Dynamics and Distribution of Cyanophages and Their Effect on Marine *Synechococcus* spp.†

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Cyanophages infecting marine *Synechococcus* cells were frequently very abundant and were found in every seawater sample along a transect in the western Gulf of Mexico and during a 28-month period in Aransas Pass, Tex. In Aransas Pass their abundance varied seasonally, with the lowest concentrations coincident with cooler water and lower salinity. Along the transect, viruses infecting *Synechococcus* strains DC2 and SYN48 ranged in concentration from a few hundred per milliliter at 97 m deep and 83 km offshore to ca. 4×10^5 ml^{-1} near the surface at stations within 18 km of the coast. The highest concentrations occurred at the surface, where salinity decreased from ca. 35.5 to 34 ppt and *Synechococcus* concentrations were greatest. Viruses infecting strains SNC1, SNC2, and 838BG were distributed in a similar manner but were much less abundant (<10 to >5 × 10^3 ml^{-1}). When *Synechococcus* concentrations exceeded ca. 10^3 ml^{-1}, cyanophage concentrations increased markedly (ca. 10^2 to >10^5 ml^{-1}), suggesting that a minimum host density was required for efficient viral propagation. Data on the decay rate of viral infectivity d (per day), as a function of solar irradiance I (millimoles of quanta per square meter per second), were used to develop a relationship ($d = 0.2610I - 0.00718$; $r^2 = 0.69$) for conservatively estimating the destruction of infectious viruses in the mixed layer of two offshore stations. Assuming that virus production balances losses and that the burst size is 250, ca. 5 to 7% of *Synechococcus* cells would be infected daily by viruses. Calculations based on contact rates between *Synechococcus* cells and infectious viruses produce similar results (5 to 14%). Moreover, balancing estimates of viral production with contact rates for the farthest offshore station required that most *Synechococcus* cells be susceptible to infection, that most contacts result in infection, and that the burst size be about 324 viruses per lytic event. In contrast, in nearshore waters, where ca. 80% of *Synechococcus* cells would be contacted daily by infectious cyanophages, only ca. 1% of the contacts would have to result in infection to balance the estimated virus removal rates. These results indicate that cyanophages are an abundant and dynamic component of marine planktonic communities and are probably responsible for lysing a small but significant portion of the *Synechococcus* population on a daily basis.

It has been established, by using a number of independent approaches, that viruses can be significant agents of mortality for heterotrophic marine bacteria (3). For example, estimates based on the proportion of visibly infected bacterioplankton suggest that 3 to 31% of free-living bacteria are infected by viruses (16). Also, high removal rates of virus particles in seawater imply high rates of viral production and hence significant mortality due to viral infection (4). Similarly, rates of destruction of viral infectivity in the absence of sunlight suggest that 4 to 13% of bacteria are infected on a daily basis (23). If the loss of infectivity as a result of solar radiation is also included, these estimates might increase twofold when integrated over a 10-m coastal water column. Empirical measurements of viral production in a variety of coastal and offshore environments also imply that viruses are a significant source of mortality for marine bacteria (20).

Cyanobacteria of the genus *Synechococcus* are important primary producers and account for a substantial portion of the carbon fixation in the world's oceans (6, 8, 25). Although viruses which infect freshwater cyanobacteria have been known for some time (17), attempts to isolate cyanophages from seawater have been relatively rare (12, 22), and it has only

recently been realized that cyanophages that infect *Synechococcus* cells make up an extremely abundant (21, 24) and genetically diverse (27) component of marine planktonic communities. Despite their potential importance, there have been few estimates on the impact of viruses on *Synechococcus* mortality. The initial interpretation of observations that ca. 1.5% of *Synechococcus* cells contained virus-like particles was that viruses might be responsible for 30% of cyanobacterial mortality (15). However, interpretation of the data is very sensitive to the portion of the lytic cycle during which virus particles are visible, and the actual percentage of cells that were infected was probably overestimated (3, 24).

Given the evidence that a significant proportion of *Synechococcus* cells may be lysed by viruses, studies were initiated to examine the abundance of infective cyanophages in the Gulf of Mexico near the coast of Texas. The goals were to determine seasonal and spatial patterns of cyanophage abundance, as well as the potential impact of viruses on *Synechococcus* mortality in nature. As virus production rates must approximate virus loss rates over time, the number of *Synechococcus* cells lysed can be estimated from the number of cyanophages removed divided by the burst size (4, 23). In this paper, estimates of cyanophage destruction rates and virus-host contact rates calculated from transport theory are used to ascertain the impact of viruses on *Synechococcus* mortality. These data further emphasize the role of virus-mediated processes in the cycling of nutrients and energy in marine ecosystems.

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MATERIALS AND METHODS

Study sites and sampling. The abundance of cyanophages that infect specific *Synechococcus* strains was determined at nine stations along a transect in the Gulf of Mexico on 20 June 1992 and seasonally in seawater samples collected from the pier of the Marine Science Institute.

The transect originated at the Marine Science Institute in Port Aransas, Tex. (27°50′N, 97°02′W), and extended 83 km offshore (27°21′N, 97°27′W). Stations 1 through 9 were numbered consecutively from the most inshore to the most offshore station, corresponding to depths of 11 and 99 m. Depth profiles were collected at four stations (stations 3, 5, 7, and 9) located at 16.7, 42.6, 64.8, and 83.3 km along the transect, corresponding to bottom depths of 23, 45, 73, and 99 m, respectively. Cyanophage and cyanobacterium concentrations were estimated directly from seawater samples collected from the surface in buckets or from depth in Go-Flo bottles mounted on a rosette equipped with a Sea-Bird CTD (model SBE 9 plus). The bottles were deployed in the closed position, and samples were collected on the upcast, based on the salinity and temperature data that were recorded on the downcast.

Seasonal data on cyanophage concentrations were obtained by assaying the concentration of infectious cyanophages in natural virus communities that were concentrated by ultrafiltration from seawater collected from the Marine Science Institute pier (21). Depending on tide, rainfall, wind, and season, the water varies from low salinity (<20 ppt) and estuarine (3.0 to 10.0 μg of chlorophyll *a* liter^{-1}) to high salinity (37 ppt) and oligotrophic (0.1 to 1.0 μg of chlorophyll *a* liter^{-1}).

Estimation of cyanophage and cyanobacterium concentrations. The concentration of viruses infecting five marine *Synechococcus* strains (SNC2, SNC1, SYN48 = WH6501, 838BG = WH8007, and DC2 = WH7803) was estimated by adding aliquots of exponentially growing cyanobacteria to each well of a 96-well microtiter plate, followed by one of several dilutions of the sample whose titer is to be determined (21). The concentration of viruses was determined directly from natural seawater samples (offshore transect) or in natural virus communities that had been concentrated from seawater by ultrafiltration (seasonal data). Each dilution was replicated eight times, and each assay was duplicated for a total of 16 replicates at each dilution. Controls received no virus addition. The plates were incubated under continuous irradiance (10 to 15 μmol quanta m^{-2} s^{-1}) at 25°C, and the wells were monitored for 7 to 10 days for evidence of lysis. Material in wells that did not clear after 10 days was propagated into fresh exponentially growing cultures and monitored for another 7 days. Cultures which did not clear after propagation were scored as negative for the presence of lytic viruses. Examination of representative wells at the highest dilutions by transmission electron microscopy always confirmed that lysis was attributable to viruses (21). The number of wells in which lysis occurred, or did not occur, was scored, and the concentration of infective units and the error associated with the estimates were determined by using a BASIC program (5). Estimates obtained from concentrated natural virus communities were converted to ambient abundances by assuming that the viruses were concentrated from seawater with 100% efficiency, because concentration efficiencies were not available for most dates. Comparisons of cyanophage abundances in seawater and in ultrafiltration retentate indicated that the recovery efficiency of the cyanophages varied between 13.3 and 60.4%; hence, the cyanophage concentrations in water collected from the Institute pier are probably underestimated by 1.7 to 7.5 times.

Cyanobacteria used in the assays were isolated from the coastal waters of Texas (SNC1 and SNC2) or obtained from the culture collection at Bigelow Laboratory (DC2, SYN48, and 838BG). DC2, SYN48, and SNC2 are red strains possessing phycoerythrin, whereas 838BG and SNC1 are blue-green strains with phycocyanin as the dominant pigment.

Cyanobacteria in the water samples taken during the offshore transect were enumerated by using epifluorescence microscopy (21). The samples were heavily dominated by yellow-autofluorescing cyanobacteria (red strains); hence, prochlorophytes (red fluorescence) would not have biased estimates. The concentrations of cyanobacteria in surface waters were not estimated, because the autofluorescence of cyanobacteria in these samples was extremely weak and variable.

Virus decay rates. The effect of solar radiation on virus decay rates was determined by aliquoting marine bacteriophages (LMG1-P4, PWH3a-P1, LB1VL-P1b, and H40/1) and a cyanophage (S-PWM1) into natural or ultrafiltered (molecular weight cutoff, 30,000) seawater and incubating the samples at ambient temperature in sunlight. With the exception of H40/1, which was isolated from the North Sea (2), the viruses were isolated from the coastal waters of Texas. Detailed protocols are described elsewhere (23). Briefly, the viruses were exposed to full sunlight or to sunlight attenuated by neutral-density screening. The concentration of infective viruses was monitored over time by a plaque assay for the bacteriophages (23) or by a most-probable-number assay for the cyanophage (see above). Incident quantum irradiance (400 to 700 nm) was integrated over each period by using a Licor Li-1000 quanta data logger and cosine collector.

The effect of solar radiation on virus decay rates in situ was calculated by estimating the decay rate at the surface from the incident irradiance averaged over 24 h and assuming that decay at depth was proportional to the penetration of biologically damaging radiation (23); therefore, the decay rate d at depth z as a result of sunlight can be approximated from d_0e^{-kz} where d_0 is the decay rate at the surface and k is the attenuation coefficient for damaging radiation. k was estimated from water transparency by using a relationship for "typical oceanic" waters (1) in which k^{-1} is 0.215 times the depth at which a Secchi disc disappears. The decay rates over 24 h were integrated over the mixed depth to determine the impact of sunlight on cyanophages in the surface layer.

Near the shore, where water transparency was much lower, removal rates would be dominated by processes associated with particles (23). Consequently, it was assumed that nearshore decay rates of infectivity would be similar to those that we had measured in similar water types (range, 0.26 to 0.67 day^{-1}; average, 0.45 day^{-1}) and which are in agreement with reports of others (3).

Effect of cyanophages on *Synechococcus*. The impact of viruses on *Synechococcus* strains was estimated by assuming that the removal of infectious viruses was balanced by virus production over 24 h. The number of cells that would have to be lysed in order to balance the virus removal rate was calculated by dividing the number of viruses produced over 24 h by a burst size of 250, which was based on data for a marine cyanophage (21).

The rate at which infectious cyanophages encountered *Synechococcus* cells was also calculated, by using transport theory (13); it was assumed that every contact resulted in infection. This provided an upper limit to the number of cells that could be infected per day for measured *Synechococcus* and cyanophage abundances. Contact rates, R (contacts per milliliter per day), were calculated as $(Sh2\pi wD_v)VP$, where Sh is the Sherwood number (dimensionless) for *Synechococcus* strains
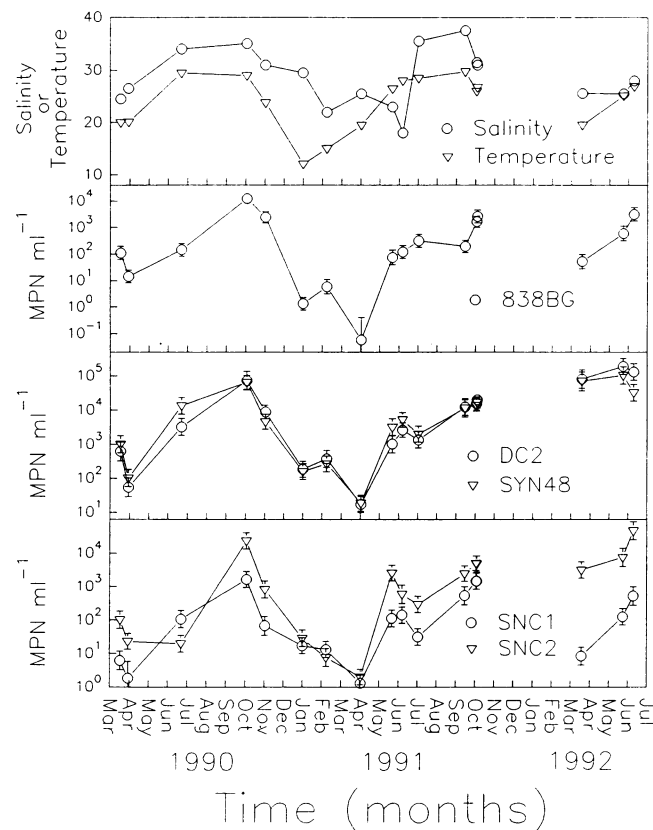
FIG. 1. Temperature, salinity, and most probable number of cyanophages infecting five marine *Synechococcus* strains (838BG, DC2, SNC1, SNC2, and SYN48) in coastal seawater collected from the pier of the Marine Science Institute.

(1.01), w is the cell diameter (1.5 × 10^{-4} cm), D_v is the diffusivity of the viruses (3.456 × 10^{-3} cm^2 day^{-1}), V is the concentration of infectious viruses (per cubic centimeter), and P is the concentration of *Synechococcus* cells (per cubic centimeter). The origins of the parameter estimates are summarized elsewhere (13).

RESULTS

Cyanophage abundance and distribution. Infective cyanophages were present in every sample assayed at concentrations ranging from a few to 4 × 10^5 ml^{-1}, although the abundance depended strongly on the host that was screened (Fig. 1 and 2).

Seasonally, infective cyanophages were most abundant when temperature and salinity were highest but occurred over the entire range of salinity (18 to 37.5 ppt) and temperature (12 to 30°C) assayed (Fig. 1). Viruses infecting the red oceanic isolates (DC2 and SYN48) were always present in the highest concentrations and ranged from a few to 1.9 × 10^5 ml^{-1}. In contrast, viruses infecting a red strain isolated inshore (SNC2) were about 1 order of magnitude less abundant and were present in similar concentrations to those of one of the green strains (SNC1).

In the Gulf of Mexico the concentration of viruses generally decreased with increasing distance offshore and below depths of 20 to 30 m (Fig. 2), where an increase in salinity and decrease in temperature resulted in a pronounced pycnocline

(Fig. 3). As was observed for pier samples, viruses that infected DC2 and SYN48 were most abundant and ranged in titer from ca. 10^2 ml^{-1} at 97 m deep and 83 km offshore to 4 × 10^5 ml^{-1} at the surface 11 km offshore. Viral abundance was related to the distribution of *Synechococcus* cells, with the highest concentrations of both associated with water of slightly lower salinity near the shore. There was a threshold of about 10^3 *Synechococcus* cells ml^{-1}, above which the abundance of infectious cyanophages increased sharply (Fig. 4). For example, concentrations of viruses infecting DC2 and SYN48 increased from about 10^2 to 10^5 ml^{-1}.

Virus decay rates. The effect of sunlight on the decay rates of five different marine viruses (four bacteriophages and one cyanophage) was determined from 106 independent estimates, collected at different times of the year and over a range of irradiances (Fig. 5). The decay rate d (per day) of infectivity was dependent on irradiance I (millimoles of quanta per square meter per second), although there was considerable scatter in the relationship ($d = 0.2610I - 0.00718$; $r^2 = 0.69$). Much of this variation probably stemmed from differences in decay rates among different viruses at a given irradiance (23) and occurred because the effect of temperature on viral decay and the effect of seasonal and hourly changes in solar angle on the reflectance of sunlight from the water's surface were not accounted for. Since the decay rate was related to the radiation received, the decay rate in situ was estimated from the solar radiation at the surface and the extinction coefficient for biologically damaging radiation. The average quantum irradiance (400 to 700 nm) over 24 h at the surface on the date of the cruise was 685 μmol m^{-2} s^{-1}, corresponding to an average daily decay rate for viral infectivity at the surface of 0.17 h^{-1} or 4.08 day^{-1}. Variations in the attenuation coefficient of the seawater and the depth of mixing resulted in a range of calculated decay rates along the transect. Solar radiation would have been responsible for decay rates of viruses in the mixed layer that ranged from 0.12 day^{-1} in the relatively turbid inshore waters (station 3) to 2.01 day^{-1} at the farthest offshore station (station 9) (Table 1).

Synechococcus infection rates. The percentage of *Synechococcus* cells infected daily was estimated from the mortality that would be required to support the inferred virus production rates and from calculated contact rates between *Synechococcus* cells and infectious cyanophages.

Estimated average viral decay rates in the mixed layer attributable to solar radiation varied from 2.01 day^{-1} at the farthest offshore station to 0.12 day^{-1} inshore, corresponding to the removal of 30,753 and 30,192 cyanophages ml^{-1} day^{-1}, respectively (e.g., 2.01 day^{-1} × 15,300 viruses ml^{-1}). To support these decay rates, assuming a burst size of 250 (21), 123 and 121 *Synechococcus* cells ml^{-1} (6.6 and 0.2% of the population, respectively) would be lysed daily (Table 1). Smaller burst sizes would require a proportionately larger percentage of the population to be lysed. These are minimum estimates, because they are based on the conservative assumptions that the concentrations of viruses which lyse DC2 are representative of the entire assemblage of infectious cyanophages and that solar radiation is the only mechanism responsible for the decay of infectivity.

At each station where depth profiles were taken (stations 3, 5, 7, and 9) the proportion of *Synechococcus* cells that could be infected per day by phages was calculated from transport theory (Table 2), assuming that the titer of viruses infecting DC2 approximated that of the entire cyanophage community and that all *Synechococcus* cells were susceptible to infection by viruses which lyse DC2. At offshore stations 7 and 9, the calculated proportion of cells that could be infected each day,
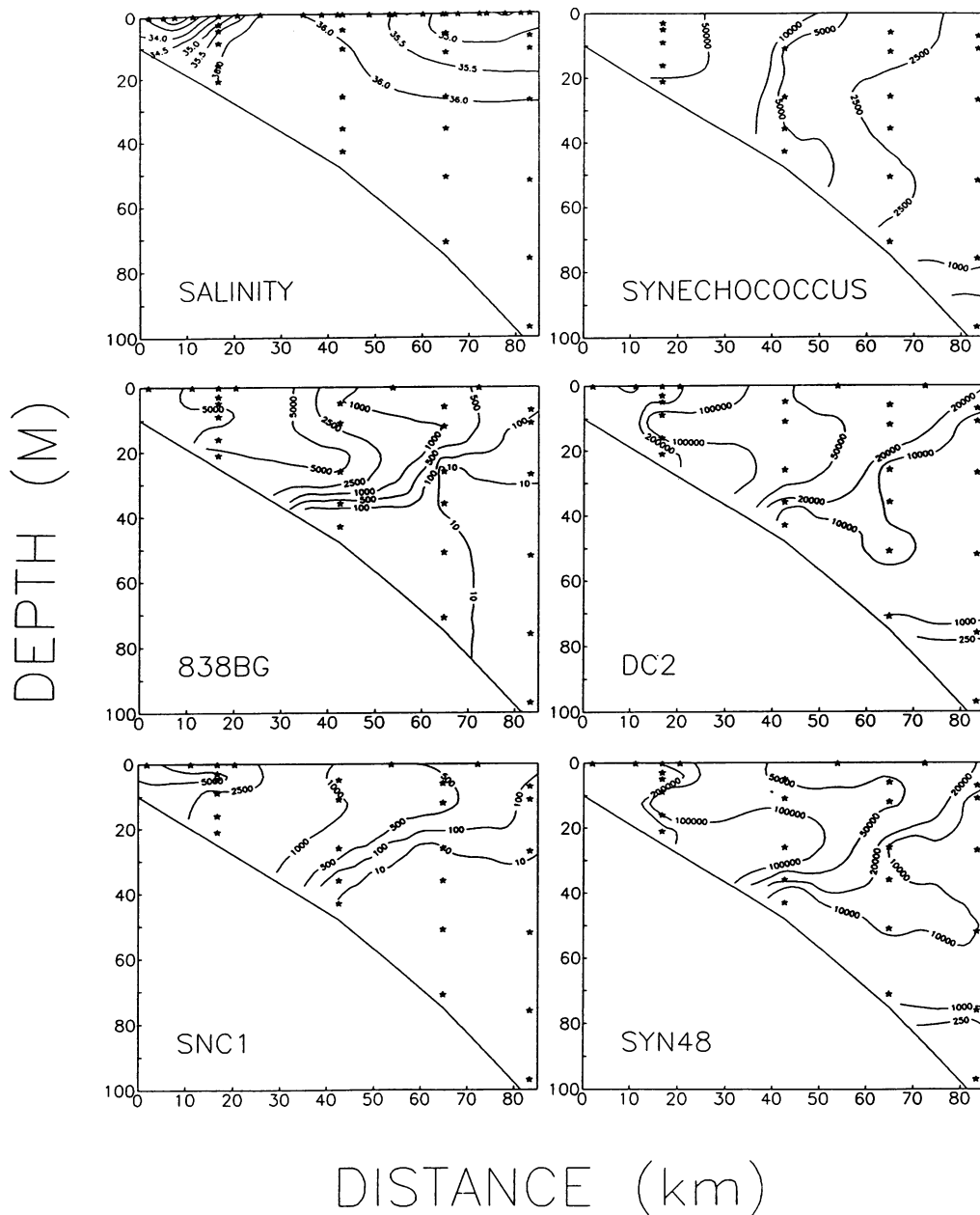
FIG. 2. Salinity, *Synechococcus* concentration (cells per milliliter), and abundance of cyanophages (viruses per milliliter) infecting four *Synechococcus* strains (838BG, DC2, SNC1, and SYN48) along a transect in the western Gulf of Mexico. Stars indicate the locations and depths at which samples were taken.

in the mixed layer, was 14 and 5%, respectively. In the surface waters at the most inshore station, >80% of *Synechococcus* cells were estimated to encounter infectious cyanophage each day.

Burst size. Since the concentrations of *Synechococcus* cells and cyanophages were known, as well as the contact rates (Table 2), it was possible to calculate the burst size required to produce enough infectious virus to balance the estimated decay rate. For example, in the surface waters of offshore stations 7 and 9, 39,997 and 30,753 viruses ml^{-1} day^{-1}, respectively, would have to be produced to balance the estimated virus decay due to solar radiation (0.94 and 2.01 day^{-1},

respectively). Calculated contact rates between infectious cyanophages and *Synechococcus* cells over the mixed depth at these stations were 437 and 95 collisions ml^{-1} day^{-1}, respectively (Table 2). Consequently, the burst size required to balance the estimated decay is 92 and 324 viruses, respectively. In inshore waters the decay rates are much less certain, precluding the estimation of burst sizes.

DISCUSSION

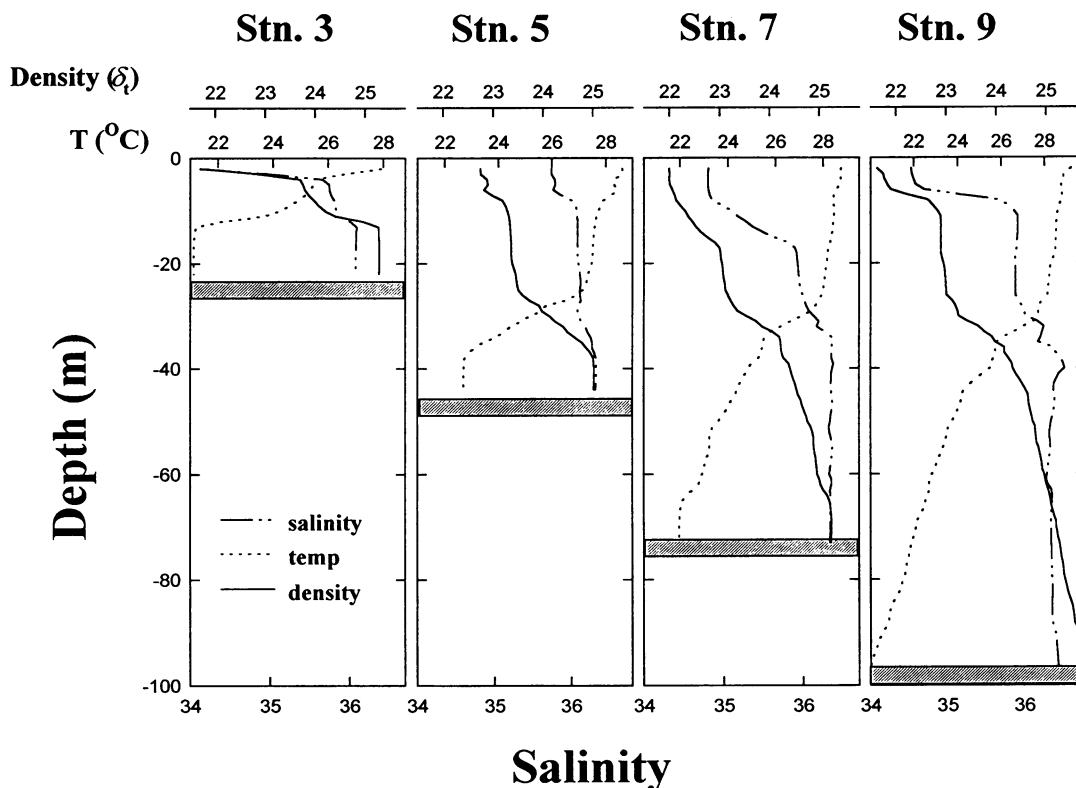The data presented in this paper further demonstrate that infectious cyanophages are an abundant and dynamic compo-

FIG. 3. Salinity, temperature, and density of the water at each station where subsurface samples were collected for enumerating *Synechococcus* cells and infectious cyanophages. The bottom is indicated by the bars.

nent of marine waters and suggest that there is a threshold in *Synechococcus* abundance beyond which concentrations of cyanophages increase dramatically. Moreover, calculations based on decay rates of infectious viruses in seawater and contact rates between *Synechococcus* cells and cyanophages imply that viruses can be a significant source of *Synechococcus* mortality. Because *Synechococcus* species are major primary producers in coastal and oceanic environments, these data provide evidence that cyanophages are important players in marine biogeochemical cycles.

Abundance and distribution of cyanophages. The highest concentrations of cyanophages (Table 2) were >20 times those given in previous reports (21, 24). The abundance also depended on the *Synechococcus* strain used as an assay organism and was highest for offshore isolates that possess phycoerythrin as their dominant pigment (DC2 and SYN48). As previously found (21), the titers were generally highest for SYN48, although results obtained with SYN48 and DC2 were very similar, suggesting that the subset of the cyanophage community that was assayed by using each host was also very similar. However, the titers were not identical, because the host ranges of viruses isolated for each strain do not necessarily overlap (21), and the estimated concentration of cyanophages determined by using each host were significantly different on occasion (95% confidence intervals did not overlap). Since DC2 and SYN48 are open-ocean isolates, whereas the others are not, this suggests that oceanic strains may be less resistant to viral infection. This makes intuitive sense; as the concentrations of infectious cyanophages and *Synechococcus* cells are much lower in offshore than onshore environments (Fig. 2), the rate at which viruses will contact host cells is much lower

(Table 2), resulting in less selection for the *Synechococcus* strains to be resistant. Cyanophage concentrations were temporally and spatially variable and, as has been previously shown (21, 24), most abundant when water temperatures were high (Fig. 1). Also, the abundance decreased with increasing distance offshore and below the thermocline, but infectious cyanophages were found even at the deepest point sampled (97 m deep).

Consistent with other observations, the highest concentrations of cyanophages were found where the *Synechococcus* abundance was greatest (Fig. 2); however, the ratio of infectious cyanophages to *Synechococcus* cells was much higher than previously reported (21, 24). Prior data indicated that cyanophage densities were about 1/10 those of *Synechococcus* cells (21, 24); however, in this study the concentration of viruses exceeded that of *Synechococcus* cells by as much as six to eight times. Consequently, it is not necessary that natural communities of *Synechococcus* strains, and the phages that infect them, mimic steady-state chemostat phage-host systems in which phage-resistant strains ultimately dominate (cf. 24).

Furthermore, there was a threshold in the *Synechococcus* abundance of ca. 10^3 cells ml^{-1}, above which the concentration of phages infecting DC2 and SYN48 increased about 100-fold (Fig. 4). A similar relationship was also observed for data collected during a previous cruise in the Gulf of Mexico (21), where the concentrations of cyanophages decreased about 10-fold when the *Synechococcus* abundance decreased by about half (6 × 10^4 to 3 × 10^4 ml^{-1}). Intuitively, lytic viruses should persist when the average time required to contact and infect a host is less than the average time for virus decay, and data from other natural systems suggest that viral propagation
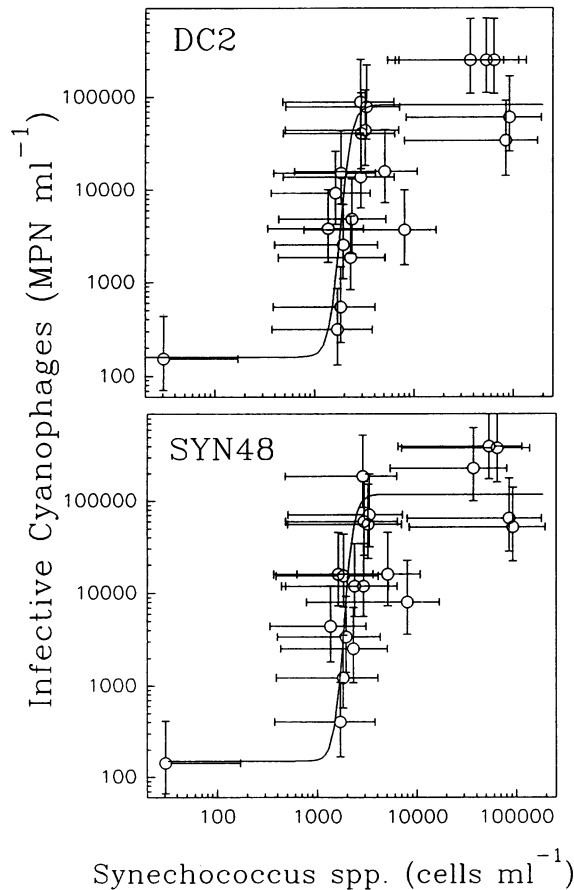
FIG. 4. Concentrations of *Synechococcus* cells and cyanophages that infect *Synechococcus* strains DC2 and SYN48 along the transect in the Gulf of Mexico. The figure is a compilation of data from all stations and depths.
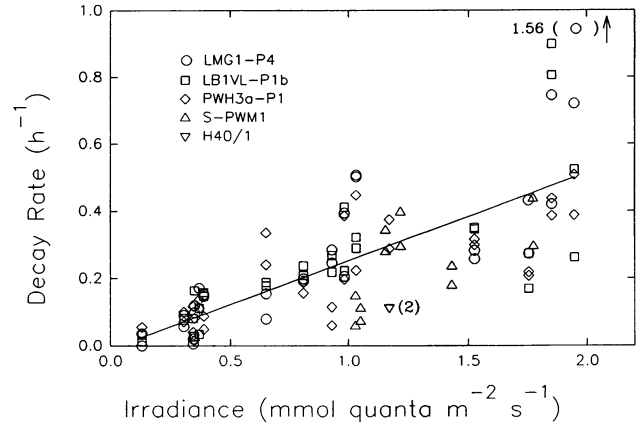

FIG. 5. Effect of sunlight on the decay rate of infectivity of a variety of marine bacteriophages (LMG1-P4, LB1VL-P1b, PWH3a-P1, and H40/1) and a cyanophage (S-PWM1); $(d = 0.2610I - 0.00718; r^2 = 0.69; n = 106)$.

can occur at host densities as low as 10 ml^{-1} (7). In contrast, in some studies with cultured bacteriophage, rapid viral propagation did not occur until host densities exceeded 10^4 cells ml^{-1} (26). For the natural community of *Synechococcus* strains investigated, the results suggest that host densities of $>10^3$ ml^{-1} allowed for efficient viral propagation.

Impact of cyanophages on *Synechococcus* cells in nature. Because there are relationships between viral decay rate and solar radiation (Fig. 5) (23) and for the attenuation of biolog-

ically damaging radiation with depth (1, 19), it is possible to calculate the proportion of *Synechococcus* cells that must be infected daily to balance the inferred viral decay rates in the mixed layer, given an average daily quantum flux (14, 23). For the farthest offshore station (station 9), solar radiation was probably the major process responsible for the decay of infectivity, because waters of high transparency have a low concentration of particulate material, which appears to be the other factor primarily responsible for viral decay (9–11, 18, 23). For station 9 the calculated average daily decay rate in the mixed layer (7.5 m) for viruses was 2 day^{-1} (Table 1). Given that there were 15,300 viruses ml^{-1} in the mixed layer which infected DC2, it follows that the rate of viral production required to balance the daily removal of viruses would be ca. 15,300 × 2, or 30,600, viruses ml^{-1} day^{-1}. If the burst size is 250 viruses (21), 122 cells ml^{-1} day^{-1} would have to be infected to balance the rate of virus removal. Since there were 1,878 *Synechococcus* cells ml^{-1}, this implies that in a steady-state system, 6.5% of the *Synechococcus* cells would have to be infected daily. This is remarkably similar to the estimate of 5% based on contact rates between cyanophages and *Synechococcus* cells at station 9 (Table 2) and for waters of the northwestern Atlantic (24).

These results also agree with independent estimates based on electron-microscopic observations of visibly infected cells (15). Since 0.8 to 2.8% (mean, 1.5%) of *Synechococcus* cells contained mature viruses, Proctor and Fuhrman (15) suggested

TABLE 1. Calculated percentages of *Synechococcus* cells that must be infected daily to balance viral decay from solar radiation, over the estimated depth of mixing, for a range of assumed burst sizes

Station	k^a (m^{-1})	$Z_m{}^b$ (m)	*Synechococcus* concn (cells ml^{-1}, 10^3)	Cyanophage concn[c] (viruses ml^{-1}, 10^3)	Decay rate[d] (day^{-1})	% of cells infected/day at burst size of:		
						50	100	250
3	4.65	7.5	59	252	0.12	1.0	0.5	0.2
5	0.52	30.0	3.1	84	0.28	15.1	7.5	3.0
7	0.31	14.0	3.1	43	0.94	25.7	12.9	5.1
9	0.22	7.5	1.9	15	2.01	32.8	16.4	6.6

[a] Estimated attenuation coefficient for biologically damaging radiation.
[b] Estimated depth of mixing.
[c] Concentration of cyanophages that infect *Synechococcus* strain DC2.
[d] Average decay rates over 24 h for cyanophages in the mixed layer resulting from solar radiation (average surface irradiance over 24 h was 0.685 mmol m^{-2} s^{-1}).

TABLE 2. Percentage of *Synechococcus* cells contacted by
infectious cyanophage each day, estimated from the abundances of
Synechococcus cells and cyanophages which infect strain DC2

Station	Depth (m)	*Synechococcus* concn (cells ml^{-1}, 10^3)	Cyanophage concn[a] (viruses ml^{-1}, 10^3)	R[b]	% of *Synecho- coccus* cells contacted/day
3	3	64	252	53.110	82.9
	5	53	251	44.110	82.6
	9	84	34	9.478	11.2
	16	92	61	18.430	20.0
	21	37	252	30.670	82.9
5	11	3.3	79	0.857	25.9
	26	2.9	89	0.854	29.3
	36	5.2	16	0.273	5.2
	43	8.0	3.7	0.099	1.2
7	6	3.3	44	0.478	14.5
	12	2.9	41	0.395	13.5
	26	2.0	2.6	0.017	0.8
	36	1.6	9.3	0.049	3.0
	51	2.9	14	0.132	4.5
	71	1.9	0.54	0.0034	0.2
9	7	1.9	15	0.095	5.0
	11	2.3	1.8	0.014	0.6
	27	1.4	3.8	0.017	1.3
	52	2.4	4.9	0.038	1.6
	76	1.8	0.31	0.0019	0.1
	97	0.03	0.15	0.00002	0.1

[a] Concentration of cyanophages that infect *Synechococcus* strain DC2.

[b] Calculated contact rates between cyanophages and *Synechococcus* cells (10^3 contacts per milliliter per day).

that about 15% of the cells would be lysed by phage. However, if a more appropriate conversion factor is used (3, 24), ca. 1.5 to 6% of the cells are estimated to be infected. When taken together, the results from these studies suggest that in relatively transparent waters ca. 2 to 7% of *Synechococcus* cells are infected by viruses on a daily basis.

An important conclusion is that most *Synechococcus* cells at the offshore stations were susceptible to infection by viruses which infect DC2, otherwise contact rates would be inadequate to produce enough virus to balance viral removal rates. If a small proportion of *Synechococcus* cells were susceptible to infection, much larger burst sizes, much lower viral decay rates, or much higher contact rates would be required. It is unlikely that burst sizes could be much larger; even a burst size of 325 reduces the percentage of cells that must be infected by only 1.5%. If the burst size was less than 250, contact rates would not be adequate to support the required infection rate, even if all *Synechococcus* cells were susceptible to infection; hence, 250 is probably a reasonable estimate of burst size in natural *Synechococcus* communities. It is also unlikely that decay rates were much lower, because solar radiation was the only decay mechanism included in the calculations. If other processes were important, burst sizes would have to be larger or contact rates would have to be higher. Finally, the transport theory used to calculate contact rates is relatively well understood (13). Consequently, a scenario in which a small subset of the *Synechococcus* community is responsible for most of the cyanophage production seems untenable for the offshore population (24).

A different conclusion is possible for inshore *Synechococcus* populations. Rates of viral decay in the mixed layer as a result of solar radiation greatly decrease in the more turbid nearshore water; however, the percentage of cells that would have

to be lysed in order to support the decay rates remained similar at stations 5 and 7, because of the increase in the concentration of cyanophages (Table 1). At the most inshore station (station 3), decay rates would probably be similar to those measured in water of similar type, or ca. 0.45 day^{-1} (23). Consequently, ca. 113,220 viruses ml^{-1} day^{-1} (0.45 day^{-1} × 251,600 viruses ml^{-1}) would have to be produced to balance the decay rate estimates. Assuming a burst size of 250 viruses, 453 cells ml^{-1} day^{-1} (or 0.2% of the *Synechococcus* cells) would have to be infected daily. Given that ca. 83% of *Synechococcus* cells would be contacted by infective cyanophages each day (Table 2), this implies that ca. 1% of the contacts would result in infection, suggesting that most cells are probably resistant to the most abundant phages. This may explain the results of Waterbury and Valois (24), who found that the majority of *Synechococcus* strains isolated were resistant to infection by the most abundant viruses.

These results emphasize the abundance and dynamic nature of cyanophage communities in the sea and suggest that through lytic infection they are important factors in the cycling of nutrients and energy in the ocean. By directly affecting major primary producers, they short-circuit the flow of fixed carbon to higher trophic levels, and in the process they convert particulate organic carbon to dissolved organic carbon via cell lysis.

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