Effects of temperature and photosynthetically active radiation on virioplankton decay in the western Pacific Ocean

Wei Wei1, Rui Zhang1, Lulu Peng1, Yantao Liang1,2 & Nianzhi Jiao1

In this study, we investigated virioplankton decay rates and their responses to changes in temperature and photosynthetically active radiation (PAR) in the western Pacific Ocean. The mean decay rates for total, high-fluorescence, and low-fluorescence viruses were 1.64 ± 0.21, 2.46 ± 0.43, and 1.57 ± 0.26% h⁻¹, respectively. Higher temperatures and PAR increased viral decay rates, and the increases in the decay rates of low-fluorescence viruses were greater than those of high-fluorescence viruses. Our results revealed that low-fluorescence viruses are more sensitive to warming and increasing PAR than are high-fluorescence viruses, which may be related to differences in their biological characteristics, such as the density of packaged nucleic acid materials. Our study provided experimental evidence for the responses of natural viral communities to changes in global environmental factors (e.g., temperature and solar radiation).

The fifth report of the Intergovernmental Panel on Climate Change (IPCC) predicts that under the conditions of Representative Concentration Pathways 8.5 (RCP 8.5), global average temperatures will increase by 2.6–4.8 °C (average 3.7 °C) by the end of this century, relative to temperatures from 1986–20051. This estimated global temperature change will directly warm the marine surface water and increase vertical stratification in the oceans2. Increased vertical stratification will then slow the mixing of surface and deep water, increasing the exposure of microorganisms in the marine surface waters to high temperatures and solar irradiation2. Numerous studies have investigated how changes in temperature and light irradiation will affect the physiology and ecology of marine macro-organisms and, recently, microorganisms1. Observatory, experimental, and modelling studies have indicated that the responses of microbes to these global change factors will impact marine biogeochemical cycling3,4.

Viruses are the most abundant microorganisms in the marine environment, with ca. 4 × 10³⁰ viral particles5,6. Many studies have shown that viruses play an important role in marine microbial food webs3,7–9. The viral lysis of cells causes the release of progeny viruses and host cell components into the water, significantly increasing the cycling of dissolved organic matter (DOM). The DOM from bacterial lysates is thought to promote bacterial growth10–12 and affect bacterial diversity and community structures13–15. Recently, Jover et al. analysed the contents and proportions of carbon, nitrogen, and phosphorus in viral particles and proposed that the elements in viral particles cannot be ignored in marine biogeochemical cycles16. The decay of viral particles leads to the release of elements from the viral components to the marine DOM pool, which can be utilized by bacterioplankton and cycled within the microbial loop17. However, whether and how the global climate change factors (e.g., changes in temperature and light irradiation) affect natural viral population (virioplankton) is not clear18.

The decay processes of viruses include loss of infectivity (inactivation) and disappearance of viral particles. The rupture of the capsid shell is the major cause, leading to failure of viral adsorption onto the host cells and the ability to infect the host cells. Subject to further degradation, a decrease in the number of viral particles can be detected by flow cytometry or other techniques. In the marine environment, viral decay is dependent upon complex physicochemical and biological factors, including solar radiation, temperature, adsorption to particles,
extracellular enzymes, and capsid thickness and genome size of the virus. Solar radiation is considered one of the most important factors affecting viral inactivation and degradation. Previous studies have shown that the ultraviolet radiation (UV) in solar radiation is the underlying factor that destroys the capsid protein and nucleic acids of viral particles. Studies have also shown that increasing temperature enhances decay (or inactivation) rates of viral isolates in the laboratory. For example, the decay rates of Escherichia coli viruses are positively correlated with temperature. However, very limited information is available for the responses of natural viral communities to environmental (e.g., temperature or solar radiation) changes.

Therefore, for a better understanding of the role of viruses in marine microbial food webs in the ocean in the future, we investigated virioplankton decay rates and examined whether and how warming and solar radiation affects their decay. To do this, we set up three levels of experimental temperature treatments: in situ temperature and a 2 °C and 4 °C increase in temperature, which correspond to the present temperature and those approximately predicted for the middle and end of this century. Because it is relatively well-known that UV causes both isolates and natural viral populations to decay, in the present study, we focused on photosynthetically active radiation (PAR), which has seldom been investigated in previous studies. PAR treatments at four intensities, which may reflect the possible exposure to solar irradiation of viruses in the euphotic waters, were set up in the current study.

Results and Discussion
In this study, virus and picoplankton abundances in the western Pacific Ocean were investigated with flow cytometry. Viral decay rates and their responses to changes in temperature and photosynthetically active radiation were explored with filtration techniques.

Environmental parameters and picoplankton abundances. The western Pacific Ocean is a typical oligotrophic marine environment. The in situ environmental variables at the 16 stations including temperature, salinity, conductivity, density, chlorophyll a concentration (chl a), and turbidity are shown in Table S1. The chl a concentration was low throughout the region investigated, ranging from 0.0468 to 0.1367 mg m$^{-3}$. The temperatures ranged from 28.10 °C to 29.80 °C, and salinity ranged from 32.07 to 34.08.

The heterotrophic bacterioplankton abundance during the investigation was $7.17 \pm 0.98 \times 10^5$ ml$^{-1}$ ($n = 16$, ±SE), while autotrophic Synechococcus and Prochlorococcus abundances were $4.57 \pm 5.85 \times 10^3$ ml$^{-1}$ ($n = 16$, ±SE) and $1.93 \pm 1.68 \times 10^4$ ml$^{-1}$ ($n = 16$, ±SE), respectively. The concentration of picoeukaryotes was $4.85 \pm 3.21 \times 10^2$ ml$^{-1}$ ($n = 16$, ±SE). The highest viral abundance was observed at the southern Station P13 ($1.21 \pm 0.06 \times 10^7$ ml$^{-1}$), whereas the lowest value was recorded at Station P7 ($2.07 \pm 0.06 \times 10^6$ ml$^{-1}$). As shown in Fig. S1, the flow cytometry analysis allowed two viral groups to be distinguished: high- and low-fluorescence viruses. Low-fluorescence viruses formed the majority of the total viruses in the western Pacific Ocean, accounting for $83.93 \pm 5.88\%$ ($n = 16$, ±SE), on average (Table S1). The total, high-fluorescence, and low-fluorescence virus abundances were significantly positively correlated with chl a concentration and heterotrophic bacterial abundance ($n = 16$, $P < 0.05$, Pearson’s correlation; Table S2), suggesting that viral dynamics are closely related to and interact with both autotrophic and heterotrophic plankton.

Spatial variations in viral decay. The decay experiment for natural virioplankton was performed at six stations in the western Pacific Ocean (Fig. 1, red dots). The viral decay rates obtained were low and were similar to those observed in other marine oligotrophic ecosystems. Spatially, the total virus decay rates (Fig. 2)
ranged from 1.08 ± 0.18% h⁻¹ (corresponding to 5.92 ± 0.98 × 10⁴ ml⁻¹ h⁻¹; n = 3, ±SE) at Station N18-3 to 2.20 ± 0.52% h⁻¹ (9.90 ± 2.34 × 10⁴ ml⁻¹ h⁻¹; n = 3, ±SE) at Station P5, with an average of 1.64 ± 0.21% h⁻¹ (9.11 ± 1.17 × 10⁴ ml⁻¹ h⁻¹; n = 6, ±SE). The multivariate multiple regression analysis (DistLM-forward) revealed that the total virus decay rate was not significantly correlated with any of the biological or environmental factors in the investigation. Low-fluorescence virus decay rates ranged from 0.98 ± 0.13% h⁻¹ (4.29 ± 0.56 × 10³ ml⁻¹ h⁻¹; n = 3, ±SE) to 2.38 ± 0.51% h⁻¹ (8.23 ± 1.76 × 10⁴ ml⁻¹ h⁻¹; n = 3, ±SE) (average 1.57 ± 0.26% h⁻¹, 6.92 ± 0.56 × 10⁴ ml⁻¹ h⁻¹; n = 6, ±SE). The high-fluorescence virus decay rate ranged from 1.45 ± 0.19% h⁻¹ (1.73 ± 0.23 × 10⁴ ml⁻¹ h⁻¹; n = 3, ±SE) to 4.01 ± 0.21% h⁻¹ (6.05 ± 1.05 × 10⁴ ml⁻¹ h⁻¹; n = 3, ±SE), with a mean value of 2.46 ± 0.43% h⁻¹ (3.28 ± 0.50 × 10⁵ ml⁻¹ h⁻¹; n = 6, ±SE). The DistLM-forward analysis revealed that chl a explained 71.70% of the variability in high-fluorescence virus decay rate in the surface waters of the western Pacific Ocean (n = 6, P < 0.05, Table S3). This indicates a close relationship between autotrophic plankton and high-fluorescence viral dynamics because chl a concentration is expressed as the biomass of autotrophic plankton.

Figure 2. The total, high-, and low-fluorescence viruses are affected by warming (in situ temperature, 2 °C and 4 °C increases) in six viral decay experiment stations. Error bars indicate the standard errors calculated from triplicate sample measurements.
in general. This is consistent with previous studies that autotrophic plankton (such as alga) viruses exhibit relatively higher fluorescence signals in flow cytometry analysis. No biological or environmental factor was correlated with low-fluorescence virus decay rate, indicating that the decay behaviour of low-fluorescence viruses may be correlated with other factors, which were not measured in this investigation and need to be studied in the future. We found that the decay rates were not significantly correlated with either latitude (an indicator of solar radiation intensity) or temperature. This might be attributable to the large diel variations in solar radiation and the narrow changes in temperature in tropical and subtropical areas. Therefore, irradiance with PAR and temperature were artificially manipulated in the following experiments.

Viral decay rates with experimental increases in temperature. The experiments of different temperature treatment impacting viral decay were performed at six stations (Fig. 1, red dots). Higher decay rates were observed for total, high-, and low-fluorescence viruses under the 4 °C increase compared with the in situ temperature (Fig. 2). The maximum increase was recorded in low-fluorescence viruses at Station S1-2 (265%), and the minimum increase appeared in high-fluorescence viruses at Station P5 (22%). Significant differences in the viral decay rates were observed under + 4 °C compared to the in situ temperature (Fig. 3, ANCOVA, P < 0.01 for total and low-fluorescence virus, P = 0.05 for high-fluorescence virus). The magnitude of the increase is consistent with previous studies based on pure cultures and a meta-analysis of field observations.

Extracellular proteases and nucleases are considered to contribute to viral decay. For example, Dell’Anno et al. found that the viral decay rates were positively correlated with extracellular proteolytic activity, indicating proteases can destroy viral capsids, which are composed of proteins. Within an appropriate range, extracellular enzymes are more active at higher temperatures, at which viral decay rates are higher. Although viruses may be more resistant to thermal stress than their hosts, many isolated viruses are very sensitive to temperature, and the inactivation events occur at a low temperature. Previous studies using pure strains have also shown that the viral inactivation rates increase rapidly under warming conditions in response to thermal degradation. However, the exact mechanism by which temperature affects virus inactivation and decay is not clear. Limited information suggested that temperature may directly affect protein stability and biomolecule elasticity of viral capsid proteins or lipid membranes, and slight alteration of temperature may affect the folding and binding of proteins and nucleic acids.

Viral decay rates under an experimental PAR gradient. In the PAR gradient experiments (Fig. 1, red dots), total, high-, and low-fluorescence viruses exhibited significantly higher decay rates under the highest PAR condition than under the lower PAR conditions and in the dark (Fig. 4), and the result of statistical analysis is shown in Fig. 5 (ANCOVA, P < 0.01 for total, high-, and low-fluorescence virus). The maximum variation appeared in low-fluorescence viruses at Station S1-2 (4.64-fold), and the minimum was observed in high-fluorescence viruses at Station P15 (0.27-fold). The UV in solar radiation is a principal factor in viral variation appeared in low-fluorescence viruses at Station S1-2 (4.64-fold), and the minimum was observed in high-fluorescence viruses at Station P15 (0.27-fold). The UV in solar radiation is a principal factor in viral decay. However, few studies have shown that viral decay is also affected by PAR.

Although more energy is produced by shorter wavelengths of light (such as UV), all photons carry energy, and PAR may carry enough energy to directly affect viruses by reducing their infectivity and degrading their proteins and nucleic acids, which is supported by previous studies in which blue light (434 nm) can damage DNA. Traving et al. suggested that the light with 400–700 nm wavelengths was the major contributor to the loss of infectivity in cyanophage S-PM2. Additionally, compared with the dark condition, cool white fluorescent illumination caused a significant increase in the inactivation of Heterosigma akashiwo virus and Heterocapsa circularisquama virus. Beyond inactivation (i.e., infectivity loss), our study demonstrates for the first time that PAR increases marine virioplankton decay rates in natural environments.

To compare our data with previous studies (e.g., Wilhelm et al.), we calculated the light-induced viral decay rates by subtracting the viral decay rate in the dark from that at light conditions. The light-induced (here, PAR-induced) viral decay rates were, on average, 0.53 ± 0.19% h⁻¹ (n = 6, ±SE), 1.40 ± 0.20% h⁻¹.
Different responses of high- and low-fluorescence viruses. Higher increases in viral decay rates were always observed for low-fluorescence viruses in our experiments (Figs 3 and 5). When the +4°C condition...

Figure 4. The total, high-, and low-fluorescence virus decay rates are affected by different PAR levels (dark, 400, 640, and 880 μmol m$^{-2}$ s$^{-1}$). Error bars indicate the standard errors calculated from triplicate sample measurements.
mental factors is necessary for a better understanding of the response of virioplankton to global climate changes. 

... with consideration of virus-host interactions and their adaption/evolution to gradually changed environment on a decadal scale. Viruses and their hosts may adapt or coevolve during this process. Therefore, further investigation with consideration of virus-host interactions is essential to understanding viral dynamics in the marine microbial food web. The data reported herein show that warming and increased PAR can accelerate marine viroplankton decay rates. This enhanced decay will reduce the viral infection rate on their hosts, which will have significant biological, ecological, and biogeochemical consequences for marine microbial ecosystems. The increase in viral decay will also shunt more viral elements into the marine DOM pool, which contributes to the nutrient supply for surface bacterioplankton. We also observed that the acceleration of viral decay is more significant for low-fluorescence than for high-fluorescence viruses. Although the mechanism underlying this phenomenon requires further research, our study indicates that the responses of different viral groups to climate change factors differ, and, consequently, their impacts on microbial interactions in marine ecosystems differ. It is worthy to note that although the protocol for determination of viral abundance in natural samples was optimal\(^{44,45}\), the use of a preservative may affect the counting of viruses and then the decay rate. Furthermore, in a natural environment, viral decay is affected by biological factors such as protozoan grazing and virus-host interaction (e.g., viral production and cell lysis) in addition to temperature and PAR, which was artificially excluded in our study by filtration by 0.22 μm pore-size filter\(^{2,20}\). Additionally, the sudden manipulation of temperature and PAR in our experiment may not have simulated the actual conditions likely to occur in the future. The changes of temperature and other environmental factors are expected to be gradual, with fluctuation on a decadal scale. Viruses and their hosts may adapt or coevolve during this process. Therefore, further investigation with consideration of virus-host interactions and their adaption/evolution to gradually changed environmental factors is necessary for a better understanding of the response of viroplankton to global climate changes.

**Materials and Methods**

**Study site and sampling.** Sixteen stations were investigated during the National Natural Science Foundation of China cruise in the western Pacific Ocean (Fig. 1, black and red dots). At each station, water samples were collected from the surface (5 m depth) using a carousel water sampler carrying 12 Niskin bottles (12 litres). The samples were prefiltered through 20 μm mesh filters to remove large particles and zooplankton. A CTD profiler (SBE9/11 plus, Sea-Bird Electronics, Inc., USA) was used to obtain salinity, temperature, conductivity, density, chl a concentration, and turbidity data at all stations.

**Determination of picoplankton abundance.** Seawater samples (2 ml) were taken to determine picoplankton abundances. Subsamples were fixed with glutaraldehyde (0.5% final concentration), incubated at 4°C...
for 15 min in the dark, flash frozen in liquid nitrogen, and then stored at −80°C. Autotrophic picoeukaryote, Synechococcus, and Prochlorococcus abundances were determined by flow cytometry (Epics Altra II, Beckman Coulter), with scatter diagrams of side scatter vs. red fluorescence and orange fluorescence vs. red fluorescence, according to previously published methods. To obtain heterotrophic bacterial abundance, the samples were stained with 1.0 × 10^-4 SYBR Green I (v/v, final concentration, Molecular Probes), incubated for 15 min in the dark, and then analysed in a scatter diagram of red fluorescence vs. green fluorescence. The heterotrophic bacterial abundance equaled the total microorganism abundance, subtracting the autotrophic microorganism (i.e., picoeukaryotes, Synechococcus and Prochlorococcus) abundance. The samples for virus counting were diluted with Tris–EDTA buffer (pH 8.0; Sigma), stained with 5.0 × 10^-5 SYBR Green I (v/v, final concentration), incubated at 80°C for 10 min, cooled to room temperature, and then analysed by flow cytometry. The yellow-green fluorescent beads with 1 μm in diameter (Molecular Probes) were added as an internal reference. All data analysis was performed with the FCS Express V3 software (De Novo Software).

**Viral decay experiment.** The virioplankton decay rates were determined according to Noble and Fuhrman, after the water samples had been filtered through 0.22μm pore-size polycarbonate filters to exclude bacteria and particles >0.22μm. The filtered water (150 ml) was then dispensed in triplicate into 50 ml aseptic tubes and incubated under each experimental condition. A temperature gradient was established with dry bath incubators (MK-20, Hangzhou Allsheng, China), in situ temperature, 2°C increase, and 4°C increase, and the light experiment was performed in a light incubator (PGX-80C, Ningbo Saifu, China) with fluorescent lamps at the in situ temperature. To obtain different light intensities, the tubes were either covered with aluminium foil, a sheet of white paper, or three layers of Ziploc bags or not covered. Detection by the sensor of GER1500 was performed with the FCS Express V3 software (De Novo Software).

Subsamples (1 ml) were collected every 3h between 0 and 24h, and the viral and bacterial abundances were determined by the method described above. Since a clear growth of bacteria at 15h were observed in some of the incubations, which might result in infection of viruses and effect the estimation of viral decay rate, only data of 12 h incubations were applied to calculate viral decay rates in this study. The viral decay rate was calculated as the slope of the linear fitted curve of the decline in ln-transformed viral abundance during the 0–12 h experiment (see Fig. S3 for an example). The decay rate was expressed as a percent per hour by multiplying the slope by 100. The decay rate calculation was applied for total, high-fluorescence, and low-fluorescence viruses.

**Statistical analysis.** Pearson’s correlation analysis was used to assess the degree of correlation among the parameters investigated in SPSS Statistics 19 software (SPSS Inc., Chicago, IL, USA). Linear regression analysis was performed to assess viral decay rates at different temperatures and PAR intensities. ANCOVA analysis was performed to assess significant difference among average total, high-, and low-fluorescence viruses affected by warming (in situ temperature, 2°C and 4°C increases), and by different PAR levels (dark, 400, 640, and 880 μmol m^-2 s^-1). The distance-based multivariate analysis for a linear model using forward selection (DistLM-forward) was applied to test the relationships between viral decay rates and biotic and abiotic environmental parameters in Primer 6 software with the PERMANOVA + package (Primer-E, Plymouth, United Kingdom). The response variable was logarithmically (base 10) transformed, and the resulting data were converted into Euclidian distance similarities matrices. Fifteen variables were used to explain the variation of decay rates of high- and low-fluorescence viruses, including longitude, latitude, salinity, temperature, conductivity, density, chl a, turbidity, bacterial abundance, Synechococcus abundance, Prochlorococcus abundance, picoeukaryote abundance, total virus abundance, high-fluorescence virus abundance and low-fluorescence virus abundance.

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Author Contributions
R.Z. and N.J. conceived and designed the experiments. W.W. and L.P. performed the experiments. W.W., R.Z. and Y.L. analysed the data. W.W., R.Z., L.P., Y.L. and N.J. contributed reagents/materials/analysis tools. W.W. and R.Z. wrote the manuscript. All authors reviewed the manuscript.

Additional Information
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