al*., 2005; Liu *et al*., 2011), virivory (Suttle & Chen, 1992; Gonzáles & Suttle, 1993), solar radiation (Suttle & Chen, 1992; Noble & Fuhrman, 1997), chemical inactivation (Kapuscinski & Mitchell, 1980) and adsorption to particulate matter (Hewson & Fuhrman, 2003; Brussaard, 2004) associated with sedimentation. Other factors contributing to virus–host dynamics in pelagic environments include water currents and aquatic snow. These factors can disturb established steady states by dislocating both host cells and viruses (Weinbauer *et al*., 2009).

(2) Poorly productive environments

In less-productive (oligotrophic) environments, the scenario is different. As there is less prokaryotic production, viral production is likely to be low. This may lead to different viral lifestyles, such as lysogeny, which may be advantageous when cellular abundance, as well as temperature or nutrient concentration, is low (Stewart & Levin, 1984; Maurice *et al*., 2010; Thomas *et al*., 2011) or when rates of viral particle degradation are too high for lytic infection (Lenski, 1988). Other lifestyles include pseudolysogeny or chronic infection commonly observed among archaeal viruses and often referred to as the 'carrier state' (Pina *et al*., 2011). In less-productive environments low viral contact rates could arguably lead to higher VPR values as free viruses remain in the system searching for suitable hosts. As in the above scenario (Section V.1), high prokaryotic diversity would also lead to low viral infection rates. However, as prokaryotic production is also limited, the VPR would increase overall, provided that viral decay rates are low (Williamson *et al*., 2007; Winter *et al*., 2009; Maurice *et al*., 2010; De Corte *et al*., 2012). Note, therefore, that low viral abundance does not necessarily generate a low VPR. By contrast, oligotrophic habitats with low viral production and high viral decay rates would be expected to have low VPR values. High viral decay rates would decrease contact rates, intuitively leading to a decline in viral populations (Murray & Jackson, 1992; Wommack & Colwell, 2000). An example of this type of environment is hot springs, where mean viral and prokaryotic abundances and VPR values are all low, presumably due to the extreme nature of this environment. Another example is the oligotrophic open ocean surface waters, where the incidence of lysogeny is probably high (Weinbauer & Suttle, 1999), contact rates low (Wilhelm *et al*., 1998; Seymour *et al*., 2006) and decay rates high due to temperature (Rowe *et al*., 2012) and solar radiation (Suttle & Chen, 1992; Wommack *et al*., 1996; Wilhelm *et al*., 1998). Yet another factor contributing to viral loss in oligotrophic waters is grazing by heterotrophic nanoflagellates (Bettarel *et al*., 2005). By contrast, saline conditions may have lower viral decay rates (Danovaro *et al*., 2005; Kellogg, 2010). Haloviruses are reported to be stable at high salinity (Witte *et al*., 1997; Pietilä *et al*., 2012, 2013) and their continuous chronic or lytic production and accumulation of viral particles would thus lead to high VPR values in saline sites.

In the above discussion we emphasize that although it appears logical that high viral abundances and low prokaryotic abundances will generate high VPR values and low viral/high prokaryotic abundances produce low VPR values, data on either VA or PA alone are not enough to predict VPR values. High or low VPR values can occur in both highly or poorly productive environments. Moreover, what might appear counterintuitive is the fact that in highly productive environments, high host diversity

(and high resistance to viruses) favours a low VPR, whereas in poorly productive environments it tends to increase the VPR. The crucial difference lies in prokaryotic production. VPR is negatively correlated with microbial production. In productive environments as microbial diversity and resistance increases, so does host abundance, as viruses lag behind in ability to infect new strains. This is not the case in less-productive environments, where prokaryotic production is both weaker and probably also more local, in hot spots of greater nutrient availability. This results in a restricted and regional production of host cells, instead of the prolific and generalized production in nutrient-rich environments, and affects competition for space and nutrients which can be expected to be more localized. Therefore, in poorly productive environments we would expect relative accumulation of viral particles and hence a higher VPR, assuming viral decay to be limited. However, when viral decay is high, as in near-surface waters subject to high ultraviolet radiation, or when lysogeny is favoured (as might be the case for hot springs or nutrient-depleted zones), the VPR be lower.

VI. CONCLUSIONS

(1) The VPR is often presented as useful information with viral and prokaryotic abundances when new environments are investigated. Some studies have attempted to infer virus–host relationships using the VPR. High ratios have been interpreted as indicating elevated and ongoing viral production and low values have been attributed to reduced viral dynamics as well as high decay rates.

(2) Our overview of VPR values within a series of ecosystems shows that they can cover a wide range in most habitats, although some general trends can be identified. Our meta-analysis also confirms the suspicion that this ratio is complex and influenced by a multitude of factors related to virus–host dynamics (Peduzzi & Schiemer, 2004). Our analyses suggest a stronger dependence of the VPR on prokaryotic production than on viral production. It is likely that viral decay and contact rates, as well as prokaryotic mortality will also affect the VPR, although there were insufficient data available to investigate these variables.

(3) As the variables influencing the VPR differ in impact among ecosystems, it is clear that caution must be exercised when using the VPR to infer relationships between viruses and their hosts. High VPR values can be indicators of ongoing viral processes if the studied habitat allows high viral production, for example in the case of lytic production in eutrophic habitats. Yet, high values can also be a sign of low viral loss rates, when there is continuous production by lysogeny and/or chronic infection in oligotrophic habitats. By contrast, low VPR values can be related to high viral decay rates (e.g. in harsh environments) or to high prokaryotic production (e.g. in eutrophic habitats).

(4) Whereas measurements of viral and cellular abundances can be used to assess the status of microbial and viral communities within a studied ecosystem, the VPR is less useful. We suggest that the VPR can only express the numerical dominance of free viral particles over their cellular hosts at a specific site and time. Its interpretation will always require additional information on the investigated ecosystem, thus its role in the study of viral ecology loses importance and may cause confusion. We therefore recommend extreme caution when inferring relationships (such as the importance of viral dynamics and lysis) between viruses and their hosts using the VPR.

(5) As for any ratio, the VPR can be a useful tool when most processes are known or controlled. Using simple *in vitro* models, effects of stressors, such as application of different inducing agents, could be studied with simple host–virus systems. The VPR could also be used in the calculation of other indices, such as burst size, where other variables have been determined. Thus the VPR may retain a role in monitoring virus–host dynamics *in vitro*.

VII. ACKNOWLEDGEMENTS

We are grateful to Y. Bettarel, M. O. Gessner, I. Hewson, K. Šimek, C. Säwström, N. L. Patten, M. Filippini, J. Colombet, M. Middelboe, P. Peduzzi and D. F. Bird for kindly providing detailed data from their publications. We are also grateful for T. F. Thingstad for his critical reading of a previous version of this manuscript.

VIII. APPENDIX A1: LIST OF ABBREVIATIONS AND TERMS

BS, Burst size: number of virions released per host cell; Chl *a*, Chlorophyll *a*; FIC, Frequency of infected cells; FLC, Frequency of lysogenic cells: proportion of cells containing a prophage/provirus in a prokaryotic cell population; FMVL, Fraction of mortality from viral lysis: fraction of a cellular population that dies from viral infection; FVIC, Frequency of visibly infected cells: proportion of cells infected by viruses, as determined by observation with a microscope; GR, Grazing rate: rate at which prokaryotic cells are ingested by protozoa; KoM, 'King of the Mountain' model: a model proposing that (numerically) dominant prokaryotic strains are able to retain their dominance by genetic recombination, which offers a positive-feedback mechanism that maintains their superiority in nutrient uptake (and therefore resource competition); KtW, 'Killing the Winner' model: a model describing the maintenance of prokaryotic diversity by viral lysis of the (numerically) dominant strains; LP, Latent period: period between viral attachment to host and virion release; PA, Prokaryotic abundance; PP, Prokaryotic production; RQH, 'Red Queen hypothesis'; VA, Viral abundance; VBR, Virus-to-bacterium ratio (term that pre-dates the 'virus-to-prokaryote ratio', used when the numerical importance of archaea was not yet recognized); VCR, Virus–host contact rates; VDR, Viral decay rate; VIBM, Virus-induced bacterial mortality (equivalent to

the 'fraction of mortality from viral lysis' – FMVL); Viral shunt, Prokaryotic mortality from viral lysis, disabling their predation by higher trophic levels (therefore 'shunting' the food chain); VLP, Virus-like particle: a particle likely to be a virion but whose infectivity has not been established [note that this definition is not the same as the more restricted use of VLP in clinical microbiology as a particle devoid of viral genome constituting a potential vaccine candidate]; VP, Viral production; VPR, Virus-to-prokaryote ratio; VT, Viral turnover: the time needed for (or rate at which) a viral population is replaced by a new generation of viruses.

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(*Received 23 April 2015; revised 18 February 2016; accepted 1 March 2016*)