Viral abundance in aquatic systems: a comparison between marine and fresh waters

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ABSTRACT: In order to investigate the factors controlling viral abundance, 22 lakes in Québec were surveyed. We measured viral and bacterial abundance, bacterial production, chlorophyll a, total phosphorus and DOC (dissolved organic carbon) concentrations. Regression models built with these data were compared to models based on literature data, which to date have been collected largely from marine sites. Positive empirical relationships were found between viral abundance and (1) chlorophyll a concentrations, (2) bacterial abundances, (3) bacterial production, and (4) total phosphorus concentration. There was little to no trend in the virus-to-bacteria ratio with increasing trophy. Analysis of covariance revealed significant differences between relations in marine and freshwater systems. The virus-to-bacteria ratio was significantly higher in freshwater (mode = 22.5) than marine environments (mode = 2.5), and there were significantly more bacteria per unit chlorophyll in our freshwater samples. We suggest that this difference is related to the increased dependence of freshwater bacteria on allochthonous material relative to marine systems, as well as the increased relative importance of photosynthetic cyanobacteria in lakes.

KEY WORDS: Virus · Bacteria · Chlorophyll a · Bacterial production · Marine · Freshwater · Empirical relationships

INTRODUCTION

In the past decade and a half, considerable effort has been applied to identify the factors which control bacterial populations in aquatic systems. This effort stems from the realization of the important role of heterotrophic bacteria as decomposers and remineralizers, and as a source of particulate organic carbon (POC) through their rapid assimilation of dissolved organic carbon (DOC) and their efficient conversion of this DOC into bacterial biomass. From this recognition of bacteria as an integral component of aquatic systems, Azam et al. (1983) formulated the microbial loop hypothesis wherein significant amounts of DOM (dissolved organic matter) produced by algae are assimilated by bacteria. Bacteria are in turn grazed upon by flagellates, ciliates and macrozooplankton, thereby reintroducing 'lost' carbon back into the food chain (Fenchel 1982). Empirical studies support several predictions about bacterial abundance and activity brought forth by the microbial loop hypothesis. Included among these relationships is the increase in bacterial abundance with increasing chlorophyll a and total phosphorus concentrations (Bird & Kalff 1984, Del Giorgio & Peters 1993). In addition, an increase in bacterial production has been observed with an increase in (1) net primary production (Cole et al. 1988), (2) bacterial abundance (White et al. 1991), and (3) DOC concentration (Tranvik 1992). Generally these relationships are consistent across aquatic systems.

Until recently, bacterial community size was thought to be controlled largely by flagellate and ciliate grazing (Fenchel 1982, McManus & Fuhrman 1986, Pace 1988). However, there appear to be inconsistencies with the budgets which attempt to balance bacterial production and the removal of bacteria by grazers (Sherr et al. 1989), suggesting that bacteria are also being removed from the system by other means.

Viruses have recently been identified as dynamic components of aquatic environments. High viral abun-

dances have been observed in marine (Bergh et al. 1989, Proctor & Fuhrman 1990), coastal (Suttle et al. 1990, Paul et al. 1991), and fresh waters (Klut & Stockner 1990), marine (Paul et al. 1993) and freshwater sediments (Maranger unpubl. data) and in polar sea ice (Maranger et al. 1994). The high concentration of viruses in aquatic systems along with rapid changes in viral abundances (Børsheim et al. 1990, Bratbak et al. 1990), rapid viral decay rates (Heldal & Bratbak 1991) and the direct demonstration of virus particles within bacterial cells imply that viruses play an important role in the control of bacterial populations. Suttle & Chen (1992) estimated that viruses are responsible for 8 to 26% of bacterial mortality; Proctor et al. (1993) set it at 6 to 62%.

Despite increasing research on aquatic viruses, little work has been done to model changes in virus concentrations with the important parameters affecting bacterial production and abundance. In this study, we sampled 22 lakes in Québec and collected literature data in an attempt to model changes in viral abundance with (1) chlorophyll *a* concentrations, (2) bacterial abundance, (3) total phosphorus and (4) DOC concentrations, all of which are parameters known to control bacterial production. We also determined whether viral abundance could be predicted empirically from bacterial production.

MATERIALS AND METHODS

Sampling sites and methods. Samples were collected from 22 lakes with a total of 23 sites, 12 located in the Laurentian Mountains and 11 in the Eastern Townships, north and east of Montréal, Québec, respectively between July 24 and August 8, 1993.

Samples for measuring bacterial production were taken at 1 m using a Van Dorn bottle. All other water samples were taken using an integrated water column sampler (Tygon tubing, inner diam. 3 cm). The integrated sample was taken from 0 m to twice the Secchi depth, in order to sample most or all of the euphotic zone.

Virus and bacteria samples were fixed with glutaraldehyde (2.5% final concentration); total phosphorus samples were taken directly, and DOC samples were prefiltered in the field through a 0.45 μm syringe-tip filter and acidified with concentrated nitric acid. All samples were stored at 4°C until processed which was within 1 mo of collection. Total phosphorus concentration was determined using an Alpkem auto-analyzer and DOC concentrations were determined using a Dohrmann carbon analyzer.

Enumeration of bacteria and viruses. Bacteria were stained with DAPI (1 μg ml^{-1}) and filtered onto black 0.2 μm pore size polycarbonate filters (Porter & Feig 1980). Samples were enumerated using epifluorescence microscopy at a final magnification of 1250×.

Viruses were pelleted directly onto 400-mesh formvar-coated Cu grids at 100000 × *g* for 30 min using an EM-90 rotor in a Beckman airfuge (Hammond et al. 1981). Grids were air dried, stained with 2% uranyl acetate for 5 min and counted directly using a Phillips EM 300 transmission electron microscope at high magnification (90000 ×). Recovery efficiency of the EM-90 rotor was established at 96% (± 19%) by pelleting a known concentration of T5 phage, an extensively studied lytic phage of *Escherichia coli*. Viral abundance was estimated by observing 50 fields per EM grid. Two grids per sample were prepared in which viruses from 5 fields in 10 grid squares were enumerated. The coefficient of variation of the mean count per sample using this method was less than 10%. Viruses from the Québec lakes were grouped into the following size classes as determined by head capsid diameter: <50 nm, 50–70 nm, 70–100 nm, and >100 nm.

Bacterial production. Production was measured in the field using ^{3}H leucine (Kirchman et al. 1985). Two replicates and 2 controls killed with cold trichloroacetic acid (TCA; 5% final concentration) and formaldehyde (1% final concentration) were incubated for 2 h at ambient lake water temperature. Approximately 10 nmol l^{-1} final of labelled leucine with a specific activity of 54 Ci mmol^{-1} was added to 15 ml samples. Incubation was terminated with the addition of TCA and formaldehyde to final concentrations of 5 and 1% respectively. Fixed samples were kept at 4°C and were filtered onto 0.2 μm-pore cellulose nitrate filters immediately upon returning to the lab. These filtered samples were washed 3 times with 5% TCA and twice with 80% ethanol (Wicks & Robarts 1988). Filters were dissolved in ethyl acetate and assayed for radioactivity by liquid scintillation.

Algal biomass. Chlorophyll *a* samples were collected in brown bottles and kept at 4°C. These were filtered on glass fiber filters immediately upon returning to the lab and kept frozen until processed. Filters were ground in cold acetone (80%) and centrifuged in a clinical centrifuge. Supernatants were read spectrophotometrically using the trichromatic method as outlined in Wetzel & Likens (1991).

Literature data collection. Simultaneous observations of viral and bacterial abundances and chlorophyll *a* concentrations were assembled from the recent literature. Chlorophyll data was not always available. The data were obtained from 14 separate studies covering a relatively wide geographic range of sites (Bergh et al. 1989, Bratbak et al. 1990, Hara et al. 1991, Heldal & Bratbak 1991, Smith et al. 1992, Wommack et al. 1992, Boehme et al. 1993, Cochlan et al. 1993, Paul et al. 1993, Wein-

bauer et al. 1993, Jiang & Paul 1994). New observations included the present study and unpublished data collected as part of the Long Term Ecosystem Research (LTER) program in the Antarctic, and from the Hawaiian Ocean Time-series. All values were restricted to the photic zone and in cases where coastal profiles were available, a mean value for bacterial and viral abundance was taken from the upper 30 m. Single site observations were not excluded. Although data from all seasons were included for marine sites, only summer observations were found for freshwater.

Statistical analysis. The data were analysed by ordinary least squares regression analysis and analysis of covariance (ANCOVA) using SAS (SAS Institute Inc. 1987). The data were log transformed to meet the normality assumptions of least squares regression analysis and to equalize the variance over the range of observations.

RESULTS

Bacterial abundance was a moderately good predictor of viral abundance (Fig. 1). The overall relationship is:

$$\log vir = 0.90 + 0.99 \log bac \quad r^2 = 0.64, n = 176 \quad (1)$$

where *vir* is the number of virus-like particles (ml^{-1}) as determined by transmission electron microscopy and *bac* is number of bacteria (ml^{-1}). However, many of the observations which fell below the line of prediction came from studies in which the the virus samples were concentrated by ultrafiltration before enumeration by electron microscopy (Fig. 1). It has been reported in several studies that recovery is not 100% efficient when samples are concentrated, but may be between 13 and 60% (Suttle & Chan 1994) or higher (Paul et al. 1991). An analysis of covariance (ANCOVA) revealed a significant difference in the intercept but not in the slope of the viral and bacterial relationship when virus samples were concentrated as compared with the relationship when virus samples were not concentrated $(r^2 = 0.69, n = 149, F \text{ prob. } 0.0001)$. This comparison was done only between concentrated and non-concentrated virus samples from marine observations. These relationships were:

Non-concentrated:
$$\log vir = 1.32 + 0.93 \log bac \quad n = 94$$
$$\tag{2}$$
Concentrated:
$$\log vir = 0.87 + 0.93 \log bac \quad n = 55$$

Based on the adjusted mean difference between the methods for determining viral adundance from the ANCOVA, for a given bacterial abundance, viral abundances from the non-concentrated counts were about
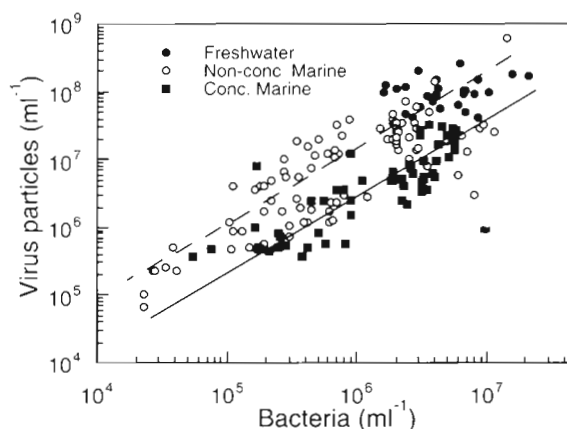

Fig. 1. Relationship between viral and bacterial abundance in fresh- and salt water habitats. An ANCOVA revealed a significant difference in the intercept between estimates based on non-concentrated marine virus samples (represented by the fitted dotted regression line), and those that had first been concentrated (represented by the fitted solid line). Concentrated virus samples gave significantly lower counts for a given bacterial abundance

2.9 times greater than those from the concentrated counts. To account for the possible underestimation of viruses in concentrated samples, we multiplied those viral abundances, which accounted for 55 observations, by a correction factor of 2.9 as determined by the higher intercept of the non-concentrated samples in the analysis of covariance. All subsequent analyses were done with the corrected viral abundances.

Repeating the analysis with corrected virus values, a fairly strong relationship was found between bacterial and viral abundance (Fig. 2). The overall relationship is ·

$$\log vir = 0.95 + 1.01 \log bac \quad r^2 = 0.72, n = 176 \quad (3)$$

An analysis of covariance revealed a significant difference in both the slope and the intercept of the viral and bacterial relationship in freshwater as compared with marine sites $(r^2 = 0.77, n = 176, p < 0.0001)$. These relationships were:

Marine: $\log vir = 1.33 + 0.93 \log bac \quad n = 149$
Freshwater: $\log vir = 6.88 + 0.16 \log bac \quad n = 27$ $\quad (4)$

Viral abundance increased with increasing bacterial numbers in marine environments. According to our values, this relationship, as determined through ordinary least squares regression analysis, was not significant for freshwater. Mean abundance of virus-like particles did not change with increasing bacterial numbers in our study lakes (Fig. 3).

An unexpected result was that chlorophyll *a* concentration was a slightly better predictor of viral abundance in all habitats than was bacterial number. We
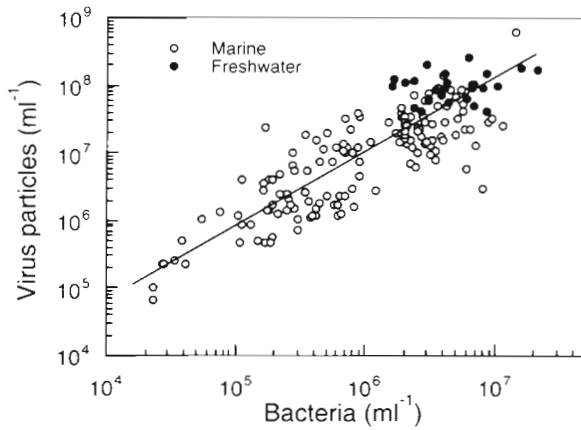
Fig. 2. Relationship between viral and bacterial abundance in fresh- and salt water habitats, including the corrected values for the concentrated marine virus samples. The fitted line represents the regression equation we derived from the overall relationship
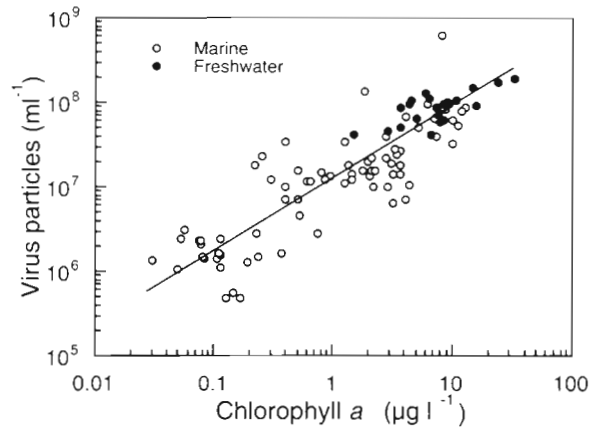

Fig. 4. Scatter diagram of the points obtained from the literature and this freshwater study used to derive regressions of viral abundance and chlorophyll a concentration. Solid line represents the overall regression equation we derived

found moderately strong empirical relationships between viral abundance and chlorophyll (Fig. 4). The overall relationship is:

$$\log vir = 7.09 + 0.80 \log chl\,a \qquad r^2 = 0.77,\ n = 101 \quad (5)$$

where chl a is the concentration of chlorophyll a ($\mu g\ l^{-1}$). However, based on our results, the relationship is different in the 2 environments. An analysis of covariance revealed a significant difference in the intercept but no significant difference in the slopes between marine and freshwater environments ($r^2 = 0.79$, n = 101, p < 0.0001). The 2 models are:

Marine: $\log vir = 7.05 + 0.73 \log chl\,a$ n = 78
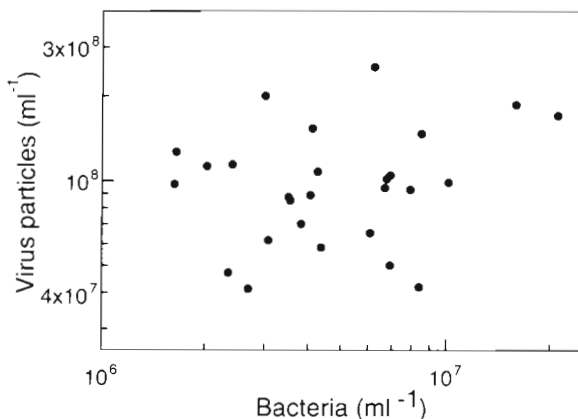Freshwater: $\log vir = 7.30 + 0.73 \log chl\,a$ n = 23   (6)

Fig. 3. Viral and bacterial abundances in 22 Québec freshwater lakes. The relationship was statistically non-significant

Based on the adjusted mean between the 2 groups from the ANCOVA, for a given chlorophyll concentration, viral abundance is expected to be about 1.8 times greater in freshwater than marine environments.

Both chlorophyll a concentration and bacterial abundance were significant factors in explaining viral abundance when included in a multiple regression. The model is:

$$\log vir = 4.97 + 0.51 \log chl\,a + 0.34 \log bac$$
$$r^2 = 0.80,\ n = 101 \qquad (7)$$

There was no significant relationship between viral abundance and bacterial abundance in our lakes (Fig. 3, model 3: Freshwater), however there were significant positive relationships between viral abundance and (1) chlorophyll a concentration, (2) bacterial production and (3) total phosphorus for these same sites (Fig. 5A to C). The models for these relationships are:

$\log vir = 7.55 + 0.45 \log chl\,a$ $r^2 = 0.52$, n = 23   (8)
$\log vir = 9.37 + 0.47 \log prod$ $r^2 = 0.52$, n = 18   (9)
$\log vir = 7.50 + 0.44 \log TP$ $r^2 = 0.57$, n = 18   (10)

where prod is bacterial production ($\mu g\ C\ ml^{-1}\ h^{-1}$), and TP is total phosphorus ($\mu g\ l^{-1}$). No significant multivariate models were found with our freshwater parameters. No significant relationship was found between viral abundance and DOC.

The size class distribution of the viruses of the 23 sites we sampled varied among sites, although viruses <70 nm in diameter were dominant (more than 80% of the observed virus particles) in all cases (Fig. 6). No statistically significant trend in size class distribution in viruses was detected with increasing trophy.
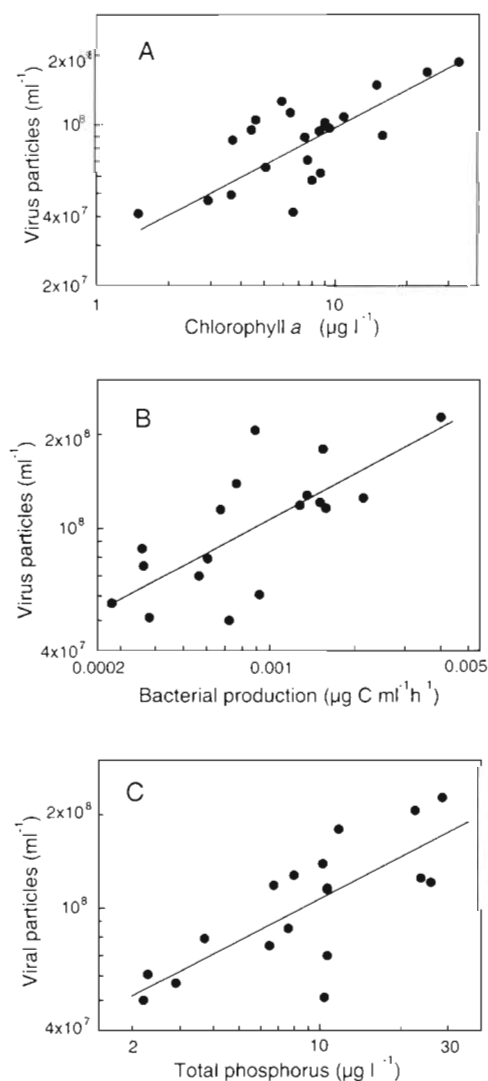
Fig. 5. (A) Relationship between viral abundance and chlorophyll *a* concentration in our freshwater lakes. (B) Relationship between viral abundance and bacterial production as determined through leucine incorporation in the Québec lakes. (C) Scatter diagram of viral abundance and total phosphorus concentration in freshwater lakes. Solid lines represent the regression equation we derived for each relationship
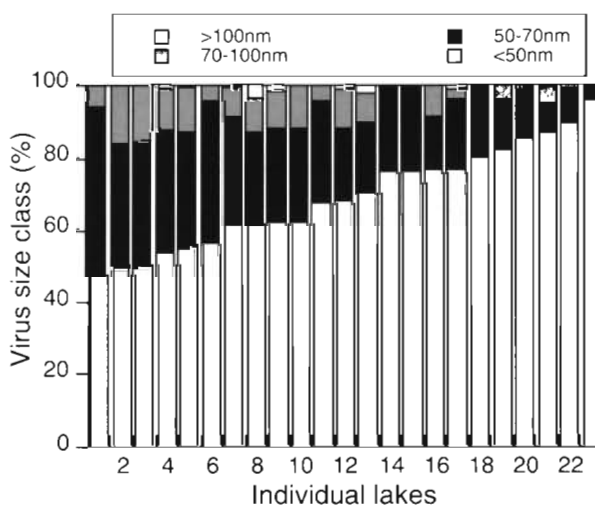

Fig. 6. Viral size class distribution, represented as a percentage of each size class, from the 22 lakes surveyed; 2 bays from Lake Memphremagog were sampled accounting for the 23 sites represented here

Interestingly, the virus-to-bacteria ratio (VBR) was significantly higher in freshwater than marine waters (*t*-test, p < 0.0001). Mean, maximum, and minimum values of viral and bacterial abundances, VBR, and chlorophyll *a* concentrations for both marine and freshwater habitats are listed in Table 1. The difference in VBR between environments is evident from the frequency distribution of the VBR (Fig. 7). We did detect a very slight, but significant (p < 0.05) trend of chlorophyll *a* on the VBR (Fig. 8). The overall relationship is:

$$\log VBR = 1.01 + 0.14 \log chl\ a \quad r^2 = 0.08, n = 101 \quad (11)$$

We did not detect any significant trend in the overall relationship of VBR with increasing bacterial abundance (Fig. 9). There was a significant negative trend of the VBR with increasing bacterial abundance in our freshwater sites (Fig 9). Chlorophyll *a* concentration and bacterial abundance were significant parameters in a multiple regression explaining changes in the VBR. Based on the coefficients of this model, increasing chlorophyll concentration has a significant positive effect on the VBR when the influence of bacterial abundance is removed, while increasing bacterial abundance has a significant negative effect when the influence of chlorophyll *a* is removed. The overall relationship is:

$$\log VBR = 4.82 + 0.56 \log chl\ a - 0.63 \log bac$$
$$r^2 = 0.18, n = 101 \quad (12)$$

When this multiple regression was included in an ANCOVA in order to determine any differences between marine and freshwaters, we found a significant difference in the intercepts between the 2 habitats ($r^2 = 0.32, n = 101, p < 0.001$). The 2 models are:

Marine: $\log VBR = 4.97 + 0.51 \log chl\ a - 0.66 \log bac$
$$n = 78 \quad (13)$$
Freshwater: $\log VBR = 5.20 + 0.51 \log chl\ a - 0.66 \log bac$
$$n = 23$$

Hence, based on the adjusted mean difference, when we remove the influence of both bacterial abundance and chlorophyll concentration on the VBR, the VBR is on average 1.7 times higher in freshwater as compared with marine sites.

Table 1. Summary statistics. Mean, maximum, and minimum values of virus abundance (virus particles ml⁻¹), bacterial abundance (cells ml⁻¹), chlorophyll *a* concentrations (µg l⁻¹) for fresh and marine systems. The virus-to-bacteria ratio (VBR) is expressed in terms of mode with maximum and minimum values. na: not applicable

| | Freshwater | | | Marine | | | Overall |
	Mean	Max	Min	Mean	Max	Min	mean
Virus abundance	1.1×10^8	2.5×10^8	4.1×10^7	1.7×10^7	7.1×10^7	6.7×10^4	3.0×10^7
Corrected				2.2×10^7	8.0×10^7		3.5×10^7
Bacterial abundance	5.8×10^6	1.6×10^7	2.4×10^6	2.0×10^6	1.4×10^7	2.3×10^4	2.6×10^6
Chlorophyll *a*	9.2	32.8	1.5	2.5	12.8	0.05	5.0
VBR	20–25	77.5	4.9	1–5	53.8	0.38	na

In order to demonstrate the slight effect of trophy on the VBR we have plotted the rate of increase of viral abundance with chlorophyll concentration (model 4) and the rate of increase of bacteria with increasing chlorophyll (Fig. 10). The relations are similar, although the relationship with viruses has a slightly steeper slope. The overall relationship for bacteria and chlorophyll is:

$$\log bac = 6.08 + 0.66 \log chl\ a \qquad r^2 = 0.79,\ n = 101 \quad (14)$$

When viruses were added to this relationship in a multiple regression, both parameters had a positive, significant influence on bacterial abundance. The model for this relationship is:

$$\log bac = 4.48 + 0.48 \log chl\ a + 0.22 \log vir$$
$$r^2 = 0.81,\ n = 101 \qquad (15)$$

DISCUSSION

The objective of this study was to model changes in viral abundance with the parameters which affect bacterial production. Viral abundance and algal biomass appear to be quantitatively linked in aquatic ecosystems. Chlorophyll *a* explained more of the variation in viral abundance in aquatic systems than did bacterial abundance. Cochlan et al. (1993) found bacterial abundance to be a superior predictor variable over chlorophyll *a*. In their models, bacterial abundance explained 69% of the variation in viral abundance as opposed to chlorophyll explaining only 45%. A greater range in both trophic status and bacterial abundance is considered in this study, due to the addition of freshwater data, making chlorophyll *a* a slightly better predictor variable in estimating changes in viral abundance.

According to our values, there was no significant change in viral abundance with increasing bacterial abundance in freshwater, whereas there was a positive relationship with chlorophyll *a*. A simple explanation for this observation is that the observed increase in virus numbers with algal biomass is due to an increase in the number of algal or cyanobacterial viruses. Unfortunately there is currently no method to distinguish between algal or cyanobacterial, and bacterial viruses by microscopy. The importance of algal viruses
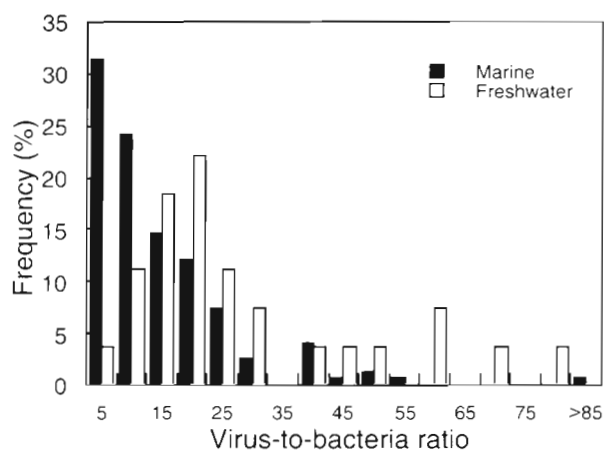

Fig. 7 Frequency distribution, represented as a percentage, of virus-to-bacteria ratios observed in marine and freshwaters
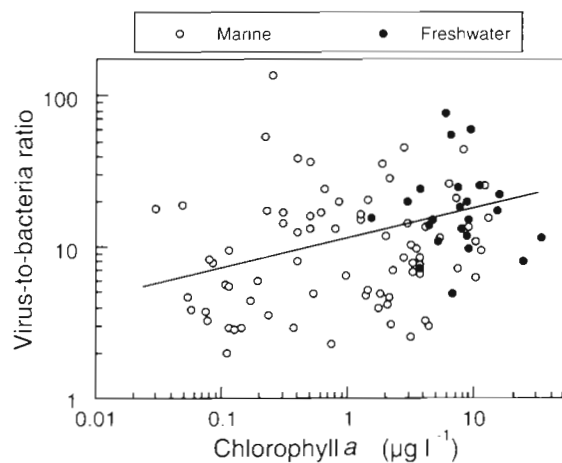

Fig. 8. Overall relationship between the virus-to-bacteria ratio and chlorophyll *a*. Solid line represents the regression equation ($r^2 = 0.08$, n = 101, p < 0.05)
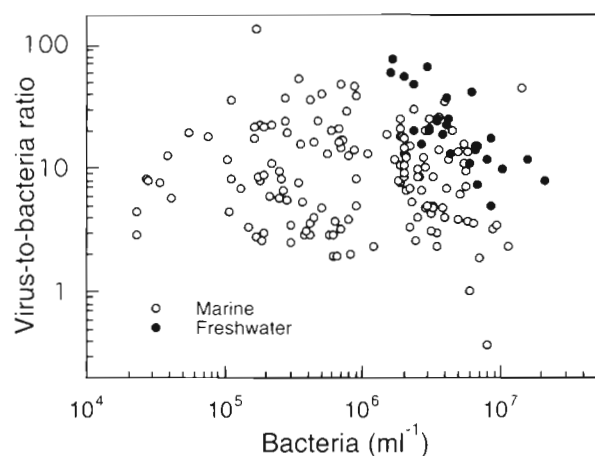
Fig. 9. Scatter diagram of VBR and bacterial abundance. This relationship was not statistically significant

remains an important open question. Alternatively, the explanation we favor is that the temporal dynamics of the relationship between bacteriophage and bacteria within a lake is inevitably negative in the short term hiding the quantitative dependence of viruses on bacteria. Hence a large trophic range is necessary in order to demonstrate a positive trend among systems. The positive effect of increasing algal biomass on the abundance of both bacteria and viruses reflects the availability of resources for bacterial production, whereas the fundamental interdependence of viruses and their bacterial hosts is obscured.

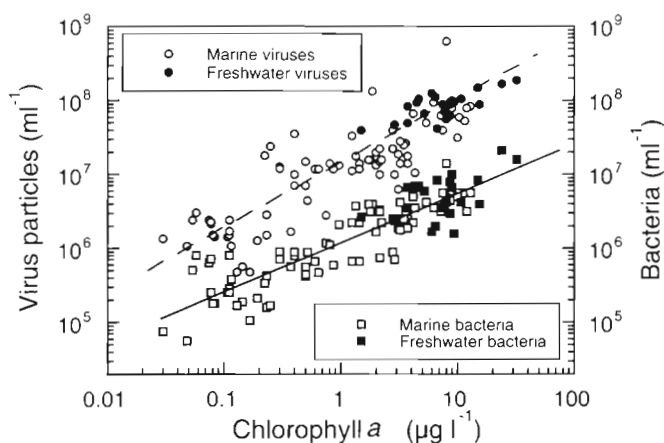The effect of trophy on viral densities is probably related to an increase in bacterial production, and

Fig. 10. Relationships between viral and bacterial abundances and chlorophyll a. The dotted line represents the line of best fit for the virus-chlorophyll a relationship, the solid line represents the line of best fit for the bacteria-chlorophyll a relationship ($r^2 = 0.79$, n = 101, p < 0.0001) for the same sites

many of the viruses produced in areas with elevated algal biomass may be the product of the more active members of the bacterial community. We observed an increase in viral abundance with increasing bacterial production. It is also interesting to note that the rates of increase in viral abundance in relation to chlorophyll a, bacterial production and total phosphorus are virtually identical in freshwater, suggesting that these parameters all represent the same influence, the suitability of the lake for nurturing bacterial growth.

It is useful to compare these empirical results with the current quantitative understanding of phage-bacterial dynamics. Weinbauer & Peduzzi (1994) estimated average infection rates and burst sizes for different morphological types of bacteria in the Adriatic Sea, that were not much different from others reported in the literature. Summing, over morphological types, the product of the proportion of each type in the community, times the proportion observably infected, times mean burst size, times the correction factor relating total infection rate to observable infection rate, one can calculate that about 300 viruses are released per 100 native bacteria per lytic cycle. In general, the lytic cycle of viruses corresponds to the bacterial generation time or slightly longer (Proctor et al. 1993). At steady state, the VBR is equal to the ratio of viral production to viral loss. Therefore the steady state VBR would be 3 if viral loss per bacterial generation time were 100%. The mean marine VBR was 10, suggesting that the turnover time for viruses is roughly 3 times bacterial generation time. The much greater VBRs seen in some environments must stem from greater infection rates or lower losses — up to 25 times longer persistence in the environment than their bacterial hosts.

It is possible that more active bacteria, growing in favorable conditions, for example those growing in areas of elevated concentrations of phytoplankton, are more susceptible to lysis induced by viral infection than those growing in nutrient-poor environments. It has been demonstrated, for the lambda phage of Escherichia coli, that the lytic pathway is favored over lysogeny under ideal growth conditions because of the reduction in the production of phage repressor protein (Watson et al. 1987). Viral control of active bacterial populations may be partly responsible for the competitive disadvantage of bacteria with respect to phytoplankton for nutrients in more eutrophic sites (Currie & Kalff 1984). Viruses may be responsible for maintaining the bacterial population at relatively low densities by lysing its more active members, making the entire community less competitive for inorganic phosphorus. Selective viral attack on the fastest-growing bacteria has been suggested to be responsible for observations of viral abundance peaks that precede bacterial peaks in time (Maranger et al. 1994).

Temperature may also be an important factor in controlling viral abundance in that temperature controls bacterial growth rates and has a significant positive effect on bacterial production (White et al. 1991). Jiang & Paul (1994) found a positive correlation between viral abundance and temperature. *In situ* temperature was taken in our study and had no effect on viral abundance, but temperature only varied by 3°C among lakes during this study.

Increasing viral abundances in areas of higher trophic status may be related to an increase in burst size per lytic event, since the VBR is loosely related to increasing chlorophyll a concentrations. By simulating richer environmental growth conditions, Kokjohn et al. (1991) demonstrated that more progeny phage were produced per lysed cell than in nutrient-poor conditions. Also, Weinbauer et al. (1993) directly observed an increase in the number of assembled phage particles inside bacteria growing in more eutrophic sites.

An interesting possibility is that the consistently lower VBR observed in marine systems as compared to freshwater is an effect of salinity differences. A negative relationship appears to exist between viral abundance and increasing salt concentrations (Paul et al. 1993, Jiang & Paul 1994). There is evidence that bacterial production also declines with increasing salinity (White et al. 1991) which might explain a part of the difference. However, we suggest that the decline results from elevated loss rates due to nonspecific adsorption to particles, which is apparently a major viral loss factor in marine coastal waters (Suttle & Chen 1992) and which increases in direct proportion to salinity (Valentine & Allison 1959). If this idea is correct, then the VBR in saline lakes will be found to be very low.

Alternatively, there is another major difference between marine and freshwater environments: the greater relative abundance of cyanobacteria in lakes. Prokaryotic photosynthetic biomass in marine systems is primarily picoplanktonic (*Prochlorococcus* and *Synechococcus* spp.), and may be included in the heterotrophic bacterial counts. Freshwater prokaryotic biomass can be predominantly algal, especially in richer lakes, so that the VBR in lakes is an overestimate because not all of the 'bacteria' are included in the bacterial count.

There was no apparent trend in viral size class distribution with changes in trophic status. In each of the lakes surveyed, greater than 80% of the viruses were <70 nm in diameter. This may be due to grazing pressure on larger virus particles (González & Suttle 1993). Since the viral community is a product of the host community because of the specificity of viral infection, differences in the community of viruses may imply differences in the bacterial community. It is highly probable

that there were differences in the composition of the bacterial community or in the dominant active bacterial species between lakes. However, this aspect of bacterial and viral dynamics in aquatic systems cannot be studied by direct microscopy, but must await the development of novel, perhaps molecular biological, approaches to bacterial and viral identification.

That the relationship between viral and bacterial abundances in freshwater was statistically non-significant does not necessarily imply that these parameters are not related. First of all, viruses are apparently more variable in abundance than bacteria. A greater variability in viral abundance as compared with bacteria has been demonstrated in daily (Bratbak et al. 1990) and diel (Jiang & Paul 1994) time-series studies. This greater variability in viral abundance may be due in part to a virus population's lysogenic response to given environmental conditions. Some 90% of known viruses have been identified as being temperate, and it has been suggested that (1) a depletion in nutrients in the growth medium and (2) a high multiplicity of infection (Friefelder 1987) would stimulate viral lysogenic incorporation. This would imply that lysogeny would be favored in oligotrophic sites and when the VBR is very high. The nature of the virus-host relationship, which is essentially a predator-prey relationship, may also explain the higher variability of viral abundance than of bacteria. Because viral proliferation results in host cell lysis, one would expect to observe a negative relationship between viral and bacterial abundances on a short enough time scale. Furthermore, the lysis of 1 infected bacterial cell in aquatic assemblages may produce anywhere from 6 to 124 (average 48) progeny phage (Weinbauer & Peduzzi 1994). Hence the loss of relatively few bacterial cells to lysis induced by viral infection would produce considerably more virus particles. Virus-to-bacteria ratios vary diurnally and as a result, the viral contribution to bacterial mortality should be estimated on a time scale of hours (Jiang & Paul 1994). Thus single observations per site would not fully describe the relationship between viral and bacterial abundance.

The relationship between viral abundance and DOC was not statistically significant in this study. However, DOC variation does not appear to influence bacterial production, which is more dependent on chlorophyll a and phosphorus concentrations in Québec lakes (Del Giorgio & Peters 1993). DOC has a strong influence on bacterial production in Swedish lakes (Tranvik 1992). The range of DOC observed in these lakes is much greater than what is observed in Québec lakes, and the inverse is true for chlorophyll and phosphorus concentrations. It is therefore possible that viral abundance is correlated with DOC concentration in lakes where DOC has a strong influence on bacterial production.

The models presented here confirm that viral abundance is a predictable parameter among aquatic systems over a large trophic range. Abundances appear to be strongly affected by those parameters which control bacterial production. There is some indication that viruses are more abundant in freshwater than salt, than can be explained by differences in system productivity. The virus-to-bacterium ratio varies widely, from roughly 1 to 80, raising the interesting possibility that viruses may be the most dynamic component of aquatic communities. Current data suggest that there is very little systematic change in this ratio along a trophic gradient, however, indicating that viruses may be equally important to bacterial dynamics in all aquatic environments.

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