Marine viruses and their biogeochemical and ecological effects

Jed A. Fuhrman

Viruses are the most common biological agents in the sea, typically numbering ten billion per litre. They probably infect all organisms, can undergo rapid decay and replenishment, and influence many biogeochemical and ecological processes, including nutrient cycling, system respiration, particle size-distributions and sinking rates, bacterial and algal biodiversity and species distributions, algal bloom control, dimethyl sulphide formation and genetic transfer. Newly developed fluorescence and molecular techniques leave the field poised to make significant advances towards evaluating and quantifying such effects.

The importance of microbial processes in the sea has become increasingly apparent over the past two decades. Although heterotrophic microbes (see Box 1) in the water column used to be generally ignored, it is now clear that a large proportion (often half or more) of the total flux of matter and energy in marine food webs passes through such organisms by means of dissolved organic matter[1,2]. Heterotrophic prokaryotes are abundant[1], representing a significant proportion of the biomass in the euphotic zone (the surface waters), and an even larger proportion in deeper waters[3]. The concept of the microbial loop in marine food webs has been developed and refined in recent years, but until a decade ago, viruses were ignored in such studies. However, a series of reports then showed that viruses are not only remarkably abundant in marine plankton, but are also likely to be significant agents in the control of bacteria and phytoplankton[4–7].

Viruses are small particles, usually about 20–200 nm long, consisting of genetic material (DNA or RNA, single- or double-stranded) surrounded by a protein coat (some also have lipids). They have no intrinsic metabolism and function only as parasites through the cellular machinery of a host organism. In theory, all cellular organisms are susceptible to infection, often by more than one type of virus, implying that viruses are probably the most diverse creatures on Earth. A given type of virus usually has a restricted range of hosts—often a single species, although some viruses infect only certain subspecies, whereas others may infect more than one related species or even genus[8]. Viruses contact their hosts by passive diffusion and use their host's exposed cellular structures, often transporter proteins, as attachment and entry points to the cell.

There are three basic types of virus reproduction. (1) Lytic infection: the virus attaches to a host cell and injects its nucleic acid into the cell, directing the host to produce numerous progeny viruses; these are released by fatal bursting of the cell, allowing the cycle to begin again. (2) Chronic infection: the progeny virus release is non-lethal and the host cell releases them by extrusion or budding over several generations. (3) Lysogeny: the nucleic acid of the viral genome becomes part of the genome of the host cell, and reproduces as genetic material (called a prophage or provirus) in the host cell line. In this case, an induction event, such as stress to the host, can trigger a switch to lytic infection.

A less well-defined type of virus–host interaction is termed pseudolysogeny, in which the viral nucleic acid may remain within a host cell for some time (possibly for a few generations[9]) before lysis, or cell destruction, occurs. Pseudolysogeny may be related to host starvation, in which the virus adopts an inactive state, unable to initiate viral gene expression owing to the low energy state of the cell; normal viral activity returns when the cell is fed[10]. Alternatively, pseudolysogeny may be regarded as a transient state of host immunity, apparently induced by an immunizing agent (perhaps a polysaccharide depolymerase) released from infected cells, and helping to foster coexistence of host and virus[11,12]. Viruses or virus-like particles may also be involved in killing cells by mechanisms that do not result in virus reproduction[13].

Here I synthesize the accumulated evidence regarding the nature of marine viruses and their ecological and biogeochemical effects. The principal conclusion is that viruses can exert significant control on marine bacterial and phytoplankton communities, with respect to both biological production and species composition, influencing the pathways of matter and energy transfer in the system.

Virus abundance

Viruses infecting specific marine organisms have been studied[14] for several decades, initially from studies of pure cultures that focused on organisms rather than ecological systems; but viruses were not regarded as quantitatively important components of marine food webs until they were shown by direct counts to be highly abundant. The small size of viruses renders them invisible to ordinary light microscopy, and although they can be visualized by transmission electron microscopy (TEM), special procedures are required to concentrate them from sea water.

The ultracentrifugation and TEM method that is commonly used to collect and visualize marine viruses and prokaryotes is adapted only slightly from the original technique published[15] in 1949—had this technique been applied then to natural waters, the discovery of highly abundant prokaryotes and viruses in aquatic habitats would have been pushed forward by about 25 and 40 years, respectively. This could have redirected the development of modern biological oceanography and limnology, which have only recently moved significantly towards microbiological studies.

The first reports of high viral abundance, exceeding the typical bacterial abundance of 10^9 per litre (refs 4–6), awakened interest in this topic. Many subsequent studies[16–25] have shown that viruses are consistently the most abundant biological entities in the sea—nearshore and offshore, tropical to polar, sea surface to sea floor, and in sea ice and sediment pore water. Viral abundances are typically 10^{10} per litre in surface waters (about 5–25 times the bacterial abundance), and follow the same general abundance patterns as bacteria. These patterns include a decrease of about one order of magnitude between rich coastal waters and oligotrophic (nutrient poor) open ocean, a decrease of between five- and tenfold from the euphotic zone to the upper midwaters (for example, 500 m depth), and a further decrease several-fold to

Box 1 Glossary of marine organisms

Archaea. One of the two groups of *prokaryotes*, whose cultivated members are often methane-producing or tolerant to unusually high temperature or salinity. Uncultivated archaea, such as those from the deep sea, may have other physiologies.

Autotrophs. Organisms that use CO_2 as their source of carbon. (All green plants are autotrophs.) See also *heterotrophs*.

Bacteria. One of the two groups of *prokaryotes*; also used as a generic term describing organisms that appear to be *prokaryotic* but are otherwise unidentified (some may be *Archaea*).

(Bacterio)phages. Viruses that infect bacteria.

Cyanobacteria. Type of *bacteria* that contain chlorophyll *a* and undergo photosynthesis, generating oxygen. This phylogenetic group includes the marine *prochlorophytes*.

Cyanophages. Viruses that infect *cyanobacteria*.

Eubacteria. Another name for the *Bacteria* group.

Eukaryotes. Organisms with membrane-bound nuclei; all animals and higher plants are eukaryotes. (See also *protists*.)

Eukaryotic algae. Photosynthetic *protists*.

Flagellate. A type of *protist* that moves by beating flagella (long, strand-like structures present on the surfaces of some cells).

Heterotrophs. Organisms that derive energy from preformed organic matter. (All animals are heterotrophs.) See also *autotrophs*.

Metazoa. Multicellular animals.

Microbe. Microscopic organism, occurring as a single cell or simple colony.

Phytoplankton. Photosynthetic plankton, including *cyanobacteria* and *eukaryotic algae*.

Prochlorophytes. Small photosynthesizing *bacteria* that possess divinyl chlorophyll *a* and *b* (see also *cyanobacteria*).

Prokaryotes. Cells without membrane-bound nuclei. They include two broadly different groups, the *Bacteria* and *Archaea*.

Protist. A type of *eukaryote* that is single-celled, or lives in simple colonies of single cells. Protists are generally larger and less abundant than *bacteria*. (See also *flagellates*, *eukaryotic algae* and *protozoa*).

Protozoa. Old name for non-photosynthetic *protists*. (Protozoa are distinct from the *metazoa*.)

Zooplankton. Eukaryotic (animal) plankton. Macrozooplankton are visible to the naked eye and feed on the largest prey organisms. Microzooplankton are just below the visible size range (~20–200 μm), and may include very small *metazoa*, animal larvae and large *protists*. Nanozooplankton include the smallest *protists* (~2–20 μm) and eat the smallest prey, such as *bacteria*.

abyssal depths. As occurs with bacteria, sea ice is highly enriched in viruses compared with the water beneath it[21], and sediment pore waters are highly enriched compared with overlying water[19,24].

Viral abundances are dynamic, being particularly responsive to changes in ecological conditions such as algal blooms[7,26]—this provides strong evidence that viruses are active members of the community rather than inert particles. Pronounced fluctuations[27] in virus abundance over timescales of minutes to hours may be indicative of synchronized host-cell lysis and rapid degradation of a large portion of the progeny viruses. On scales of hundreds of kilometres, virus abundance is usually strongly correlated to bacterial abundance and less so to particulate chlorophyll *a* (refs 18–20), indicating that most marine viruses infect bacteria. The term 'bacteria' refers here and throughout to prokaryotes in general because most studies do not distinguish true Bacteria (also called Eubacteria) from Archaea (see Box 1); in some environments (particularly the deep sea), half of the prokaryotes may belong to the Archaea[28].

It is possible to use epifluorescence microscopy with nucleic acid stains such as 4,6-diamidino-2-phenylindole (DAPI), YoPro and SYBR Green to count viruses accurately, rapidly and inexpensively compared with TEM[25,29–32]. Viruses stained with SYBR Green can be observed (Fig. 1) and counted by ordinary epifluorescence microscopy in the field (for example, on board ship) within an hour of sampling; the counts obtained[25] are similar or slightly higher (by ~1–1.5 times) than those obtained by TEM. Viruses stained with SYBR Green may also be counted by flow cytometry[33], further expediting aquatic virus studies; again the counts are higher than TEM values.

The reasons for the higher fluorescence counts compared with those obtained from TEM preparations are uncertain, but fluorescence counts may include viruses that are otherwise obscured by other dark-stained particles in TEM preparations; fluorescence also enables counts of filamentous viruses or other types that are not recognized easily by TEM. The new stains that permit fluorescence imaging or flow cytometric detection of a diverse array of viruses may prove useful in biomedical studies, representing a spin-off of marine virus research.

Beyond simply counting viruses, marine viral diversity has also been examined by morphology and size distribution; cultures and natural samples show a broad array of shapes and sizes[34]. A new technique, pulsed field electrophoresis[35,36], has revealed the size diversity of viral genomes extracted directly from marine samples. The method provides a minimum estimate of the diversity of the most abundant viruses. There are typically 15–40 visibly distinct genome sizes in a given sample, and the composition is variable in space and dynamic over seasons.

Viral activities

One of the initial reasons that biological oceanographers looked for abundant viruses was that there seemed to be excess bacterial production that had a missing sink, and viruses seemed good candidates for that sink[2]. Viruses can limit bacterial abundance to several orders of magnitude below the resource-limited level, demonstrating a potential for control[9]. As soon as it was learned that viruses have consistently high abundances in sea water, it was hypothesized that these viruses must be infecting native marine microbes at significant rates in order to persist[5].

Microbes, particularly prokaryotes, seemed to be the major host candidates because they are by far the most abundant potential hosts (~10^9 marine bacteria per litre)—viruses reach their hosts by random diffusion and interception, with contact proportional to host abundance and size, so that a virus is much more likely to contact a bacterium than a protist or metazoan[37]. The relationship between viruses and bacteria seems corroborated by field data showing that bacterial abundance is the best predictor of virus abundance, explaining about 67% of the variance[18]. However, given the presumed host specificity of viruses and the largely unknown species diversity and composition of marine microbial assemblages[28], quantitative evaluation of contact between marine viruses and hosts still involves much guesswork.

In the decade since the discovery of high viral abundance, several lines of evidence have converged to the conclusion that viruses are significant agents in both the mortality of aquatic microbes and in the structuring of aquatic communities. That evidence is summarized below.

Infection frequency. Direct evidence that viruses infect native bacteria and cyanobacteria originally came from TEM studies of thin-sectioned plankton, in which the assembled viruses were observed in about 0.8–4.3% of the cells from a variety of marine habitats: high-nutrient coastal waters, oligotrophic open ocean[6] and sinking material collected in offshore sediment traps[38]. Similar percentages are typical in freshwater samples[39], although some freshwater work has found lower percentages, occasionally undetectable[26].

As viruses are visually detectable inside hosts only at the last stage of lytic infection, the visibly infected cells represent only a small proportion of the total number of infected cells. A model based

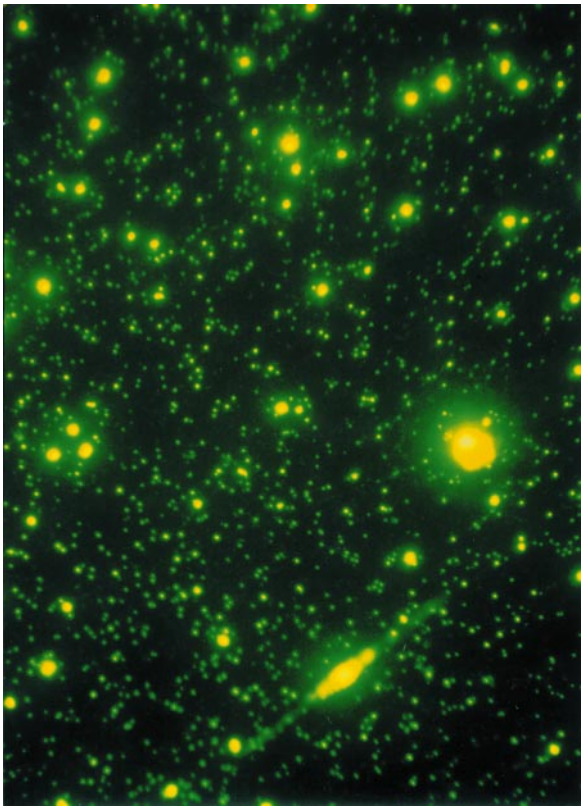

Figure 1 Fluorescence imaging of marine viruses. Epifluorescence photomicrograph of viruses (small, numerous green dots) and prokaryotic cells (rarer, larger green dots), collected from 5 m depth in the San Pedro Channel near Santa Catalina Island, California, on 29 May 1998 and prepared with SYBR Green I stain[25]. Photographed on Kodak Ektachrome 400 film, 30 s exposure. The two largest stained objects are a pennate diatom (oblong) and an unidentified protist.

upon culture studies[6,40] was used to calculate the total mortality from visibly infected cells. The model's predictions indicated that the apparent percentage infected should be multiplied by a conservative factor of about 10 (but ranging from 7.4 to 14.1) to estimate the total percentage of mortality due to infection. This analysis indicates that viruses are responsible for about 8–43% of the bacterial mortality in the sites mentioned above. However, this may be an overestimate, because an infected cell may be grazed before it lyses[41].

Some studies[39,42–44] simplify the examination of infected cells by looking through intact cells, rather than preparing thin sections. The former procedure is much faster, and requires only a few millilitres of sample. A slightly higher percentage of visibly infected cells is often found compared with the thin-section method[45], probably because viruses are more likely to be seen when observing an entire cell, rather than just a portion. However, visualization through a whole cell is less sharp than through thin sections, so intracellular components such as storage products may be more likely to be mistaken for viruses with this method. Nevertheless, the approach has yielded similar estimates of the virus contribution to mortality in aerobic waters (~10–50%), and it has also been used in anaerobic fresh water where percentages have been much higher[45] (~45–100%).

A simple way to examine the possible impact of viruses on planktonic systems is to artificially increase the number of viruses. This has been done experimentally using natural viral concentrates produced by ultrafiltration of sea water[46–51]. As little as a 20% increase in the 2–200-nm size fraction of viruses can reduce photosynthetic rates by as much as 50% (refs 46, 48). In a variety of coastal and open-ocean samples with added viruses, growth inhibition (by about half) of the total bacterial community, thought to be primarily heterotrophs, was observed[47]. Although the concentrates increase the mortality of bacteria, they may also stimulate[51] the growth of the surviving bacteria, as predicted by models[2,7] and experimental studies[50,52]. Heat-treating the concentrates removed the inhibitory effect in these studies, which is consistent with a viral effect. This has significant biogeochemical implications (see later).

Viral decay. Although studied for a variety of reasons, historically for sanitary engineering, viral decay is an important parameter in the investigation of the ecological roles of viruses. The term 'decay' is ambiguous, sometimes referring to degradation, sometimes to disappearance of detectability (including removal by adsorption); in most cases, the definition is an operational one set by the mode of study. In cultures, decay is usually measured as the decrease over time in the number of measurable, infectious viral particles (or rather centres of infection), after spiking a sample with culturable viruses. In whole viral communities, decay is measured as the disappearance over time of directly countable viral particles (new virus production must be stopped to measure this[42]).

In general, measurements of viral decay have led to estimates of viral turnover times (the time it takes for the standing stock to be replaced as it decays) of about an hour to a few days[42,53–55]. Decay rates tend to follow the overall biological richness of the water, with the fastest decay occurring in rich nearshore waters and the slowest decay in low-nutrient offshore waters.

Studying loss of infectivity and the mechanisms leading to such loss requires cultures of the hosts, so the largely unknown nature of marine microbial diversity has resulted in relatively few marine viruses being studied. A virus undergoing decay might lose its infectivity well before it decays beyond recognition, hence loss of infectivity provides a more sensitive measure of the decay rate[56].

The most prominent single decay factor identified in several marine studies so far is sunlight-induced damage[53,57] (mostly from UV-B, but also from other wavelengths). However, susceptibility to damage from full sunlight varies greatly, with some viruses decaying rapidly at a rate of 40–80% per hour[53,57], whereas others decay at a slow rate of only 5–10% per hour[14,55,56]; thus, generalizations are hard to make. Viruses from sunnier regions apparently show more resistance to light damage, an adaptation that one might expect[55]. Because light is attenuated with depth, solar-induced decay is most important near the sea surface and has no effect in the deep sea. But even with light attenuation, the particularly susceptible viruses in the surface mixed layer may be more affected by solar radiation than by any other decay mechanism[53].

In the presence of light, host bacteria can often repair light-damaged viral DNA, restoring infectivity[58]. This photoreactivation (or other light-dependent repair mechanism in host cells) has led to the new conclusion that most viruses in the lighted portion of the water column are infective[58], contrary to the previous common supposition that they are inactive.

Solar radiation is by no means the only major mechanism of viral decay; other mechanisms can be more important in many instances (even in sunlight). Significant decay has been observed[55] from heat-labile substances that occur in particulate form (that is, bacteria, protists and some aggregates), and also in the form of high molecular mass (>30 kilodaltons) dissolved material such as hydrolytic enzymes. Often the particulate and dissolved material work synergistically with sunlight. Typical overall decay rates are about 3–10% per hour, implying turnover times of about 0.5–1.5 days. The slowest decay rates, typically 0.5–1% per hour, are observed when viruses are placed in heat-treated sea water in the dark.

Early culture experiments[59] showed that the rate of decline of infectivity in sea water can slow with time, leaving a slowly decaying low level of infectious viruses. A possible explanation is that there are

refuges in sea water that protect the viruses. Alternatively, natural variation among viruses, even within a burst, may lead to some that are assembled perfectly and so are more resistant to decay than their siblings, which may have minor flaws in the protein coat. Therefore, in modelling viral processes, it may be invalid to apply a single decay rate parameter, perhaps even for a single viral type.

Virus production. The quantitative significance of viral activity can be investigated by estimating the rate at which viruses are produced, because this necessarily implies infection of host cells. Assuming that the infection is lytic, burst size (the number of progeny viruses produced from a single ruptured host cell) can be used to estimate the host mortality rate from the viral production rate. Viral production has been estimated in three ways, as described below.

The first method involves comparing viral community decay rates (where production has been halted) to untreated controls in which production continues[42]. Net decay rates reflect turnover and replenishment of viruses; these processes tend to balance out, as virus abundance is relatively steady. The earliest studies used cyanide to inhibit production of new viruses, and found[42] viral turnover times of about 1–2 h. Some experiments reported surprisingly high viral decay rates, implying losses that were incompatible with independent measurements[60] of bacterial production, so there is uncertainty in interpreting this approach. But calculations that assume more conservative infectivity decay rates can still lead to estimates of significant viral production. For example, a tenfold excess of viruses over bacteria can be maintained with a lytic burst size of 50 and viral turnover time of one day, if 20% of the bacteria lyse daily.

The second method of determining viral production is more direct, involving the incorporation of radiolabelled thymidine or phosphate tracers in viral nucleic acid[61]. This approach has been used in California and Arctic surface waters[24,54,61], revealing that viruses account for about 10–40% of the total mortality rate there.

The third method uses fluorescently labelled viruses (concentrated from sea water, stained and then added back). By means of calculations that are analogous to those used for isotope dilution studies, it is possible to calculate simultaneously the production and decay rates of viruses by measuring the change in total and labelled virus counts[62]. This method has been used to measure virus turnover times of about 1–2 days in Southern California nearshore and offshore waters, probably accounting for most of the total bacterial mortality[62].

Bacterial mortality. It is generally thought[14,24,45,54,63,64] that viruses are responsible for about 10–50% of the total bacterial mortality in surface waters, and 50–100% in environments that are unfriendly to protists, such as low-oxygen lake waters. Multiple correlation analysis of the abundances of bacteria, viruses and flagellates indicates[41] that virus-induced mortality of bacteria can occasionally prevail over flagellate grazing, especially at high bacterial abundances.

A few studies have directly compared virus-induced mortality with other causes, and balanced total mortality and loss rates with independent estimates of bacterial production. For example, a study[54] of California coastal waters found evidence that the total mortality balanced production to within 30% (and that viruses were responsible for about 40% of the total mortality). In the Bering and Chukchi (Arctic) Seas, evidence has been found[24] that viruses and protists are responsible for a similar amount of bacterial mortality, with protists dominating in some water samples and viruses in others; however, the total mortality estimates typically fell short of balancing production estimates, often by more than 50%. It was also suggested[24] that the viral effect is probably larger in eutrophic waters than in oligotrophic waters, although there are still not enough data to make firm conclusions.

Not every study has concluded that viruses have a significant effect on bacterial mortality: relatively little infection was observed[65] in a Mediterranean saltern pond, and cyanobacterial mortality was hardly affected by viruses in one particular study area[66] (see later). Therefore, some groups of organisms and some habitats may display relatively little virus induced mortality; the effect may be seasonal, local or sporadic.

Overall, it seems reasonable to conclude that viruses often, but not always, have a significant effect on bacterial mortality, sometimes even greater than grazing. Several studies indicate that viruses are relatively more important in nutrient-rich waters.

Phytoplankton mortality. This topic is not as extensively studied as infection of heterotrophic bacteria, but its potential impact is great, given that phytoplankton form the base of the marine food web. A few studies have looked at the overall effect of viruses. As mentioned earlier, a relatively modest 20% enrichment of sea water with a native virus concentrate leads to 50% reduction of phytoplankton biomass and primary production, with the effect eliminated by heat treatment[46,48]. This is strong presumptive evidence that abundant viruses actively infect a significant proportion of the phytoplankton community.

In contrast to these whole-community studies, other reports have focused on particular host organisms. Studies of viruses that infect ecologically important phytoplankton are facilitated because some of these species are available in pure culture, and are also readily recognizable by microscopy (unlike heterotrophs).

Marine phytoplankton are composed of prokaryotes (cyanobacteria and prochlorophytes) and eukaryotes; both of these major types show evidence of viral infection in nature. Highly abundant viruses (often 10^2–10^4 per millilitre in nearshore and offshore waters, and sometimes exceeding 10^5 per millilitre) can infect[66,67] specific strains of marine cyanobacteria (*Synechococcus* sp.). Electron microscopy shows that 1–3% of the native cyanobacteria from a variety of locations contain assembled viruses apparently near the end of the lytic cycle[6].

However, cyanophages are probably not responsible for a large proportion of the cyanobacterial mortality: about 5–15% of the cyanobacteria in the Gulf of Mexico (Texas coast) are lysed by cyanophages daily[67], with an even smaller percentage[66] (up to about 3% per day) of *Synechococcus* lysed in temperate Atlantic waters (near Woods Hole, Massachusetts, and also offshore). As culturable cyanobacteria tend to be resistant to co-occurring viruses, it was concluded[66] that cyanophages affect the species or strain composition of the community more than they affect the total abundance. This has numerous implications for the importance of viruses in affecting community structure, and perhaps ultimately community function as well (discussed later).

Viruses infecting eukaryotic marine phytoplankton are also found in the marine euphotic zone, and are sometimes quite abundant. Marine viruses[34,38,68–72] are capable of infecting diatoms, chrysophytes, pyrmnesiophytes, haptophytes, rhaphidophytes and cryptomonads; probably every kind of phytoplankton can be infected. Several viruses that are pathogenic to the common marine prasinophyte *Micromonas pusilla* (a small flagellate) have been isolated from sea water in several locations[68,73,74]; their abundance can exceed 10^5 per millilitre in coastal waters and the turnover time is about 1.3 days, with infection leading to 2–10% of the host population lysing per day[74].

The viruses can be diverse: five genotypes of the *M. pusilla* virus were identified[75], using sequence analysis of DNA polymerase genes, from one region (offshore Gulf of Mexico). Significant diversity was also found[71] among 14 virus strains isolated and tested against 18 cultured strains of *Heterosigma akashiwo*.

Given the dependence of viral infection on host population density, such infection is expected to be most prominent in algal blooms, where hosts are abundant. Several reports indicate that viral infection may be important in the control of algal blooms. Phytoplankton that are susceptible to viral infection include the chrysophyte *Aureococcus anophagefferans*[4,70,76] (which causes costly and devasting 'brown tides' in US northeastern embayments), the toxic rhaphidophyte *Heterosigma akashiwo*[71] (which kills fish in

both aquaculture and natural settings), the common *Phaeocystis pouchetii*[77] and *Emiliana huxleyi*[69,78,79] (an organism distributed worldwide that often forms large, dense blooms in mid-latitudes). Up to 50% of all *E. huxleyi* cells in a decaying natural bloom were observed[79] using TEM to be infected, and viral lysis can account for 25–100% of the net mortality of *E. huxleyi* during the decline of blooms in experimental mesocosms[69]. *E. huxleyi* is one of the planet's major producers of calcite (in the form of coccoliths), causing a large amount of carbon to be transported to sediments[80]; it also generates dimethyl sulphide (DMS), a gas important in regulating climate[81].

Biogeochemical effects

Given that viruses often cause a large proportion of bacterial and even phytoplankton mortality (particularly during blooms), what are their effects on the system as a whole? Lytic infection converts cells into viruses plus cellular debris. This debris is made up of dissolved molecules (monomers, oligomers and polymers) plus colloids and cell fragments[82], most of which is operationally defined as dissolved organic matter.

What is the likely fate of this material? The most reasonable assumption is that most or all of the lysis products will immediately or eventually become available to bacteria[2,6,7]. This has recently been confirmed experimentally[52,62,76], although a very small proportion of the viruses may be grazed directly by heterotrophic flagellates[83], and some cellular debris may resist degradation.

If the cell lysed is a bacterium, then the availability of the lysis products to bacteria represents a semi-closed trophic loop, whereby bacterial biomass is consumed primarily by other bacteria. The loop is fed externally by release of dissolved organic matter from phytoplankton and grazers (Fig. 2). Given that there are respiratory losses and inorganic nutrient regeneration associated with use of dissolved organic substances, this loop has the net effect of oxidizing organic matter and regenerating inorganic nutrients[2,7] (Fig. 2). However, given that protists would otherwise consume the bacteria[1], this bacterial consumption of matter originating from other bacteria has the interesting property of essentially robbing production from the rest of the food web, and sequestering the biomass and activity into the dissolved and smallest particulate forms[2].

Similarly, viral lysis of phytoplankton would rob the larger grazers and move material into these small forms. The net effect has been illustrated by a model[2] showing that, compared to a system with no viruses, an otherwise identical food web with 50% of bacterial mortality from viruses has 27% more bacterial respiration and production, and 37% less bacterial grazing by protists, which culminates in a 7% reduction in macrozooplankton production.

However, the original steady-state model[2] assumed that only bacteria are infected and that all the viral matter is consumed by bacteria. A modification of that model, including a small amount of viral infection of phytoplankton (7% loss) and also flagellate grazing of 3% of the virus production, has essentially the same net effect of increasing bacterial production and respiration (by 33%), and reducing protist and animal production (Fig. 3).

Sequestration of materials in viruses, bacteria and dissolved matter may lead to better retention of nutrients in the euphotic zone in virus-infected systems, because more material remains in these small non-sinking forms[2]. This may be particularly important for nutrients that potentially limit productivity (such as N, P and Fe), which are relatively concentrated in bacteria compared to eukaryotes[2]. Reduced viral activity may result in more material in larger organisms, which either sink themselves or as detritus, transporting carbon and inorganic nutrients from the euphotic zone to the deep sea.

Lysis of organisms and release of their cell contents to the water have other potential geochemical effects, because of the chemical and physical properties of the released materials and the location in the water column where the lysis occurs. For example, released polymers contribute to the 'gel' characteristics of sea water that influence many biological and microscopic physical–chemical processes, and may facilitate aggregation and sinking of material from the euphotic zone[38,49]. Alternatively, viral lysis of microorganisms within sinking aggregates may effectively dissolve the particles, converting some sinking particulate matter into non-sinking dissolved material and colloids at whatever depth the lysis occurs[38].

Viruses may also be important in shaping global climate because they induce the release of DMS, a gas that influences cloud nucleation. Experiments show that infection causes the total release of intracellular DMS precursor (DMSP) from the phytoplankter *M. pusilla*[84], and viruses are also known to infect major DMSP-containing bloom organisms such as *E. huxleyi*[79] and *P. pouchetii*, causing enhanced release of DMS[85]. Complex interactions within the food web (from microbes to zooplankton) determine whether appreciable DMS can be released to the atmosphere, or whether it is transformed and degraded primarily by microbes or animals in the water[86]. Thus, it is important to understand physical mixing processes and the activities and complex interactions of viruses (both algal and bacterial), protists and zooplankton grazers in order to predict the extent of oceanic DMS release compared with its degradation.

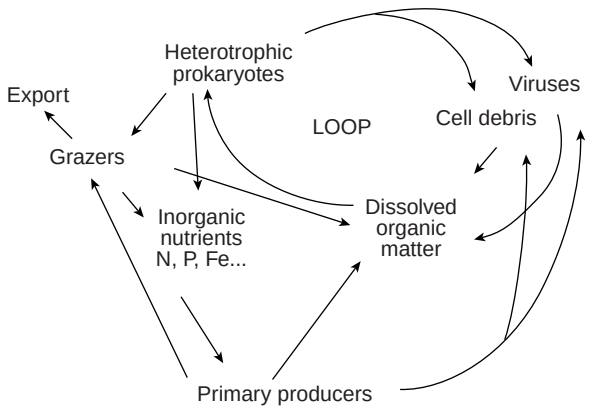

Figure 2 The planktonic viral loop. Schematic diagram of an aquatic food web, emphasizing the semi-closed loop connecting prokaryotes, viruses and dissolved organic matter (curved lines). Note that the loop has the net effect of converting organic matter into dissolved inorganic nutrients. The viruses and cell debris are separated for illustration, but are often defined operationally as dissolved organic matter.

Ecological effects

Diversity regulation. Marine prokaryote diversity is beginning to be understood with the advent of 16S ribosomal RNA sequence-based techniques, which enable analysis of most organisms that are resistant to conventional cultivation[87]. As evident from their effect on algal blooms and cyanobacteria, viruses are in a unique position to influence community species compositions. Theoretical analysis[88] indicates that the maximum possible number of bacterial populations in a spatially homogeneous environment is equal to the number of unique resources plus the number of unique virus types.

Even if viruses cause only a small proportion of the mortality of a group of organisms, they still can have a profound effect on the relative proportions of different species or strains in the community[66,89]. Similar ideas have been known from experimental systems for some time[9]. An important consideration is that viral infection is thought to be both density dependent and species specific (or nearly so). Because viruses must diffuse randomly from host to host, rare hosts are less susceptible to the spread of infection than more common ones. Lytic viruses can only increase in abundance when the average time to diffuse from host to host is shorter than the average time they remain infectious. Thus, when a

particular species or strain becomes more densely populated, it is more susceptible to infection. This may have direct relevance to solving Hutchinson's 'paradox of plankton'[90], which asks how so many different kinds of phytoplankton can coexist on only a few potentially limiting resources, when competition theory predicts just one or a few competitive winners. Although several possible explanations may contribute to the answer[91], viral activity probably assists because the competitive dominants become particularly susceptible to infection, whereas rare species are relatively protected[14].

Models[92] of the potential factors that control the biomass and species composition of microbial systems include growth limitation by organic carbon, inorganic phosphate or nitrogen (inorganic or organic), and cell losses due to grazing by protists and viral lysis. Even when bacterial abundance is assumed to be controlled by protist grazing, the models consistently show that viruses control the steady-state diversity of the bacterial community, whether bacterial growth-rate limitation is by organic or inorganic nutrients. Consistent with this picture are studies[35,93] of viral genome sizes and hybridization analyses of total viral communities, which show episodic and spatial changes in viral community composition. Thus, both empirical and theoretical analyses indicate that viruses are important in regulating patterns of diversity.

Resistance. The development of resistance to viral infection is well known from non-marine experimental studies[9], and is an important assumption in models[88,92] describing the regulation of diversity by viruses. Resistance has been shown[66] to occur in marine cyanobacteria. The incidence of resistance in marine heterotrophic bacterial populations has not been evaluated directly; doing so would be difficult because most have not yet been grown in culture. However, the high rates of virus production in many marine locations seem to be inconsistent with a community dominated by virus-resistant organisms.

Why is it that viral resistance does not appear to be a dominant factor, even though theory and some field data indicate that it should be? Unfortunately, there are no obvious explanations. One possibility is that resistance is dominant and virus production is not as high as it seems to be, but this would require explanations for the varied observations that are consistent with high virus production. Another possibility is that resistance costs too much physiologically, in that it often occurs through the loss of some important receptor and thus confers a competitive disadvantage[9]. Theory suggests that if the cost is too high, resistant organisms cannot persist in competition with sensitive ones, even when viruses are present[88]. Chemostat experiments[94] indicate that even with a cost that allows persistence of resistant cells, they may still be rare unless the cost is low or limiting resources are abundant. However, laboratory experiments indicate that low-cost or no-cost resistance is possible[9], so uncertainty remains.

It seems likely that the explanation lies outside the realm of simplified theoretical or laboratory systems. The real ocean has many species of bacteria and viruses living together, with a variety of alternative mortality mechanisms and low, but variable, nutrient conditions. Perhaps there are advantages to being sensitive to infection. In a typical (oligotrophic) marine environment, bacterial growth may be limited by nitrogen, phosphorus or organic carbon; unsuccessful viral infection (perhaps stopped intracellularly by a restriction enzyme, or with a genetic incompatibility) is then of significant nutritional benefit[6] to the host organism, because the virus injection of DNA is rich in C, N and P. Even the viral protein coat, left attached to the outside of the cell, is probably digestible by cell-associated exoenzymes[95]. In this situation, one might even imagine bacteria using decoy virus receptors for viral strains that cannot successfully infect them; if an infectious one occasionally gains entry, the strategy might still confer a net benefit to the cell line.

Could a model with so many aborted infections still be consistent with high virus production rates? It would seem so given that, in the steady-state, only one virus out of every burst from cell lysis (typically producing 20–50 progeny viruses) successfully infects another host. A large proportion of the remaining 95–98% unsuccessful viruses could end up infecting the 'wrong' hosts, giving these hosts an incentive to continue permitting virus attachment and injection. This is a consistent model, but highly speculative.

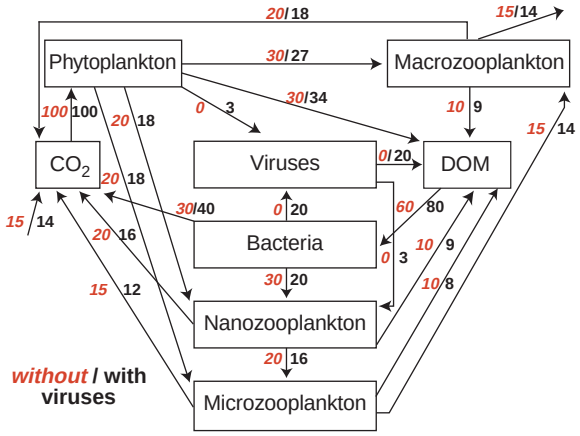

Figure 3 Modelling viral effects on carbon flow. Steady-state model of carbon flow in a hypothetical aquatic microbial food web with and without a virus component that is responsible for 50% of bacterial mortality and 7% of phytoplankton mortality. The numbers represent units of carbon transferred between boxes in a given time unit. The virus compartment includes bacterial cell debris from lysis, and lysed cells are assumed to degrade to dissolved organic matter (DOM). Nanozooplankton are heterotrophic protists 2–20 μm in diameter and microzooplankton are protists or small metazoa ~20-200 μm in diameter (see Box 1 for further descriptions). Note that the viruses lead to an increase in bacterial production and respiration, and a decrease in zooplankton production and respiration. See text and ref. 2 for details.

Another consideration that argues against resistance relates to the results of system models (Fig. 3) which show that, as a group, the heterotrophic bacteria benefit greatly from viral infection (as infection boosts their production significantly, essentially by taking carbon and energy away from large organisms). Viruses also boost the entire system biomass and production by helping to maintain nutrients in the lighted surface waters. However, these explanations would additionally require some sort of group-selection theory to explain how individuals would benefit from not developing resistance (for example, why not cheat by developing resistance and letting all the other organisms give the group benefits of infection?).

It seems that most explanations of why resistance is not a dominant factor are either unsatisfactory or highly speculative. Whether lack of comprehensive resistance is due to frequent development of new virulent strains, rapid dynamics or patchiness in species compositions, or to a stable coexistence of viruses and their hosts has yet to be determined. As a final note, the extent of resistance may somehow be related to pseudolysogeny (in the sense of a transient immunity to infection induced by infected hosts[11,12]), but this is not well understood.

Lysogeny. Lysogeny has been suggested as a survival strategy of viruses living at low host density; at the same time, it may confer advantages to the host, including immunity from infection by related viruses and the acquisition of new functions coded by the virus genome (known as conversion[9]). In seawater and lakewater samples[96,97], lysogens (bacteria that harbour prophage and can be induced to produce lytic viruses) were found to be common, with variable abundances ranging from undetectable to almost 40% of the total bacteria, and this variability can be seasonal[98]. About 40% of cultured marine bacteria are lysogens[99].

There may be certain natural events that induce such lysogens to enter the lytic cycle, and reports[96,100] show that common pollutants such as hydrocarbons, polychlorinated biphenyls (PCBs) and Aroclor can do so. However, growth experiments with native mixed bacterial communities grown in filtered sea water indicate that under typical natural conditions, including exposure to bright sunlight, induction of lysogens is rare, and the vast majority (97% or more) of viruses observed in sea water are probably the result of successive lytic infection[101]. The same conclusion was reached from a quantitative interpretation of induction experiments[99,102]. Lysogenic induction may be occurring at a low level all the time, or may occur sporadically. Although induction may be rare, the widespread existence of lysogeny probably has major implications for genetic exchange and evolution (see below).

Genetic exchange. Probably the most uncertain aspect of marine viruses is their role in genetic exchange among microorganisms, and its effect on short-term adaptation, population genetics and evolution. There can be direct effects such as transduction[103,104], in which a virus picks up DNA from one host and transfers it to another (often a lysogen). The overall effect over large scales of space and time would be to homogenize genes among the susceptible host populations. Although the traditional view is that transduction usually operates within a highly restricted host range, one report[13] indicates that some marine bacterial viruses are capable of nonspecific horizontal gene transfer. Another consideration is that viral lysis causes release of DNA from host organisms, which could be transferred to another organism through natural transformation. Dissolved DNA is readily found in sea water, and it has been reported[19] that viral lysis may be a major source mechanism.

The latter two processes (generalized viral horizontal gene transfer and transformation) would have the effect of mixing genes among a broad variety of species, with wide-ranging effects on adaptation and evolution. Although transfers of these sorts may be extremely rare, the typical bacterial abundances of 10^9 per litre in the euphotic zone and the huge volume of the sea (3.6×10^7 km^3 in the top 100 m; ref. 105), coupled with generation times on the order of a day, implies that an event with a probability of only 10^{-20} per generation would be occurring about a million times per day. Thus, these process may have major effects on the genetic structure and evolution of the global population of marine bacteria. They should also be considered in the evaluation of the potential spread of genetically engineered microbial genes, or of antibiotic resistance introduced by intensive fish farming.

Looking ahead

New molecular methods that enable the diversity of viruses and their hosts to be studied within their natural habitats will make it easier to unravel the complex web of interactions in marine communities. Once we gain a better understanding of diversity issues and important factors such as host range, we can start to follow, and maybe even predict, the complex relationships between viruses and the rest of the ecosystem. □

Jed Fuhrman is in the Department of Biological Sciences and Wrigley Institute for Environmental Studies, University of Southern California, Los Angeles, California 90089-0371, USA.

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Correspondence and requests for materials should be addressed to J.F. (e-mail: fuhrman@usc.edu).