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Viruses in the sea

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Viruses exist wherever life is found. They are a major cause of mortality, a driver of global geochemical cycles and a reservoir of the greatest genetic diversity on Earth. In the oceans, viruses probably infect all living things, from bacteria to whales. They affect the form of available nutrients and the termination of algal blooms. Viruses can move between marine and terrestrial reservoirs, raising the spectre of emerging pathogens. Our understanding of the effect of viruses on global systems and processes continues to unfold, overthrowing the idea that viruses and virus-mediated processes are sidebars to global processes.

For years viruses were known to exist in seawater, but reports 15 years ago caused great excitement by demonstrating not only that viruses are abundant, but that they infect the dominant organisms in the ocean[1–3]. These observations occurred against the backdrop of a major shift in thinking among oceanographers to acknowledge that bacteria and microbial processes are important players in the oceans. For example, because viruses are significant agents of microbial mortality, they have an effect on nutrient cycling[4–6]. Moreover, the narrow host range of most viruses suggests that infection is important in controlling the composition of planktonic communities[6–8]. Interest in the vast viral communities in the sea continues to expand as the relevance of viruses to evolution, pathogen emergence and even exobiology begins to be explored.

Several excellent reviews[4–7] have captured the advances in our understanding of marine viruses and their role in the ocean. This review focuses on areas where our knowledge is changing rapidly, where methodological problems have impeded progress and where new data are altering perceptions. Without doubt, viruses are the most abundant and genetically diverse 'life forms' in the ocean. They are major pathogens of planktonic organisms and consequently are significant players in nutrient and energy cycling. As well, they are pathogens of higher organisms and there is good evidence that some viruses move between marine and terrestrial reservoirs. Recognition that viruses play a major role in marine ecosystems has added a significant new dimension to our understanding of biological oceanographic processes.

Total virus abundance has been underestimated

Viruses are extremely abundant in aquatic systems. The first observations by transmission electron microscopy (TEM)[1,2] indicated that, typically, there are ~10^7 viruses ml^{-1} and that abundance decreases with depth and distance from the shore[9,10]. In general, abundance correlates with system productivity and is highest where bacteria and chlorophyll are greatest[11,12]. In marine sediments, abundances are even higher, with 10^8–10^9 viruses cm^{-3} typical in nearshore surface sediments[1,10,13]. Even 100 m below the sediment surface[15] viruses can be plentiful, although at the sediment surface in the deep ocean they seem to be less abundant[16].

Epifluorescence microscopy (EM) is now the preferred method for counting viruses because of its higher accuracy and precision[17,18], although flow cytometry shows promise as a high-throughput method[19]. The shift to EM-based techniques has not been without problems, including significant differences in results among methodologies[20], concerns about reproducibility and effects of sample storage[19,21]. For instance, estimates of viral abundance performed using samples not immediately processed or frozen in liquid nitrogen can be an order of magnitude too low. Currently, our best estimates range from ~3×10^6 viruses ml^{-1} in the deep sea[22,23] to ~10^8 viruses ml^{-1} in productive coastal waters. Assuming the volume of the oceans is 1.3×10^{21} l and the average abundance of viruses is 3×10^9 l^{-1}, then ocean waters contain ~4×10^{30} viruses. Because a marine virus contains about 0.2 fg of carbon and is about 100 nm long, this translates into 200 Mt of carbon in marine viruses. If the viruses were stretched end to end they would span ~10 million light years. In context, this is equivalent to the carbon in ~75 million blue whales (~10% carbon, by weight[24]), and is ~100 times the distance across our own galaxy. This makes viruses the most abundant biological entity in the water column of the world's oceans, and the second largest component of biomass after prokaryotes.

But total viral abundance alone does not give us an indication of infectivity. Most viruses in seawater seem to be infectious[25], and some can remain infectious in sediments for long periods, from decades to a hundred years or more[26,27]. Estimating the abundance of infectious viruses is complicated by strain specificity; yet in offshore waters most collisions between bacteria and viruses seem to result in infection, suggesting that selection for resistance is low[28]. Even so, viruses infecting specific hosts can be extremely abundant. For example, viruses infecting single strains of the cyanobacterium *Synechococcus*[28] or the photosynthetic flagellate *Micromonas pusilla*[29] can occur in excess of 10^5 infectious units ml^{-1}. Yet, even the most permissive hosts are not sensitive to infection by all viruses that infect a given species; therefore, even these are underestimates. Ultimately, high strain specificity combined with poor representation in culture of the dominant microbes in the sea means that the absolute abundance of infectious viruses must be deduced by inference.

Unexplored diversity

Virus form provides insight into function

Not only are viruses abundant in oceans but, as is becoming clear, they also harbour enormous genetic and biological diversity. TEM studies of marine viral communities[1] and phage isolates[30] reveal a plethora of morphotypes, whereas host-range studies show complex patterns of resistance and susceptibility[31].

Among marine phages (Fig. 1), those with contractile tails (such as myoviruses and T4-like viruses) and long flexible tails (such as siphoviruses and lambda-like viruses) are most frequently isolated[32–34], even though TEM suggests that phages with short non-contractile tails (such as podoviruses and T7-like viruses) and without tails are most abundant. Because 'lifestyles' among tailed phages differ, morphology provides clues about host range and viral replication. For example, myoviruses are typically lytic and often have a broader host range than other tailed phages, even infecting different species of bacteria[33,34]. By

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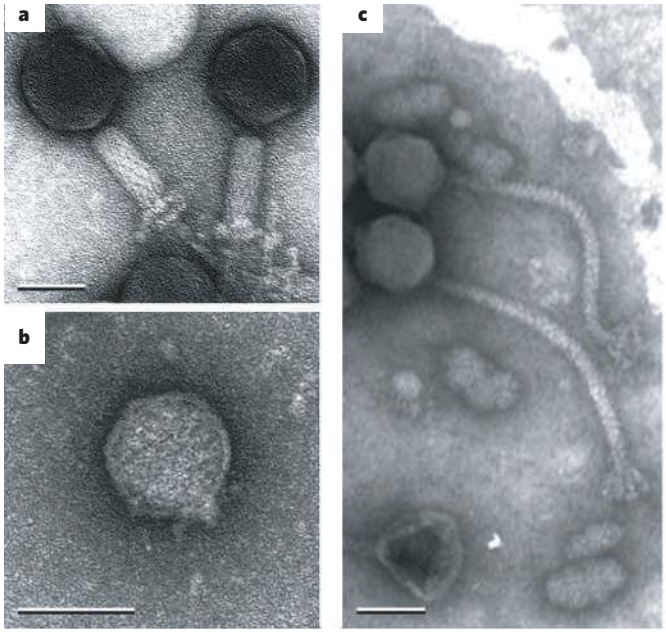

Figure 1 | The three families of tailed dsDNA viruses (phages) that infect bacteria. **a**, Myoviruses are often the most commonly isolated phage from natural marine viral communities. They have contractile tails, are typically lytic and often have relatively broad host ranges. **b**, Podoviruses have a short non-contractile tail, are also typically lytic and have very narrow host ranges. They are less commonly isolated from seawater. **c**, Siphoviruses have long non-contractile tails. They are frequently isolated from seawater, often have a relatively broad host range, and many are capable of integrating into the host genome. Scale bar, 50 nm.

contrast, the host range of podoviruses is generally narrowest, with siphoviruses being intermediate. However, many siphoviruses can integrate into the host genome and be passed from generation to generation.

Morphotype provides some insights into the selective pressures facing virus communities. Myoviruses with their broader host ranges can quickly take advantage of increases in host populations, consistent with *r*-selection (short generation times and high reproductive rates). By contrast, many siphoviruses can archive their genomes in host cells, tying their replication rate to that of the host, until an environmental cue triggers the lytic cycle. This suggests that siphoviruses are more *K*-selected (longer generation times and lower reproductive rates). There is much to learn about the biological and genetic diversity of marine phages. In culture, the phages used are unlikely to be representative of the dominant phages in the ocean, because the most abundant prokaryotes have proven very difficult to grow in culture, although this is beginning to change.

The story with respect to viruses infecting eukaryotes is even more complex. The first virus isolates infecting eukaryotic phytoplankton belonged to a group of large double-stranded (ds) DNA viruses, the *Phycodnaviridae*, and included viruses that infected important taxa of marine primary producers including toxic bloom formers and macroalgae[26,35]. The scene is changing rapidly as the isolation of many previously unknown viruses (Table 1) greatly enriches the taxonomic and phylogenetic space of known viral 'life'. For instance, a single-stranded (ss)RNA virus that infects a toxic bloom-forming alga (*Heterosigma akashiwo*) led to the creation of the *Marnaviridae*[36], probably the first of many new marine virus families. Other examples include a previously unknown dsRNA virus that infects the photosynthetic flagellate, *Micromonas pusilla*[37], a ssRNA virus that infects a thraustochytrid fungus[38], and a nuclear-inclusion virus (NIV) that has both ss and dsDNA and infects the diatom alga *Chaetoceros salsugineum*[39], and has virtually no similarity to known viruses. Other NIVs that infect *H. akashiwo*[40] (Fig. 2) and *Cheatoceros*[41], as well as a large dsDNA virus that infects a marine heterotrophic protist[42], remain to be characterized. The largest virus genome belongs to Mimivirus[43], which also infects a protist, whereas viruses that cause disease in shrimp[44] and crabs[45] are distantly related to other viruses, suggesting that heterotrophs will also yield an untapped oasis of unexplored viral diversity.

Viral genetic diversity is extremely high

Culture-independent approaches indicate that we are just scraping the surface of viral life in the oceans. No gene is found universally in viruses but some genes are representative of specific subsets of the viral community. The first studies used the DNA polymerase genes of *Phycodnaviridae*[46] to reveal enormous genetic variation that was not represented in cultures, and showed that very similar sequences were present in distant oceans[47]. Even more striking results were obtained in studies targeting a subset of myoviruses: tremendous diversity occurred on large[48] and small[49] spatial scales, with most of the sequences falling into groups with no cultured representatives[50,51]. Even more remarkable is that nearly identical sequences at the nucleotide level occurred in environments as far-reaching as the Southern Ocean, the Gulf of Mexico and a melt-water pond on an Arctic ice shelf[51]. Similarly, a study that targeted podoviruses found that indistinguishable sequences were ubiquitous in the environment[52]. These results indicate that in phycoviruses, podoviruses and myoviruses, and by inference probably other groups, co-infection results in viral genes being sequentially passed in many small 'steps' through a series of organisms. This emphasizes that, from a viriocentric perspective, bacteria and protists operate as vehicles for viral 'sex', allowing viral genes to spread widely. This remarkable diversity is not confined to dsDNA viruses infecting bacteria and protists. A study

Table 1 | Some unusual aquatic viruses whose genomic sequences have recently been completed

Virus	Family of proteins	Nucleic acid	Genome size (bp)	Number
Chaetoceros salsugineum nuclear inclusion virus[39]	Unassigned	ssDNA / dsDNA	6,005 ssDNA 997 dsDNA	Unknown
Emiliania huxleyi virus86 (ref. 63)	*Phycodnaviridae*	dsDNA	407,339	472
Heterosigma akashiwo RNA virus[36]	*Marnaviridae*	ssRNA	8,600	6 or 7
Micromonas pusilla[37]	*Reoviridae*	dsRNA	26,000 on 11 segments	Unknown
Ectocarpus siliculosus virus[85]	*Phycodnaviridae*	dsDNA	335,593	240
Mimivirus[43]	*Mimiviridae*	dsDNA	1,181,404	911
White spot syndrome virus[44]	*Nimaviridae*	dsDNA	305,107	531

All infect eukaryotic phytoplankton with the exception of the *Ectocarpus*, Mimivirus, and white-spot viruses, which infect a brown alga, freshwater protist and penaeid shrimp, respectively.

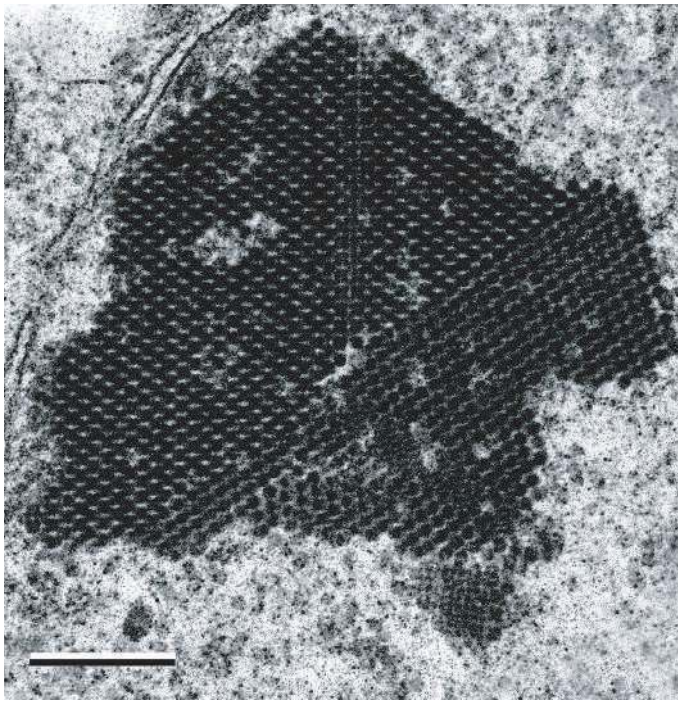

Figure 2 | The nuclear inclusion virus of *Heterosigma akashiwo*. There are a large number of viruses infecting marine protists that have only partially been characterized. Shown is a nuclear inclusion virus that infects the toxic bloom-forming alga *H. akashiwo*. Scale bar, 250 nm.

that targeted the RNA-dependent RNA polymerase of picorna-like viruses found a putative four new families in a few seawater samples[53]. This is particularly remarkable given the few existing families of known picorna-like viruses.

The extent of viral diversity in the sea is driven home by metagenomic studies in coastal waters[54] and sediments[55] that show there are perhaps several thousand viral genotypes in 200 l of seawater and a million in 1 kg of sediment[56]. The communities have an uneven distribution, with the most abundant genotypes making up less than 5% of the communities, whereas the majority of genotypes comprise < 0.01 of the communities[56,57]. The genetic richness of the communities is revealed by the fact that 60–80% of the sequences were not similar (E-value > 0.001) to those in databases[57]. By contrast, ~90% of putative genes from metagenomic data for prokaryotic communities had recognizable similarity to database sequences[57]. Clearly, metagenomic data indicate that marine viral communities contain much greater genetic richness than their prokaryotic counterparts, and are much less sampled.

Host cells are vessels for viral sex

Sequencing of virus isolates also reveals how little we know. The first marine phages to be sequenced infect *Roseobacter*[58], *Vibrio parahaemolyticus*[59] and *Synechococcus* sp.[60]. The results showed that these viruses appear to be ancestral to their terrestrial relatives; however, most of the putative genes had no significant similarity to those in databases. Subsequently, other marine phages have been sequenced[61,62], revealing that most of the putative genes code for proteins of unknown function, although some are homologous to genes of surprising metabolic function, such as those associated with photosynthesis, carbon metabolism and phosphate stress[62]. Similarly, analysis of the first viral genome to be completely sequenced that infects eukaryotic phytoplankton revealed that of 472 putative coding sequences, only 14% have significant similarity to database sequences[63].

One of the most surprising observations to stem from genomic analysis of cyanophages was that they contain homologues to *psbA* and *psbD* genes, which encode key components of photosynthesis[64]. At least one of these, and a varying number of other related genes, occur in phages that infect *Synechococcus*[65], *Prochlorococcus*[66] or both. These genes are widespread, with most marine cyanophage isolates carrying *psbA*[65]. Sequence analysis reveals that lateral gene transfer has occurred, probably numerous times, between cyanobacteria and their viruses[67]; however, these exchanges appear to be infrequent, particularly with respect to *Synechococcus*. The result is that photosynthesis genes in cyanophages have their own evolutionary history, with phage *psbA* genes forming distinct monophyletic groups[67,68]. This example demonstrates that viruses capture genes of host origin and exchange them among viral progeny, resulting in photosynthetic genes that are now clearly viral.

Accurate estimates of virus-mediated mortality are elusive

At least a half-dozen approaches have been used to infer the impact of viruses on microbial mortality. All of them suffer from poorly constrained assumptions. The elegant approach of inferring mortality from the percentage of visibly infected cells[2] requires estimates of the proportion of the lytic cycle during which cells are visibly infected. Few data exist for marine microbes and those that do are highly variable. Extrapolations from increasing the abundance of viruses only demonstrate the potential effect of viral infection[3]. Inferring mortality from decay rates of virus communities[69] or viral tracers[70] assumes that viral production and removal are balanced, that tracers are representative of *in situ* communities and that the number of viruses produced per lytic event (burst size) is known. Calculations on the basis of fluorescent-virus tracers[71] also require an estimate of burst size, and this method is not amenable to use in sunlight. Extrapolation from synthesis rates of viral DNA[72] requires conversion factors and is sensitive to contamination from bacterial DNA. Converting from directly measured rates of viral production to microbial mortality rates[73] requires an estimate of burst size and considerable sample manipulation. Finally, measurements based on changes in net growth rate as a function of viral dilution[74] require sample manipulation and assumptions about the relationship between infection and host-cell mortality.

Although it is discouraging that no method gives high precision and accurate estimates of virus-mediated mortality, it is encouraging that these diverse approaches, many of which rely on assumptions that are independent of each other, consistently indicate that viruses cause significant microbial mortality in a wide range of environments. Yet, many environments remain inadequately sampled. This includes the

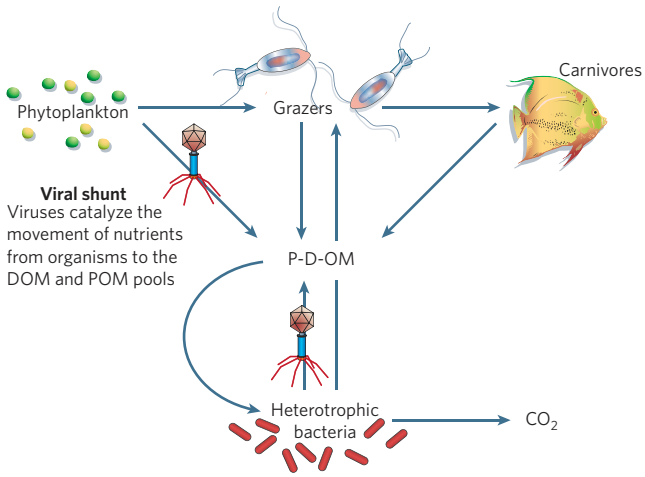

Figure 3 | Viruses are catalysts for biogeochemical cycling. Viruses short-circuit the flow of carbon and nutrients from phytoplankton and bacteria to higher trophic levels by causing the lysis of cells and shunting the flux to the pool of dissolved and particulate organic matter (D-P-OM). The result is that more of the carbon is respired, thereby decreasing the trophic transfer efficiency of nutrients and energy through the marine foodweb.

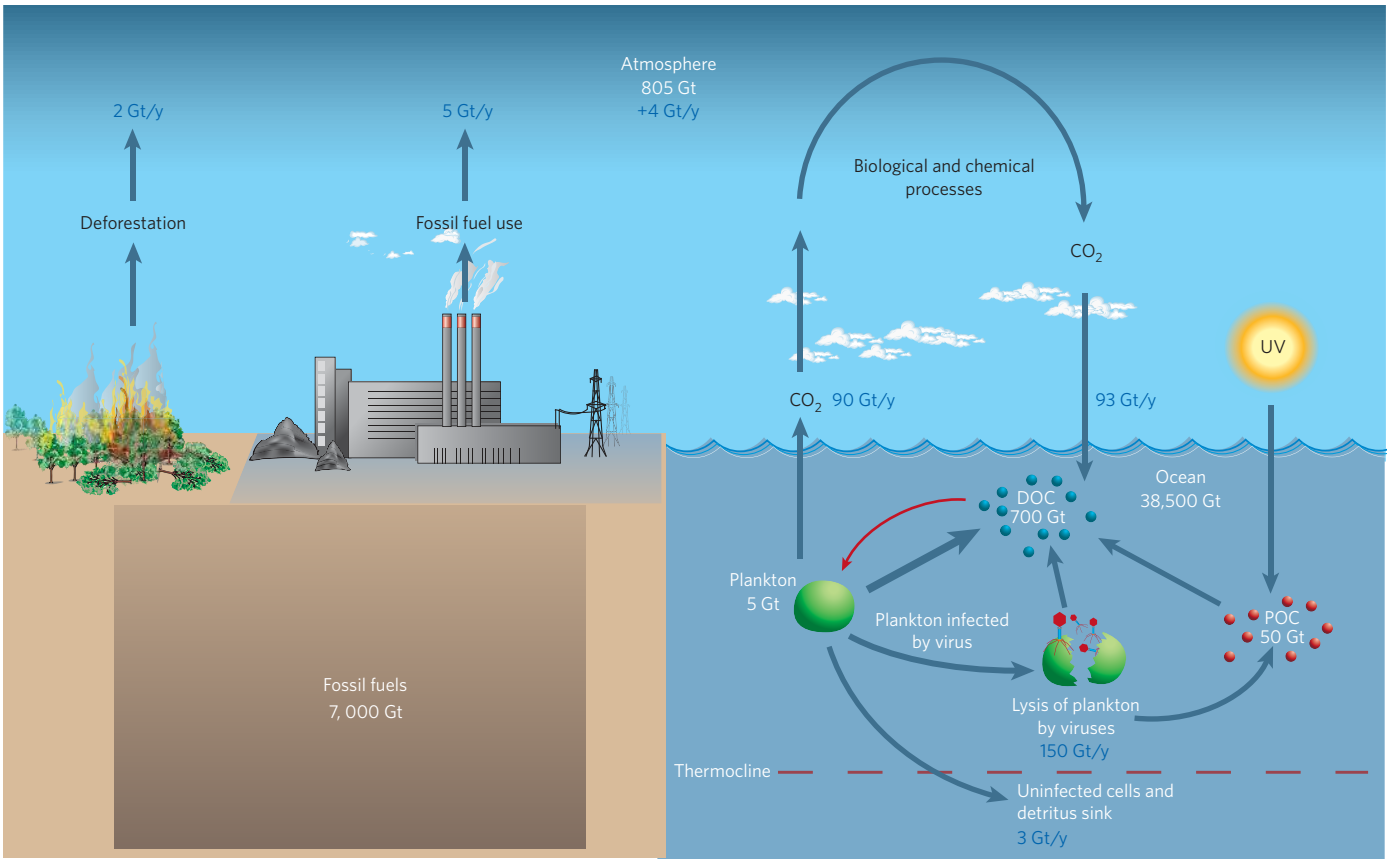

Figure 4 | Viruses can affect the efficiency of the biological pump. Viruses cause the lysis of cells, converting them into particulate organic carbon (POC) and dissolved organic carbon (DOC). This reduces the rate atwhich C sinks from the surface layer into the deep ocean where the carbon is trapped for millennia (biological pump). Instead the carbon is retained in the surface waters where it is photo-oxidized and respired, in chemical equilibrium with the atmosphere. The net effect is a faster rate of CO_2 build-up in the atmosphere than would occur if the POC were 'exported' to the deep ocean.

oligotrophic ocean, where slow rate processes make it difficult to obtain reliable data except from visibly infected cells or viral decay rates. Moreover, the assumptions for calculating mortality rates are particularly poorly grounded for open-ocean species. Even within relatively productive environments, estimates of the contribution of viruses to total mortality range from undetectable to 100%. In many cases, the wide range in estimates is probably real, and reflects differences between locations and times. Nonetheless, accurate estimates of virus-mediated mortality remain elusive, and we are not much further ahead than a decade ago when viruses were estimated to kill ~20–40 % of marine bacteria on a daily basis[75] and contribute to microbial mortality at a level similar to that of grazing by zooplankton[76].

Viruses are catalysts of global nutrient cycles

Given that viruses cause a significant, albeit variable, amount of marine microbial mortality, it implies that they also play an important role in marine geochemical cycles. Simple models[4,5] and model systems[77] demonstrate that viruses are catalysts that accelerate the transformation of nutrients from particulate (living organisms) to dissolved states, where it can be incorporated by microbial communities (Fig. 3). A net effect of this shunt is to increase community respiration and decrease the efficiency of carbon transfer to higher trophic levels. In addition, cell lysis converts particulate organic carbon (POC) into dissolved (therefore lower levels of cellular carbon) sinks (Fig. 4), resulting in more carbon being respired in the surface waters. This is significant for global carbon cycling because sinking of POC results in the net transfer of about 3 Gt of carbon between near-surface and deep waters thus the build-up of CO_2 in the atmosphere is only about half of what it otherwise would be. An exception occurs in some phytoplankton, where virus-infected cells sink rapidly[78], potentially increasing the transport of cells to deeper waters[27]. Nutrients other than carbon are also released by viral lysis[79,80]. As these nutrients are largely organically bound, this can affect their availability and pathways of cycling. In some cases, released nutrients such as iron can fill a major portion of the requirements of other organisms[80]. As well, the small size of viruses makes them excellent nucleation sites for mineralization of iron and perhaps other metals[81].

Marine viruses and disease

Viruses are not only players in microbial mortality and geochemical cycling, they are also progenitors of disease in higher organisms. Our limited knowledge of viral diseases in non-microbial marine organisms stems almost entirely from effects on fisheries from obvious instances of visible disease or large mortality events. Although viruses infect marine organisms ranging from crustaceans to whales, we know little about modes of infection and transmission, or the reservoirs of these viruses in nature. Some of these viruses pose potential health risks to humans. For example, calici and distemper viruses are thought to cycle between marine and terrestrial mammals, and some marine caliciviruses are thought to cause disease in humans[82,83]. Similarly, there is evidence that marine birds harbour avian flu, particularly the dangerous H5N1 strain[84]. We have very little understanding of the natural reservoirs of viruses that are carried by, or cause disease in, marine animals and know even less about their potential to spread to terrestrial systems. Our emerging knowledge of the enormous diversity of viruses in the marine milieu suggests that the oceans are potential reservoirs of many unknown causative agents of disease.

A look to the future

Our understanding of viruses and virus-mediated processes in the oceans is developing rapidly. From a very few studies in the early 1990s indicating that viruses were abundant and active in the sea, we have reached a point where it is clear that viruses constitute the greatest genetic diversity in the ocean, are important agents of mortality and major players in global geochemical cycles. Yet, for the most part, the depth of understanding is extremely limited in all of these areas. We have not determined the genetic richness of the marine viral metagenome within an order of magnitude, estimates of mortality are highly variable and poorly constrained, and the ways in which viruses influence global geochemical cycles are only beginning to be elucidated. Moreover, the role of marine viral communities in emerging and established diseases in marine and terrestrial ecosystems and the cycling of viruses between these reservoirs is largely unknown. Knowledge of marine viruses and their role in the global ecosystem will influence the spectrum of our thinking, ranging from quantitative models of global geochemical cycling to the localization of aquaculture facilities. Few would have predicted that the observation a decade and a half ago of high viral abundances in seawater[1,2] would have had such a profound influence on our understanding of biological oceanographic processes, evolution and geochemical cycling. ■

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