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Viral dynamics II: a model of the interaction of ultraviolet light and mixing processes on virus survival in seawater

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ABSTRACT: Viruses are an important component in the functioning of marine ecosystems. They are especially vulnerable at the stage when they are free particles seeking a new host. A major factor in viral mortality during this phase is the presence of ultraviolet (UV) radiation. UV radiation penetrates only a short distance into the water column because of a very high attenuation coefficient. Processes that move viruses to the surface change their UV exposure. We have modelled the mortality of viruses subject to UV radiation by means of a Lagrangian Monte-Carlo type model that incorporates viral movements within the mixed layer. For viruses with a given UV-induced surface mortality, mixed-layer depth and UV attenuation coefficient are important factors in their water column mortality. Other more subtle factors can also affect viral mortality: nature of the diurnal thermocline; type of mixing; and the time of day that they are released into the water. Viruses not subject to mixing have their mortality rate enhanced by internal wave motion, although the absolute mortality rates may remain low. Increased UV irradiance associated with atmospheric ozone depletion could significantly change viral mortality in polar environments. UV-induced mortality can be comparable to that from biological factors such as virucidal bacteria.

KEY WORDS: Mixed layer · Model · UV · Virus mortality

INTRODUCTION

Viruses have recently been recognised as important members of the ocean's ecological community. Viruses are particularly vulnerable when suspended in seawater because they are unable to repair themselves or reproduce in the absence of a host cell. Physical processes that move them around in the life-stage between their release from infected organisms and their infection of new hosts can affect their mortality. Simulation studies help to clarify the nature of these interactions.

Marine viruses have been found in seawater at concentrations high enough to greatly reduce primary and bacterial production (Bergh et al. 1989, Proctor & Fuhrman 1990, 1992, Suttle 1992). Viruses infect larger organisms from zooplankton (Bergoin et al. 1984) to sea mammals (Haebler & Moeller 1993) but the dominant hosts are bacteria and cyanobacteria (Proctor & Fuhrman 1990). There are transport considerations that could explain the dominance of small organisms as hosts (Murray & Jackson 1992). Viruses are an important element of the marine ecosystem whose dynamics need to be understood if we are to understand the system.

Viruses can be rendered non-infective by several means. Virucidal organisms, principally bacteria, can destroy them (Fujioka et al. 1980). Heavy metals can bind to them and inactivate them in a process most important in polluted water (Bitton 1980). Viruses can bind to non-living particles, which may cause them to sediment out of the water column (Metcalfe et al.

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1974). Viruses can also be disrupted by high energy photons. The different inactivation processes vary in relative importance for controlling viral populations but they all can control viral activity. We have already considered the interaction of viruses with particles (both living and non-living), which is an essential process in the transmission of infection and a potentially important means of viral destruction (Murray & Jackson 1992). Sunlight, particularly ultraviolet radiation (UV), can also be very important for viral inactivation in the upper water column (Harm 1980, Suttle & Chen 1992). This near-surface zone is the region of the ocean in which most biological activity occurs. It is also the region where physical processes most actively move water up and down. Vertical movement of a virus changes its UV exposure. Because UV irradiance depends so much on depth, an assessment of viral inactivation should include descriptions of viral position in the water column through time.

In this paper we examine viral inactivation caused by UV. We develop a Monte-Carlo type model simulating the movement and survival of large numbers of viruses as a function of mixing regime, UV penetration and viral response to resulting UV exposure. We use the model to study expected viral mortality under different conditions.

THE MODEL

A random encounter model to describe the destruction of viruses by solar UV radiation needs to describe UV irradiance as a function of viral depth and time, UV penetration as a function of depth and time, viral inactivation as a function of UV irradiance and viral depth as a function of time (Calkins et al. 1978). We developed a Lagrangian model of viral particle motion in the mixed layer subject to different mixing regimes to provide the basic physical framework. By simulating the movement and mortality of large numbers of viruses we calculated viral survival probability over 24 h under a range of physical and ecological conditions. (Variables used in this paper are summarised in Table 1.)

Water motion

Woods & Onken (1982) developed a Lagrangian model to simulate the depth of a particle in a surface layer mixed by the separate processes of Langmuir circulation (LC) and eddy diffusion (ED). We have divided changes in viral position in the water column into changes caused by these 2 processes. The change

Table 1. Notation

Symbol	Description	Values	Units
A	Internal wave amplitude		m
d	Direction of motion in Langmuir cell	+1, −1	
D	Daylight period	12	h
D_z	Diffusion coefficient	40, 160, 640	$m^2 h^{-1}$
F	Diurnal thermocline depth factor		
k	UV attenuation coefficient	0.15, 0.5, 2	m^{-1}
M	Noon surface viral mortality rate	4.6, 1.6, 0.51, 0.05	$U_{max}^{-1} h^{-1}$
P_v	Probability of viral mortality		
r	Random direction of turbulent motion	−1, +1	
s	Integer $Z_t/\Delta L$ rounded down		
t'	Time from dawn		h
T_L	Langmuir cell cycle period	0.5, 1, 2	h
U_0	Surface UV level relative to noon level		
U_{max}	Surface noon UV level		
U_z	Relative UV at fixed depth z		
$\overline{U}_z$	Mean UV received with internal wave		
z	Depth		m
Z_{max}	Depth of permanent thermocline	10, 30, 50	m
Z_{min}	Shallowest diurnal thermocline	$0–Z_{max}$	m
Z_t	Depth of thermocline		m
ΔE	Depth change, eddy turbulence		m
ΔL	Depth change, Langmuir circulation		m
Δs	Turbulent motion step		m
Δt	Model time step	1.25, 2.5, 5	min
ΔZ	Total depth change		m
ζ_1	Random direction of turbulence	−1, +1	
ζ_2	Random mortality check	0–1	

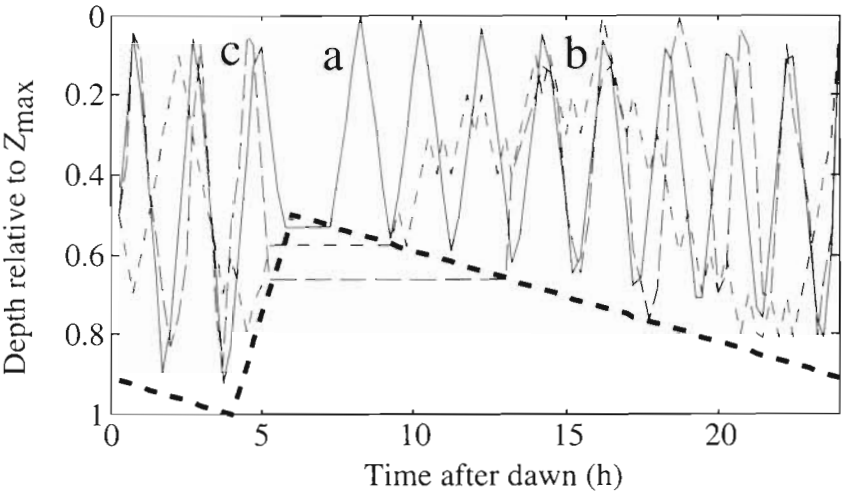

Fig. 1. Typical particle motion under (a) Langmuir circulation (——), (b) eddy mixing (– –) and (c) combined mixing (— —). Diurnal thermocline depth for $Z_{min} = 0.5\,Z_{max}$ (▬ ▬); viruses trapped beneath this line remain stationary. Notice that eddy mixing can produce long periods in which depth changes relatively little. If mortality is high then viruses remaining at depth through periods of highest UV intensity have highly enhanced survival

in viral depth ΔZ over a period Δt is given by the sum of displacements (Fig. 1)

$$\Delta Z = \Delta L + \Delta E \qquad Z_v < Z_t \qquad (1a)$$

$$= 0 \qquad Z_v \leq Z_t \qquad (1b)$$

where ΔL is displacement caused by LC, ΔE is the displacement caused by ED, Z_v is the viral depth, and Z_t is the mixed layer depth. A virus below the diurnal thermocline does not move (Fig. 1).

Langmuir circulation forms vertical circulation cells in the mixed layer that are driven by the wind (Langmuir 1938). We approximated it as consisting of 2 flows which carry a virus from the sea surface to the bottom of the mixed layer and back up. Direction reversals occurred when a virus hit the sea surface or the bottom of the mixed layer. Virus velocity was calculated as $0.5\,Z_t\,T_L^{-1}$ where T_L is the period of transport through a complete Langmuir circulation cell. Change in viral position over a small time Δt, based on the Wood-Onken model, was given by:

$$\Delta L = d\,0.5\,Z_t\,T_L^{-1}\,\Delta t \qquad (2)$$

where direction is indicated by the sign d.

Eddy diffusion is the vertical movement provided by relatively random vertical displacements. We approximated it by using a random step of constant length up or down calculated at each time interval, with the choice of direction determined by a random variable ζ_1 equally likely to be +1 or −1.

$$\Delta E = \zeta_1 \Delta s \qquad (3)$$

The step length is a function of the vertical eddy diffusion coefficient and the model time step. This is related to a diffusion coefficient D_z by (Berg 1983):

$$\Delta s = (2\,D_z\,\Delta t)^{-0.5} \qquad (4)$$

The standard diffusion coefficient in this study, taken from the Woods-Onken model, was $D_z = 160$ m^2 h^{-1} (0.044 m^2 s^{-1}). This is a representative diffusion coefficient for mixed layers (e.g. order 10^{-2} m^2 s^{-1}; Nihoul 1975).

The no-flux boundary conditions at the surface and at the bottom of the mixed layer was expressed by reflecting a particle that strikes either boundary and reversing the sign of d.

The thickness of the surface mixed layer varies through the day (Fig. 1). The mixed layer bottom is determined by the diurnal thermocline depth Z_t. We developed an equation to reproduce the patterns generated by the Woods-Onken (1982) model.

$$Z_t = Z_{max} + F(Z_{min} - Z_{max}) \qquad (5)$$

where

$$F = \frac{2D - 6t'}{144 - D} \qquad \text{if } 0 \leq t' \leq \frac{D}{3} \qquad (6a)$$

$$= \frac{6t' - 2D}{D} \qquad \text{if } \frac{D}{3} \leq t' \leq \frac{D}{2} \qquad (6b)$$

$$= \frac{2D + 144 - 6t'}{144 - D} \qquad \text{if } \frac{D}{2} \leq t' \leq 24 \text{ h} \qquad (6c)$$

where Z_{max} is the depth of the permanent or seasonal thermocline and Z_{min} is the depth of minimum mixing. The depth is controlled by F, which is a function of time from dawn t' and daylight period D in hours. Depth of the thermocline rises sharply between $t' = D/3$, (10:00 h for 12 h daylight) and noon ($t' = D/2$) and then gradually deepens throughout the rest of the day (Fig. 1).

UV irradiance

We assumed that ultra-violet irradiance at the sea surface, U_0, varied sinusoidally as a proportion of noon

UV irradiance U_{max} during daylight hours (Caldwell et al. 1980):

$$U_0 = U_{max} \sin\left(\frac{t'\pi}{D}\right) \quad \text{if } 0 < t' \leq D \quad (7a)$$

$$= 0 \quad \text{if } D < t' \leq 24 \text{ h} \quad (7b)$$

UV decreases exponentially through the water column (Gordon et al. 1984):

$$U_z = U_0 e^{-kz} \quad (8)$$

where U_z = intensity of UV irradiance at depth z and k is the UV attenuation coefficient. The value of k is 0.5 m^{-1} unless stated otherwise.

Viral mortality

Viral mortality rate for a given wavelength is linearly dependent on the dose of UV received (Rontó et al. 1992, Suttle & Chen 1992). Dose is the product of UV irradiance and exposure length. The probability P of viral mortality during a short time interval is:

$$P = M U_z \Delta t \quad (9)$$

where M is the surface noon viral mortality rate constant and $U_z \Delta t$ is the dose of UV to which the virus is exposed. P was used to determine mortality at each time step by comparison with a random variable (ζ_2) uniformly distributed between 0 and 1. A virus died if $\zeta_2 < P_t$. We used values of M = 4.6, 1.6, 0.51, and 0.05 h^{-1} to represent the mortality rate constants of UV-sensitive enteric viruses, typical marine viruses, UV-tolerant, and extremely UV-tolerant marine viruses. The significance of these values is discussed later.

Assembling the model

We simulated the movement of 10 000 viruses initially distributed randomly in the mixed layer for each case studied. Each individual virus was observed until it was destroyed or until 24 simulation hours elapsed. Viruses released at dawn were followed for only the 12 h daylight period because there was no nocturnal mortality. Position of a virus was recalculated after every time step using Eq. (1). At the same time, its survival was tested using Eq. (9) and a newly generated random number. The result of these calculations was the fraction (probability) of viruses surviving 24 h for given initial conditions.

Eq. (9) assumes that $M U_z \Delta t$ is small enough so that the population change over Δt is small. If $M U_z \Delta t = 0.1$ then mortality is only 5% exaggerated. As viral movements are discrete over Δt, numerical problems may also occur in the motion equations if this time step is too large. We have represented viral movement for LC in terms of a discrete length ΔL. One result is that a model virus has its shallowest position at a depth between 0 and $\Delta L/2$, where it stays for Δt. The mortality during this interlude can vary greatly depending on exactly what this depth is. Viruses in the real ocean move continuously over all depths and are not exposed to the highest mortality for the period Δt. This potential error caused by using discrete time steps is decreased by using small Δt. The diffusion step Δs, which also depends on Δt, must be small relative to Z_{max}. If Z_{min} tends to zero, Δs will become large relative to it. We have chosen Δt to minimise these calculation errors unless otherwise noted.

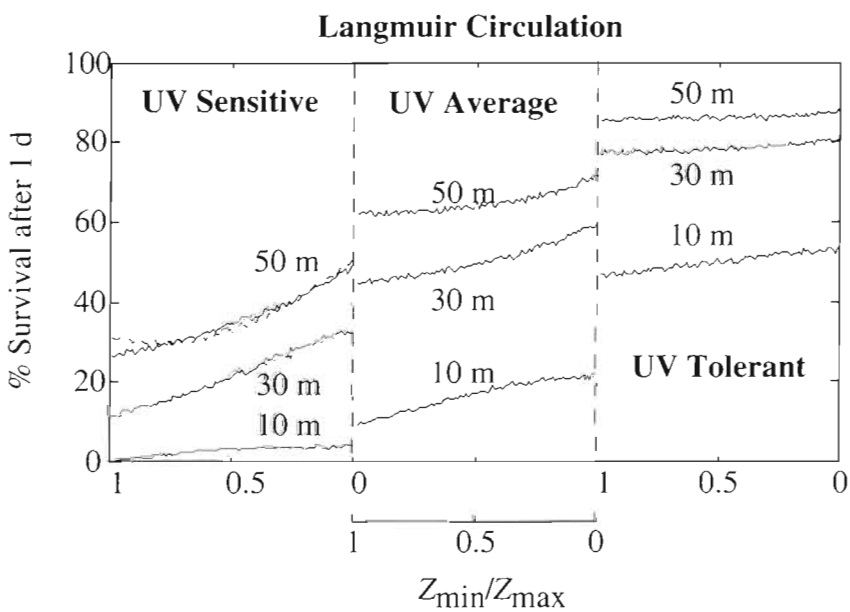

Fig. 2. Survival over 24 h of UV-sensitive, average and tolerant viruses under pure Langmuir cell mixing conditions with 10, 30 and 50 m permanent thermoclines (indicated on figure). Diurnal thermoclines vary from 1 to 0 of the permanent thermocline, i.e. from no change relative to permanent thermocline to a diurnal thermocline minimum that reaches the surface. Dashed lines separate examples of different viral UV sensitivities. Note that Δt = 1/24 h (2.5 min), except in the case of sensitive viruses with a 50 m permanent thermocline (for which 1/48 h is used). Enhanced survival of sensitive viruses for small relative changes in diurnal thermocline depth with a 2.5 min time step (shown dashed) is an artifact of time step length. Survivorship for the extremely tolerant virus is 0.93, 0.97 and 0.98 for the 10, 30 and 50 m permanent thermocline cases

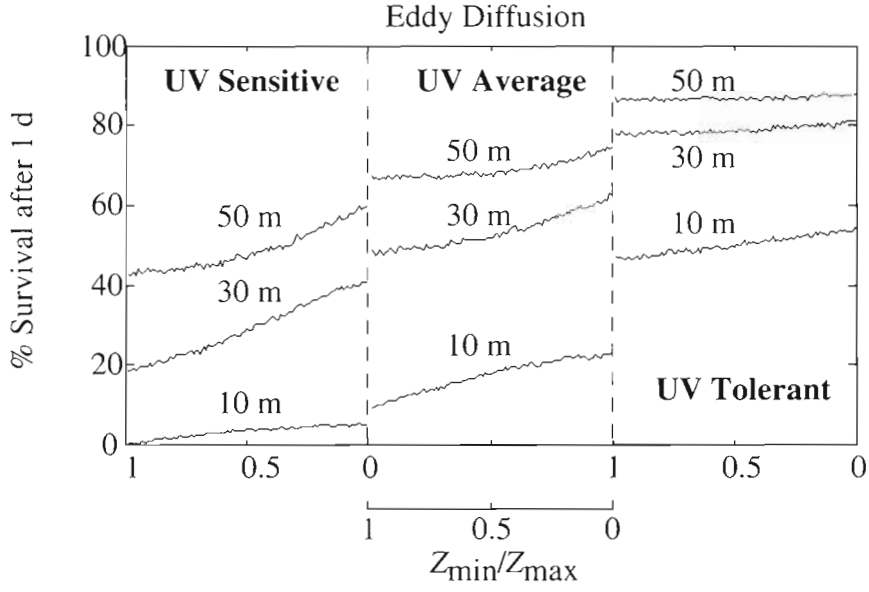

Fig. 3. Survival of viruses under pure eddy diffusion. Notation and conditions as in Fig. 2. Survivorship for the extremely tolerant virus is 0.93, 0.97 and 0.99 for the 10, 30 and 50 m permanent thermocline cases. Notice survival is much greater and the importance of the diurnal thermocline for survival is less for sensitive viruses than in Figs. 2 or 4

RESULTS

Typical viral survival in the presence of Langmuir circulation increased with mixed layer depth (Fig 2). For a 50 m deep thermocline, the minimum survival fraction after 1 d was slightly more than 0.6. This decreased to about 0.4 when the thermocline was 30 m and less than 0.1 when the thermocline was 10 m. When the diurnal thermocline became a smaller fraction of Z_{max}, survival increased to about 0.7, 0.6 and 0.2 for the 50, 30 and 10 m cases. This represents a doubling of the survival in the case of the 10 m mixed layer but only a small relative increase for the 50 m mixed layer. UV-tolerant virus' survival was higher and showed relatively little effect of changes in diurnal thermocline depth. The extremely UV-tolerant case showed so little effect of release time that the results are not graphed, with survivorship ranging from 0.93 for the 10 m to 0.98 for the 50 m permanent thermoclines. The UV-sensitive case showed relatively low survival, even for viruses in a 50 m mixed layer. There were relatively large changes in the survival of the sensitive virus for greater diurnal changes in the thermocline. The minimum viral survival probability, 0.001, was for the UV-sensitive virus with a 10 m mixed layer. Thus, the physical dynamics have large effects on viral survival that are greatest for UV-sensitive forms.

Viral survival increased when the physical mechanism for virus movement was eddy diffusion (Fig. 3). This was particularly true for sensitive viruses. With a 50 m mixed layer and no diurnal thermocline change, sensitive viral survival probability was 0.45, compared to 0.25 for LC.

When the 2 processes of LC and ED were combined, results closely resembled results from LC alone (Fig. 4).

The preceding simulations used a Langmuir cell cycle time T_L = 0.5 h, as did those of Woods & Onken (1982). Simulation results were almost identical for T_L = 1 and 2 h. There was only a very slight (1 to 2%) decrease in mortality with mixed circulation for small Z_{min} and for Z_{max} = 30 m in the T_L = 2 h case. When very large values of T_L, > 4 h, were used the model showed increased survival of viruses. Such large values are for periods much longer than normal Langmuir circulation times (Langmuir 1938). For small values usually associated with LC, the results are relatively constant.

When the value of Δs was doubled or halved, survival probability under pure turbulent diffusion was strongly affected. That of sensitive viruses in a deep mixed layer nearly doubled from 0.35 to 0.65 when D_z ranged from 640 to 40 $m^2\ h^{-1}$. This sensitivity to ED rates was greatly reduced in the presence of LC. There was no significant change in viral survival for different Δs in any case where LC is present. Tolerant virus survival did not significantly change over the range of D_z considered.

Virus survival was heavily influenced by variations of the UV attenuation coefficient over the range k = 0.15 to 2 m^{-1}. Sensitive virus survival over a day in a deep mixed layer increased from < 0.05 to 0.75. Even the tolerant virus was strongly affected: survival increased from 0.15 to 0.8 in a shallow mixed layer. Survival for the extremely tolerant virus ranged from 0.81 to 0.98 under the same conditions.

The viral release time was an important parameter determining viral survival (Fig. 5). Viral release

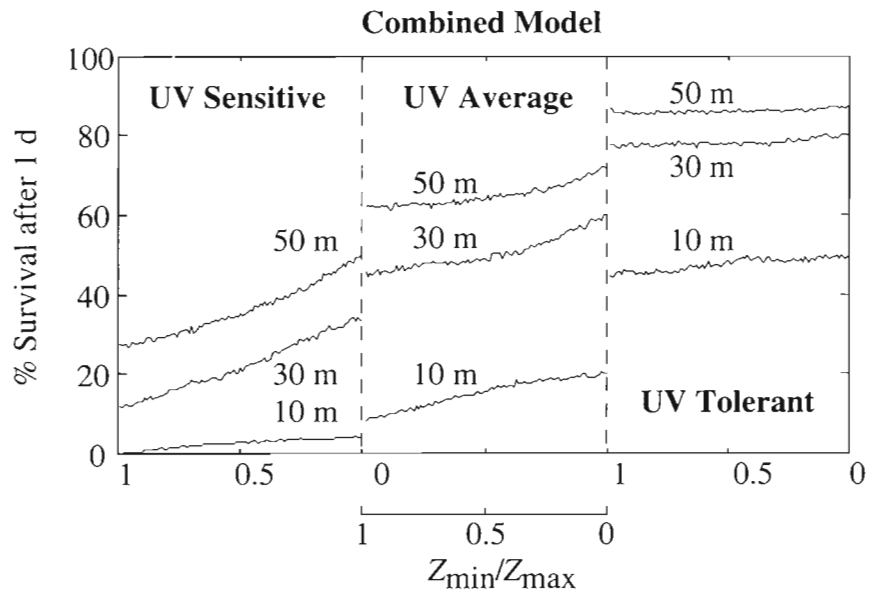

Fig. 4. Survival of viruses under combined Langmuir circulation and eddy diffusion. Notation and conditions as in Fig. 2. Notice this figure is very similar to Fig. 2. Tolerant virus in a 10 m mixed layer had slightly lower survival rates. Survivorship for the extremely tolerant virus is 0.93, 0.98 and 0.99 for the 10, 30 and 50 m permanent thermocline cases

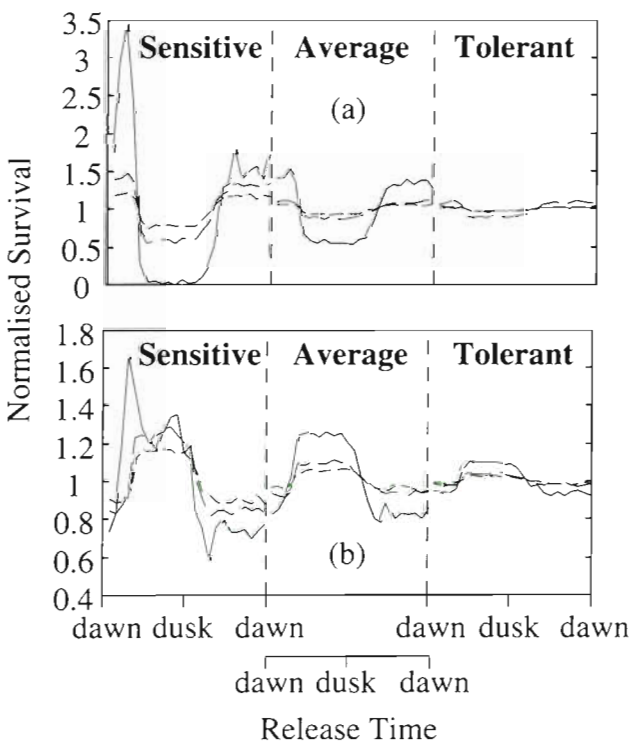

Fig. 5. Effect of viral release time on 24 h survival in (a) top half of mixed layer and (b) bottom half of mixed layer for $Z_{min} = 0.5\,Z_{max}$, Δt = 2.5 min. Depths and other aspects of the graphs are as for Fig. 2. Calculations are normalised to the mean survival for that viral type and environment. Sensitive virus survival is very strongly controlled by time and depth of release. Average virus survival is strongly affected by depth of release if this occurs during daylight. Tolerant viruses are not affected much, although release below the thermocline during the day in a shallow mixed layer does enhance survival

occurred at dawn in previous simulations. If $Z_{min} = 0.5\,Z_{max}$, sensitive virus survival was negligible in the top half of a 10 m water column for afternoon release. Survival of sensitive viruses released in the afternoon was a third of that for viruses released at dawn even in a 30 m water column. The effect of release time was negligible for tolerant and extremely tolerant viruses. Viruses released in the bottom half of the mixed layer had higher survival if release occurred in the afternoon. This change in release time led to a doubling of survival of sensitive virus. A release just prior to the formation of the diurnal thermocline resulted in viruses being rapidly trapped at the base of the thermocline. As a result, sensitive viruses had a 60% survival increase over that for dawn release, whether release occurred in the top or bottom half of a 10 m water column.

Under pure Langmuir circulation, large values of Δt can produce errors, particularly in the survival of sensitive viruses (Fig. 2). Usually, $\Delta t < 5$ min was sufficiently small to avoid these errors and Δt = 1.25 min was small enough to avoid error for all the cases considered.

DISCUSSION

The UV radiation spectrum has been subdivided into UV-A (400 to 320 nm), UV-B (320 to 280 nm) and UV-C (190 to 280 nm). Viral inactivation probability by a given dose of radiation increases rapidly with decreasing wavelengths (Rontó et al. 1992). Conversely, solar irradiance declines over the same range. The resulting interaction is a peak deactivation of nucleic acid by

sunlight in the UV-B band at about 300 nm wavelength (Caldwell 1971, Setlow 1974; Fig. 6). Mortality rate of marine viruses has been measured at 0.4 to 0.8 h^{-1} in full sunlight (Suttle & Chen 1992), with UV-B accounting for 50 to 90% of this rate. Mortality caused by UV-A accounted for the rest. UV-C had an insignificant effect. Mortality rate for marine viruses is linearly related to UV irradiance (Suttle & Chen 1992). To determine viral population decay rates it is only necessary to know the surface mortality of the virus and the intensity of UV, particularly UV-B, through the water column relative to that at the surface.

Downward solar irradiance at the outer edge of the atmosphere is described by a semisinusoidal curve (Kirk 1983). The radiation actually received at the ocean surface is lower in the morning and evening than the sine curve describes because of atmospheric attenuation (Caldwell et al. 1980). The effect is not great and barely affects UV-A. The fact that predicted viral mortality rates are low early and late in the day minimises the effect of the discrepancy between Eq. (7) and observed radiation.

Maximal UV-B irradiance at the earth's surface is around 4 W m^{-2} (Frederick & Lubin 1988) and is greatest in the tropics. Temperate regions can receive a large proportion of this in summer. For example, values of 1 to 3 W m^{-2} have been measured in southeast England (Webb 1992). Polar regions currently receive less UV-B than other areas (Stordahl et al. 1982). Besides latitude, UV radiation received at the surface of lakes is also strongly controlled by elevation (Caldwell et al. 1980).

Sea ice is relatively transparent to UV radiation. Antarctic sea ice of about 2 m thickness can transmit up to 10% of incident UV in spring (Ryan 1992). Because under-ice viruses cannot move to deeper UV-shaded water and Antarctic under-ice community turnover rates are often low (Ryan 1992), viral mortality from UV radiation could be important in this ecosystem.

Shorter wavelength UV has greater water column attenuation than that of longer wavelength fractions. Despite this the actual wavelength that causes greatest viral damage shifts only slightly with depth (Smith & Baker 1979). This wavelength is about 305 nm. Because of the single wavelength dominance in water, a single attenuation coefficient works well to describe ultraviolet radiation distribution and the damage it inflicts.

Humic acids, abundant in fresh and coastal water, and nitrate are particularly effective UV filters (Armstrong & Boalch 1961, Calkins 1982). Other compounds, such as chlorophyll, preferentially absorb visible light. Changes in the ratio of UV to photosynthetically active radiation could change the relative importance of viral mortality. Coastal regions with high concentrations of UV adsorbers could be regions were viruses are protected from UV. Moreover, viral dynamics could be enhanced by large host populations there, which would decrease the time that a virus needed to find a host and thereby decrease the time it is exposed to UV damage. A similar protective effect could occur when a layer of water that is relatively transparent to light but absorbs UV overlies a region of plankton production.

High attenuation would appear to make UV an insignificant element in mortality of viruses found beneath the mixed layer. However, movement of the thermocline by internal waves could significantly increase exposure of a deep layer to radiation (Lande & Yentsch 1988). For horizontal sinusoidal waves (Pond & Pickard 1983) the average amount of radiation received by viruses $\overline{U}_z$ over a wave cycle at average depth z subject to internal waves of amplitude A would be:

$$\overline{U}_z = \frac{U_0}{2\pi} \int_{-\pi}^{\pi} e^{-k[z + A\sin(t)]}\, dt$$

$$= \frac{U_0\, e^{-kz}}{2\pi} \int_{-\pi}^{\pi} e^{-kA\sin(t)}\, dt \qquad (10)$$

The relative exposure enhancement is

$$\frac{\overline{U}_z}{U_z} = \frac{1}{2\pi} \int_{-\pi}^{\pi} e^{-kA\sin(t)}\, dt \qquad (11)$$

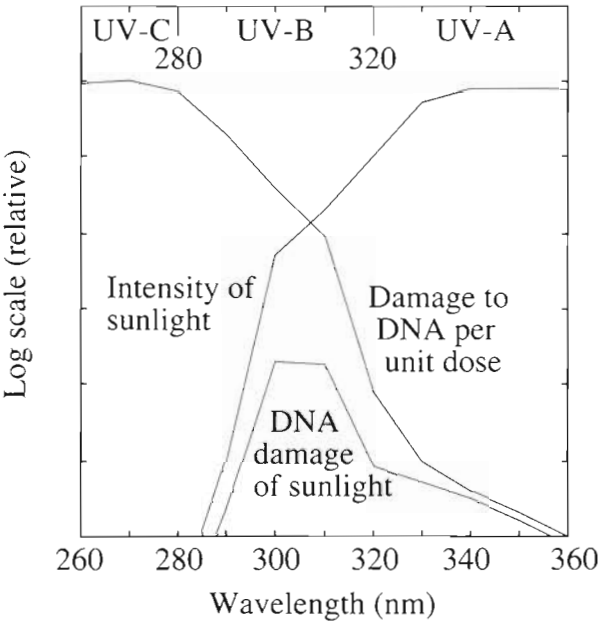

Fig. 6. Interaction between solar UV spectrum and DNA damage. Shown are sea-level irradiance as a function of wavelength, relative DNA damage per quantum as a function of wavelength and relative DNA damage for the sea-level spectrum (after Setlow 1974). Also shown are the regions of UV-A, -B and -C

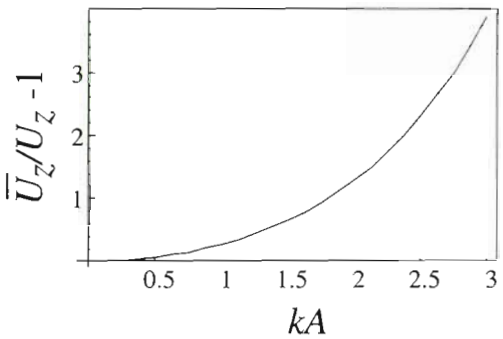

Fig. 7. Enhancement of UV exposure of viruses in a thermocline subject to internal waves. Results calculated by numerical integration of Eq. (11). A: wave amplitude; k: UV attenuation coefficient

The relative exposure to UV increases by about 0.25 when $kA = 1$ and doubles when $kA = 2$ (Fig. 7). However, the large values of k that make for large values of kA and high relative exposure imply low absolute exposure unless the oscillations are very large or take place near the surface.

Physical processes controlling the movement of viruses in a mixed layer of given depth strongly influence their survival. Turbulent eddy diffusion is less effective than Langmuir circulation in exposing viruses to UV. Eddy diffusion coefficient and the period of Langmuir mixing appear to be relatively unimportant if both factors contribute to mixed-layer water motion. Two other factors, diurnal thermocline formation and time of virus release, can be important influences on mixed-layer virus survival.

The more sensitive a virus is to UV the more important the diurnal thermocline cycling is for its population dynamics. Survival probability over a day for sensitive virus ($M = 4.6\ h^{-1}$) more than doubles as the minimum diurnal thermocline becomes shallower. For a virus with $M \leq 0.51\ h^{-1}$, the diurnal thermocline can be completely ignored unless the permanent thermocline is very shallow. The clear skies and rapid warming of surface waters associated with diurnal thermoclines are usually associated with high UV irradiance. Diurnal thermoclines are more likely to form on days when viral mortality is highest.

Woods & Onken (1982) used a simulation of solar heating to determine the diurnal thermocline minimum depth. Price et al. (1986) showed that the diurnal thermocline minimum depth off Baja California was extremely variable from day to day. The model results discussed are here for periods of 1 d or less. Viral survival for longer periods of time would need to account for this longer period variability.

Viruses can influence their survival if they can control their release times. A virus released during daylight below a diurnal thermocline has an increased 24 h survival probability. If release occurs above the thermocline then survival is highest for nighttime release. Very sensitive viruses do have enhanced survival if released in the late morning just as the diurnal thermocline starts to form. This enhancement amounts to a doubling of a very low survival probability. It could explain a counterintuitive observation that virus populations in Norwegian water increase as sunlight irradiance increases during the day (Heldal & Bratbak 1991).

Some viruses can tolerate much greater exposure to UV radiation than others because they possess the ability to repair nucleic acid damage. There are essentially 3 known ways viruses can compensate for UV damage (Harm 1980): use host repair mechanisms; carry their own repair genes; share genes in a multiphage infection. Van Etten et al. (1991) listed 9 eukaryotic algal viruses as containing double-stranded DNA and only 1 with single-stranded RNA. The exception was a virus of the multicellular benthic alga *Chara corallina*. Double-stranded DNA has been shown to be the basis of cyanophages (Bitton 1980) and appears to be for bacteriophages (Wommack et al. 1992).

Marine viruses usually have been reported with UV-induced population mortality rates of 0.4 to 0.8 h^{-1} (Suttle & Chen 1992), values in the range typical of UV-tolerant viruses, e.g. 0.7 h^{-1} (Harm 1980). Mortality of viruses can be higher. T4 viruses lacking specific UV repair genes have full sunlight induced mortality rates of 0.99 to 0.999 h^{-1}, values in the range of our sensitive viruses (Harm 1980). The lower mortality rate for marine viruses suggests that UV tolerance has been a naturally selected viral characteristic, indicating that UV can be an important cause of viral mortality in the real world as well as in the model.

Fuhrman & Suttle (in press) have recently found 2 virus strains with mortality rates in full sunlight on the order of 0.05 d^{-1}. This is the value used for the extremely tolerant case. Our results suggest that survivorship after 24 h for this strain is high enough so that the mortality is negligible unless the water is extremely transparent to UV and/or the water is very shallow.

The calculations presented here have implicitly assumed the present values of sea-level UV irradiance. Depletion of atmospheric ozone could increase the total UV reaching the ocean surface. Predicted general reduction in ozone will probably have a relatively minor effect on UV irradiance. A 16.5% increase in damaging UV at 45° N by the year 2040 has been forecast if CFC release continues (Stordahl et al. 1982). Local ozone holes can already cause enhancement in 300 nm UV levels by a factor of 4 to 10 in polar regions (Trodahl & Buckley 1990). Maximum Antarctic ozone depletion in 1988 was only half that of either 1987 or

1989 (Trodahl & Buckley 1990). This large degree of interannual variation could prevent ecosystems from adapting to changing UV irradiance. The biologically damaging dose may increase even faster than average UV-B because of a disproportionate increase of irradiance in shorter wavelengths (Stordahl et al. 1982). Because of the way we formulated the model, using surface viral mortality rate rather than UV irradiance, a tripling of the surface damaging UV irradiance is equivalent to replacing a UV-tolerant virus with a virus of typical tolerance or a typical virus with a UV-sensitive one. Because the probability of viral survival changes drastically with such a shift, ozone depletion could dramatically affect viral dynamics and the associated interactions of the entire food web at high latitudes.

The UV dose received by a virus is the product of irradiance and exposure time. Thus, viruses of common organisms will probably receive lower UV doses before finding a host than will viruses of rare organisms. Viral contact with individual picoplankton is less likely than contact with larger plankton, but the very large numbers of picoplankton found in the environment mean that they are as a population much more likely to intercept viruses (Murray & Jackson 1992). The exact time taken to find a host will depend on the product of contact rate, host population, and inverse probability of infection per contact. For example, populations of motile spherical organisms of 1, 5, 25 and 125 µm diameter present at 10^{-6} biovolume/water volume have contact rates of 9.1×10^{-5}, 7.2×10^{-6}, 6.3×10^{-7} and 6.2×10^{-8} s^{-1} (Murray & Jackson 1992). The inverse of contact rate is the average virus:host contact times of 3 h 3 min, 38 h 35 min, 18 d 9 h and 186 d 16 h for these cases. UV irradiance has time to inflict substantial mortality on viruses, except those with the smallest hosts. Larger hosts can produce many more new viruses per lysis and so could tolerate higher levels of mortality. Some viruses should survive mixed-layer UV exposure unless host population densities are very low. Genetic diversity within populations can reduce the effective population of hosts for a given virus. The large numbers of bacteria:phage systems in the North Sea (Moebus 1992) indicate that viral survival can be high.

UV-associated mortality is only one of the potential sources of viral mortality. Other organisms can inactivate viruses by a variety of mechanisms (e.g. Zachary 1976, Fujioka et al. 1980, Toranzo et al. 1982). Bacterial exoenzymes can be photolytically cleaved by UV-B (Herndl et al. 1993), possibly reducing their ability to inactivate viruses. An earlier analysis that included some of these processes but not UV inactivation suggested that viral mortality over 24 h depended on bacteria:virus collision rates but could be on the order of 10 to 99% (Murray & Jackson 1992). The UV-associated mixed-layer mortality probabilities presented here vary but are in the same range. The comparable size of these mortality rates suggests that both processes are important, although one or other may dominate a particular environment. Viral mortality in humic-rich coastal waters are probably dominated by biological effects. Viral UV-induced mortality rates should be highest in the surface waters of oligotrophic seas where contact times are long. Where production is concentrated in a chlorophyll deep maximum the total UV exposure of viruses may be small. Relative particle concentrations and UV irradiances should determine the dominant mechanisms of viral inactivation throughout the ocean.

Viral mortality associated with ultraviolet radiation is influenced by a range of physical and chemical processes in the ocean. The model results presented here offer a means to integrate the laboratory of viral mortality rates with assessments of the effects of mixed-layer dynamics and of changes in UV absorbance. Furthermore, they show the effects of possible changes in viral properties, such as the UV response characteristics and the viral release time. One important conclusion is that UV mortality could be a significant component of viral mortality in the environment. A complete assessment of viral population dynamics would need to incorporate other mortality causes as well. Experimental studies and mathematical models of the processes involved need to be integral parts of such an assessment.

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