REVIEW ARTICLE

Marine viruses and global climate change

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Abstract

Sea-surface warming, sea-ice melting and related freshening, changes in circulation and mixing regimes, and ocean acidification induced by the present climate changes are modifying marine ecosystem structure and function and have the potential to alter the cycling of carbon and nutrients in surface oceans. Changing climate has direct and indirect consequences on marine viruses, including cascading effects on biogeochemical cycles, food webs, and the metabolic balance of the ocean. We discuss here a range of case studies of climate change and the potential consequences on virus function, viral assemblages and virus–host interactions. In turn, marine viruses influence directly and indirectly biogeochemical cycles, carbon sequestration capacity of the oceans and the gas exchange between the ocean surface and the atmosphere. We cannot yet predict whether the viruses will exacerbate or attenuate the magnitude of climate changes on marine ecosystems, but we provide evidence that marine viruses interact actively with the present climate change and are a key biotic component that is able to influence the oceans' feedback on climate change. Long-term and wide spatial-scale studies, and improved knowledge of host–virus dynamics in the world's oceans will permit the incorporation of the viral component into future ocean climate models and increase the accuracy of the predictions of the climate change impacts on the function of the oceans.

Introduction

Climate change and its expected impacts on marine ecosystems

Over the past 250 years, atmospheric carbon dioxide (CO_2) levels have increased by nearly 40%, from preindustrial levels of approximately 280 parts per million volume (p.p.m.v.) to nearly 384 p.p.m.v. in 2007 (Solomon *et al.*, 2007). This rate of increase is at least one order of magnitude faster than has occurred for millions of years (Doney & Schimel, 2007), and the current concentration is higher than experienced on Earth for at least the past 800 000 years (Lüthi *et al.*, 2008). The Arctic Climate Impact Assessment (ACIA, 2005), using an intermediate scenario, predicts a doubling of CO_2 within the next *c.* 80 years. According to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change (IPCC, 2007), the global mean surface air temperature increased by 0.74 °C while the global mean sea-surface temperature rose by 0.67 °C over the last century (Trenberth *et al.*, 2007). Models predict that the effect of the increased climate-altering gases will lead, within the end of this century, to an increase in air temperature ranging between 1.4 and 5.8 °C (IPCC, 2007).

Comprising > 70% of the surface of the Earth, the oceans have the capacity to store > 1000 times the heat compared with the atmosphere (Levitus *et al.*, 2005). The oceans and shelf seas play a key role in regulating climate by storing, distributing and dissipating energy from solar radiation and exchanging heat with the atmosphere. The oceans are also the main reservoir and distributor of heat and salt and contribute to the formation, distribution and melting of sea ice. They regulate and modulate the evaporation and precipitation processes. Moreover, the oceans act as

the primary storage medium for, and are able to absorb large quantities of the greenhouse gas CO_2 (~37 000 Gt; Falkowski *et al.*, 2000). Since the beginning of the 19th century, the oceans are estimated to have taken up ~50% of fossil fuel emissions and ~30% of all anthropogenic emissions (including those from land-use modifications), thereby reducing the build-up of CO_2 in the atmosphere. CO_2 at the surface of the ocean is in equilibrium with the air and moves freely across the interface. Chemically transformed into dissolved and particulate forms of carbon, it is transferred to the deep ocean by shelf export, by mixing or via an active biological system, and the consequent sinking of particulate carbon from the plankton that plays a crucial role in the carbon cycle.

In turn, the oceans are affected by global climate change that influences temperature, sea level, ocean circulation and the frequency and intensity of storms. Stronger storms affect vertical mixing and wave regimes, which can influence marine ecosystems and habitats. Precipitation will be more variable, with more frequent intense rainfall leading to extensive flooding. Past estimates of the global integral of ocean heat content anomaly indicate an increase of 14.5×10^{22} J from 1955 to 1998 from the surface down to 3000 m depth (Levitus *et al.*, 2005) and 9.2 (± 1.3) $\times 10^{22}$ J from 1993 to 2003 in the upper (0–750 m) ocean (Willis *et al.*, 2004). Climate models exhibit similar rates of ocean warming, but only when forced by anthropogenic influences (Gregory *et al.*, 2004; Barnett *et al.*, 2005; Church *et al.*, 2005; Hansen *et al.*, 2005). Several coupled atmosphere–ocean models have shown global warming to be accompanied by an increase in vertical stratification (IPCC, 2001). Elevated summer temperatures will strengthen near-surface stratification and decrease winter mixing of the water column (e.g. McClain *et al.*, 2004; Llope *et al.*, 2006). A fast temperature rise has been reported for the deep ocean interior (Levitus *et al.*, 2005) and intense warming of sea-surface temperature over the last two decades has been documented for different marine regions with major changes (up to 1.35 °C) occurring in semi-enclosed European and East Asian Seas (Belkin, 2009). Recent observations indicate that the returned subsurface flow associated with the meridional overturning circulation has slowed down due to reduced convective sinking (Bryden *et al.*, 2005). Continuing increases in the length of the melt season are expected to result in an ice-free Arctic during summer within the next 100 years (due to the changing balance between evaporation and precipitation; Johannessen *et al.*, 1999; Laxon *et al.*, 2003). The wide spatial differences observed in temperature and salinity generate density-driven circulation patterns that redistribute water masses between the equator and the poles.

Acidification of the ocean is another effect of global change linked to CO_2 emissions. Since preindustrial times, the average pH of the ocean surface has fallen from approximately 8.21 to 8.10 (Raven, 2005). The pH of the ocean surface water is expected to decrease by a further 0.3–0.4 pH units by the end of the century (Orr *et al.*, 2005), if atmospheric CO_2 reaches 800 p.p.m.v. (scenario of the IPCC). Ocean acidification will alter the chemistry of the CO_2/carbonate system, and is expected to decrease calcium carbonate ($CaCO_3$) saturation states and increase dissolution rates, so that ocean alkalinity and the ocean's capacity to take up more CO_2 from the atmosphere will increase (Doney *et al.*, 2009). If the biological production of carbonate was shut down by ocean acidification, atmospheric CO_2 would decline by approximately 10–20 p.p.m.v. (Gruber *et al.*, 2004). In the near term, this may be observed first in coastal regions where coral reef calcification rates could decrease by as much as 40% by the end of this century (Andersson *et al.*, 2005, 2007). However, over the same timeframe, the uptake rate of CO_2 from the atmosphere could completely overwhelm these natural buffering mechanisms, so the ocean's efficiency for taking up carbon will probably decline with time over the next two centuries (Doney *et al.*, 2009). If predictions on the CO_2 sequestration capacity of the oceans are still uncertain, how marine ecosystems (e.g. in term of food web structure and biogeochemical cycles) can respond to ocean acidification is even more unpredictable (Riebesell *et al.*, 2007; Fabry, 2008; Fabry *et al.*, 2008).

Climate change is expected to have a range of effects on marine ecosystems, including their function and biodiversity. Some effects may be related to changing water temperatures, circulation and/or changing habitat, while others occur through altered pathways within biogeochemical cycles and food webs. Changes in species biogeography (i.e. the poleward movement of native species and an increased risk of invasions of non-native species) have been linked to the North Atlantic Oscillation (Fromentin & Planque, 1996; Beaugrand *et al.*, 2002a), the latitudinal position of the Gulf Stream north wall (Hays *et al.*, 1993; Taylor, 1995) and the Northern Hemisphere temperature (Beaugrand *et al.*, 2002a, b). An increase in precipitation-driven flooding will particularly affect estuaries through enhanced river runoff and changes in nutrients and salinity. In estuaries and upwelling areas, changes in strength and seasonality of circulation patterns can affect the dispersion mechanism of plankton and planktonic larvae (Gaines & Bertness, 1992). In open marine ecosystems, the population dynamics of many marine species are driven by recruitment processes, which are generally synchronized with seasonal production cycles of phytoplankton. If warming results in advancement of the timing of reproduction of these species, this may result in a mismatch with the presence of their main food sources, with a consequent decrease in recruitment success (Hays *et al.*, 2005).

Climate change is expected to change nutrient availability via ocean currents, fluctuations in the depth of the surface mixed layer and period of vertical stratification. Increasing temperatures and enhanced stratification could affect biomass and production of phytoplankton, but in different and even contrasting ways in different oceanic regions. Phytoplankton accounts for approximately 50% of the total photosynthesis on Earth (Field *et al.*, 1998), and contributes to the removal of CO_2 via sinking or transfer by food webs of fixed carbon to the deep ocean. Because pelagic primary producers transfer carbon to higher trophic levels, changes in the timing, abundance or species composition of these organisms will affect food webs. At higher latitudes, atmosphere–ocean general circulation models usually associate a warming climate with an increase in net primary production (NPP) owing to improved mixed-layer light conditions and extended growing seasons (Boyd & Doney, 2002; Le Quéré *et al.*, 2003; Sarmiento *et al.*, 2004). In stratified oceans, different prognostic models consistently yield a decrease of NPP (Bopp *et al.*, 2001; Boyd & Doney, 2002; Le Quéré *et al.*, 2003; Sarmiento *et al.*, 2004). Although with large regional differences, satellite observations at a global scale, provide evidence of a decrease of NPP from 1999 to 2004 (largely driven by changes occurring in the expansive stratified low-latitude oceans; Behrenfeld *et al.*, 2006).

Viruses likely infect every organism on Earth. In aquatic systems, viruses are thought to play important roles in global and small-scale biogeochemical cycling, influence community structure and affect bloom termination, gene transfer, evolution of aquatic organisms and (re)packaging of genetic information. Global change will likely influence all ecosystem components (Genner *et al.*, 2004) including bacteria, archaea and protista. Viruses are the most numerous 'lifeforms' in aquatic systems, with about 15 times the total number of bacteria and archaea. Given that the vast majority of the biomass [organic carbon (OC)] in oceans consists of microorganisms, it is expected that viruses and other prokaryotic and eukaryotic microorganisms will play important roles as agents and recipients of global climate change. Despite the extensive research on the potential effects of increasing CO_2 concentration and global warming on ecosystems (Hughes, 2000; Hays *et al.*, 2005; Doney *et al.*, 2009), our knowledge on the impact of climate change on marine microorganisms and the role of viruses in such change is largely absent.

Marine viruses could have a wide range of potentially simultaneous direct and/or positive and negative feedback type effects on climate change, but the magnitudes of these are still inadequately assessed. This lack of knowledge is probably one of the reasons that the viral component is missing from most climate change models. Viruses can be influenced by climate change in many different ways. They can be influenced indirectly by the changing climate through: (1) potential impact of altered primary production and phototrophic community composition on viruses, their life cycles and virus–host interactions; (2) changes in prokaryotic metabolism and shift in prokaryotic community compositions; and (3) shift from lysogenic to lytic infections or an alternative shift in life strategy. The direct effects of climate change on viruses include the potential effect of rising temperatures and ocean acidification on viral decay.

This review is organized to provide the reader with: (1) an examination of the global relevance of marine viruses, including their effects on biogeochemical cycles, viral diversity and impacts of viruses on autotrophic organisms; (2) a presentation of case studies that synthesize information and provide possible scenarios given our current understanding; and (3) a summary of the salient points of the presented review, and identification of the highest priority research areas for targeted research.

Global relevance of marine viruses

Viruses are the most abundant biological entities in global ecosystems, and comprise $\sim 10^{30}$ particles in the world's oceans (Suttle, 2005, 2007), or about 10-fold greater than prokaryotic abundance (Whitman *et al.*, 1998; Karner *et al.*, 2001), and equivalent to the carbon in ~75 million blue whales (~10% of prokaryotic carbon by weight; Suttle, 2005). Studies conducted in the last two decades have made it increasingly evident that marine viruses play critical roles in shaping aquatic communities and determining ecosystem dynamics (Bergh *et al.*, 1989; Proctor & Fuhrman, 1990; Suttle *et al.*, 1990).

Viral abundance in marine waters ranges from about 10^7 to $10^{10}\,L^{-1}$, and 10^7 to $10^{10}\,g^{-1}$ of dry weight in marine sediments. Therefore, on a volumetric basis, abundances in surface and subsurface sediments exceed those in the water column by 10–1000 times (Paul *et al.*, 1993; Maranger & Bird, 1996; Steward *et al.*, 1996; Danovaro & Serresi, 2000; Danovaro *et al.*, 2002, 2008a, b).

Investigations in both coastal waters and sediments show that viral abundance varies substantially over short time scales and distances (Corinaldesi *et al.*, 2003; Middelboe *et al.*, 2006; Danovaro *et al.*, 2008a). Large differences in viral abundance exist among different environments, with the highest viral abundances typically occurring in coastal and low-salinity waters (Culley & Welschmeyer, 2002; Corinaldesi *et al.*, 2003; Clasen *et al.*, 2008), and surficial coastal sediments (Danovaro *et al.*, 2008b), whereas abundances may be up to three orders of magnitude lower in deep waters (Hara *et al.*, 1996; Magagnini *et al.*, 2007) and in some deep-sea sediments that are largely disconnected from continental material inputs (Danovaro *et al.*, 2008a, b). Nonetheless, care must be taken when interpreting absolute values of viral

abundance, as many data have been collected using electron microscopy and epifluorescence microscopy on preserved samples, both of which can lead to large underestimates of virus abundance (Weinbauer & Suttle, 1997; Wen *et al.*, 2004).

Several studies carried out in different pelagic systems worldwide provided evidence that changes in physiochemical characteristics of the water masses greatly influence viral abundance and distribution. For instance, higher virioplankton abundance has been reported at the thermocline (Cochlan *et al.*, 1993; Weinbauer *et al.*, 1995; Hwang & Cho, 2002; Riemann & Middelboe, 2002), halocline (Winter *et al.*, 2008), chemocline (from oxic to anoxic waters; Taylor *et al.*, 2001, 2003; Weinbauer *et al.*, 2003) or in frontal systems (Wommack *et al.*, 1992; Wilhelm *et al.*, 2002).

Physical and chemical characteristics of surface waters (e.g. temperature, salinity, turbulence and mixing regimes, nutrient availability) are important covariates influencing viral dynamics in marine ecosystems, because they have a major effect on the abundance, distribution and metabolism of planktonic host cells (Wommack & Colwell, 2000; Weinbauer, 2004). To this regard, several studies carried out at a regional scale pointed out that virioplankton abundance decreases moving from productive coastal to oligotrophic off-shore surface waters (Wommack & Colwell, 2000; Corinaldesi *et al.*, 2003; Payet & Suttle, 2008; Evans *et al.*, 2009). Similarly, the distribution of benthic viruses appears dependent upon pelagic primary production and vertical particle flux, indicating a possible causal relationship between viriobenthos and trophic state (Hewson *et al.*, 2001a; Danovaro *et al.*, 2002). Moreover, other studies provided evidence that viral abundance, and the importance of viruses in prokaryotic mortality increases with the productivity of water bodies, with the highest percentage of infected cells in highly eutrophic ecosystems (Weinbauer *et al.*, 1993, 2004). This is supported by the significant positive correlation between viral production and prokaryotic respiration, and between viral production and prokaryotic growth rate observed in different studies (e.g. Glud & Middelboe, 2004; Mei & Danovaro, 2004; Middelboe *et al.*, 2006; Danovaro *et al.*, 2008b).

The analyses of available literature information indicate that the abundance of marine viruses is closely linked with the abundance of their hosts, so that any change in the abundance, metabolic state and doubling time of the prokaryotic host populations will affect viral abundance (Fig. 1a). Comparing the slopes of the regressions between viral and prokaryotic abundance for the water column and sediments suggests that changes in the abundance of pelagic hosts are likely to have a significantly higher effect in terms of viruses released per host cell (due to different lysis rates and burst size; ANCOVA, $P < 0.01$). Similarly, the existence of a link between the autotrophic biomass (expressed as chlorophyll *a* concentration) and viral abundance is evident (Fig. 1b), which suggests the influence of primary producers that might influence viral dynamics, either directly (as hosts) or indirectly (by providing organic sources for heterotrophic prokaryotic metabolism). In this case,

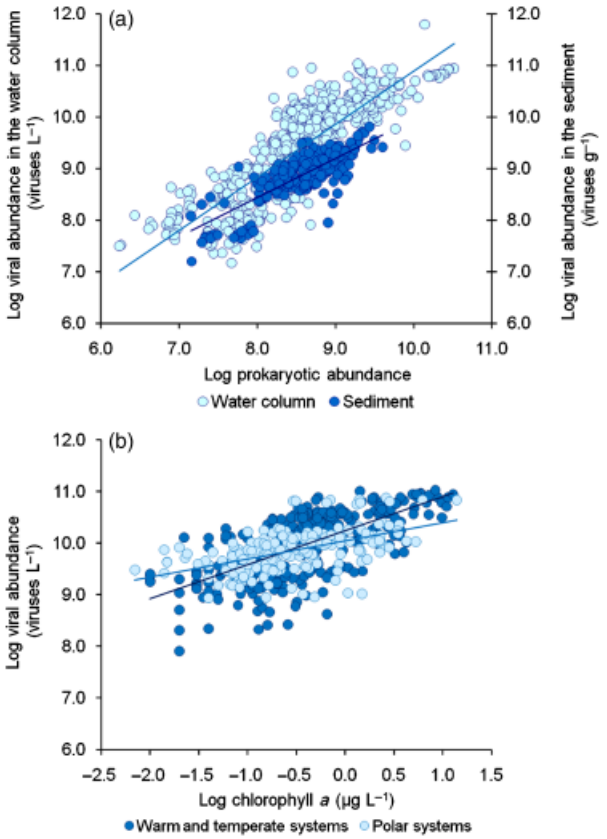

Fig. 1. Relationships between viral and prokaryotic abundance in surface waters and sediments world wide (a) and between viral abundance and chlorophyll *a* (as a proxy of phytoplankton biomass) in surface waters of warm and temperate systems and polar seas (b). All data are log transformed. Prokaryotic abundances are expressed as cells L^{-1} for the water column or cells g^{-1} for the sediment. (a) The equations of the fitting lines are: $y = 1.03x + 0.660$ for the water column ($n = 631$, $R^2 = 0.698$, $P < 0.001$) and $y = 0.761x + 2.35$ for the sediment ($n = 305$, $R^2 = 0.641$, $P < 0.001$). (b) The equations of the fitting lines are: $y = 0.661x + 10.24$ for temperate and tropical ecosystems ($n = 291$, $R^2 = 0.456$, $P < 0.001$) and $y = 0.353x + 10.44$ for polar systems ($n = 195$, $R^2 = 0.271$, $P < 0.001$). Data of the water column are summarized from Maranger & Bird (1995), Steward *et al.* (1996), Guixa-Boixereu *et al.* (2002), Wilhelm *et al.* (2002), Corinaldesi *et al.* (2003), Bongiorni *et al.* (2005), Magagnini *et al.* (2007), Clasen *et al.* (2008), Payet & Suttle (2008), Boras *et al.* (2009) and Evans *et al.* (2009). Unpublished data collected in the Adriatic Sea (Mediterranean Sea) have been also included. Data of sediments are summarized from Danovaro & Serresi (2000), Hewson & Fuhrman (2003), Middelboe & Glud (2006), Mei & Danovaro (2004), Danovaro *et al.* (2005), Middelboe *et al.* (2006), Danovaro *et al.* (2008b) and Danovaro *et al.* (2009). Only data sets, where synoptic measurements are available, have been utilized.

significant differences can be observed among different pelagic systems, such as cold and temperate pelagic waters (ANCOVA, $P < 0.01$; this issue will be discussed later in this review).

Despite the fact that these correlation analyses confirm and reinforce the general idea that viral abundance is tightly dependent upon the abundance of potential host cells in marine ecosystems (Wommack & Colwell, 2000; Weinbauer, 2004; Danovaro *et al.*, 2008a), our understanding of the controlling factors of viral abundance over larger spatial scale in the oceans (thousands of kilometer) is still poor. Wilhelm *et al.* (2003) pointed out that the high variability of viral abundance in surface waters collected along a ~5000 km latitudinal transect in the south-eastern Pacific Ocean may be dependent upon changes in sunlight regimes (influencing the loss of viral infectivity) and oceanographic features (influencing viral transport and residence time in surface waters). Recent investigations carried out from the oligotrophic Sargasso Sea to the more productive northern Atlantic indicate that viral abundance increases with increasing trophic state (Rowe *et al.*, 2008). However, despite a high variability being observed in trophic regimes among the North Atlantic samples investigated, virioplankton abundance remained within a narrow range, suggesting the presence of a dynamic equilibrium between factors influencing viral production and decay (Wilhelm *et al.*, 1998; Weinbauer *et al.*, 1999). Detailed statistical analyses carried out on the data set collected in the two systems lead also to hypothesize that viral distribution is more predictable in rather constant environments such as the Sargasso Sea (due to the relatively constant biological productivity and environmental conditions, such as sunlight regimes) than in highly dynamic systems such as the North Atlantic (experiencing wide changes in biological productivity and complex hydrodynamic regimes). These findings underlie the need of understanding changes in physical, chemical and biological characteristics occurring across different oceanic realms for a better assessment of factors and processes influencing virioplankton distribution.

The role of viruses in marine food webs and biogeochemical cycles

Viral infections are frequently followed by death of the host cells, thus representing an important source of mortality of marine microorganisms. Despite the fact that viruses can cause spectacular epidemics within a wide range of organisms, most marine viruses infect prokaryotes and microalgae, the most abundant organisms in the ocean (Fuhrman, 1999; Weinbauer, 2004). Virus-mediated mortality of prokaryotes, in both water column and sediments, is often in the range of 10–30% and can reach 100% (Heldal & Bratbak, 1991; Suttle, 1994; Fuhrman & Noble, 1995; Wommack & Colwell, 2000; Corinaldesi *et al.*, 2007a, b). In addition, viruses infect microzooplankton (González & Suttle, 1993; Massana *et al.*, 2007) and have been implicated in phytoplankton mortality and the decline of phytoplankton blooms (Suttle *et al.*, 1990; Suttle, 1992; Bratbak *et al.*, 1993; Nagasaki *et al.*, 1994; Fuhrman, 1999; Brussaard, 2004a). The integration of viruses into microbial food web models has shown, moreover, that viral lysis of microbial cells enhances the transfer of microbial biomass into the pool of dissolved organic matter (DOM) (Thingstad & Lignell, 1997; Noble & Fuhrman, 1999; Middelboe *et al.*, 2003a, b; Miki *et al.*, 2008). This in turn can influence nutrient cycling, alter pathways of OC use by prokaryotes (Fuhrman, 1999; Wilhelm & Suttle, 1999; Wommack & Colwell, 2000; Weinbauer, 2004; Suttle, 2005), and divert microbial biomass away from higher trophic levels (Fuhrman, 1992; Bratbak *et al.*, 1994). These viral-induced alterations of organic matter flows, within microbial food webs, have been termed 'viral shunt' (Wilhelm & Suttle, 1999). The viral shunt has also a profound impact on microbial population sizes and biodiversity and horizontal transfer of genetic materials (Suttle, 2005). Because prokaryotes and autotrophic and heterotrophic protists play pivotal roles in biogeochemical cycles and global ocean functioning, viral infections of these groups of organisms have important ecological consequences.

Epidemiological models predict that viral infection rates increase with increasing host cell density (Wiggins & Alexander, 1985) because infection is a direct function of the encounter rate between a pathogen and its host. However, in aquatic systems, this encounter rate is driven by a myriad of processes, including adsorption to particles, and mixing along a range of scales. Encounter rates have been somewhat studied in aquatic systems (Murray & Jackson, 1992; Suttle & Chan, 1994; Wilcox & Fuhrman, 1994), and it is thought that host density drives much of the lytic response.

However, Weitz & Dushoff (2008) suggest that viruses cannot invade high-density host populations due to low rates of host growth and viral lysis, whereas viruses can invade low-density host populations. At high encounter rates between viruses and host cells, there is a strong selection for resistant host cells (Waterbury & Valois, 1993; Tarutani *et al.*, 2000). As distances between cells in sediments are shorter and virus abundances are higher, the probability of contact between viruses and prokaryotic cells is greater than in the water column. Hewson *et al.* (2001b) added viral concentrates to marine benthic microcosms and observed a net decrease in prokaryotes and an increase in aggregates, probably from uninfected cells growing on the products of viral lysis. This result suggests that viral lysis stimulates DOM recycling in sediments. Virus-induced carbon production was equivalent to 6–11% of the average

carbon sedimentation rate (Hewson *et al.*, 2001a). In estuarine sediments, viral-mediated release of dissolved organic carbon (DOC) could sustain 1–8% of the total prokaryotic carbon demand (Glud & Middelboe, 2004; Middelboe & Glud, 2006).

Recent studies reported that viral production in deep-sea benthic ecosystems worldwide is extremely high, and that viral infections are responsible for the abatement of 80% of prokaryotic heterotrophic production (Danovaro *et al.*, 2008b). Virus-induced prokaryotic mortality increases with increasing water depth, and beneath a depth of 1000 m nearly all of the prokaryotic heterotrophic production is transformed into organic detritus. The viral shunt, releasing on a global scale, 0.37–0.63 Gt carbon $year^{-1}$, is an essential source of labile organic detritus in the deep-sea ecosystems (Danovaro *et al.*, 2008b). This process sustains a high prokaryotic biomass and provides an important contribution to prokaryotic metabolism, allowing the system to cope with the severe organic resource limitation of deep-sea ecosystems.

After host cell lysis, the released viruses might infect other hosts or decay. The factors controlling viral decay may provide selective pressures that influence the composition of viral communities. Such changes in viral community structure may also have consequences on the flow of energy and nutrients in aquatic ecosystems (Wommack & Colwell, 2000). Thus, viral decay plays an essential role in the dynamics of microbial food webs and the flow of genetic information within microbial communities.

Theoretical modeling suggests that if the main control of prokaryotic abundance is via protozoan grazing, most of the carbon will be channeled to higher trophic levels in the food web (Wommack & Colwell, 2000). Conversely, if viral infection accounts for most prokaryotic losses, the flow of carbon and nutrients can be diverted away from larger organisms (Bratbak *et al.*, 1990; Proctor & Fuhrman, 1990; Fuhrman, 1992, 1999), thus accelerating the transformation of nutrients from particulate (i.e. living organisms) to dissolved states. Studies comparing the impact of viruses and protozoan grazers on prokaryotes in different pelagic ecosystems have revealed that viral lysis can be a source of prokaryotic mortality comparable or even higher than bacterivory by protists (Fuhrman & Noble, 1995; Guixa-Boixereu *et al.*, 1996, 1999, 2002; Steward *et al.*, 1996; Wells & Deming, 2006c; Bonilla-Findji *et al.*, 2009a). In particular, in deep waters viral lysis of prokaryotes can prevail over protozoan grazing (Wells & Deming, 2006c; Fonda Umani *et al.*, 2010). If this finding can be generalized, it would suggest that viruses could potentially stimulate prokaryotic production and respiration, and increase nutrient regeneration through the liberation of products from cell lysis (i.e. soluble cytoplasmic components and structural materials, extracellular DNA and nutrients). This in turn might have important ecological and biogeochemical consequences, particularly in systems characterized by limited external nutrient loading (Dell'Anno & Danovaro, 2005; Corinaldesi *et al.*, 2007a).

Different studies have emphasized that viral lysis of host cells, releasing labile cellular contents including high-rich phosphorus and nitrogen compounds (such as proteins and nucleic acids), enhances prokaryotic heterotrophic metabolism and nutrient turnover (Middelboe *et al.*, 1996; Noble & Fuhrman, 1999; Wilhelm & Suttle, 1999; Middelboe & Jorgensen, 2006). Among the cell products released by viral lysis, extracellular DNA might represent a particularly important source of nutrients for prokaryotic metabolism (Danovaro *et al.*, 1999; Dell'Anno & Corinaldesi, 2004; Dell'Anno & Danovaro, 2005; Corinaldesi *et al.*, 2007b) or a direct source of exogenous nucleotides for *de novo* synthesis of DNA (Paul *et al.*, 1988, 1989). Recent studies carried out in deep-sea anoxic systems reported that extracellular DNA released by viral lysis had the potential to fulfill an important fraction of the nitrogen and phosphorus requirements of prokaryotes, suggesting that viral lysis may represent an important nutrient source, especially in systems characterized by reduced external supply (Dell'Anno & Danovaro, 2005; Corinaldesi *et al.*, 2007a). At the same time, viral-mediated mortality of host cells, enhancing nutrient regeneration processes, has the potential to contribute in sustaining phytoplankton growth in surface waters of the oceans (Brussaard *et al.*, 1996a, 2007; Gobler *et al.*, 1997; Bratbak *et al.*, 1998; Middelboe *et al.*, 2003b). By this transfer, cellular lysates may function as a nutrient link to noninfected phytoplankton (Bratbak *et al.*, 1994; Poorvin *et al.*, 2004; Brussaard *et al.*, 2007, 2008). Thus, in addition to the direct impact on the mortality of individual algal species, viral lysis may stimulate growth of competing algal populations in the community by supplying regenerated nutrients. However, at present there exist only few direct estimates of the role of viruses for the recycling of nutrients and stimulation of algal production. Gobler *et al.* (1997) provided the first evidence that the inoculation of viral lysates in nutrient-limited diatom cultures caused alleviation of the nutrient limitation. However, viral lysis was not associated with mineralization of nitrogen and phosphorus and the observed stimulation of algal growth rate and biomass in response to algal lysis was proposed to be mainly in the form of organic nutrients (Gobler *et al.*, 1997). Recently, a model system with two autotrophic flagellates (*Phaeocystis pouchetii* and *Rhodomonas salina*), a virus specific to *P. pouchetii* (PpV) and bacteria and heterotrophic nanoflagellates (HNF) was used to investigate effects of viral lysis on algal population dynamics and nitrogen and phosphorus remineralization processes (Haaber & Middelboe, 2009). Lysis of *P. pouchetii* by PpV had strong positive effects on bacterial and HNF abundance, and the mass balance of

carbon, nitrogen and phosphorus suggested an efficient transfer of organic material from *P. pouchetii* to bacterial and HNF biomass through viral lysis. At the same time, the degradation of *P. pouchetii* lysates was associated with significant regeneration of inorganic N and P, corresponding to 78% and 26% of lysate nitrogen and phosphorus being mineralized to NH_4^+ and PO_4^{3-}, respectively. These results showed that the turnover of viral lysates in the microbial food web was associated with significant nitrogen and phosphorus mineralization, supporting the general view that viral lysis by promoting the regeneration of inorganic nutrients can be an important mechanism to sustain primary productivity in pelagic ecosystems.

Viral lysis can be also an important mechanism involved in the recycling of essential organically bound trace elements such as iron (Rue & Bruland, 1995; Hutchins *et al*., 1999), whose availability influences primary and secondary production in large sector of the oceans (i.e. high-nutrient, low-chlorophyll areas, HNLC), including the subarctic Pacific, the equatorial Pacific and the Southern Ocean (Martin *et al*., 1994; Boyd *et al*., 2007).

First empirical data on the potential relevance of viruses in iron cycle were provided analyzing the release and subsequent bioavailability of iron from the lysis of the marine chrysophyte *Aureococcus anophagefferens* (Gobler *et al*., 1997). They found that elevated levels of dissolved iron released during viral lysis was followed by a rapid transfer of iron to the particulate phase, leading to hypothesize that the transfer of iron was the result of rapid assimilation by heterotrophic prokaryotes. Subsequently laboratory experiments, using other model planktonic organisms, have shown that viral lysis resulted in the release of a range of dissolved to particulate iron-containing components and that this iron can be rapidly assimilated by other plankton (Poorvin *et al*., 2004). Mioni *et al*. (2005) found that organic iron complexes released during viral lysis are much more efficiently assimilated by prokaryotes than Fe(III). All of these studies provide evidence that viral lysis can be important in the regeneration of bioavailable iron species, thus potentially providing an important fraction of total bioavailable iron sustaining primary and secondary carbon production. These experimental evidence were confirmed by field studies, which showed that virus-mediated iron release can support as much as 90% of the primary production in HNLC systems, such as the subtropical equatorial eastern Pacific Ocean (Poorvin *et al*., 2004) and further supported during mesoscale experiments of iron fertilization carried out in subarctic Pacific Ocean (Higgins *et al*., 2009). Therefore, because as much of the primary production in the World's oceans is at least sporadically limited by iron availability (Moore *et al*., 2002), the effect of viruses on iron recycling should be taken into adequate account for a better assessment of the global carbon cycle and their implications on climate regulation and feedback (Strzepek *et al*., 2005).

Impact of viruses on photosynthetic organisms

Before the identification of marine viruses as agents of mortality of photoautotrophic organisms, the death of primary producers was ascribed to zooplankton grazing and sedimentation below the photic zone. This view changed rapidly after the discovery that viruses infected both prokaryotic and eukaryotic phytoplankton, and were associated with the reduction of primary productivity (Suttle *et al*., 1990; Suttle, 1992). Subsequently, high titers of viruses (10^2–10^5 mL^{-1}) infecting specific taxa of phytoplankton were measured in near-shore and off-shore waters (Waterbury & Valois, 1993; Suttle & Chan, 1994; Cottrell & Suttle, 1995a; Tomaru *et al*., 2004), implying that viruses were likely significant mortality agents of phytoplankton. Among the highest concentrations measured were viruses infecting strains of marine cyanobacteria (*Synechococcus* sp.), which is consistent with observations that 1–3% of the native cyanobacteria from a variety of locations contain assembled viruses apparently near the end of the lytic cycle (Proctor & Fuhrman, 1990).

However, to quantify the impact of viruses on photosynthetic organisms, it is essential to determine the extent to which they impose mortality on their host. Studies addressing this issue have been conducted during conditions of high host cell abundance, such as during bloom events when the probability of collision between a host and a virus increases; hence, viruses may propagate rapidly through the host population. This may result in bloom collapse if the viral lysis rates exceed the specific host growth rates. This type of interaction has been referred to as control by 'reduction' (Brussaard, 2004b). Compelling evidence that viruses are involved in algal bloom demise, comes from observations that high proportions (10–50%) of cells were visibly infected (using transmission electron microscope) at the end of blooms of *A. anophagefferens* (Sieburth *et al*., 1988; Gastrich *et al*., 2004), *Heterosigma akashiwo* (Nagasaki *et al*., 1994; Lawrence *et al*., 2002) and *Emiliania huxleyi* (Bratbak *et al*., 1993, 1996; Brussaard *et al*., 1996b). Further evidence for viral control of phytoplankton comes from dividing empirical estimates of virus production by burst size, which indicates that viral lysis can be responsible for 7–100% of the mortality of *Phaeocystis globosa* (Brussaard *et al*., 2005a; Ruardij *et al*., 2005) and *E. huxleyi* (Jacquet *et al*., 2002) during bloom events.

Fewer studies have investigated the potential role of viruses in regulating non-blooming photoautotrophic populations. Studies on viral-mediated mortality of the picoeukaryote *Micromonas pusilla* indicated a turnover of the

host standing stock of between 2 and 25% day^{-1} (Cottrell & Suttle, 1995a; Evans *et al.*, 2003), while the cyanobacterium *Synechococcus* has been reported to lose < 1–8% of the host population daily (Waterbury & Valois, 1993; Suttle & Chan, 1994; Garza & Suttle, 1998). These results suggest a stable host–virus coexistence, where the viruses maintain host populations at nonblooming levels; this type of regulation has been referred to as a 'preventive' viral control (Brussaard, 2004b).

Our understanding of the global significance of viral lysis on phytoplankton mortality and primary productivity is far from complete. High phytoplankton lysis rates have been reported not only during bloom termination in eutrophic ecosystems (Brussaard *et al.*, 1995, 1996a), but also under more oligotrophic conditions (Agusti *et al.*, 1998). These results have been supported by reports from Baudoux *et al.* (2007, 2008) of comparable or even higher mortality rates in specific photoautotrophic groups in the oligotrophic subtropical northeastern Atlantic Ocean (0–0.20 day^{-1} in surface water and up to 0.80 day^{-1} at the deep-chlorophyll maximum) than in the eutrophic North Sea (0.01–0.35 day^{-1}). The highest rates of viral lysis observed at the deep-chlorophyll maximum in the oligotrophic system were tightly dependent upon the smallest picoeukaryotic groups rather than the numerically dominating cyanobacteria *Synechococcus* and *Prochlorococcus*.

Although several studies have demonstrated that viruses can cause substantial mortality on their photoautotrophic hosts (reviewed in Mann, 2003; Brussaard, 2004a), the quantitative relevance of viral-induced lysis in the oceans is not well understood. One reason for this is that rates of viral-induced mortality are determined using different approaches; therefore, results from these studies are not necessarily comparable. More importantly, all of the methodological approaches used to quantify viral-mediated mortality rely on different assumptions and conversion factors (e.g. Steward *et al.*, 1992; Noble & Fuhrman, 2000; Wilhelm *et al.*, 2002). Apart from the methodological problems, information on the impact of viruses on primary producers is largely scattered in time and space and very few studies have addressed the impact of viruses at community levels. However, observations indicate that a modest 20% enrichment of seawater with native virus concentrates can reduce phytoplankton biomass and primary production by 50% (Suttle *et al.*, 1990; Suttle, 1992). Therefore, although it is recognized that viruses can infect a broad range of photosynthetic hosts, the lack of straightforward and reliable approaches for estimating the rates of mortality imposed by viruses on phytoplankton (Kimmance & Brussaard, 2010), and the lack of information at wide spatial and temporal scales represent the major obstacles for incorporating viral-mediated processes into global models of carbon cycling.

Potential factors influencing virus-mediated mortality

Host defense

A number of factors can regulate the dynamics of virus-mediated mortality including host abundance, morphology, physiology and their potential to develop defense mechanisms. Encounter rate between viruses and host cells is a stochastic process that depends on host and virus abundance as well as characteristics such as host size and motility (Murray & Jackson, 1992). At a given virus concentration, contact rates will be higher with host cells that are larger and motile. Other host morphological characteristics can influence viral infection rate. For example, in mesocosm studies *P. globosa* cells embedded in a colonial matrix tended to escape viral infection (Brussaard *et al.*, 2005a; Ruardij *et al.*, 2005). The most efficient defense against viral infection is resistance. The incidence of resistant host strains has been reported for algal viruses in culture (Thyrhaug *et al.*, 2003) as well as in the field (Waterbury & Valois, 1993; Tarutani *et al.*, 2000). Theory based on bacterial host–phage interactions suggests that resistance has a physiological cost for host cells; resistant cells may have a competitive disadvantage against susceptible hosts (Levin *et al.*, 1977). So far, the importance and the mechanisms underlying resistance of phytoplankton against viruses remain largely unclear. It has been suggested that acrylic acid (AA) and dimethyl sulfide (DMS) negatively effects titers of *E. huxleyi* virus (Evans *et al.*, 2006), and that strains of *E. huxleyi* with high lyase activity [i.e. capable of efficient conversion of β-dimethylsulfoniopropionate (DMSP) into AA and DMS] are resistant to infection. It was suggested that the cleavage of DMSP into DMS and AA during cell lysis of *E. huxleyi* may reduce the titers of *E. huxleyi* viruses, and therefore decrease the probability of infection of further cells. These observations led to arguments that the DMSP system in phytoplankton may operate as a chemical defense against viral infection. This study supported the hypothesis that virucidal compounds can be produced alongside viruses during viral infection, and in turn, can reduce infection rates of other algal cells (Thyrhaug *et al.*, 2003). Another recent study also reported that *E. huxleyi* can escape viral attack by switching its life cycle from a diploid to haploid stage (Frada *et al.*, 2008). The motile, noncalcifying haploid stage is resistant to the specific coccolithoviruses of *E. huxleyi* (i.e. EhV-86; Schroeder *et al.*, 2002; Wilson *et al.*, 2005a; Allen *et al.*, 2006b, 2007), which conversely infects and lyses the diploid calcifying phase. Besides this, *E. huxleyi* strains, which are virus resistant, showed higher DMSP-lyase activity than strains that are susceptible to virus infection (Schroeder *et al.*, 2002).

Another example of potential host defense strategy includes the enhanced sinking rates of *H. akashiwo* cells when infected by a virus (Lawrence & Suttle, 2004). Infected cells rapidly sink out of the euphotic zone, which, in turn, may prevent viral infection of conspecifics. As obligate parasites, viruses depend on the host cellular machinery to propagate. Several studies have shown that the algal host growth stage may significantly influence the lytic viral growth cycle. Reduction in viral burst size (Van Etten *et al.*, 1991; Bratbak *et al.*, 1998) and even prevention of viral infection were observed (Nagasaki & Yamaguchi, 1998) during the algal host stationary growth phase. The algal host cell cycle stage may also influence the production of algal viruses (Thyrhaug *et al.*, 2002). Viral infection of *Pyramimonas orientalis* at the onset of the light period led to higher viral production than when infected at the beginning of the dark period. Conversely, *P. pouchetii* infection was independent of the host cell cycle. Different environmental variables known to influence phytoplankton growth rates (i.e. light, nutrients and temperature) may, furthermore, affect viral development. For instance, darkness prevents viral infection or replication in different prokaryotic and eukaryotic photoautotrophic hosts (MacKenzie & Haselkorn, 1972; Allen & Hutchinson, 1976; Waters & Chan, 1982). Temperature may alter the susceptibility of host species to virus infection as shown for *H. akashiwo* (Nagasaki & Yamaguchi, 1998). Nutrient limitations were found to have variable effects; phosphate depletion resulted in a reduction of the burst size of *P. pouchetii* and *E. huxleyi* viruses (Bratbak *et al.*, 1993, 1998) or delayed cell lysis in *Synechococcus* (Wilson *et al.*, 1996). Under nitrogen depletion, the production of *E. huxleyi* viruses was delayed (Jacquet *et al.*, 2002) and a reduction in viral burst size was observed for *P. pouchetii* (Bratbak *et al.*, 1998). In the ocean, phytoplankton cells experience wide fluctuations in environmental conditions (e.g. light, nutrient, temperature). For instance, light intensity and nutrient concentrations can vary during phytoplankton blooms and across the water column. These variations may thus affect the impact of viruses on host populations. Furthermore, contrasting nutrient conditions found in oligotrophic vs. eutrophic environments may determine different rates of viral lysis of phytoplankton in these ecosystems.

Viral lysis vs. other sources of phytoplankton mortality

Whether phytoplankton biomass sinks, is preyed upon, or undergoes lysis, it has implications on the structure of phytoplankton communities. As stated by Kirchman (1999), 'how phytoplankton die largely determines how other marine organisms live'. The fraction of autotrophic carbon escaping lysis or grazing in the photic layer of the oceans can sink in the deep ocean interior to the sea floor where it is mostly converted to CO_2 by heterotrophic degradation and utilization and partially buried in the sediment (Buesseler *et al.*, 2007; Smith *et al.*, 2008). In contrast, zooplankton grazing channels phytoplankton biomass to higher trophic levels, whereas the release of cell constituents mediated by lysis directly affects the standing stock of DOC and particulate organic carbon (POC), forcing the food web toward more regeneration pathways (Wilhelm & Suttle, 1999; Brussaard *et al.*, 2005b). By the simplest approximation, cell lysis switches biomass into organic debris (dissolved and particulate) (Suttle, 2005) and increases the availability of carbon for heterotrophic prokaryotic metabolism, thereby leading to an increase in bacterial production and respiration rates and a decrease in the carbon-transfer efficiency to higher trophic levels (Fuhrman, 1999; Wommack & Colwell, 2000; Suttle, 2005, 2007). Different field studies have reported that organic matter released by viral-induced algal lysis was sufficient to fulfill most of the heterotrophic prokaryotic carbon demand (Brussaard *et al.*, 1996b, 2005b). The quantification of cell lysis in relation to sinking and grazing rates is, therefore, essential for assessing the flow of matter and energy in marine ecosystems, and for comprehending the carbon cycle and its implications on climate regulation.

In addition to viruses, other mechanisms can cause phytoplankton lysis. For example, some pathogenic bacteria and fungi kill phytoplankton (Fukami *et al.*, 1992; Mayali & Azam, 2004). Another example includes allelopathic interactions between phytoplankton species. In this type of interaction, the production of a metabolite by a phytoplankton species has an inhibitory effect on the growth or physiological function of another phytoplankton species that may result in cell lysis (Vardi *et al.*, 2002; Legrand *et al.*, 2003). Nonpathogenic forms of algal cell lysis are also reported. For example, chemostat cultures of the diatom *Ditylum brightwellii* under nitrogen-controlled conditions experience a type of 'intrinsic mortality' (Brussaard *et al.*, 1997). Another form of autocatalyzed cell death was shown to share similarities with the programmed cell death (PCD) observed in higher plants and metazoans (Berges & Falkowski, 1998; Vardi *et al.*, 1999; Berman-Frank *et al.*, 2004). PCD, unlike 'natural cell death' or 'necrosis' refers to an active, genetically controlled degenerative process, which involves a series of apoptotic features such as morphological changes (e.g. cell shrinkage, vacuolization) and complex biochemical events (e.g. activation of PCD markers like caspases) that ultimately lead to cell lysis. Laboratory studies suggest that a wide range of phytoplankton is programmed to die in response to adverse environmental conditions (see review by Franklin *et al.*, 2006). The PCD pathway in phytoplankton was found to operate under environmental stresses such as intense light (Berman-Frank *et al.*, 2004), darkness (Berges & Falkowski, 1998), nutrient depletion (Berman-Frank *et al.*, 2004), CO_2 limitation and oxidative

stress (Vardi *et al.*, 1999). Some apoptotic features, possibly related to PCD, have also been detected upon viral infection (Lawrence *et al.*, 2001). The complete genome sequence of a virus infecting *E. huxleyi* has recently revealed the presence of genes encoding the biosynthesis of ceramide, which is known to suppress cell growth and is an intracellular signal for apoptosis (Wilson *et al.*, 2005b).

Although the extent of 'spontaneous' cell lysis is still a matter of debate (Bidle & Falkowski, 2004), the few available data suggest that viral-induced cell lysis can prevail (Baudoux *et al.*, 2007). Also information on the extent of mortality on phytoplankton assemblages induced by viral lysis and zooplankton grazing under different trophic conditions is extremely limited. However, this information is of paramount importance for understanding how planktonic food webs can respond to shifts in nutrient regimes potentially induced by climate change. Estimates of mortality rates induced by viral lysis and microzooplankton grazing on specific groups of phytoplankton have been recently reported for eutrophic and oligotrophic ecosystems. Baudoux *et al.* (2006) estimated the mortality rates on *P. globosa*, a widely distributed bloom-forming phytoplankton that can sustain a large fraction of primary productivity in some areas (Lancelot & Billen, 1984). During two spring blooms in the North Sea, viral mortality of *P. globosa* cells was comparable with that of microzooplankton grazing (up to 0.35 and 0.4 day^{-1}, respectively), although there was high variability in their relative importance over the course of the blooms and between years. In the oligotrophic subtropical northeastern Atlantic, viral lysis was estimated to be responsible for between 50% and 100% of the total cell losses for the smallest picoeukaryotes (group I), with rates ranging from 0.1 to 0.8 day^{-1} (Baudoux *et al.*, 2008). Although the available information does not allow to draw general conclusions, these studies pinpoint that viral lysis may divert a significant fraction of the photosynthesized carbon pool into organic debris both in oligotrophic and eutrophic marine ecosystems.

The mortality of benthic microalgae is generally attributed to grazing, burial, resource exhaustion or apoptosis (Fenchel & Staarup, 1971). Only in the last few years has viral infection been recognized as an additional important source of microalgal mortality in sediments. Hewson *et al.* (2001b) tested experimentally the effect of viral infection on microphytobenthos abundance in sediments by enriching the natural community with viral concentrates. A 90% decrease was reported in the biomass of benthic pennate diatoms and *Euglenophytes* decreased by 20–60%. These data indicate that viruses may have a strong influence on benthic microalgae. Virus-induced mortality of bacteria associated with benthic microalgae may be instrumental for the maintenance of dissolved organic nitrogen flows from heterotrophic bacteria to benthic microalgae (Cook *et al.*, 2009).

Viral diversity

Given the sensitivity of the composition of prokaryotic and protistan communities to environmental conditions, and that viruses typically have a narrow host range, significant climate change would have a dramatic impact on viral diversity. Investigations using both metagenomic and gene-targeted approaches have revealed that the genetic diversity of viruses in aquatic ecosystems is enormous, and constitutes the largest reservoir of unknown genetic diversity in the oceans (Suttle, 2005). Although error estimates are large, metagenomic analyses indicate that there are thousands of different dsDNA viruses in tens to hundreds of liters of water (Breitbart *et al.*, 2004b; Angly *et al.*, 2006), and that even the most abundant viral genotypes comprise a small percentage of the entire assemblage. In contrast, although the diversity of RNA viruses is also high, these communities appear to be much less even with a few genotypes dominating the community (Culley *et al.*, 2006). Similarly, studies targeting marker genes from specific groups of viruses reveal enormous genetic variation. Even within closely related groups of phage, the genetic variation within highly conserved genes encoding structural proteins indicates that the diversity is not only enormous, but that there are numerous groups of phages for which there are no cultured representatives (Fileé *et al.*, 2005; Short & Suttle, 2005; Comeau & Krisch, 2008; Labonté *et al.*, 2009). Despite the enormous levels of genetic richness in natural assemblages of viruses, there are distinct differences in the composition of viral communities associated with environmental differences. For example, there are distinct differences in the genotypic composition of viruses among different oceanic provinces (Angly *et al.*, 2006). Although some marine virus genotypes appear to be widespread across a wide range of biomes (Short & Suttle, 2002, 2005; Breitbart *et al.*, 2004b; Breitbart & Rohwer, 2005), others are much more restricted in distribution. For example, homologues to cyanophage sequences were much rarer in the Arctic Ocean than in temperate oceans, which in turn were lower than in subtropical seas.

In the pelagic realm, high levels of genetic diversity are found in viruses infecting both heterotrophic and autotrophic prokaryotes and eukaryotes. Significant genetic variation can be found even within closely related viruses infecting a single strain of phytoplankton (Cottrell & Suttle, 1995b; Schroeder *et al.*, 2002), and the diversity can be enormous with viruses infecting broad genera such as *Synechococcus* or *Prochlorococcus* (Zeidner *et al.*, 2005; Sullivan *et al.*, 2006; Chénard & Suttle, 2008; Chen *et al.*, 2009). Moreover, the diversity of viruses is even larger than might be expected when one considers that viruses from unrelated families can infect the same species of phytoplankton including *H. akashiwo* (Nagasaki & Yamaguchi,

1997; Lawrence *et al.*, 2001; Tai *et al.*, 2003) and *M. pusilla* (Cottrell & Suttle, 1991; Brussaard *et al.*, 2004).

The diversity of the viriobenthos has received less attention; however, metagenomic approaches have indicated that there are 10^4–10^6 different viral genotypes in 1 kg of nearshore marine sediment (Breitbart *et al.*, 2004a). These empirical estimates are similar to results obtained in Monte-Carlo simulations, which suggest that sediments containing 10^{12} viruses could host at least 10^4 viral genotypes (Breitbart *et al.*, 2004a). To the extent that host communities are different between the water column and sediments, the *in situ* viral communities can also be expected to differ. However, sinking of particles and infected cells means that viruses that obligately infect pelagic hosts can also routinely be recovered from sediments (Suttle, 2000; Lawrence *et al.*, 2002). Comparisons among viral sequences indicate that although there are distinct differences among phage genotypes in sediment and water-column samples, the same major phylogenetic groups occur in both environments (Labonté *et al.*, 2009). Although some viral genotypes appear to be widely dispersed in very different environments (Short & Suttle, 2002, 2005; Breitbart & Rohwer, 2005), the overall genetic composition of viral communities is spatially and temporally dynamic over a wide range of spatial and temporal scales (Wommack *et al.*, 1999; Steward *et al.*, 2000; Frederickson *et al.*, 2003; Short & Suttle, 2003; Mühling *et al.*, 2005; Hewson *et al.*, 2006), as would be expected given the sensitivity of microbial community composition to environmental conditions.

Overall, it is becoming clear that although some genetically similar viruses are widespread across a wide range of habitats, most are constrained to the specific environmental conditions where their hosts can survive and reproduce. Changing climate will affect these conditions, change the distribution of host organisms, and hence, the viruses as well. In some instances, we are likely to lose entire microbial communities and their associated viruses. For example, as ice shelves disappear the microbial communities that thrive in brine pools within the ice (Wells & Deming, 2006b), in epishelf lakes above the ice and ice-dam lakes behind the ice will also disappear (Vincent *et al.*, 2009) leading to a loss in biodiversity and potentially metabolic function.

Interactive roles of marine viruses in global climate change

Case study 1: Examining the potential impact of rising temperatures on viral interactions

Climate change is expected to cause an increase in sea-surface temperature (Belkin, 2009), with the greatest increases expected in the Norwegian, Greenland and Barents Seas (*c.* 4–6 °C), and the higher latitudes of the Northern Hemisphere (~7 °C) (ACIA, 2005). In turn, these changes will have significant effects on the associated microbial communities. Although there is considerable scatter in the relationship, the available data (Fig. 2b) show that prokaryotic heterotrophic production increases with rising temperature until approximately 15 °C (Apple *et al.*, 2006). The cap on increasing production with rising temperature is linked to the concomitant increase in respiration, and the resulting reduction in growth efficiency (Fig. 2), which decreases by about 2.5% per 1 °C of temperature increase (Rivkin & Legendre, 2001).

As viral replication and life cycle are closely linked with host metabolism, increases in temperature will likely influence the interactions between viruses and the cells they infect. For example, as growth rate increases in prokaryotes, the length of the lytic cycle decreases and burst-size increases (Proctor *et al.*, 1993; Hadas *et al.*, 1997); this should lead to higher rates of virus production. Increases in burst size with increases in production have been observed in natural systems, although evidence across systems is limited (Parada *et al.*, 2006).

Evidence that rising sea-surface temperatures will affect the associated virus communities can be inferred from examining the relationships between viral abundance and temperature for different oceanic regions (Fig. 3a). The strongest temperature effect was observed in temperate-open oceans (ANCOVA, $P < 0.01$), where a temperature increase of only a few degrees was associated with a doubling of viral abundance. However, the fraction of the total variance explained by these relationships was generally low, indicating that factors influencing virioplankton distribution are more complex than those predicted by temperature alone. Although we observed positive relationships between viral abundance and temperature in different oceanic regions, overall a decreasing pattern of viral abundance with increasing temperature can be identified when global data are grouped together. Patterns between viriobenthos abundance and temperature (Fig. 3b) were different than those revealed by the analyses of the water-column data, but the data set is too limited to draw firm conclusions. The inverse pattern of virioplankton abundance with temperature suggests that latitudinal changes, which influence sunlight regimes and trophic characteristics can have cascade effects on the host growth rates and consequently on viral infectivity. To this regard, multivariate analyses conducted on a large data set from the Atlantic Ocean revealed that temperature was an important variable controlling virioplankton distribution in the Sargasso Sea, but not in the northeastern Atlantic (Rowe *et al.*, 2008). At the same time, recent experimental studies revealed that, over short time scales, changes in temperature do not directly influence viral abundance (Feng *et al.*, 2009).

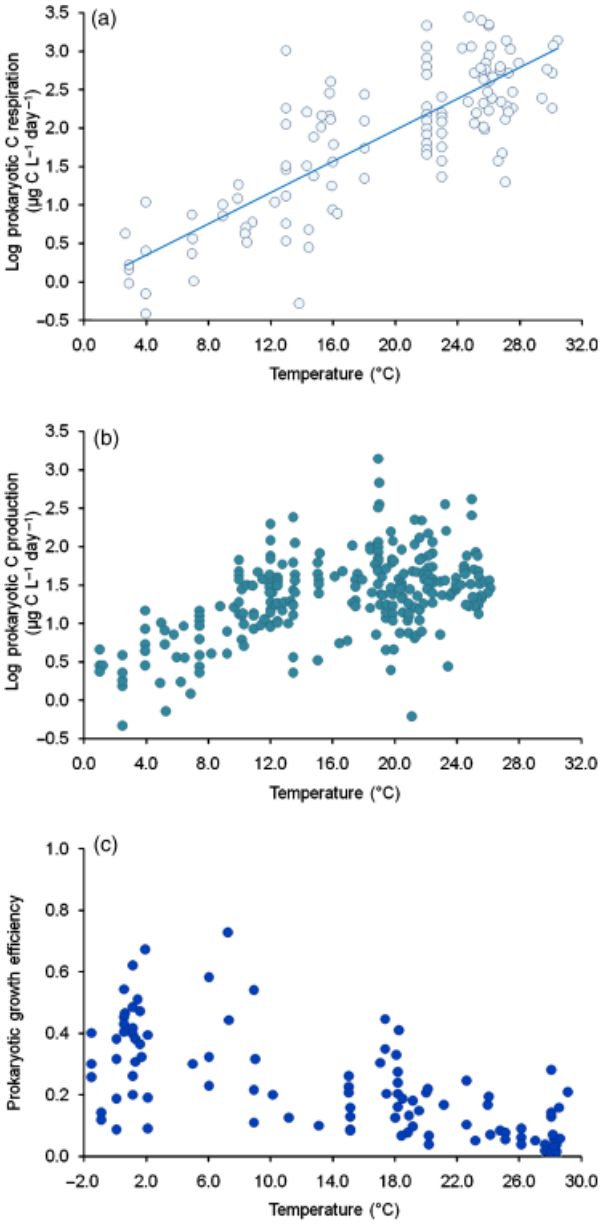

Fig. 2. Relationships between temperature and prokaryotic carbon respiration (a), prokaryotic carbon production (b) and prokaryotic growth efficiency (c). The equation of the fitting line between temperature and prokaryotic carbon respiration is: $y = 0.102x - 0.059$ ($n = 121$, $R^2 = 0.648$, $P < 0.01$). The data sets of synoptic measurements of temperature and prokaryotic carbon production and respiration have been collected from different estuarine systems and have been summarized from Apple *et al*. (2006). Data on temperature and prokaryotic growth efficiency have been collected in marine ecosystems world wide and have been summarized from Rivkin & Legendre (2001).

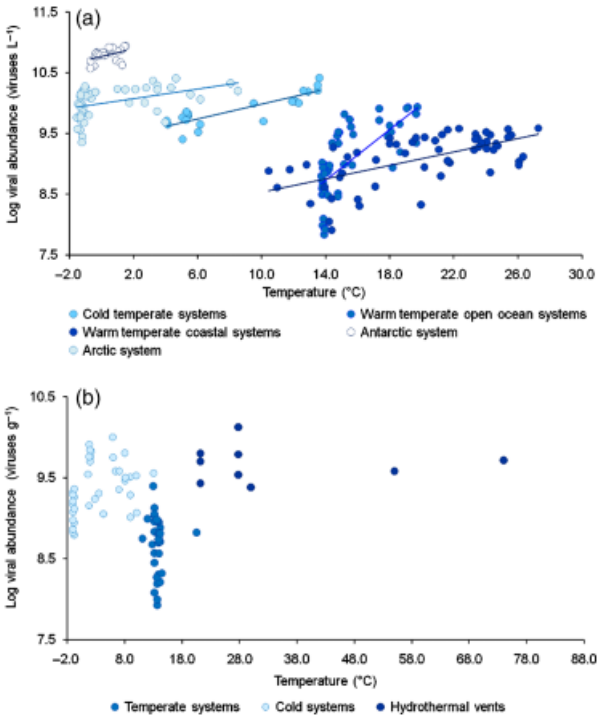

Fig. 3. Relationship between viral abundance (log transformed) and temperature in surface waters (a) and sediments (b) collected world wide. (a) The equations of the fitting lines are: $y = 0.063x + 10.77$ for Antarctic system ($n = 24$, $R^2 = 0.183$, $P < 0.05$), $y = 0.0398x + 9.99$ for Arctic system ($n = 48$, $R^2 = 0.192$, $P < 0.01$), $y = 0.061x + 9.37$ for cold temperate systems ($n = 21$, $R^2 = 0.728$, $P < 0.001$), $y = 0.208x + 5.82$ for warm temperate open ocean systems ($n = 50$, $R^2 = 0.485$, $P < 0.001$) and $y = 0.055x + 7.97$ for warm temperate coastal systems ($n = 57$, $R^2 = 0.380$, $P < 0.01$). Data of the water column are summarized from Steward *et al*. (1996), Guixa-Boixereu *et al*. (2002), Magagnini *et al*. (2007), Payet & Suttle (2008) and Evans *et al*. (2009). Unpublished data collected in the Adriatic Sea (Mediterranean Sea) have been also included. Data of sediments are summarized from Danovaro *et al*. (2008b) and Manini *et al*. (2008). Only data sets, where synoptic measurements are available, have been utilized.

The relationships between viral production in surface waters and temperature (Fig. 4) also show that, in cold-water systems, higher viral production rates are associated with warmer temperatures. The relationship for warm-temperate systems (i.e. systems at tropical and mid-latitudes) is less clear, although there is a downward trend with increasing temperature. These results are consistent with the scenario that different oceanic regions will respond differently to changes in surface temperatures caused by climate change. The data on viral production and temperature in the benthic compartment are very unevenly distributed making any inferences weak; however, in cold-water systems, there is a trend for viral production rates to be higher at warmer temperatures (Fig. 4b).

The inferred changes in viral abundance and production associated with higher temperatures are secondary effects stemming from changes in host cell communities, but physical effects such as changes in virus-decay rates with temperature (Nagasaki & Yamaguchi, 1998; Wells &

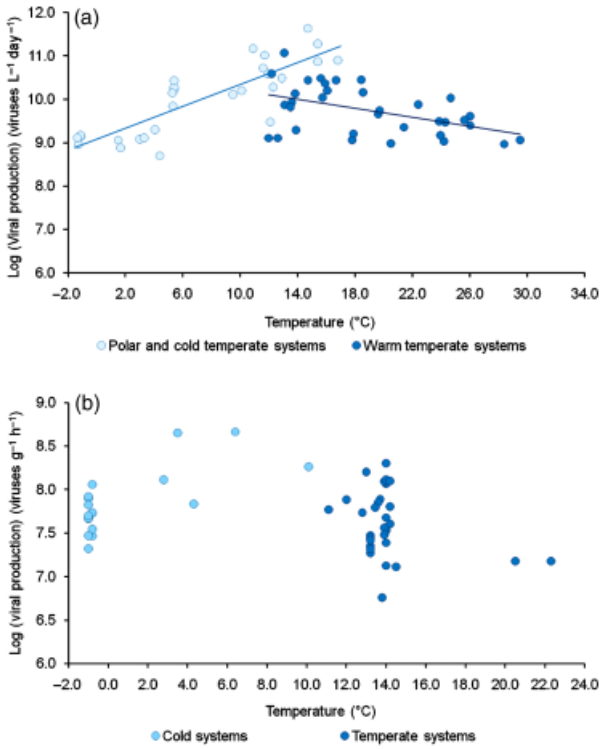

Fig. 4. Relationship between viral production (log transformed) and temperature in the water column (a) and in the sediment (b) of different marine systems. (a) The equations of the fitting lines are: $y = 0.126x + 9.09$ for polar and cold temperate ecosystems ($n = 34$, $R^2 = 0.709$, $P < 0.01$) and $y = -0.052x + 10.728$ for warm temperate ecosystems ($n = 34$, $R^2 = 0.230$, $P < 0.05$). Data of the water column are summarized from Steward *et al.* (1996), Wilhelm *et al.* (2002), Bongiorni *et al.* (2005), Boras *et al.* (2009), Evans *et al.* (2009) and Motegi *et al.* (2009). Data of sediments are summarized from Danovaro *et al.* (2008b). Only data sets, where synoptic measurements are available, have been utilized.

Deming, 2006a) can also play a role. Ultimately, higher productivity of host cells results in increased burst sizes and decreases in the latent period, resulting in higher virus production rates. This effect is revealed in overall relationships between host cell and viral abundances (Maranger & Bird, 1995; Clasen *et al.*, 2008; Wilhelm & Matteson, 2008) and in the seasonal dynamics observed in host cell and viral abundances ranging from warm-temperate waters such as the Gulf of Mexico (Suttle & Chan, 1993; Cottrell & Suttle, 1995a; McDaniel *et al.*, 2002), cold-temperate environments (Waterbury & Valois, 1993; Nagasaki *et al.*, 2004a, b) and the Arctic Ocean (Payet & Suttle, 2008). The observed dynamics are driven by seasonal changes in productivity; what is not clear is whether predictable seasonal changes in hosts and viruses are good proxies for the large-scale and long-term changes that would be anticipated from a changing climate. Ultimately, the effects from long-term changes in surface temperatures on physical and chemical features, such as stratification and the availability of resources, will have the greatest impact on viral production rates and abundances. If primary productivity decreases, we can expect cascading effects on viral production.

One effect that can likely be extrapolated is that the relative importance of lysogeny could change in response to alterations in the productivity of systems as the result of a changing climate. A pattern is emerging where the relative importance of lytic infection decreases as the proportion of lysogens in the population increases in response to lower levels of productivity. This pattern generally holds whether comparing productive coastal with oligotrophic off-shore environments (Jiang & Paul, 1996; Weinbauer & Suttle, 1999), or seasonal changes in temperate (McDaniel *et al.*, 2002) or Arctic environments (Payet & Suttle, 2009). In the Arctic, the high proportion of lysogens that is observed during periods of low productivity is also reflected in the disproportionate number of prophage homologues found in viral metagenomic data from the Arctic Ocean (Angly *et al.*, 2006).

Case study 2: Examination of the effects of temperature, salinity and nutrient changes on viral life strategies

Among viral life cycles in marine systems, lytic and lysogenic infections are most frequently observed (Ripp & Miller, 1997; Wommack & Colwell, 2000; Weinbauer, 2004), despite chronic, pseudolysogenic and polylysogenic infections being possible (Hurst, 2000; Weinbauer, 2004; Chen *et al.*, 2006). Different studies reported that a high proportion of marine bacterial isolates can be lysogenic (Moebus & Nattkemper, 1983; Ackermann & DuBow, 1987; Jiang & Paul, 1994, 1998; Chen *et al.*, 2006; Paul, 2008) and that inducible lysogens can represent a large fraction of the community (Ortmann *et al.*, 2002; Weinbauer *et al.*, 2003). However, this fraction, as determined by prophage induction due to mitomycin C, can vary widely among different marine environments from undetectable up to 100% (Weinbauer & Suttle, 1996, 1999; Weinbauer, 2004).

Temperate phages can enter into either a lysogenic or lytic interaction with their host cell, which is termed the lysogenic decision (Williamson & Paul, 2006). The environmental factors that influence the lysogenic decision are currently not well understood, and the molecular mechanisms behind whether a phage enters into a lysogenic or lytic interaction with its host cell are still largely unclear (Long *et al.*, 2008). Understanding these aspects can help to clarify and potentially forecast the effects of changing environmental conditions induced by present climate changes (e.g. temperature rise, shifts in nutrient availability, freshening

of surface waters) on viral–host interactions and viral life strategies in the oceans.

Current models in thermohaline circulation suggest that in some areas of the oceans the increased vertical stratification due to climate changes can result in severe nutrient limitation (Sarmiento *et al.*, 2004). Such nutrient shifts can have important consequences on viral life strategies through changes occurring in heterotrophic and autotrophic hosts. Several studies have suggested that nutrient availability may have an important influence upon whether viruses enter into lytic or lysogenic interactions with their host cells (Bratbak *et al.*, 1993; Tuomi *et al.*, 1995; Wilson *et al.*, 1996; Wilson & Mann, 1997; Hewson *et al.*, 2001a; Williamson & Paul, 2004; Long *et al.*, 2008). Wilson & Mann (1997) proposed that lysogenic interactions are favored when nutrient concentrations are low and the virus to host cell ratio is high. This is consistent with observations from the Arctic Ocean showing that the highest proportion of lysogens occur in well-mixed cold oligotrophic waters, when up to 34% of the cells could be induced (Payet & Suttle, 2009). Moreover, eutrophic benthic systems, showing a low virus to host cell ratio, were characterized by a low fraction of lysogens (from undetectable to 14%, Glud & Middelboe, 2004; Mei & Danovaro, 2004). However, this is not a general rule because a higher incidence of lysogens has been also reported in nutrient-rich marine systems when compared with oligotrophic environments (Jiang & Paul, 1994). Inorganic nutrient limitation, particularly PO_4^{3-}, has been suggested as a possible constraint on viral replication and/or prophage induction (Wilson & Mann, 1997). Wilson *et al.* (1996) found that 100% of *Synechococcus* sp. WH7803 cells underwent viral lysis under phosphate-enriched conditions, while only 9.3% of the cells were lysed by viruses under phosphate-limited conditions. During mesocosm experiments, Gons *et al.* (2006) showed that in phosphate-limited conditions cyanobacterial population strongly declined, but when the systems were supplied with phosphorus, the cells grew quickly concomitantly with an increase of the viral replication rates. Bratbak *et al.* (1993) also provided evidence that phosphate limitation resulted in the inhibition of viral replication in the marine coccolithophorid *E. huxleyi*. Williamson & Paul (2004) reported that nutrient amendments to marine water samples stimulated lytic phage production as a consequence of the increased host-cell growth. Similarly, Long *et al.* (2008) reported along a gradient of trophic conditions from the Mississippi river plume to off-shore waters located in the Gulf of Mexico that cyanophage and bacteriophage induction decreased with the increase of the productivity of their hosts (i.e. heterotrophic bacterial and *Synechococcus* assemblages). The effects of changes in nutrient availability can also cause a reduction of genome sizes of host cells resulting in a protection against persistent viral infection (Brown *et al.*, 2006). Overall, these findings suggest that shifts in nutrient availability in the oceans potentially induced by present climate change trends could have large-scale implications on heterotrophic and autotrophic organisms with cascade effects on viral–host interactions and viral life strategies. If low-nutrient conditions promote lysogeny, coastal and estuarine environments may shift to more strongly favor lytic over lysogenic lifestyles. As well, increased vertical stratification in surface waters of the open oceans may cause severe nutrient limitation, owing to higher levels of lysogeny. This suggested pattern of increasing nutrient limitation due to stronger stratification may occur first in the polar oceans, promoting a stronger influence of lysogeny over prokaryotic assemblages and a decreased prokaryotic role in nutrient regeneration. Ultimately, this could reduce the importance of the 'viral shunt', thus increasing the transfer of organic matter to the higher trophic levels.

The potential effects of temperature on viral life strategy are still unclear. Cochran & Paul (1998) conducted a seasonal study on lysogeny in Tampa Bay (Gulf of Mexico) and detected prophage induction only at temperatures $> 19\,^{\circ}C$. Similarly, Wilson *et al.* (2001) reported induction by elevated temperatures of virus-like agents from symbiotic zooxanthellae cells isolated from sea anemones. Conversely, Williamson *et al.* (2002) detected a higher fraction of lysogenes in winter (when water temperature was low). Other studies carried out in the Gulf of Mexico reported that prophage induction occurs within a temperature range of 14 to 29 °C and the lack of correlation between the percentage of lysogenized bacteria and temperature (Weinbauer & Suttle, 1996, 1999). Moreover, results from the Baltic Sea and from deep waters in the Mediterranean Sea suggest that lysogeny can be higher than 50% at temperatures $< 15\,^{\circ}C$ (Weinbauer *et al.*, 2003). The effects of temperature on the transition from lysogenic to lytic lifestyles have been also investigated by specific experiments using different phage–host systems. Williamson & Paul (2006) reported that a rapid temperature shift did not cause any effect on the production of phages by the 7HSIC/*Listonella pelagia* phage–host system.

Also, salinity can influence viral life strategies (Husson-Kao *et al.*, 2000; Lunde *et al.*, 2005; Gnezda-Meijer *et al.*, 2006; Williamson & Paul, 2006). In lysogenic strains of *Streptococcus thermophilus*, sodium chloride concentrations beyond the cell's normal range resulted in almost immediate cell lysis. Bacteriophage fragments were observed by electron microscopy, indicating that the bacteriophages produced were not stable (Husson-Kao *et al.*, 2000). On the other hand, increased concentrations of sodium chloride led to an increase in the phage latent period during induction experiments with *Vibrio* sp. (Gnezda-Meijer *et al.*, 2006). Induction studies with CTXφ, a phage that infects *Vibrio cholerae*, showed that when the salinity was at or $< 0.1\%$ w/v, the

majority of transducing particles were inactivated. CTXφ was most stable at salinities $> 0.5\%$. Additionally, the frequency of spontaneous induction of φLC3, a *Lactococcus lactis* phage, significantly decreased as the osmolarity of the solution was increased from 0% to 1.0%. This indicates that the stability of the prophage can increase concomitant to salinity increase (Faruque *et al.*, 2000; Lunde *et al.*, 2005). Williamson & Paul (2006), using the φHSIC/*L. pelagia* marine phage–host system, reported that growth and phage production were stimulated at high salinities and depressed at low salinities. Moreover, using the same phage–host system, it has been reported that HSIC-1a cells growing at salinity of 11 p.p.t. produce *c.* 2 orders of magnitude less phages than those growing at salinity of 39 p.p.t. (Long *et al.*, 2007). Moreover, in low-salinity conditions a lysogenic-like relationship in the HSIC-1a pseudolysogen is favored through the modulation of phage gene expression (Long *et al.*, 2007).

Environmental conditions influencing host physiology can have also important consequences on the burst size of infected cells by viruses (Brown *et al.*, 2006). There is robust evidence that the largest burst sizes are encountered in waters characterized by high salinities (250 p.p.t., burst size = 200, Guixa-Boixareu *et al.*, 1996). At the same time, burst sizes in freshwater systems (burst size on average 28–40) are significantly higher than in marine waters (burst size on average 20–25; Wommack & Colwell, 2000; Parada *et al.*, 2006). Because freshwater and marine viral populations are thought to be genetically distinct (Sano *et al.*, 2004; Angly *et al.*, 2006; Wilhelm *et al.*, 2006; Bonilla-Findji *et al.*, 2009b; Clasen & Suttle, 2009), it is likely that the mixing of the two environments will cause important shifts in host growth rates and availability of DOM, thereby altering burst size. If climate change will increase the nutrient concentrations, especially poleward (Shaver *et al.*, 1992; Falkowski *et al.*, 1998), it could be expected that lytic life strategies could be increased in importance through a shift toward increasing burst size, accompanied by increased rates of infection due to the addition of both freshwater viruses to the system and increased rates of growth for bacteria and phytoplankton alike (and increased DOM availability with senescing cells).

If we pair examinations of burst size with taxonomic distribution studies, it can be suggested that myoviruses have the possibility to expand their coverage to the global marine systems (Williamson *et al.*, 2008). With highly adapted systems to infect a range of hosts, myoviruses may have an advantage over other virus types. Also, expecting the increased role (already postulated by Paerl & Huisman, 2009) of cyanobacterial populations, it is possible to hypothesize an increased prevalence of myoviral infection. Williamson *et al.* (2008) showed that the majority of virus sequences with database homologues recovered from the Global Ocean Survey metagenomic data set belonged to myoviruses. In part, this is likely because more marine myoviruses (primarily cyanophages) have been isolated (Suttle & Chan, 1993; Waterbury & Valois, 1993; McDaniel *et al.*, 2006) and their genes and genomes have been sequenced (Millard *et al.*, 2004, 2009; Sullivan *et al.*, 2005, 2006; Zeidner *et al.*, 2005) than viruses from other taxonomic groups. For example, of 35 cyanophage isolates infecting *Synechococcus*, 33 were myoviruses (McDaniel *et al.*, 2006). Some of these cyanophages could infect almost 70% of the 25 host isolates tested. Interestingly, the cyanophages isolated from surface waters were much more likely to infect across wide host range than their subsurface counterparts, and almost half of the cyanobacteria contained inducible prophages.

The concept of cascades of predator–prey interactions raises the possibility for global shifts in viral lifestyles caused by changes in environmental conditions. Increases in global temperatures, indeed, will favor increased rates of prokaryotic production and growth rates (Rivkin & Legendre, 2001). A shortening of doubling times, and increasing prokaryotic production will cause two processes that may cancel each other out to some extent. First, prokaryotic cell lysis will release components that are known to be highly active in the process of viral decay. Nucleases, proteases, high-molecular-weight organic compounds and cell debris will be released into aquatic environments causing rates of viral decay to increase (Noble & Fuhrman, 1997; Wommack & Colwell, 2000). Increasing resources for the prokaryotes from the viral 'shunt' will contribute to larger burst sizes, and potentially for enhanced rates of adaptation and evolution. The extent of both sets of processes is, of course, in question, so that the relative importance remains to be seen.

As ocean temperatures rise, precipitation patterns change and freshening of the surface oceans occur, there will be simultaneous, intermingled and difficult-to-predict cascading effects.

Until field scenarios are studied in this context, modeling efforts are overly simplistic and more study is required before model predictions can be useful in predicting what will occur in the field (Gons *et al.*, 2006).

Ultimately, viruses and the hosts they infect are highly coevolved systems that have coexisted for billions of years. These systems can respond rapidly to environmental changes on time scales from days to decades, and collectively will be highly adapted to the relatively minor environmental changes, in an evolutionary sense, that will result from climate change. However, the potentially cascading effects caused by possible shifts in viral life-cycle strategies in respond to changing climate could have much more dramatic effects on global biogeochemical cycles.

Case study 3: Viral adaptation with global climate change: the changing freshwater–marine continuum

A global examination of excursions from freshwater to marine and return is challenging due to a wide array of unknowns such as, extent of polar (especially Arctic) freshening, rate of sea-level rise and modeling of heat balance. To assess the possible viral adaptation with global climate change related to freshening, stratification and salinity regimes, we must turn to studies that have examined excursions between the freshwater and marine biomes.

A brief examination of what we currently know of viral dynamics across the freshwater–marine transition proves necessary as a foundation. A recent review by Wilhelm & Matteson (2008) provides an excellent overview of the commonalities and differences between marine and freshwater viruses. The salient points of summarized material in that body of work are that: (1) viruses play an important role in the microbial food webs of both systems, (2) trophic status appears to be a more important determination of burst size, rates of virus production and prevalence of lysogeny/pseudolysogeny than salinity, (3) viruses in both systems are important controlling factors of microbial community structure, (4) freshwater and marine viruses appear to be taxonomically distinct (also some viruses are shared across systems) and (5) host range is a major unknown. However, the effects of modifications of salinity, osmotic stress and pH on marine viruses are still largely unknown.

The analysis of literature data reveals that systems characterized by lower salinity generally display higher viral abundance (Fig. 5a). However, the data set is largely influenced by the availability of data from few estuarine systems (such as the Chesapeake Bay) and these results could reflect the more specific characteristics of the sampling areas rather than a general trend. This applies also to the benthic compartment for which the data set available is still rather limited (Fig. 5b). In addition, it is possible that the positive effect on viral abundance is linked to the different nutrient availability and trophic state in estuarine systems supporting a higher host abundance and metabolism rather than to a direct effect on the viral assemblages. The response of benthic viruses to changes in salinity is unclear, as the apparent increase of viral production with decreasing salinity is statistically weak (Fig. 6) and data on viral-decay rates (from which viral production, according to Fischer *et al.*, 2004 can be estimated) are too limited to provide a comprehensive view of the potential effects of changing salinity on viral dynamics.

Changes in the salinity and pH can influence virus survival by influencing the extent of virus adsorption to particles (Harvey & Ryan, 2004). In particular, cation

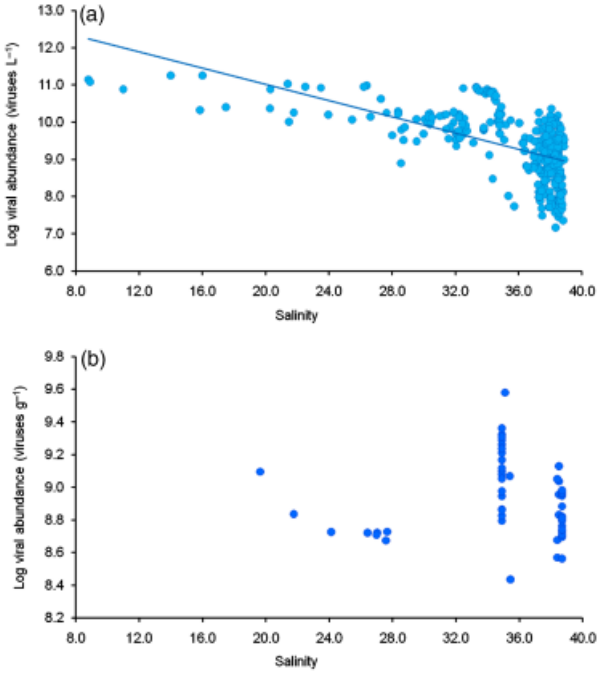

Fig. 5. Relationships between salinity and viral abundance in surface waters (a) and in surface sediments (b). All data of viral abundance are log transformed. (a) The equations of the fitting lines are: $y = -0.109x + 13.20$ ($n = 333$, $R^2 = 0.350$, $P < 0.01$). Data of the water column are summarized from Steward *et al.* (1996), Guixa-Boixereu *et al.* (2002), Wilhelm *et al.* (2002), Corinaldesi *et al.* (2003), Bongiorni *et al.* (2005), Magagnini *et al.* (2007), Payet & Suttle (2008) and Evans *et al.* (2009). Unpublished data collected in the Adriatic Sea (Mediterranean Sea) have been also included. Data of sediments are summarized from Danovaro *et al.* (2008b). Only data sets, where synoptic measurements are available, have been utilized.

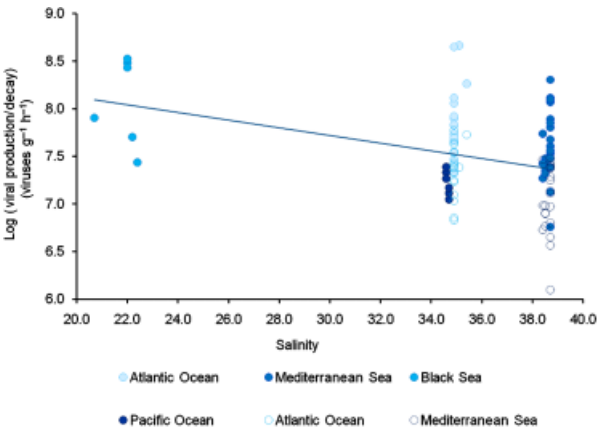

Fig. 6. Relationship between salinity and viral production (filled dots) (a) and viral decay (empty dots) (b) in surface sediments collected world wide. The equation of the fitting line is $y = -0.044x + 9.06$ ($n = 94$, $R^2 = 0.15$, $P < 0.001$). Data are summarized from Danovaro *et al.* (2008b) and Corinaldesi *et al.* (2010). Only data sets, where synoptic measurements are available, have been utilized.

concentrations and pH of the medium affect virus adsorption by determining the thickness of the diffuse layer, thus promoting or inhibiting van der Waals attraction forces (Gerba, 1984; Lukasik *et al.*, 2000; Harvey & Ryan, 2004). The adsorption of viruses generally increases as pH decreases and as cation concentrations, especially the concentrations of multivalent cations, increase (Gerba, 1984; Lance & Gerba, 1984; Grant *et al.*, 1993; Penrod *et al.*, 1996). Salt concentrations have been demonstrated to play a significant role in the adsorption of poliovirus type 1 and echovirus type 7 to 11 solid waste components (Sobsey *et al.*, 1975). Viruses were shown to adsorb efficiently in the presence of high concentrations of dissolved salts, while in the absence of dissolved salts an efficient viral adsorption was not observed.

Unknowns that hinder speculation about suggested transitions of freshwater to marine and mixing of marine and freshwater populations, especially for microbial-loop components must include the traits that permit adaptation of both viruses and bacteria to different salinities, host range across salinity regimes and genus, and the importance of other concomitant changes associated with global climate change that can affect successful viral infection (e.g. penetration of UV light, the heightened role of nucleases and proteases with increasing temperature). For viruses, the complex details of the story relating to host range and adaptation (i.e. packaging and transfer of genes) are only beginning to be unraveled. There are a few studies from which we can glean some insight into likely changes.

Freshening of the poleward oceanic environments has been discussed at length in a range of documents (e.g. Peltier *et al.*, 2006). A synthesis conducted by Peterson *et al.* (2002) suggests that freshwater increases from sources to the Arctic Ocean are linked to a 'freshening' of the North Atlantic. It has been suggested that the high-latitude freshwater cycle is one of the best sentinels for changes in climate and broad-scale atmospheric dynamics related to global climate change. The research conducted by Peterson *et al.* (2002) focuses on increases in glacial melting, precipitation and runoff to the Arctic Ocean and North Atlantic. The reason for the concern over freshening of the Arctic Ocean and North Atlantic is that a significant increase of freshwater flow to the Arctic Ocean could slow or halt the Atlantic Deep Water formation, a driving factor behind the great 'conveyor belt', also known as thermohaline circulation (e.g. Broecker & Denton, 1989). This is the oceanic current responsible for upward distribution of nutrient-rich waters, redistribution of salinity and assimilation of thermal energy. The atmosphere is warmed over the North Atlantic when thermohaline circulation is dominant and strong. It has been theorized that freshening of the Arctic and North Atlantic could cause a breakdown in the ocean circulation pattern of the Gulf Stream, resulting in a much cooler European continent (Fig. 7). In addition to determining the changes in the overall volumes of freshwater that contributed to the high-latitude oceans, a main challenge has been to determine the mechanisms of freshwater delivery, the significance of each freshwater source and how it has changed through time. Other scientists have described the importance of both polar oceans in global meridional circulation (Lumpkin & Speer, 2007). The polar waters impact the entire global ocean in a myriad of ways (Johnson *et al.*, 2008). Main details related to the freshwater–saltwater transition are a continuation of observations in the waters of Antarctic origin, showing decadal warming and freshening of abyssal waters. Certain basins (e.g. South Atlantic) have shown more warming trends than others (Coles *et al.*, 1996; Johnson & Doney, 2006).

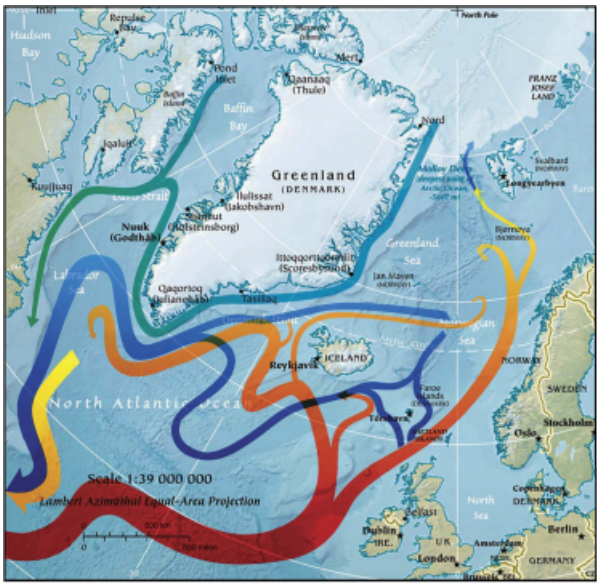

Fig. 7. Schematic illustration of the North Atlantic Current. Pathways associated with the transformation of warm subtropical waters into colder subpolar and polar waters in the northern North Atlantic. Along the subpolar gyre pathway, the red to yellow transition indicates the cooling to Labrador Sea Water, which flows back to the subtropical gyre in the west as an intermediate depth current (yellow).

Given the scenario of freshening at the poles, one likely process is the heightened contribution of freshwater viral and bacterial taxa into marine systems, and the increased opportunity for crossing over of marine and freshwater taxa. A seminal study conducted by Sano *et al.* (2004) showed the possibility for freshwater viruses to cross into the marine biome and replicate normally. Suttle & Chan (1993) highlighted the fact that marine cyanophages isolated from Texas and New York coastal waters belonged to the same three families of viruses that have been observed to infect freshwater cyanobacteria (e.g. *Siphoviridae*, *Myoviridae* and

Podoviridae). Lytic cyanophages isolated by Suttle & Chan (1993) caused lysis of a wide array of green and red strains of *Synechococcus* and occurred over a wide range of temperatures. A significant finding of the work conducted by Suttle & Chan (1993) was that viruses isolated from seawater of coastal waters of New York and Texas, the Gulf of Mexico and a hypersaline lagoon were able to infect *Synechococcus* hosts from the North and tropical Atlantic. Lu *et al.* (2001) also found a wide array of morphotypes of cyanophages, many exhibiting wide cross-reactivity with host organisms, and demonstrating increasing importance with increasing salinity in estuaries. In the case of relatively abrupt freshwater–salinity changes, as in enhanced tidal mixing due to sea-level rise, saltwater intrusion, freshwater outwelling into marine systems, a study was conducted by Cissoko *et al.* (2008) to examine viral–bacterial interactions in a tropical estuary of Senegal.

Experiments were conducted to assess the impact of various types of addition of freshwater to seawater and vice versa. To start, the nutrient concentrations in the marine water used (from a coastal marine station) were up to 100-fold higher than in the freshwater reservoir. On the other hand, in the starting waters, the concentrations of viruses and bacteria were four- and twofold more abundant in the freshwater vs. the marine water. Both the seawater and freshwaters used for these experiments, however, were from a tropical region, and showed bacterial concentrations and rates of production that were higher than those commonly seen in temperate regions. However, the viruses appeared to be less abundant than those seen in temperate regions, and the virus to bacteria ratios were one of the lowest reported so far in the literature. When concentrated forms of freshwater were amended with virus-free seawater, bacterial production was dramatically negatively impacted with only 10% of the bacterial production retained. However, a startling response to the bacterial collapse was seen within 24 h, and stabilization of the bacterial community within only 48 h. The viral production in the same treatments was also negatively impacted and within 48 h, both treatments also showed a dramatic recovery to levels even higher than observed in the start of the experiment. Surprisingly, similar freshwater additions to marine samples did not show a similar effect. The marine bacteria actually benefited from the freshwater addition, probably growing on the remnants of osmotically lysed bacterial cells from the freshwater samples. Other studies have reported strong impacts of seawater on freshwater bacteria (Painchaud *et al.*, 1995; del Giorgio & Bouvier, 2002; Troussellier *et al.*, 2002). Conversely, it has been reported for some time that marine bacteria are highly adaptable to salinity changes (Forsyth *et al.*, 1971). If salinity plays a strong role in the control of community composition (Lozupone & Knight, 2007), then the processes of sea-level rise, saltwater intrusion and freshening could have strong effects on the geographical success of dominant bacterial taxa and subsequently the success of their viral counterparts.

An additional study details the work conducted to assess the coexistence of natural phage populations with susceptible hosts (Daniels & Wais, 1990). The authors and others (Linski, 1988) suggest that the existence of an available refuge in which the host is safe from phage attack can provide a means for resistance to infection and can prevent elimination of the host population. At the same time, they reported that when host viability is threatened by dilution of the environment, phages can proliferate acting as scavengers on the host population. This could be the case of relatively rapid shifts of salinity (heightened salinity of already hypersaline lakes due to localized evaporation, rapid inundation of hypersaline environments from freshwater inundation or precipitation) in extreme environmental conditions that could favor the phage proliferation. Further studies of phage–host interactions (especially including the development of lysogeny, pseudolysogeny and 'chronic infection' life strategies), over tightly controlled conditions but focused on *in situ* populations, will provide key information on small-scale interactions.

Recent work presented by Logares *et al.* (2009) investigated the excursions of the microorganism world between freshwater and marine environments. In that review, an important point made was that freshwater and marine microorganisms belonging to SAR11 lineage are not closely related, and that saline–freshwater transitions from one environment to the other have been few during the evolutionary diversification of SAR11. The increased occurrence of freshwater–marine transitions associated with the present global climate change can result in changes in host–virus relationships due to host adaptation in changing salinity environments. Increased experimental, proteomic and genomic data analysis in systems with gradients of salinity and studies of the lifestyle of viruses and their host interactions will provide important information about the likely alterations coming with global climate change. So far, the focus has been on the type of infection cycle that the viruses use for replication. Certainly, attention in the future should be paid to the distribution of specific types of viruses, RNA and DNA viruses alike and to an understanding of their hosts, as reported in Koonin *et al.* (2008).

The work of Breitbart & Rohwer (2005) included a global examination of DNA sequences within viral genomes, and showed that identical phage sequences were found in wide-ranging environments including freshwater, terrestrial and marine . If it is true that a subset of viruses found in aquatic systems globally have wide host ranges, then lytic-based viral production from these viruses will continue to dominate regardless of the system into which they are introduced. Previous studies (Sano *et al.*, 2004 and to some extent Wilcox & Fuhrman, 1994) investigated the population

density necessary for appropriate contact rates to support lytic infection and tested the idea that viruses could replicate across biomes (including freshwater, terrestrial and marine). They found that if an appropriate amount of active, infective viruses is transferred from one biome to another (e.g. from a marine to a freshwater system), the replication from lytic viral infection will continue to be a strong controlling factor across systems. Given the fact that freshening processes will carry viral and bacterial groups with the freshwater to the marine system and that a subset of cyano- and bacteriophages in those systems will have wider host ranges than others, shifts in viral assemblages and cyano- and bacterial diversity would be expected. The process of lateral gene transfer has been observed in both environments, and because new taxonomic information packaged in freshwater viruses will become increasingly available to the marine bacterial and cyanobacterial assemblages, lytic infection will continue to play an important role in gene transfer and evolution. The main controlling factor in the shifts observed along the freshwater–marine continuum will be the speed and mechanisms of change, which are currently poorly understood. As increased information becomes available regarding the physical process of freshening, it will become increasingly possible for scientists to use multiyear data to examine the shifts in viral activity and to produce simulation experiments (e.g. Cissoko *et al.*, 2008), under different scenarios of climate change and on larger spatial and temporal scales.

Case study 4: Interactive roles of marine viruses on CO_2 sequestration and biological carbon pump

The gas equilibrium at the ocean–atmosphere interface facilitates the exchange of gases in both directions. Gases such as CO_2 escape to the atmosphere when partial pressures of the gas in water are higher than those in the air. Conversely, CO_2 is taken up when partial pressures in ocean surface are lower than those in the atmosphere. Low CO_2 concentrations in surface waters are due to sequestration of carbon in intermediate and deep waters. Two main CO_2 sequestration processes in the ocean interior are known: (1) the physical pump (or solubility pump) and (2) the biological pump, accounting for about 1/3 and 2/3 of the sequestration, respectively. The physical pump is driven by chemical and physical processes (i.e. cooling and deep water formation) and it maintains a sharp gradient of CO_2 between the atmosphere and the deep oceans where 38×10^{18} g of carbon is stored (Chisholm, 2000). Thermal and density stratification separate the shallow surface water layers (approximately a few 100 m deep) from the deep water layers (approximately a few kilometers deep) across the global oceans, except in polar regions. Large-scale, three-dimensional ocean circulation creates pathways for the transport of dissolved gases, heat and freshwater from the surface ocean into the density-stratified deeper ocean, thereby isolating them from further interaction with the atmosphere for several hundreds to thousands of years, and influencing atmospheric CO_2 concentrations over glacial, interglacial and anthropogenic timescales.

Oceanic primary production represents about half of the planet's primary production, ranging from 35 to 65 Gt carbon $year^{-1}$ (Del Giorgio & Duarte, 2002), with open ocean production accounting for over 80% of the total. Primary production processes are sustained both by photoautotrophic organisms belonging to the Bacteria and the Eukarya domain, whose quantitative relevance changes in space and in time mainly in relation to temperature, light intensity and nutrient availability (Falkowski & Raven, 2007). For instance, large size classes of phytoplankton, such as diatoms, are responsible for about one-fifth of the total photosynthesis on Earth (Nelson *et al.*, 1995; Armbrust, 2009) and tend to dominate phytoplankton communities in well-mixed coastal and upwelling regions. They also dominate along the sea-ice edge where sufficient light, inorganic nitrogen, phosphorus, silicon and trace elements are available to sustain their growth (Morel & Price, 2003). Conversely, photosynthetic picoeukaryotes and cyanobacteria (belonging to the genus *Synechococcus*, *Prochlorococcus* and *Trichodesmium*), which are responsible for up to 40% of the total oceanic primary productivity, tend to dominate in the photic zone of vast oligotrophic regions of the oceans where nutrient availability is low (such as in tropical and subtropical open ocean regions, e.g. Partensky & Garczarek, 2010).

Photosynthetic processes occurring in the photic layer are not only relevant for sustaining the entire food web but also play a key role in global climate regulation. Photosynthesis, indeed, through the conversion of CO_2 into biomass and the subsequent sinking of organic particles in the ocean interior lowers the partial pressure of CO_2, thus promoting the drawdown of atmospheric CO_2 (Falkowski *et al.*, 2004). This process is known as the organic carbon pump. Much of the carbon 'fixed' within the phytoplankton during photosynthesis is converted back to CO_2 and released to the atmosphere by the respiration of phytoplankton, bacterioplankton and zooplankton grazing in the mixed surface layers. POC is exported as planktonic debris to deeper waters at a rate of $\sim$10 PgC $year^{-1}$ (Duce *et al.*, 2008), but much of the organic matter not consumed by metazoans is remineralized into DIC by microbial degradation within the top 500 m of the water column, becoming available for further photosynthesis. A small proportion of the POC (roughly 5–20%) will sink into the density-stratified deeper ocean before it decays, where it will remain, isolated from further interaction with the atmosphere for an estimated

1000 years (Chisholm, 2000) until deep ocean currents and upwelling processes return the deep water to the surface (Denman, 2008). It is estimated that only a very small amount of the planktonic debris (0.1%) ever reaches the ocean sediments and is eventually buried (Middelburg & Meysman, 2007). The food web's structure and the relative abundance of species influence the amount of CO_2 that will be pumped to the deep ocean.

Besides photosynthesis, the production of carbonate by marine calcifying organisms is the other mechanism by which inorganic carbon is utilized and exported as particulate inorganic carbon (PIC) into the ocean interior (Rost & Riebesell, 2004). This process is known as the carbonate pump. Major calcifying plankton groups in the oceans include autotrophs (coccolithophores) and heterotrophs (aragonite-forming euthecosomatous pteropods and calcite-forming foraminifera), which together account for up to 70% of the global $CaCO_3$ production (Holligan & Robertson, 1996) with coccolithophores contributing to the large extent (Westbroek *et al.*, 1993).

Although both biological carbon pumps remove carbon from the ocean surface, they exert an opposite effect on the CO_2 concentrations: photosynthesis decreases CO_2 concentrations, whereas calcification shifts the carbonate system toward higher CO_2 concentrations. The relative strength of the two biological carbon pumps, represented by the so-called rain ratio (the ratio of PIC to POC of the exported biogenic material), determines to a large extent the efficiency of the biological pump, hence the flux of CO_2 between the ocean surface and the atmosphere (Zondervan *et al.*, 2001; Rost & Riebesell, 2004). Therefore, the assessment of factors influencing the biological carbon pump in the oceans is not only relevant for the understanding of the ecosystem functioning of the largest biome on Earth but also for a better comprehension of the global carbon cycle and its implications on climate (Riebesell *et al.*, 2009).

The biological responses of marine ecosystems to the present climate change are related both to possible direct effects of rising atmospheric CO_2 through ocean acidification (i.e. decreasing seawater pH) and ocean carbonation (i.e. increasing CO_2 concentration), and indirect effects through ocean warming and changes in circulation and mixing regimes. These changes are expected to impact marine ecosystem structure and functioning and have the potential to alter the cycling of carbon and nutrients in surface oceans with likely feedback effects on the climate system (Riebesell *et al.*, 2009).

Alterations in the physical state of the ocean will affect both the solubility pump and the biological pump. Rising atmospheric CO_2 leads to higher sea-surface temperature and a concurrent reduction in CO_2 solubility. It is estimated that this positive sea-surface temperature feedback could reduce the oceanic uptake of anthropogenic carbon by 9–15% by the end of the 21st century (Sarmiento & Le Quéré, 1996; Joos *et al.*, 1999; Plattner *et al.*, 2001).

Ocean warming impacts the pelagic ecosystem both directly and indirectly in three ways: (1) through decreased supply of inorganic nutrients because of a slowdown in vertical mixing and convective overturning, (2) through increased thermal stratification, causing increasing light availability for photosynthetic organisms suspended in the upper mixed layer and (3) through the effect of increasing temperatures on the rates of biological processes. Which of these factors will dominate in a particular region and at a given time depends on the hydrographic conditions, the composition of the pelagic community and the activities of its components (Riebesell *et al.*, 2009).

The effects of sea-surface warming on the marine biota and the resulting impacts on marine carbon cycling will differ depending on the prevailing light and nutrient conditions in the upper mixed layer and the composition of the pelagic community. A number of different approaches consistently predict an overall decrease in primary and export production in the tropics and mid-latitudes and a poleward migration of geographic boundaries separating biogeochemical provinces (Crueger *et al.*, 2008; Riebesell *et al.*, 2009). A decreased nutrient supply in surface waters of oligotrophic regions of the oceans is expected to determine a decrease in primary production and a decline in the strength of the biological pump (Riebesell *et al.*, 2009). Increased nutrient-utilization efficiency at high latitudes, however, can increase the efficiency of the biological pump (Sarmiento *et al.*, 2004), thus potentially representing a negative feedback. Warming of ocean surface water can also have a direct impact on the food webs because heterotrophic processes are more temperature sensitive than autotrophic processes (Rivkin & Legendre, 2001). The higher temperature sensitivity of heterotrophic (relative to autotrophic) processes is expected to shift the balance between primary production and respiration/remineralization in favor of the latter (Wohlers *et al.*, 2009). Additionally, warming shifts the partitioning of OC between the particulate and dissolved phase toward an enhanced accumulation of DOC (Wohlers *et al.*, 2009). This shift may cause a decrease in the vertical flux of particulate organic matter and a decrease in remineralization depth. The overall effect could be a decline in the strength and efficiency of the carbon pump (positive feedback).

Beside sea-surface warming, the rising of atmospheric CO_2 concentrations also determines major changes in seawater carbonate chemistry. Although the decrease in seawater pH may pose a threat to the fitness of pH-sensitive groups, in particular calcifying organisms, the increasing oceanic CO_2 concentration will likely be beneficial to some groups of photosynthetic organisms, particularly those that operate a relatively inefficient CO_2 acquisition pathway.

Stimulating effects of ocean carbonation have been primarily observed for processes related to photosynthetic activity. These effects include CO_2-enhanced rates of phytoplankton growth and carbon fixation (Hein & Sand-Jensen, 1997), organic matter production (Zondervan *et al.*, 2002; Leornardos & Geider, 2005) and extracellular organic matter production (Engel, 2002). A decreasing pelagic calcification and the resulting decline in the strength of the carbonate pump lowering the drawdown of alkalinity in the surface layer can increase the uptake capacity for atmospheric CO_2 in the surface layer. However, the capacity of this negative feedback is expected to be relatively small. $CaCO_3$ may act as a ballast in particle aggregates, accelerating the flux of particulate material to depth (Armstrong *et al.*, 2002; Klaas & Archer, 2002). Reduced $CaCO_3$ production could therefore slow down the vertical flux of biogenic matter to depth, shoaling the remineralization depth of OC and decreasing carbon sequestration (positive feedback). Increased production of extracellular organic matter under high CO_2 levels (Engel, 2002) may enhance the formation of aggregates (Engel *et al.*, 2004) and thereby increase the vertical flux of organic matter (negative feedback, Schartau *et al.*, 2007). It is important to note that our present knowledge of pH/CO_2 sensitivities of marine organisms is based almost entirely on short-term perturbation experiments, neglecting the possibility of adaptation. Also, little is presently known regarding the effects from multiple and interacting stressors, such as sea-surface warming and changes in nutrient and light availability.

One of the questions which remains open is how and the extent to which the viral shunt influences the efficiency of biological pump and consequently the sequestration of carbon in the ocean interior. This task is difficult to solve as marine viruses are still practically ignored into global models of carbon cycling and nutrient regeneration. Moreover, the wide natural variability in the biological pump efficiency, depending on the study area, can influence the carbon flux balance making even more complex the modeling of the viral role in the ecosystem functioning (Buesseler *et al.*, 2007). However, with as much as one quarter of the primary production in the ocean ultimately flowing through the viral shunt (Suttle, 2007), there is a crucial need to integrate and explicitly incorporate the viral component in global ocean carbon models. One scenario is that viral lysis could increase the efficiency of the biological pump by enriching the proportion of carbon in particulate material that is exported from the photic zone (Suttle, 2007), as much of the nitrogen and phosphorus in viral lysis products will be in a labile form (e.g. nucleic acids and free amino acids; Noble & Fuhrman, 1997) relative to the C, which will be tied up in structural material. This could lead to an increase of the complexity of the system and consequently contribute to an increase of ecosystem resilience and might potentially resolve some of the inconsistencies in the present generation of ocean biogeochemical models (Brussaard *et al.*, 2008).

The impact of climate change on the viral-mediated controls on the biological pump also depends on the effects of rising sea-surface temperatures on the viral-induced host mortality. Analysis of the available literature suggests that in different marine systems (where synoptic data of viral-induced prokaryotic mortality and temperature were available) higher sea-surface temperatures are associated with higher rates of viral-induced prokaryotic mortality (Fig. 8). In particular, the slope of the regressions between prokaryotic mortality and water temperature is significantly higher in colder systems (ANCOVA, $P < 0.01$). If these relationships would reflect the actual response of the virus–host interaction to changing water temperatures, it could be anticipated that climate-induced warming of surface oceans could increase the mortality of the pelagic prokaryotes (and possibly of cyanobacteria and other autotrophic hosts), thus

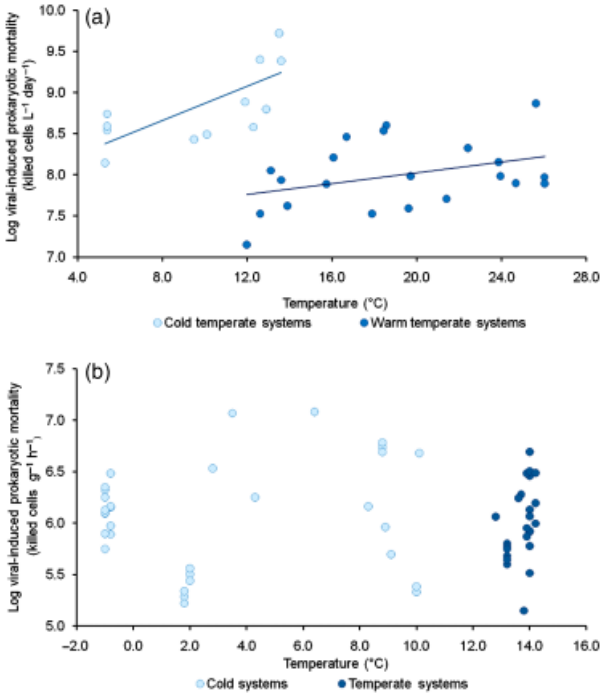

Fig. 8. Relationship between temperature and viral-induced prokaryotic mortality in the water column (a) and in the sediment (b) of different marine ecosystems. (a) The equations of the fitting lines are: $y = 0.104x + 7.83$ for cold temperate ecosystems ($n = 12$, $R^2 = 0.506$, $P < 0.01$) and $y = 0.033x + 7.37$ for warm temperate ecosystems ($n = 21$, $R^2 = 0.141$, NS). Data of the water column are summarized from Boras *et al.* (2009) and Evans *et al.* (2009), whereas those of sediments are summarized from Mei & Danovaro (2004) and Danovaro *et al.* (2008a, b). Only data sets, where synoptic measurements are available, have been utilized.

causing an increase of the release of labile DOM. However, these correlation analyses should be view with caution due to the very limited number of studies and characteristics of the sampling site investigated.

DOM includes a complex array of molecules and cell debris (as well as viral particles), which form a continuum of size spanning from a few to 10^5 Da. The actual composition of this material has important implications not only for the processes of respiration and consumption, but also for physical processes including coalescence, aggregation and the modification of the optical properties of the water column (Carlson, 2002). The processes of both viral infection and viral decay significantly support the release of DOM in the oceans, and also influence DOM composition. Recent studies suggest that the carbon released by viral decay alone can provide a significant contribution to the metabolism of prokaryotic assemblages in different benthic ecosystems (Corinaldesi *et al.*, 2010). Data summarized from the literature, including both field and experimental studies, suggest that higher temperatures are apparently associated with exponentially higher decay rates of virioplankton (Fig. 9). The increase of decay rates with increasing temperatures is expected as higher temperatures are associated with higher levels of exoenzymatic activities, which are largely responsible for the degradation of the viral capsid (Corinaldesi *et al.*, 2010). Interestingly, the benthic compartment did not display a similar relationship. We can hypothesize that such differences are due to the presence of the sedimentary matrix, which can interact with both the viral particle and the exoenzymes, thus influencing the processes of viral decay (Corinaldesi *et al.*, 2010).

A conceptual scheme of the impact of viruses on the biological carbon pump and of the potential feedback mechanisms is shown in Fig. 10. In this scheme, viral lysis along with rising CO_2 concentrations can cause significant changes in phytoplankton production and composition. The combined effect, modifying the POC produced by photosynthesis and the PIC produced by calcifying photoautotrophic organisms is expected to influence the particle export and in particular the relative ratio of PIC to POC of the exported biogenic material. This can determine to a large extent the flux of CO_2 between the ocean surface and the atmosphere, thus representing one of the possible feedback effects of the ocean on atmospheric CO_2 concentrations. At the same time, viral-induced prokaryotic mortality could increase the amount of available labile DOC that can enhance the metabolism and respiration of uninfected prokaryotic cells. Such an increased metabolism could be exacerbated by the expected sea-surface temperature rise and thus could increase carbon consumption and respiration rates.

The viral-mediated controls on the biological pump are complex and two potential scenarios can be suggested. In the first scenario, the viral shunt could negatively affect the biological pump efficiency by altering the pathways of carbon cycling in the sea as the result of cell lysis and by converting living particulate organic matter into dead particulate organic matter and DOM. In particular, DOC deriving from cell lysis will be retained to a greater extent in surface waters, where much of it will be converted to DOC by respiration or solar radiation (i.e. photolysis). In this respect, it is difficult to predict the potential consequences of the climate-induced changes in the biological pump on the functioning of deep-sea ecosystems (Smith *et al.*, 2008). The dark portion of the oceans primarily depends on the export of organic carbon produced by photosynthesis in the photic zone and any change in the amount of carbon release in the deep is likely to have major impacts on the functioning and biodiversity of the largest biome of the biosphere. Figure 11 illustrates a conceptual diagram of the potential impact of climate change on viruses and carbon cycle and its

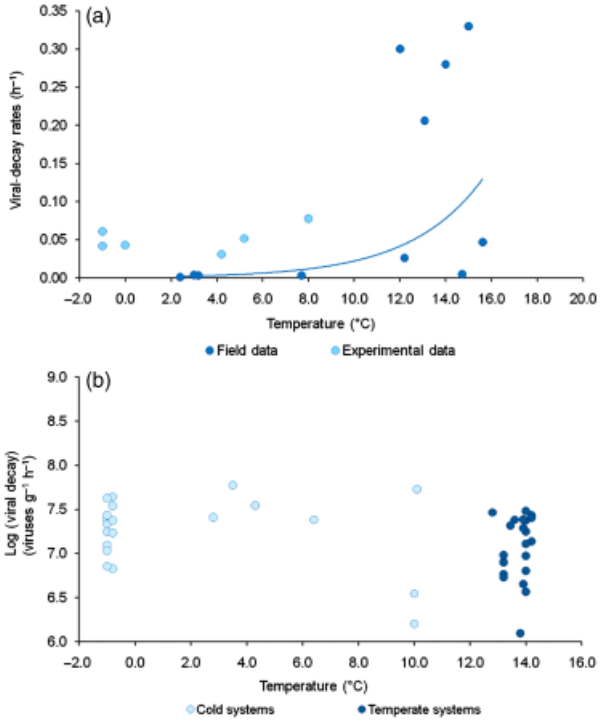

Fig. 9. Relationship between temperature and viral decay in the water column (a) and sediment (b) of different marine ecosystems. (a) Both experimental and field data. (b) Data from cold and temperate benthic systems. (a) The equations of the fitting lines referring to the field data are: $y = 0.0009^{0.32}x$ ($n = 11$, $R^2 = 0.563$, $P < 0.05$). Field data on viral-decay rates in the water column are summarized from Borsheim *et al.* (1990), Bratbak *et al.* (1990), Heldal & Bratbak (1991), Bongiorni *et al.* (2005) and Parada *et al.* (2007), whereas experimental data are summarized from Guixa-Boixereu *et al.* (2002). Data on viral-decay rates in the sediments are summarized from Corinaldesi *et al.* (2010) and R. Danovaro (unpublished data). Only data sets, where synoptic measurements are available, have been utilized.

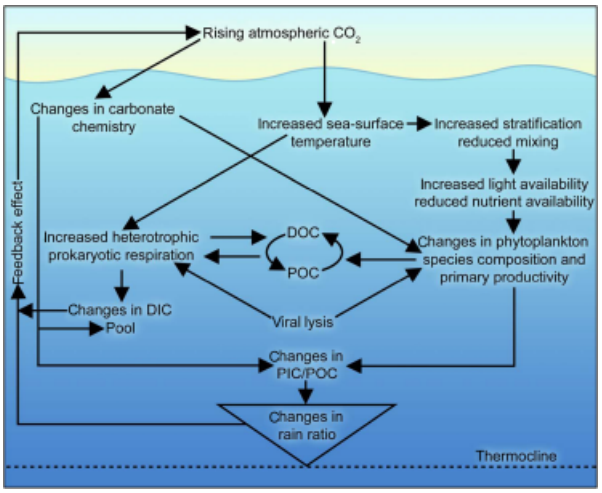

Fig. 10. Conceptual diagram of the role of viruses on the carbon pump on the light of the potential impact of climate change and their potential feedback effect. In the scheme, viral lysis together with the rising CO_2 concentrations is assumed to determine major changes in phytoplankton composition and production and to the overall prokaryotic metabolism.

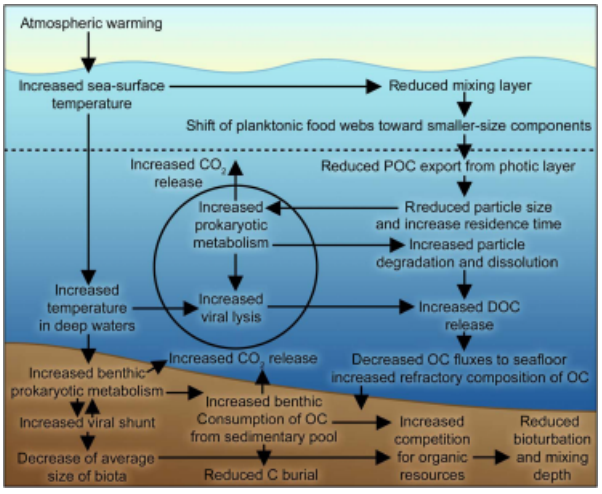

Fig. 11. Conceptual diagram of the potential impact of climate change on viruses and carbon cycling in the deep ocean. The future ocean is at least in triple trouble. Surface warming has a number of effects, as does ocean acidification. For instance, surface warming alone would probably lead to less export as given in this figure but with elevated CO_2 there may be more export (Riebesell *et al.*, 2007), which then in combination with reduced mixing and ventilation of deeper waters results in an increase in volume/area of hypoxic oceans.

consequence for the ocean interior. This simplified scheme does not take into account the potential consequences of the decreasing deep ocean ventilation and the supply of oxygen to the ocean interior (Bopp *et al.*, 2002). This process (defined as 'ocean deoxygenation'; Keeling *et al.*, 2010) could contribute to the expansion of oxygen-minimum zones (OMZs) (Stramma *et al.*, 2008; see Case study 5: Potential impact of the extension of the OMZs on viruses).

We can also hypothesize a second scenario where the viral shunt could favor carbon sequestration. As the amount of living POC in surface waters is controlled by the availability of nutrients (i.e. nitrogen, iron and phosphorous), which limit the growth of the primary producers, viruses may enhance carbon export if they increase nutrient availability or turnover.

The efficiency of the biological pump increases if the ratio of carbon relative to the amount of the limiting resource (or resources) increases. Viruses can increase the efficiency of the biological pump if they increase the export of carbon relative to the export of the limiting resource (or resources). The biological pump becomes more efficient if the ratio of exported carbon relative to the nutrient (or nutrients) that limits primary productivity is increased. There are several ways by which viruses can enrich or reduce the relative amount of carbon in exported production. For example, virus-mediated cell lysis could liberate elements that were complexed with organic molecules in approximately the same ratio as they occur in the organisms they infect. However, the chemical composition of the excretion and fecal pellets from zooplankton can differ markedly from that of the phytoplankton that they ingest, depending on the elemental assimilation efficiency (Morales, 1987; Frangoulis *et al.*, 2005). Furthermore, as lysis releases highly labile cellular components, such as amino acids and nucleic acids that can be rapidly incorporated by living organisms, this should have the stoichiometric effect of retaining more nitrogen and phosphorous in the photic zone than would occur if the cells were to sink, thereby increasing the efficiency of the biological pump by increasing the primary production.

Some authors highlighted the mechanisms how the viral shunt could contribute to the carbon export. Lawrence & Suttle (2004) revealed that viral infection can mediate the export of carbon and other organic molecules out of the photic zone by the accelerated sinking rates of virus-infected cells. Accelerated sinking, possibly due to the increase of weight of the cells or a loss of their motility, might be a mechanism that enhances the export of the smallest primary producers from surface water. Other studies highlighted that viral lysis, particularly during phytoplankton blooms, could contribute to the release of dense and refractory colloidal aggregates (Mari *et al.*, 2005). Moreover, experimental studies demonstrated that viral lysis of phytoplankton and bacterial cells increases the size of organic aggregates (Peduzzi & Weinbauer, 1993; Shibata *et al.*, 1997), suggesting that viral-mediated mortality of host cells can favor carbon export to the ocean interior.

Viral lysis influencing the type, stability and timing of formation of aggregates may either increase the retention

time of particulate organic matter in the euphotic zone (thus reducing carbon export) or increase sinking rates to the ocean interior (Weinbauer *et al.*, 2009 and citations therein).

Case study 5: Potential impact of the extension of the OMZs on viruses

While the ocean accounts for the majority of carbon in the combined atmosphere and ocean system, it accounts for $< 1\%$ of the combined atmosphere–ocean oxygen inventory. Oxygen concentrations in the ocean are governed by the balance between biological production and consumption on the one hand and physical supply processes on the other. In the thermocline and deep ocean, oxygen concentrations are highly sensitive to changes in ocean physics and biology and are projected to decrease due to climate change (Oschlies *et al.*, 2008). Ocean general circulation model predictions of average ocean oxygen decrease in 2100 vary from 3 to 12 $\mu mol\,kg^{-1}$ (Keeling *et al.*, 2010). About a quarter (range 18–50%) of the decline can be attributed to a decrease in solubility as the ocean warms. The majority of the decline is due to the combined effect of changes in ocean physics and biology (in particular, and increased stratification resulting in less oxygen supply to the ocean interior; Sarmiento *et al.*, 1998; Keeling *et al.*, 2010). Long-term observations of dissolved oxygen have already revealed systematic decreases of 0.1–0.4 $\mu mol\,kg^{-1}\,year^{-1}$ (Stramma *et al.*, 2008). Locally, in particular in coastal systems, oxygen loss may also result from cultural eutrophication (Diaz & Rosenberg, 2008; Middelburg & Levin, 2009).

Oxygen concentrations in the ocean typically show a mid-depth (200–1200 m) minimum because shallower waters are better ventilated and less oxygen is utilized in deeper waters. In areas with long ventilation times, this can result in areas with very low oxygen concentrations, sometimes close to zero. These OMZs are predicted to expand in the future ocean because of climate change, in particular when combined with CO_2 fertilization-induced enhanced export production (Oschlies *et al.*, 2008). Low dissolved oxygen concentrations have significant consequences for biogeochemical cycling, in particular of nitrogen and phosphorus (Middelburg & Levin, 2009; Keeling *et al.*, 2010). Expanding OMZs also have consequences for the distribution of organisms, in particular macroorganisms, and their sensitivity to oxygen levels is highly nonlinear (Vaquer-Sunyer & Duarte, 2008). Hypoxia can lead to changes in behavior, distribution, functioning and at very low levels to mortality of organisms. The sensitivity of organisms varies highly between taxa, but eukaryotic herbivores and bacterivores are expected to be more sensitive than prokaryotes. Consequently, virus-induced mortality of prokaryotes is likely to increase at the expense of protists and other bacterivores.

Indeed, high virus-induced prokaryote mortality has been reported in different marine anoxic systems (Weinbauer *et al.*, 2003; Corinaldesi *et al.*, 2007a, b). However, findings reported from the deep anoxic Cariaco Basin suggest that viral infection can be low in this oligotrophic anoxic marine system (Taylor *et al.*, 2003). Corinaldesi *et al.* (2007a, b) showed that marine anoxic waters have not only higher viral abundances and higher viral production but also higher viral decay implying higher viral turnover. They also proposed that viral infection plays a key role in extracellular DNA dynamics in anoxic waters.

Expanding OMZs will also lead to increase the frequency of anoxic sediments and thus potentially the global role of viruses in benthic prokaryote dynamics. However, Danovaro *et al.* (2008b) reported that viral infections are already responsible for the abatement of 80% of prokaryotic heterotrophic production and there is thus little scope for further increase in the role of viruses in benthic food webs when OMZs increase due to climate change.

Case study 6: Impact of ocean acidification on marine viruses

The ocean has already and will continue to function as a major sink of anthropogenic carbon from fossil fuel use and cement production. At present, oceanic uptake account for ~25% of current anthropogenic carbon emissions (Riebesell *et al.*, 2009; Sabine & Tanhua, 2010). The large storage capacity of anthropogenic carbon in the ocean is not only due its large volume but also to the carbonate system that buffers changes in CO_2 partial pressures. Anthropogenic CO_2 entering the ocean will result in changes in speciation among gaseous and aqueous CO_2, bicarbonate (HCO_3^-) and carbonate (CO_3^{2-}); the net reaction is most instructively presented as:

$$CO_2 + CO_3^{2-} + H_2O \rightarrow 2HCO_3^-$$

Because of the huge oceanic DIC reservoir, the oceans could, in principle, account for $> 80\%$ of all anthropogenic carbon released. However, this full (thermodynamic) potential can only be reached over timescales of oceanic turnover (from several centuries to millennia). Moreover, the uptake of anthropogenic CO_2 induces changes in speciation and pH with multiple environmental consequences. This has been termed the 'other CO_2 problem' (Doney *et al.*, 2009). Most notably, the invasion of CO_2 into the ocean causes an acidification of the surface ocean, estimated to be *c.* 0.1 unit decrease in pH over the past 200 years. Increases in atmospheric CO_2 will induce further and stronger acidification of oceanic surface waters. Models predict a decrease of 0.3–0.4 pH units going from pH 8.1 now to 7.8–7.7 in year 2100 (Caldeira & Wickett, 2003). This corresponds to a 250% increase in hydrogen ion concentrations. This ocean

acidification is a topic of concern because a decrease in the pH of seawater has multiple effects on the functioning, productivity, growth and survival of marine organisms. Much attention has been devoted to the potential consequences for calcifying organisms, in particular coral reefs, coccoliths, foraminifera and bivalves (Doney *et al.*, 2009). Ocean acidification decreases carbonate concentrations and thus lowers the seawater $CaCO_3$ saturation state. Even at small changes in pH or $CaCO_3$ saturation states, many of these organisms calcify less, show reduced growth, or even disappear. This can have important economic consequences, such as for shell fish yield (Gazeau *et al.*, 2007). Acidification of marine waters has also consequences for noncalcifying organisms, such as diatoms, nitrifiers and heterotrophic bacteria (Doney *et al.*, 2009) and for the penetration of sound in seawater (Hester *et al.*, 2008) affecting the bioacoustics of marine mammals. Overall, there is a growing recognition that ocean acidification has important effects on the performance and survival of marine communities, although marine biota may be more resistant to gradual changes than rapid perturbation during experimentation (Hendriks *et al.*, 2010).

The effects of an anticipated ocean pH changes on marine viruses is challenging to predict. It has been known for a very long time that classically studied bacteriophages, for example T2 and T7 phages of *Escherichia coli*, are indeed somewhat sensitive, with respect to survival and infectivity, to changes in pH when studied in laboratory culture (e.g. Sharp *et al.*, 1946; Kerby *et al.*, 1949). The responses of these phages to rapid pH changes within the anticipated ocean pH range are generally very small.

From the literature, it is known that some viruses are unaffected by pH = 3 or even less, whereas others are labile at pH < 7 (Krueger & Fong, 1937; Weil *et al.*, 1948; Jin *et al.*, 2005). This difference in pH stability has in some cases been argued to represent adaption to a way of life (Rueckert, 1996). Weinbauer (2004) stated that pH can affect adsorption of phages in freshwater while marine phages are typically only affected by pH values deviating from that of seawater. Cyanophages in freshwater appears to have a broader tolerance for pH (5–11) than bacteriophages in general (Suttle, 2000). Brussaard *et al.* (2004) demonstrated a loss of infection for MpRNAV-01B, a virus infecting the marine eukaryotic algae *M. pusilla*, at pH = 5.

Virioplankton community responses to increasing levels of nutrients (N and P) and CO_2 was investigated in a mesocosm experiment (Larsen *et al.*, 2008). Two specific large dsDNA viruses, infecting *E. huxleyi* and *Chrysochromulina ericina* were investigated, and while some of the viral populations did not respond to altered CO_2 levels, the abundance of two viral populations decreased.

We are not aware of similar studies for marine planktonic bacteriophages, which may be considerably more sensitive given that their native marine habitat has had a steady and narrow pH range for millions of years. It could be possible that this has led to no expected selection for pH tolerance (compared with enteric bacteriophages that encounter wide swings of pH in their animal gut habitat). But even if we had such data about marine phages, we have great uncertainty in how laboratory measurements investigated with acute pH changes provide predictive power about the ability of viruses to evolve to gradual changes in pH expected to occur over decades. Of course, this applies to cellular organisms as well to a large extent, but we know of more physiological processes in cellular organisms that are directly tied to pH and proton gradients.

While the direct effects of pH on marine viruses is uncertain, we anticipate that the most dramatic changes will be due to the effects of pH on the host organisms that the viruses rely on; bacteria, archaea, protists and metazoa. As noted above, there are many reasons to expect that some organisms will be dramatically affected, for example calcifying protists or animals, so their viruses would of course be affected as well. Such viruses include the well-studied ones that infect marine coccolithophorids, which at times occur in massive algal blooms. As the field is still in its infancy, the effects on most microorganisms are still somewhat speculative, but there are good reasons to expect strong effects on any microorganisms whose major substrates are highly pH dependent. This may in fact involve important players in major elemental cycles. For example, autotrophs, that rely on CO_2 directly for a carbon source, are widely thought to have better access to this molecule at lower pH, and would have less need to rely on carbonic anhydrase, the enzyme that converts bicarbonate to CO_2 (Hutchins *et al.*, 2009). The process of ammonia oxidation to nitrite (first step in nitrification), recently discovered to be largely performed by archaea and not just bacteria in the sea (Francis *et al.*, 2007), is quite sensitive to pH because lowered pH shifts the equilibrium away from ammonia (the enzyme substrate) toward the ammonium ion. Preliminary experiments with increasing CO_2 concentrations in seawater indicate a sharp drop in nitrification (Beman *et al.*, 2008; Hutchins *et al.*, 2009). This suggests that the viruses infecting the ammonia-oxidizing bacteria and archaea (the latter of which currently account for typically 30% of the microbial communities in ocean midwater depths; Francis *et al.*, 2007) might be strongly affected. There are also large numbers of bacterial and archaeal physiological processes that are based on a proton gradient across the membrane, including the flagellar motor (motility) and many substrate transporters. Decreasing the pH makes it harder for the cell to pump the protons out, and it is not clear what effect this may have on broad physiological processes and hence microbial communities including viruses. Furthermore, metagenomics has shown that a large fraction of the total near-surface bacterial

(and even archaeal) community possesses the pigment proteorhodopsin, which can act as a light-driven proton pump, and the examined versions of this pump are highly sensitive (inhibited) by even small decreases in the pH of the medium (reviewed by Fuhrman *et al.*, 2008). Some organisms appear to obtain substantial growth energy from this mechanism while others do not (Fuhrman *et al.*, 2008); hence, any inhibition by acidification may have a profound influence on the overall microbial communities.

Case study 7: Potential impact of viruses on cloud formation

The ocean contributes over 30% of the atmospheric sulfur budget (Nguyen *et al.*, 1978). The algal osmolyte DMSP is recognized as the major precursor of marine DMS, a volatile sulfur compound that affects atmospheric chemistry and global climate. Recent estimates of its emission flux indicate that DMS values range from 15 to 33×10^{12} g S year^{-1}, enough to make a major contribution to the atmospheric sulfur burden, and therefore, to the chemistry and radiative behavior of the atmosphere (Kettle & Andreae, 2000). The idea of marine microbiota as the principal factors in atmospheric chemistry and climate regulation has been introduced in the late 1980s and promoted the study of the links between plankton and atmospheric sulfur, yielding >1000 papers in the scientific literature.

Processes driving the synthesis, fluxes and transformations of DMSP and DMS have been extensively investigated as such research is not only being addressed from a global biogeochemistry perspective but also from the perspective of other disciplines, such as cell physiology, marine ecology and chemistry. Once emitted into the atmosphere, DMS is rapidly oxidized to generate methane sulfonic acid, SO_2 and SO_4^{2-}. The oxidation products of DMS can then be converted into sulfate aerosol particles. These particles may serve as condensation nuclei for water vapor to form cloud condensation nuclei (CCN) reflecting back the incoming solar radiation. This view provided the basis for the 'CLAW hypothesis', which states that regulation of climate by oceanic phytoplankton is possible through the production of DMS. Oxidation of DMS in the marine atmosphere produces sulfur aerosol that, either directly or by acting as CCN, scatter solar radiation thereby influencing the radiative balance of the Earth (Charlson *et al.*, 1987). These, in turn, have feedback effects on phytoplankton population and DMS production. The link between phytoplankton and climate made by the CLAW hypothesis has been proposed as one of the testable processes in the Gaia theory.

Increasing production of DMS due to global warming is expected to lead to more sulfate aerosols and subsequently to more CCN that can enhance back radiation (Kanakidou *et al.*, 2005). Gondwe *et al.* (2003) recently estimated the contribution of DMS to climate-relevant non-sea-salt sulfate (NSS SO_4^{2-}) at 43% in the relatively pristine Southern Hemisphere, confirming the potential role of oceanic DMS in climate regulation. In addition, model simulations indicate that organic matter can enhance the cloud droplet concentration by 15% to $>100\%$ and is therefore an important component of the aerosol–cloud–climate feedback system involving marine biota (O'Dowd *et al.*, 2004; Meskhidze & Nenes, 2006; Sorooshian *et al.*, 2009). DMSP is differentially produced by phytoplankton species, the greatest DMSP producers being the coccolithophores, the genus *Phaeocystis* and the dinoflagellates (Liss *et al.*, 1993). DMSP is produced as an osmolyte and a cryoprotectant (Kiene & Service, 1991; Kirst *et al.*, 1991) and its degradation products have also been shown to have antioxidant properties in marine phytoplankton (Steinke *et al.*, 2002; Van Rijssel & Buma, 2002).

DMS is generated by the enzymatic cleavage of DMSP by a bacterial or algal DMSP lyase (Challenger, 1951). The production of dissolved DMSP and DMS is also associated with the senescence of phytoplankton blooms (Turner *et al.*, 1988; Leck *et al.*, 1990) and significant correlations between DMS concentrations and zooplankton biomass have also been reported (Leck *et al.*, 1990; Yang *et al.*, 2000). Zooplankton grazing on algal cells may release DMS indirectly, by transferring DMSP to the dissolved compartment and making it available for conversion into DMS by bacteria (Belviso *et al.*, 1990; Wolfe *et al.*, 1994). Dacey & Wakeham (1986) found that a third of the DMSP originating from *Gymnodinium nelsoni* and *Prorocentrum micans* ingested by the copepods *Labidocera aestiva* and *Centropages hamatus* were released into the culture medium as DMS confirming the role of zooplankton grazing in the DMS cycle. Other processes such as cell autolysis (Nguyen *et al.*, 1988) and physical or chemical stress have been related to an increase of DMS production from different phytoplankton classes (Wolfe *et al.*, 2002).

Once produced, DMS is consumed biologically within one to several days (Kiene, 2003). Therefore, DMS concentrations transferred to the atmosphere are actually lower than DMS released in the water column (Kiene, 2003). In oxygenated seawater, the most probable consumers are methylotrophic bacteria, although several metabolisms are possible (Kiene, 2003). With the exception of very particular conditions, intracellular pools of DMSP are present in algae at approximately 0.2–0.5 M, although bulk seawater concentrations are approximately 10 nM (Kettle *et al.*, 1999). Such a narrow range suggests that losses occur in tight coupling with production processes. Indeed, not only biological consumption but also significant photolysis and ventilation rates have been found coupled to DMS production in the open ocean (Kieber *et al.*, 1996). Most studies show that bacteria are a major sink for DMS. DMSP appears

to support from 1% to 13% of the bacterial carbon demand in surface waters, making it one of the most significant single substrates for bacterioplankton so far identified (Kiene *et al*., 2000). Therefore, because bacterioplankton is involved in both DMSP and DMS utilization, factors controlling bacterial activity (such as UVB radiation, temperature, nutrients aquality of DOM; Kirchman, 2000) ultimately also play a role in controlling DMS concentration.

Viruses, by inducing the lysis of algal cells, have been reported to contribute to DMSP release to the dissolved pool where it is rapidly converted to DMS by bacteria possessing DMSP lyase (Malin *et al*., 1998; Niki *et al*., 2000). Experiments show that viral lysis causes the total release of DMSP from the *M. pusilla* (Hill *et al*., 1998) and viruses are also known to infect major DMSP-containing bloom organisms such as *E. huxleyi* (Bratbak *et al*., 1995) and *P. pouchetii* causing an enhanced release of DMS (Malin *et al*., 1998). Other experiments carried out on six *E. huxleyi* strains showed that the activity of their DMSP lyase, the enzyme responsible for cleaving DMSP to DMS and AA, varied by $>$ 6000-fold, and they were classified as either 'low lyase' and 'high lyase' strains (Steinke *et al*., 1998). Despite extensive screening, to date no viruses have been isolated that are capable of infecting the high DMSP-lyase activity strains (Evans, 2005). This has led to the suggestion that high DMSP-lyase activity may be implicated in an antivirus defense mechanism (Schroeder *et al*., 2002).

Previously, the DMSP system has been proposed to serve a number of roles including compatible solute, antioxidant and overflow for excess reduced sulfur and energy. Bratbak *et al*. (1995) reported that only during the rapid collapse of large blooms viral lysis represents an efficient mechanism of net DMSP production in seawater. In addition, DMS and AA levels released during viral lysis were lower than those obtained in parallel studies concerning grazing impact (Wolfe & Steinke, 1996), suggesting that viral infection plays a secondary role in these dynamics. The impact of viral lysis on DMS release is still to be clarified. Different scenarios have been proposed from different authors including: (1) viral lysis could increase the DMS release with direct effects on the albedo, (2) as bacterioplankton is mainly involved in both DMSP and DMS utilization, viral control on bacterial activity could indirectly regulate DMS concentration and (3) the viral impact could negatively affect DMS concentrations through the decrease of DMSP cleavage mediated by DMSP lyase (Evans *et al*., 2007).

Case study 8: Viruses and marine aerosol formation

Since the publication of the CLAW hypothesis, it has been difficult to prove or disprove this idea. Several studies have elucidated that DMS flux alone cannot explain observed particulate composition and concentration in MBL, new particle or CCN formation (O'Dowd & de Leeuw, 2007). In recent years, the role of natural organic aerosol in the marine environment has received increasing attention. Measurements indicate that the increase of the marine biological activity is accompanied by a considerable increase of the contribution of OC to the submicron marine aerosol (e.g. O'Dowd *et al*., 2004) exceeding in some cases the mass fraction of NSS SO_4^{2-} by a factor of more than two. OC in aerosols is partly in the form of primary aerosols (POC), which are directly released from bubble-bursting processes at the ocean surface (sea spray), and partly as secondary aerosols (SOC), which form in the atmosphere through chemical reactions of reactive gases released at the ocean surface to form condensable aerosol species or by oxidative transformations of POC. Aerosol represents the main vector for the transfer of prokaryotes and other primary biological particles from the terrestrial and aquatic surface to the atmosphere, representing an important mechanism for the microbial dispersion and biogeography (Bovallius *et al*., 1980; Posfai *et al*., 2003; Aller *et al*., 2005).

Prokaryotes are abundant and ubiquitous in the atmosphere (from the polar regions to the open oceans) with concentrations up to 10^{12} for m^3 of air (Kuznetsova *et al*., 2005). The presence of prokaryotes in aerosol has been defined significant for different processes that occur in atmosphere (e.g. nucleation of ice and clouds; Saxena, 1983; Vali, 1985) and the global exchange of OC between Earth's surface and atmosphere (Jurado *et al*., 2008). Because the prokaryotes are able to perform metabolic reactions, these may also be involved in chemical transformation processes of organic compounds present in the atmosphere (Deguillaume *et al*., 2008; Georgakopoulos *et al*., 2008). Marine bacteria can be dispersed and remain in the atmosphere longer than other biological particles due to their small size (Harrison *et al*., 2005). This allows us to consider a potential role of other biological particles of even smaller size, such as viruses in processes occurring in the atmosphere. However, studies relating to such biological components, both in term of concentrations and contributions to biogenic aerosol in the different types of aerosols, are very limited. As far as the knowledge of viruses in the aerosol is concerned, most of the studies have been focused on the analysis of specific pathogenic viruses and their infectivity (Ijaz *et al*., 1987; Sagripanti & Lytle, 2007). Only recently, studies on marine aerosol have reported information on the abundance of viruses and their enrichment factor in comparison with the marine microlayer (Aller *et al*., 2005; Kuznetsova *et al*., 2005). Field studies conducted in the northern Atlantic Ocean, indicate that viral and prokaryotic abundances were present in all aerosol samples collected (Aller *et al*., 2005). Both the analyzed components presented

low abundance ($\sim 10^2\ m^{-3}$), probably due to the aerosol sampling area in the open sea. Because viruses can persist in the aerosol for a long time and are small particles potentially representing nucleus of condensation, this component could have important implications also in albedo and in other physical–chemical processes occurring in the atmosphere.

Concluding remarks

There is a considerable interest in the interconnection between present climate change and consequences on marine ecosystems and their function. In the past, viruses and prokaryotes have been considered in large-scale global oceanic models as a 'black box'. We know now that viruses play a pivotal role in the functioning of both pelagic and benthic ecosystems, influencing microbial food webs, controlling prokaryotic diversity and impacting biogeochemical cycles. Clearly, a better understanding of their response to present climate change would enhance our ability to predict and adapt to the consequences of such changes. Traditionally, the evaluation of the effects of climate change on natural systems is conducted by the analysis of paleoecological and stratigraphic records or by the analysis of long-term data sets that permit the identification of the relationships between climatic conditions and the spreading or decline of specific biological components. These approaches are impossible in the field of the viral ecology as we are not aware of any evidence of fossil viruses and we do not have long-term data for this important component of marine systems. It is also unclear whether viruses, under the present scenarios of climate change, will ultimately stabilize or destabilize the dynamics of the living components of ecosystems and their biogeochemical cycles. Therefore, we cannot yet predict whether the viruses will exacerbate or smooth the impact of climate change on marine ecosystems. The approach used here, based on the presentation of specific relevant case studies, and on the meta-analysis of the available literature information on an array of changes in viral assemblages, allows us an initial examination of possible coming changes in our worlds' oceans. Given the knowledge gained from previous documented research studies, we also provide some speculation on scenarios that could occur if the current projections of climate change will result in actual changes in terms of surface and deep-water temperatures, productivity, stratification, salinity and acidification.

From the case studies analyzed, it appears that the effect of rising surface water temperatures on viruses will be significant, influencing both the metabolism and growth efficiency of the prokaryotes and altering the viral life cycles. However, it is apparent that different effects will be observed at different latitudes and in different oceanic regions. For instance, data reported here led to hypothesize that higher temperature will promote the viral component at high latitudes and depress it at the tropics. However, because the mosaic of environmental changes will be different in each biogeographic region, we stress the need of future studies aimed at addressing the impact of climate change on regional and global scales. The scenario of freshening at the poles will likely increase the input and spread of freshwater groups of viruses and bacteria into marine systems, along with increasing opportunity for crossing over of marine and freshwater taxa. Marine viruses can influence the metabolic balance of heterotrophic prokaryotes, inducing shifts in pelagic ecosystems function. However, it is unclear whether the viral shunt will ultimately have positive or negative effects on the efficiency of the biological pump, and consequently, the feedback effects of marine ecosystems on climate. In both alternatives presented here, the role of viruses on the carbon export to the ocean interior is potentially crucial and has to be considered and addressed, especially within global climate models. Viruses have the potential to interact with the climate through their contribution to the marine biogenic particles of the aerosol and by contributing to the release of DMS through the lysis of their autotrophic hosts. These processes have to be quantified and included in modeling studies dealing with the ocean–atmosphere interactions. The OMZs are predicted to expand in the future ocean because of climate change, with important consequences on biogeochemical cycling of nitrogen and phosphorus and on the distribution of organisms. Because eukaryotic herbivores and bacterivores are more sensitive than prokaryotes to the reduction in oxygen levels, it can be expected that virus-induced mortality of prokaryotes will increase at the expense of protists and other bacterivores. Expanding OMZs will also lead to an increase in the frequency of anoxic sediments and thus potentially the global role of viruses in benthic prokaryotic dynamics. The effects of ocean acidification on marine viruses are uncertain, but we can anticipate that the most dramatic changes will be due to the effects of pH on the host organisms that the viruses rely on bacteria, archaea, protists and metazoa, which are highly pH dependent. Moreover, because some key metabolic processes of the microbial communities are highly sensitive (and inhibited) by even small decreases in the pH of the medium, ocean acidification may have a profound influence on the overall functioning of the microbial communities and on virus–host interactions.

The case studies presented here suggest that marine viruses will be significantly influenced by climate change and that, in turn, viruses could influence processes contributing to climate change. Current ocean climate models are severely limited by the ability to include important biological components and to specify the temporal and spatial

scales at which biological components respond to and interact with present climate changes.

The following research priorities are vital to increase our understanding of the multifaceted role of marine viruses under present global change:

(1) Investigation of the effects of altered environmental conditions related to global change on virus–host interactions, for example small but consistent changes in pH, and empirical estimations of adaptation over changing temperature and salinity regimes.

(2) Impact of climate change on the effects of viruses on global biogeochemical cycles at varying latitudes.

(3) Specific long-term and wide spatial-scale studies on the impact of viruses on the release of climate-altering molecules and on the oceanic export of C.

(4) Investigation of the relative impact of ocean acidification on viral infectivity and life cycles in comparison with host life cycles.

These research topics, although not exhaustive, will provide a foundation that will significantly augment our understanding of the interactions of viruses with global climate change. This is, in our opinion, the first priority to increase the accuracy and reliability of future predictions of climate change impact on marine ecosystems.

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Authors' contribution

R.D., C.C., A.D., J.A.F., J.J.M., R.T.N. and C.A.S. contributed equally to this work.

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