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Mortality in the oceans: Causes and consequences

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Abstract

Microorganisms dominate the oceans and exert considerable control over fluxes of nutrients, organic matter, and energy. This control is intimately related to the life cycles of these organisms, and whereas we know much about their modes and rates of reproduction, comparably little is known about how they die. The method of death for a microorganism is a primary factor controlling the fate of the nutrients and organic matter in the cell, e.g., whether they are incorporated into higher trophic levels, sink out of the water column, or are recycled within the microbial loop. This review addresses the different sources of mortality for marine microbes including grazing, viral lysis, programmed cell death, and necrosis. We describe each mode of death, the methods used to quantify them, what is known of their relative importance in the ocean, and how these vectors of mortality differentially affect the flow of organic matter in the open ocean. We then conclude with an assessment of how these forms of mortality are incorporated into current numerical ecosystem models and suggest future avenues of research to increase our understanding of the effects of death processes in oceanic food webs.

Section 1. Introduction

Microorganisms (i.e., bacteria, protozoa, and phytoplankton) constitute a major portion of the biomass in marine environments, and because of their rapid rates of production and respiration, they contribute significantly to the flow of energy and nutrients in the ocean (Pomeroy et al. 2007). The majority of microbial production was originally thought to be consumed by grazing (Landry and Hassett 1982; Pomeroy 1974). However, researchers eventually recognized that rates of microbial mortality could not be reconciled with grazing estimates (Fuhrman and McManus 1984; Pace 1988) and speculated that viruses might be responsible for the balance of microbial mortality (McManus and Fuhrman 1988). It is now known that viral lysis is an important cause of bacterial and phytoplankton mortality in the ocean (Brussaard 2004; Fuhrman 1999), and programmed cell death (PCD) and necrosis are also emerging as previously unconsidered vectors of death for marine plankton (Bidle and Falkowski 2004; Franklin et al. 2006). Few studies have considered even two of these processes simultaneously, despite the fact that the method of cell death (e.g., lysis versus predation) largely governs the flow of nutrients between the microbial loop, higher trophic levels, and ocean sediments (Fig. 1). This chapter addresses the known sources of microbial mortality in the oceanic water column, focusing on the methods with which they are studied, their differing effects on organic matter fluxes, and the future research necessary to gain a comprehensive understanding of how these vectors of mortality affect the oceanic food web.

Section 2. Grazing

Grazing is generally the primary form of plankton mortality in the world's oceans (Calbet and Landry 2004; Landry et al. 2009; Sherr and Sherr 2002). Most phytoplankton in the ocean are consumed by either protistan or metazoan grazers, and bacterioplankton are preyed on primarily by small protozoans (Strom 2000). Fundamental differences exist among grazers, which are generally grouped into two functional categories based on size: the microzooplankton (<200 μm) and the mesozooplankton (>200 μm). This classification, while based on size, is accompanied by essential differences between the dominant grazers: the mesozooplankton includes primarily crustaceans and gelatinous organisms, and the microzooplankton is dominated by protists (Landry and Calbet 2004) although it includes smaller crustaceans. In this section, we will briefly review the different types of grazers, their grazing modes and grazing rates, and their impact on microbial production in the ocean.

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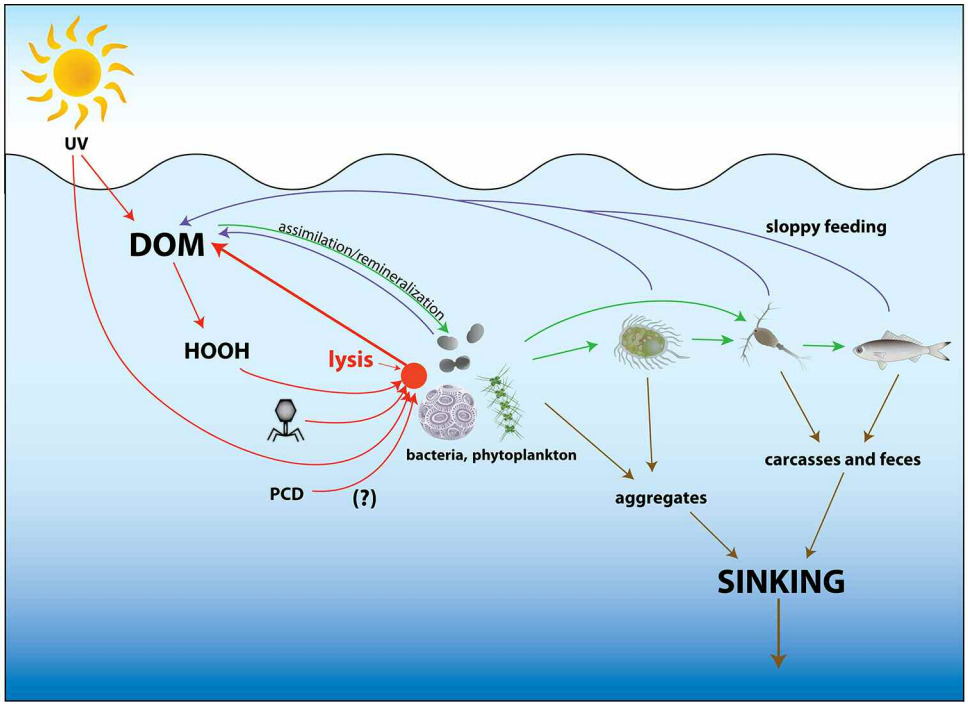

Fig. 1. Mortality processes in the ocean and their impacts on biogeochemical fluxes. *Green arrows* show trophic interactions as organic matter and remineralized organic nutrients are incorporated into higher trophic levels. *Red arrows* represent processes resulting in lysis, which result in the conversion of living organic matter to dissolved organic matter (DOM). *Purple arrows* show leakage of DOM due to exudation and sloppy feeding. *Brown arrows* represent processes that result in removal of organic matter from the mixed layer due to sinking.

Protistan grazers

Microzooplankton comprise the < 200 μm heterotrophic organisms of the plankton community, and are taxonomically diverse but dominated by phagotrophic protists (mainly flagellates and ciliates, but also amoeba, heliozoans, radiolarians, acantharians, and formanifera). Some metazoans are also included in this size category, such as rotifers and juvenile stages of crustaceans (i.e., nauplii), but their contribution is minimal compared with the protozoan component of the community (Fenchel 1988, Sherr and Sherr 2002).

The abundance of different types of protistan grazers varies based on productivity of the ecosystem. The heterotrophic flagellates, which dominate the nanoplankton, have similar average abundances in oligotrophic and eutrophic waters (~ 10^3 cells mL^{-1}; Fenchel 1982; Fenchel 1988), but can reach much higher numbers during peak seasons in more eutrophic conditions. Flagellate abundances can vary between 10^2 and 10^4 cells mL^{-1} during summer blooms in eutrophic environments (e.g., Andersen & Sørensen 1986; Verity et al. 1999).

Dinoflagellates are important members of the microzooplankton, not only because they are present in high abundances, but because they are able to consume prey as large as themselves. Sherr and Sherr (2007) found that on average dinoflagellates constituted about 50% to 60% of microzooplankton biomass, achieving higher absolute biomass in temperate springtime conditions. Their role as consumers is disproportionally important to their contribution to biomass mainly because they are major consumers of diatoms, which are usually too large to be consumed by ciliates (Landry et al. 2000; Jeong et al. 2004). In fact, dinoflagellates are hypothesized to be the main consumers of diatoms in the oceans, having a greater average impact than copepods or other larger zooplankton (Sherr and Sherr 2007).

Ciliate are usually found in high numbers in nearshore and estuarine environments (Pierce and Turner 1992). Abundances can range from 10^2 to 10^6 cells mL^{-1}, depending on conditions, and are usually on the higher end for aloricate ciliates (Dale and Dahl 1987). Mean annual abundances in temperate coastal waters are usually 10^3 cells mL^{-1}, with considerably lower abundances in oligotrophic waters (Pierce and Turner 1992).

In addition to variability in their abundance, heterotrophic marine protists are also widely diverse in the feeding modes that they display and the size range of particles they consume, which range from < 1 to > 100 μm in size (Sherr and Sherr 1992). A key characteristic of protistan grazer feeding is the high efficiency of ingestion in which little particulate or dissolved carbon is released into the water column, contrasting them from the 'sloppy feeding' exhibited by copepods (discussed below).

Small heterotrophic nanoflagellates (>5 μm) are the main consumers of bacteria in the ocean (Fenchel 1982), constituting over 80% of the bacterivore community (Strom 2000). Bacterivorous flagellates encounter bacteria by creating feed-

ing currents, then entrapping and consuming the prey cells via phagocytosis (Arndt et al. 2000; Sherr and Sherr 1991; Strom 2000).

The range of feeding modes and the sizes of prey consumed is greatest within dinoflagellates. Naked dinoflagellates are able to engulf prey of similar size to themselves via phagocytosis into food vacuoles (Lessard 1991). Some species can even feed on prey cells larger than themselves, piercing the cell walls of their prey with a specialized appendage and sucking out the cellular contents (Gaines and Elbrachter 1987). Many thecate dinoflagellates (e.g., *Protopteridinium*) exhibit 'pallium' feeding (Jacobson and Anderson 1986), which involves a large membranous organ that stretches and encloses diatom cells, with consumption proceeding via extracellular digestion and transportation of the liquefied contents back to the grazer cell (Jacobson and Anderson 1986).

Ciliates can ingest particles via raptorial feeding but are predominantly suspension feeders (Pierce and Turner 1992). They are reported to consume the gamut of available prey, including heterotrophic bacteria, pico- and nanophytoplankton, dinoflagellates, and other ciliates (Pierce and Turner 1992). Bacterivory is of lesser importance, however, and ciliates are reported to rely mainly on the larger-sized protozoans such as nanoflagellates, dinoflagellates, diatoms, and other ciliates (Fenchel 1988; Pierce and Turner 1992). Ciliates are not only important as voracious grazers, but also as a link in the transfer of carbon to the mesozooplankton, as at times they constitute an important fraction of the diet of the larger zooplankters (Calbet and Saiz 2005; Fessenden and Cowles 1994; Nakagawa et al. 2004).

Protistan grazers are the most important consumers of both the bacterio- and phytoplankton. Small heterotrophic flagellates, usually >5 μm, are thought to be responsible for most bacterivory in the ocean (Sherr and Sherr 1991; Sherr et al. 1989; Sherr and Sherr 1994; Strom 2000), although larger flagellates also consume bacteria, and ciliates can be important grazers at certain times (Pierce and Turner 1992; Sherr and Sherr 2002). The amount of bacterial production that gets consumed is variable in both space and time. The amount of water flagellates can filter per hour can be up to 10^5 times the volume of the predator (Strom 2000), and zooflagellates are capable of clearing up to 50% of bacteria in a volume of water per 24 h, although this is highly variable (Fenchel 1988). Bacterial populations often vary out of phase with that of their flagellate predators, displaying typical predator-prey cycles (Andersen and Sørensen 1986), emphasizing the importance of sampling over an entire cycle to be able to adequately quantify the consumption of bacterial production. Significant spatial variability in the balance between production and predation exists as well. A compilation of studies from different environments by Strom (2000) found that in low productivity waters bacterivory was the fate of most bacterial production. However, some studies have suggested that viral lysis can account for substantial mortality in coastal waters characterized by low productivity (Fuhrman and Noble 1995), evincing the necessity to simultaneously evaluate both sources of mortality (Strom 2000). In environments characterized by higher productivity regimes, bacterivory can still be significant, but there are other major sources of mortality, such as consumption by benthic organisms, metazoan predators, and viral lysis (Strom 2000).

Dinoflagellates and ciliates have been recognized for some time as major consumers of phytoplankton (Lessard 1991; Pierce and Turner 1992; Sherr and Sherr 1994), but it is only recently that the microzooplankton have become widely accepted as the main source of phytoplankton mortality in all marine ecosystems. A review by Calbet and Landry (2004) of the results of more than 788 studies using the dilution method (described below) found that microzooplankton consume a large and relatively constant percentage of the primary production (PP). The analysis included data from all oceanic regions, and revealed that the percentage of PP consumed ranged from 60% in coastal regions to 70% in open oceans (Calbet and Landry 2004). This is presumably due both to high ingestion rates as well as high growth rates achieved by unicellular heterotrophs. Protozoans can grow at the same rate as (and sometimes faster than) their prey (Sherr and Sherr 2002), and are therefore more capable of keeping up with increases in phytoplankton abundance than larger grazers.

Metazoan grazers

Crustacean zooplankton

The mesozooplankton (i.e., the > 200 μm fraction of zooplankton) is comprised mainly of metazoans, namely crustaceans and gelatinous organisms. The crustacean portion of the community accounts for most of the biomass, and is dominated by copepods and to a lesser extent euphausiids, with other taxa usually contributing a much smaller fraction (Lavaniegos and Ohman 2007; Mauchline 1980; Mauchline 1998; Ponomareva 1966). They are numerically much less abundant than protozooplankton, and their generation times tend to be on the order of weeks to months, in contrast to the daily turnover rates of the microzooplankton.

Herbivorous/omnivorous zooplankton have different modes of particle capture. Copepods can cruise through the water in search of prey, create a filtering current to capture particles flowing through the current, or sit motionless in the water and ambush their prey (Kiorboe et al. 2009; Koehl and Strickler 1981). The different feeding modes affect both the size of particles captured, as well as the efficiency of their nutrition. Adult copepods feed mainly on large particles, yet the consumption rates of the younger stages of copepods are less known. There is some evidence of measurable bacterivory by the naupliar stages of some marine copepods (e.g., Bottjer et al. 2010; Roff et al. 1995). Adult copepods feed mainly on particles > 5 μm (Jorgensen 1966, Mauchline 1998), with higher clearance rates on larger particles (Frost 1972). This preference, however, can shift as a function of particle abun-

dance (Cowles 1979). Most 'herbivorous' copepods are primarily suspension feeders, and therefore experience varying degrees of omnivory, which can depend on prey concentration (Landry 1981; Paffenhofer and Knowles 1980). Copepods are reported to feed preferentially on diatoms, ciliates, and dinoflagellates (Dagg et al. 2009; Frost 1972; Ohman and Runge 1994; Runge 1980). An important consequence of copepod feeding is the observation of inefficient or 'sloppy' feeding (Conover 1966b; Roy et al. 1989), which is most pronounced in conditions of high abundance of large diatoms (Roy et al. 1989), discussed further below.

Euphausiids have a feeding mode that is closer to true filter feeding (Riisgard and Larsen 2010). They use a 'filter basket' within a cavity formed by the setae of their long thoracic legs (Hamner 1988). As the filter basket expands, water is sucked in through the front, and suspended particles are trapped on the filter. The size of the particles consumed is dependent on the distance between setae, but euphausiids generally do not feed on particles < 5 μm (Mauchline and Fisher 1969; Suh and Choi 1998; Suh and Nemoto 1987). This mode of feeding allows for very high clearance rates, but with less particle selectivity and increased efficiency of food consumption compared with copepods (Mauchline 1980). Many species of euphausiids are omnivorous and can switch feeding modes to prey raptorially on zooplankton (Hamner 1988). Euphausiids exhibit many trophic strategies, but the most dominant epipelagic species tend to be herbivores and opportunistically omnivores and/or detritivores (Dilling et al. 1998; Nakagawa et al. 2001; Nakagawa et al. 2004; Ohman 1984; Ponomareva 1966). The grazing impact of the copepod community on the phytoplankton community is spatially very variable, probably mostly dependent on the size structure of the phytoplankton community. Studies in the equatorial Pacific found the copepod community removed as little as 5% of phytoplankton standing stock (Roman et al. 2002), whereas a north-south transect through the Atlantic ocean found that copepods could remove up to 100% of PP in highly productive areas, and have a substantial impact in oligotrophic gyres (Lopez and Anadon 2008). A global comparison by Calbet (2001), including studies from many different marine environments, found that, on average, copepods consume 12% of the PP.

The grazing impact of euphausiid populations is probably even more variable, because their distribution is highly patchy (e.g., Mauchline 1980; Décima et al. 2010), yet fewer studies have been conducted on their grazing impact, with most studies focused on Southern Ocean krill. Given the high densities that they can achieve and their very high clearance rates, these organisms are capable of significant local effects on phytoplankton communities. Southern Ocean krill have been calculated to remove 33% of phytoplankton standing stock per day in waters east of South Georgia (Whitehouse et al. 2009). The mesozooplankton community, as a whole, was found to sometimes exert enough grazing pressure to depress phytoplankton growth in the California Current, with grazing rates exceeding phytoplankton growth rates (Landry et al. 2009), when euphausiids comprised a significant portion of the community (Décima unpubl. data). In the Oyashio region, the euphausiid population could, at times, consume up to 24% of the PP (Kim et al. 2010), although this impact was temporally variable. The euphausiid community can therefore exhibit significant top-down control over the phytoplankton community, although this impact is highly temporally and spatially variable.

Gelatinous zooplankton

The numerically dominant herbivorous gelatinous grazers are the pelagic tunicates (e.g., salps, doliolids, and appendicularians). Planktonic tunicates differ substantially from their crustacean counterparts in their feeding modes, particle capture size range, growth rates, and biogeochemical effects. They are true filter feeders, able to feed on a very wide range of prey from bacterial-sized particles (less than 1 μm) up to a few millimeters (Alldredge and Madin 1982; Madin 1974). Bacteria can make up a substantial portion of the diet of appendicularians (King et al. 1980), and salps are able to retain particles from 4 μm to 1 mm with ~ 100% efficiency (Alldredge and Madin 1982).

Appendicularians secrete a mucus 'house,' from inside which they extract prey from the water column. Large particles, such as diatoms and dinoflagellates, are removed by a coarse mesh covering the incurrent filters, and small particles are concentrated on a complex, internal filter (particles can be as small as 0.1 μm) and ingested (Alldredge 1977; Alldredge and Madin 1982). Thaliaceans (i.e., salps, doliolids, and pyrosomes) use a mucus net to filter particles. The barrel-shaped salps pump water through a large pharyngeal cavity, and water passing through is filtered onto the net, which covers the entire cavity. The mucus is continuously created, and along with the particles adhered to it, continuously consumed. Pyrosomes and doliolids also have a mucus net, but filtering is conducted by ciliary action and filtering capacity is reduced compared with salps (Alldredge and Madin 1982). Salps exhibit very high clearance rates per individual, some species filtering up to 5 L seawater per hour (Madin et al. 1981). The ecological role of salps and appendicularians differs from that of crustaceans because planktonic tunicates can graze on bacterial-sized particles. Thus, they constitute a link between the recycling 'microbial loop' (Azam et al. 1983) and the classic food chain, transporting energy and nutrients to the higher trophic levels.

As mentioned above, the growth rates of metazoan grazers are generally lower than those of their protistan counterparts. Crustacean growth rates vary with species and temperature, but generation time is on the order of at least 3 weeks for copepods (Paffenhofer and Harris 1979) and 2 months or more for euphausiids (Ross 1982; Ross et al. 1988). In contrast, pelagic tunicates have higher growth rates (Alldredge and Madin 1982; Heron 1972), and in some cases, complex life histories. For instance, thaliaceans have episodic population bursts, leading to swarms that can extend for kilometers (Berner

1968). Large salp swarms can clear a significant portion of the water column: off the northeast coast of the US, salp swarms were estimated to extend 100,000 km^2 and to clear up to 74% of the biomass in the upper 50 m of the water column, depending on salp biomass (Madin et al. 2006; Wiebe et al. 1979). Appendicularians can also reach very high densities capable of removing ~ 50% of bacterial biomass (Scheinberg et al. 2005).

Quantifying zooplankton grazing impact

The methods for estimating grazing rates are numerous, and it is not the goal of this chapter to review them all. For a more comprehensive review on methodology, readers should consult Båmstedt et al. (2000) and references within. We will focus on the most commonly used methods for estimating both the micro- and mesozooplankton grazing impact.

Bacterivory has proven to be a hard process to measure, mainly because of the small size of the organisms involved and their tight coupling with the remaining planktonic community. Techniques used to quantify the grazing impact of microzooplankton on bacterial communities can be divided into two main classes, those that use tracer methods and those that manipulate the community in such a way as to alter encounter rates between bacteria and predators (Strom 2000). The first class includes ingestion of fluorescently labeled bacteria (FLBs), radioisotope labeling, and minicell removal; these methods focus on grazer taxa or types. The latter class includes the seawater dilution method (modified from the original method to quantify phytoplankton consumption, explained in detail below), size fractionation, and metabolic inhibitors; these methods provide estimates of whole-community grazing rates (Båmstedt et al. 2000; Strom 2000). The significance of the choice of methodology was shown by Vaqué et al. (1994), who found that methods using tracer techniques yielded consistently lower rates than the whole-community methods. Thus, the choice of methodology should be kept in mind when attempting to construct mortality budgets as different methods may result in significant error and/or variability in individual studies and in comparisons between ecosystems.

Microzooplankton grazing on phytoplankton is most commonly estimated using the dilution method, developed by Landry and Hasset (1982). This method relies on the reduction of encounter rates between microzooplankton and their phytoplankton prey. It has the advantage of simultaneously estimating both phytoplankton growth and mortality rates. Serial dilutions are carried out, mixing natural seawater with increasing fractions of filtered seawater collected under the same conditions. It is assumed that dilution does not affect the growth rate of the phytoplankton, but does decrease grazing due to the reduced likelihood of random encounters between grazers and their prey. Grazing rate is estimated as the slope of the regression of the apparent phytoplankton growth rates against the dilution factors (Fig. 2). Phytoplankton growth rate is assumed to be the growth rate extrapolated to 100% dilution (Landry et al. 1984; Landry and Hassett 1982). Criticisms center on potential violations of the assumptions of this method, including altered phytoplankton growth rates with varying dilution, differences in per-capita feeding rate in different dilution treatments, increased mortality of grazers with increasing dilution, and changes in the grazer community during incubation (Dolan et al. 2000; Dolan and Mckeon 2005). Further, the assumption that grazing is the sole source of mortality in these experiments is potentially inaccurate, as we will discuss later. Despite these concerns, the method has proven quite robust to potential violations of assumptions (Gifford 1988), and its use has steadily increased over time (Dolan et al. 2000). The method can have potentially greater biases when used for estimating bacterivory. Because bacteria are highly dependent on the community for the production of DOM for growth, dilution can have potentially greater effects on bacterial growth rate, violating basic assumptions of the method (Landry 1993a; Vaqué et al. 1994).

Mesozooplankton grazing has also been investigated by use of a variety of methods. Again, our goal is not an extensive review, but rather to introduce the most commonly used methods along with their assumptions and flaws. Because we are mainly interested in the fate of microbial production, we will focus on the methods that address this portion of the community, rather than covering the gamut of methods, which also include those used to quantify omnivory as well as consumption of other large zooplankton. Community impact on phytoplankton can be assessed by use of the gut pigment method (Mackas and Bohrer 1976), disappearance incubations based on the removal of microplanktonic prey (Frost 1972; Marin et al. 1986), radiotracer methods (Roman and Rublee 1981), and gut content analysis (see Båmstedt et al. 2000). Of these methods, the most widely used are bottle incubations that monitor prey disappearance and the gut pigment method.

The advantage of incubation experiments is that both feeding rates and selectivity can be very accurately quantified, enabling estimates of the grazing impact on different portions of both phyto- and microzooplankton community. The disadvantages of this method are that the sorting of individual grazers is time consuming, and can only be done for a subset of species since whole community incubations are typically not feasible. In addition, incubations can be biased due to bottle artifacts, such as trophic interactions within the treatments (Nejstgaard et al. 2001) or differences in algal growth between control and treatment bottles (Roman and Rublee 1980). Finally, the prey field encountered within the bottle might not adequately represent that encountered in the water column, as incubations generally underestimate mesozooplankton ingestion (Mullin and Brooks 1976).

The gut pigment method is based on quantifying chlorophyll (and its degradation products) in the guts of grazers, and can be used to estimate feeding rates on phytoplankton in the field (Mackas and Bohrer 1976). This method is popular

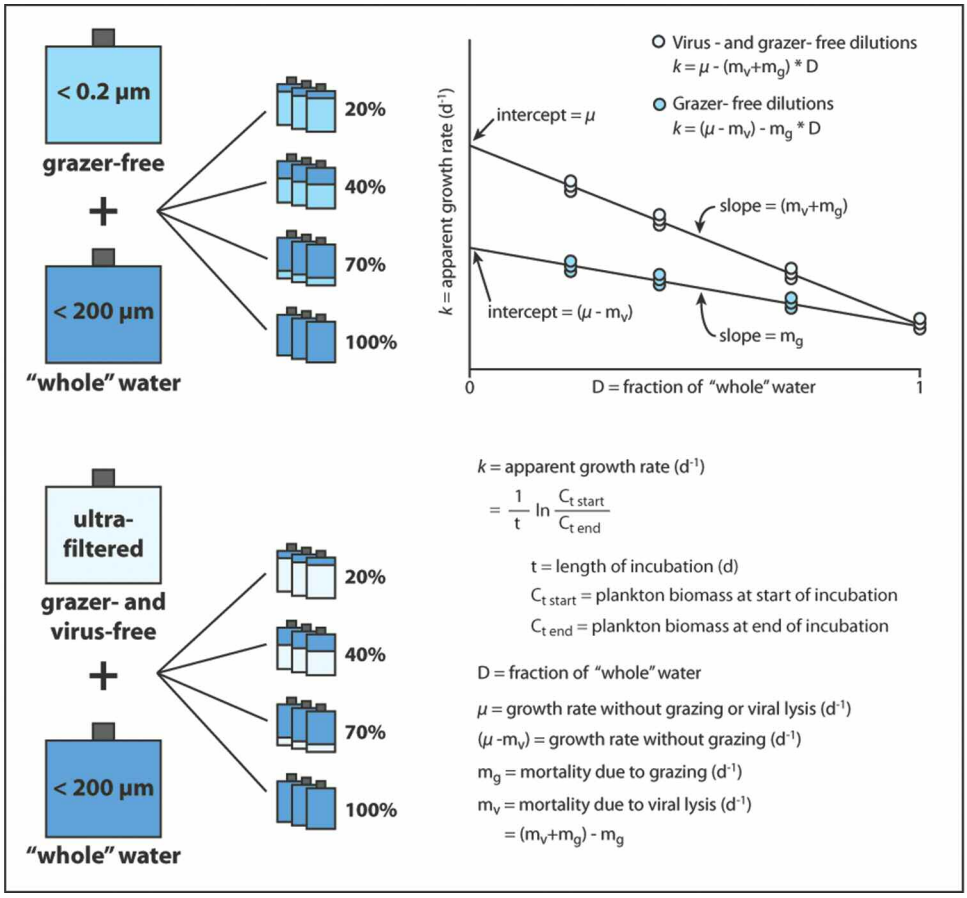

Fig. 2. Representation of the dilution method and equations used to quantify mortality of cells as a result of grazing or viral lysis. The original dilution method for microzooplankton grazing includes only the grazer-free dilution series (Landry and Hassett 1982). The fraction of whole water may also be replaced with relative grazing rate (Landry et al. 1995). Redrawn from Baudoux et al. (2006) and Evans et al. (2003).

because it is direct, does not require incubations, is very cost effective, and is analytically and conceptually simple. Individual zooplankters or whole communities are sampled from the field, immediately frozen, ground up, extracted in acetone, and gut chlorophyll and phaeopigments are quantified fluorometrically (Lorenzen 1967). Importantly, the reliance on pigment quantification means this method can only be used for estimating consumption of autotrophic prey. Potential problems with this method include underestimation of grazing due to pigment degradation when using individual animals (Dam and Peterson 1988; Kiorboe and Tiselius 1987), and/or overestimation of grazing when using whole communities due to the potential of phytodetritus contamination (Décima et al. 2011). Additional problems with this method may arise from the choice of an estimated carbon:chlorophyll ratio because this ratio is depth dependent and the depth of feeding is not usually known because mesozooplankton are integrated over the euphotic zone in this method.

In reviewing the myriad of methods used to quantify the impact of zooplankton on microbial production, it becomes clear that, as stated by Strom (2000), our understanding of these processes is colored by the methods we choose to study them. Reconciling the results of the array of methods used to quantify mortality estimates is a non-trivial issue, arising both from methodological issues regarding a single process, as well as our lack of understanding of the interaction of these different processes (discussed further below).

Section 3. Viruses

Viruses are the most abundant biological entities in the marine environment, ranging from 10^4 to 10^8 mL^{-1} with the highest concentrations found in the mixed layer and in more productive oceanic areas (reviewed by Wommack and Colwell 2000). Based purely on abundance, the hosts for the majority of these viruses are thought to be bacteria, and to a lesser extent, eukaryotic phytoplankton (Murray and Jackson 1992). In support of this, viral concentrations tend to have positive correlations with bacterial concentrations in a range of oceanic environments (reviewed by Wommack and Colwell 2000), and in some cases, closely track the concentrations of specific, numerically abundant organisms such as *Prochlorococcus* (Parsons et al. 2011). Metagenomic analyses of marine

viral populations have also shown that the majority of known viral sequences have high similarity to viruses that infect bacteria (Bench et al. 2007). Whereas these bacteriophages are probably the dominant component of the oceanic viral assemblage, many viruses that infect phytoplankton have also been isolated and can have substantial effects on phytoplankton populations (Brussaard 2004), e.g., by terminating blooms of seasonally abundant algal species such as *Emiliania huxleyi* (Jacquet et al. 2002).

Viral infections in the marine environment are primarily controlled by host specificity and contact rate. Based on tests with viral isolates, viruses typically only infect a small taxonomic range of host species or strains (e.g., Nagasaki et al. 2005; Waterbury and Valois 1993), and contact with their particular hosts is predominantly affected by the concentrations of virus and host in the environment (Murray and Jackson 1992). The host specificity of viruses results in a dynamic between viruses and their hosts described in the 'kill the winner' hypothesis (Thingstad and Lignell 1997) in which the most abundant hosts (the "winners") will have the highest rates of viral lysis, thus decreasing their abundance and limiting the ability of any specific taxon to dominate the microbial plankton. This specificity differentiates viral lysis from grazing, as grazers may preferentially feed on cells within a certain size class (Gonzalez et al. 1990) or with particular surface properties (Monger et al. 1999), but in general, do not target specific species (Paffenhofer et al. 2007).

After successful contact with their hosts, viruses can then proliferate using several strategies (reviewed by Fuhrman 2000). During lytic replication, thought to be the most common viral infection strategy in the ocean, viruses "hijack" the host's biochemical systems and redirect them to the production of virus particles, ultimately resulting in lysis, or bursting, of the host cell to release the viral progeny. Lysogenic, or temperate, infections occur when the viral genome integrates into the host genome until it is "induced" to switch to the lytic cycle. Last, some viruses are able to "bud" from the host cell, allowing viral production without causing cell death. Because this chapter deals with mortality, only lytic and temperate infections will be addressed, with the term "viral lysis" referring to the death of the host cell as a result of lytic or induced temperate viral infection.

Quantifying viral-induced mortality of marine microorganisms

Viruses are commonly quantified by epifluorescence microscopy of nucleic acid-stained particles collected on 0.02 μm-pore size filters (Noble and Fuhrman 1998), although some investigators also use flow cytometric methods (Brussaard et al. 2000). However, raw abundance estimates cannot predict rates of infection or viral productivity, and so multiple methods have been developed to estimate viral lysis of microbial cells in the marine environment. Transmission electron microscopy can be used to visualize assembled viruses within whole bacterial cells (Proctor and Fuhrman 1991) or in thin sections of cyanobacteria (Proctor and Fuhrman 1990) or eukaryotic phytoplankton (Brussaard et al. 1996). The fraction of total cells containing visible viral particles can then be used to estimate the fraction of bacterial mortality that is due to viral lysis (Proctor and Fuhrman 1990; Binder 1999), although this calculation relies on conversion factors derived from only a few cultivated virus-host systems.

Total viral production can be estimated in a variety of ways including the measurement of viral decay after addition of cyanide (Heldal and Bratbak 1991), the incorporation of ^{32}P-orthophosphate into the viral nucleic acid fraction following incubation (Steward et al. 1992), and the decay of fluorescently labeled virus tracers added to samples (Noble and Fuhrman 2000). However, the most often used method of measuring viral production is the dilution method (Weinbauer et al. 2010; Wilhelm et al. 2002; Winget et al. 2005), sometimes referred to as the "virus reduction approach" to distinguish it from the dilution studies of grazing described above (Winget et al. 2005). In this method, the majority of viruses are removed from a sample via filtration while cells are retained, and the subsequent increase in viral concentration over time is used to calculate viral production (Wilhelm et al. 2002; Winget et al. 2005). Viral production rates can then be used to estimate bacterial mortality assuming that all of the viruses are produced from bacteria and that the number of viruses produced per bacterium (burst size) is known or estimated (Wilhelm et al. 2002).

A modification of the dilution method used to measure grazing has also been developed to quantify mortality of phytoplankton populations due to viral lysis (Evans et al. 2003). In this method (Fig. 2), mortality of phytoplankton due to grazing is measured by generating a dilution series of sample water with 0.2 μm-filtered water (removing grazers, but retaining viruses), whereas phytoplankton mortality due to grazing plus viral lysis is measured by generating a dilution series of sample water with ultra-filtered water, removing both grazers *and* viruses, thus lowering the rate of viral infection in the same way that grazer dilutions lower the rate of encounter with grazers. The mortality of phytoplankton calculated from the dilution series with grazer-free water is subtracted from the mortality of phytoplankton calculated from the dilution series with grazer- and virus-free water to result in the mortality of phytoplankton due to viral lysis. This method is extremely labor-intensive and involves significant manipulation of the plankton community. Analysis of this method shows that biases associated with the sample manipulation, including alteration of nutrient regeneration pathways, trophic level interactions, and growth rates, can cause significant problems with interpretation of the results (Kimmance et al. 2007; Tijdens et al. 2008). Still, it represents an important step forward in the study of marine microbial mortality and remains one of the few experimental designs to test multiple vectors of death simultaneously.

Mortality due to viral lysis

It is now estimated that viruses cause roughly 10% to 40% of overall bacterial mortality in the oceans (reviewed by Fuhrman 2000), and that mortality due to viral lysis can be equivalent to mortality caused by grazing in some waters (Boras et al. 2009; Fuhrman and Noble 1995; Steward et al. 1996). The metabolic status of microorganisms is a significant factor in regulating viral production, with highly metabolically active bacteria producing more viruses as a result of shorter latent periods and larger burst sizes (reviewed by Weinbauer 2004). Thus, oceanic areas with greater productivity tend to have greater microbial mortality due to viral lysis (Boras et al. 2009; Steward et al. 1996; Weinbauer et al. 1993; Winget et al. 2011). This relationship does not always hold true, however. For instance, high viral productivity measurements could be a consequence of sampling single time points when there is episodically high lysis of specific bacterial subpopulations even when total bacterial production is low (Holmfeldt et al. 2010). Time series studies are thus especially helpful in resolving the overall positive relationship between bacterial production and viral lysis of bacteria (Boras et al. 2009; Winget et al. 2011).

The percent of phytoplankton mortality due to viral lysis is generally less than 10%, the estimated detection limit of the modified dilution method (Kimmance et al. 2007), and reaches approximately 20% of total mortality when statistically significant results are obtained (Baudoux et al. 2008). During blooms however, viruses can account for up to an estimated 100% of the mortality of bloom-forming phytoplankton species such as *Emiliania huxleyi* (Bratbak et al. 1993; Brussaard et al. 1996; Jacquet et al. 2002). Viral lysis of bacteria is also elevated during the decline of a bloom when bacterial abundance has increased (Guixa-Boixereu et al. 1999). Taken together, these studies show that viral lysis is especially important under non–steady-state conditions such as during phytoplankton blooms when abundance of phytoplankton and bacteria are elevated.

Section 4. Autologous cell death

In this section, we consider the various routes by which a cell can die without being actively killed by another biological entity, as well as the environmental factors that catalyze these processes. This category encompasses both mortality originating from a failure of cellular homeostasis or structural integrity (necrosis) as well as from a genetically "programmed" process of cellular suicide. In contrast to grazing and viral lysis, the effects of autologous cell death on marine microbial populations have received relatively little attention. We also review experimental methods for distinguishing autologous death from grazing and lysis, and consider ways in which these processes may influence, or be influenced by, the others described in this chapter. Last, we point out gaps in our current understanding of these processes, especially in relation to their importance in the field.

In this work, we use "programmed cell death," or "PCD," to refer specifically to programmed, non-lytic death processes that proceed through defined histological stages similar to apoptosis in multicellular animals. Necrosis, the other autologous process we consider, refers to uncontrolled cell death caused by physical damage. For our purposes, we distinguish necrotic damage from damage inflicted by predation or viral infection despite the fact that both of these processes, strictly speaking, lead to death by necrosis. Instead, we limit our definition of necrosis to damage inflicted by chemical, physical, or radiological stressors. PCD may be distinguished from necrosis primarily because PCD exhibits several prominent hallmarks that are diminished or absent in necrotic cells (Table 1). Importantly for the fate of the organic matter comprising the cells, PCD (as we above define it) does not result in lysis, whereas necrosis typically does.

Programmed cell death

PCD has been observed in bacteria, phytoplankton, and land plants, and most groups of heterotrophic eukaryotes (Bidle and Falkowski 2004; Deponte 2008; Lewis 2000; Nedelcu et al. 2011). Often, some form of environmental insult initiates the process: for instance, osmotic shock (Ning et al. 2002), oxidative stress (Darehshouri et al. 2008; Ross et al. 2006; Vardi et al. 1999), ultraviolet irradiation (Moharikar et al. 2006), nutrient starvation (Berges and Falkowski 1998; Berman-Frank et al. 2004; Vardi et al. 1999), and prolonged dark incubation (Segovia et al. 2003). Table 1 lists several of the histological hallmarks associated with PCD. Initially, the cytoplasmic volume decreases significantly, often with the appearance of large, electron-sparse vacuoles. In eukaryotes, organelles disintegrate and chromatin condensation renders the nucleus more electron-dense. Production of reactive oxygen species (ROS) increases dramatically as metabolic path-

Table 1. Distinguishing characteristics of PCD and necrosis.

	PCD	Necrosis
Morphology	Cell shrinkage	Irregular; "ghost cells"
DNA	Chromatin condensation in eukaryotes; extensive fragmentation	Depends on causative agent of cell death
Cell envelope	Remains intact, at least initially	Damage ranging from holes to complete lysis
De novo protein synthesis	Required; prominent caspase activity	None required; extant proteins are often damaged

ways shut down and reactive intermediates accumulate. ROS are capable of extensive deleterious modifications of biological molecules, including strand breaks in DNA. Last, PCD is accompanied by de novo protein synthesis that continues throughout the cell death process. A class of proteases called "caspases" is often used as an incontrovertible diagnostic marker of PCD (Bidle and Falkowski 2004). Short for "cysteine-dependent aspartate-targeted proteases," these "executioner proteins" target very specific recognition sequences inside polypeptides, cleaving certain proteins in such a way as to alter their behaviors and bring about cell death. Caspases are strongly evolutionarily conserved and widely distributed in nature, existing in bacteria, plants, fungi, animals, and disparate groups of protists (e.g., slime molds, eukaryotic algae).

There are various methods available for detecting PCD. Some researchers use intracellular ROS production alone as an indication of PCD induction. A number of simple and sensitive methods exist for measuring hydrogen peroxide (HOOH) using fluorescent or chemiluminescent substrates, allowing both ROS quantification and the detection of ROS-producing cells by epifluorescence microscopy or flow cytometry. Using ROS formation alone as an indicator of PCD is problematic, however, because ROS are also important causes of, and consequences from, necrotic cell death, attending myriad stresses ranging from desiccation to temperature shock to nutrient imbalance. Therefore, it is difficult to determine whether ROS created by "intentional" PCD, as opposed to ROS generated "accidentally" during necrosis, is responsible for cell death. This problem is exacerbated for samples exposed to light, because ROS are continually photochemically generated in natural waters (see below).

Another method used to detect PCD is terminal deoxyribonucleotide transferase-mediated dUTP nick end labeling (TUNEL). This method uses an enzyme to label free 3′ ends in DNA with a modified nucleotide that may be detected using a fluorophore-tagged antibody. As PCD is attended by extensive DNA fragmentation, dying cells are often detectable using TUNEL. However, like ROS production, DNA damage is neither exclusively the province of PCD nor is it clear whether it is a cause or a consequence of the death program. For instance, studies have shown that necrotic eukaryotic cells also stain positive using TUNEL (Graslkraupp et al. 1995). Thus, like ROS detection, a positive TUNEL signal is insufficient evidence for diagnosing PCD. Further, whereas TUNEL has been used successfully on cultured bacteria (Rohwer and Azam 2000), the multi-stage labeling process, with multiple centrifugation steps, may be problematic for dilute field populations.

The strongest evidence for PCD comes from caspase activity assays. Typically, such an assay involves a short polypeptide containing the recognition sequence for a given caspase as a blocking group covalently bound to a fluorophore. Cleavage of the peptide dramatically increases fluorescence, allowing quantitation of caspase activity. Unlike ROS and DNA fragmentation, the detection of caspase activity provides direct and specific evidence of apoptosis-like PCD. Caspases have been detected under various stressful conditions in cultured phytoplankton, including cyanobacteria (Berman-Frank et al. 2004; Ross et al. 2006), green algae (Darehshouri et al. 2008; Moharikar et al. 2006; Segovia et al. 2003), diatoms (Berges and Falkowski 1998), coccolithophores (Bidle and Falkowski 2004), and dinoflagellates (Vardi et al. 1999). Because caspase assays are generally performed on cell extracts, however, they do not readily allow identification of specific cells undergoing PCD. Thus, they are most convenient to use on dense algal blooms or axenic cultures. This is clearly an impediment to the detection of PCD in sparser, oligotrophic habitats, where mere detection of caspase activity would beg the question as to which organisms were responsible. One potential solution to this problem is to couple caspase assays with secondary PCD detection methods that allow detection of individual cells, such as ROS-sensitive fluorophores or TUNEL assays.

Evidence from laboratory cultures provides compelling evidence that phytoplankton and other microbes undergo PCD in response to environmental stresses (Bidle and Falkowski 2004), but it is unclear how important PCD is in the field. Moreover, it is conceptually surprising to find PCD commonplace in unicellular organisms, and it is not at all clear what advantage they gain from inducible suicide. There are several possible explanations for how PCD benefits a stressed population. The dying cells could become a food source for the survivors as they form resting stages (Lewis 2000). Lysis could release usually cytoplasmic protective compounds such as antioxidants (Hasset et al. 2000) and sunscreens (Roy 2000) into the environment that are capable of improving environmental conditions. Local PCD could also stymie the spread of a virus both by reducing burst size through ROS-mediated viral inactivation and by maintaining the population concentration below the critical threshold where "kill the winner" dynamics (Winter et al. 2010) lead to the crash of the broader community from unchecked infection. Indeed, strong production of ROS has been detected in *Emiliania huxleyi* cultures undergoing viral lysis (Evans et al. 2006); whereas this may be a simple consequence of a deteriorating physiology, it could also be indicative of a PCD response. This latter possibility parallels the recent idea of "cryptic escape," which suggests that some marine bacteria have evolved rarity to dissuade the evolution of specific predators, including both grazers and phage (Yooseph et al. 2010).

Alternatively, the conceptual problems underlying the evolution of PCD in unicellular organisms lead some researchers to speculate that evidence for PCD is an artifact of other, better known processes—a remnant of lysogenic viral infection, for instance, that becomes activated under stress conditions and leads to cell death (Nedelcu et al. 2011). Thus, the origin, importance, and very existence of PCD in marine microbes remains extremely controversial. It is incumbent on future researchers in oceanic mortality to measure the prevalence of

PCD in marine microbial communities. How abundant are PCD genes, at both the intra- and interspecies levels? What induces PCD in the field? And what advantages does it bestow on the populations and communities where it occurs?

Necrosis

In contrast with PCD, the mechanisms leading to necrotic death in microbes are much better understood. However, few studies have examined the relative importance of death by "natural causes" vis-à-vis grazing and viral lysis. In this chapter, we will discuss three likely sources of necrotic death (ROS, solar radiation, and chemical pollutants) as well as the conditions under which each may be of increased importance.

Environmental ROS

We have already considered how ROS can be both a cause and a consequence of PCD in microorganisms. However, ROS can also kill without a suicide response from a target cell. In the following discussion, we will focus on HOOH as opposed to other ROS (e.g., superoxide, singlet oxygen, hydroxyl radical) for several reasons. First, each of the other ROS either originates by reaction with HOOH or undergoes spontaneous reactions that form HOOH. For this reason, it may be assumed that fluxes of any ROS in excess of a system's ability to remove them will result in the presence of HOOH. Second, because of its relative stability, detection of HOOH and assessment of its dynamics are much easier than for the other ROS, particularly under field conditions. Third, laboratory experiments have shown that some marine organisms are sensitive to environmentally realistic concentrations of HOOH (Morris et al. 2011), whereas to our knowledge, no comparable experiments have yet been performed with other ROS. It is thus reasonable to use HOOH as a proxy for the ROS burden imposed on a natural system.

It has long been recognized that the formation of ROS is an unavoidable consequence of aerobic metabolism. Many enzymes capable of single-electron transfer to a substrate are also capable of reducing O_2 and the ubiquity of O_2 in aerobic environments (and even more so in photosynthetic organisms) ensures that some ROS will occur. Photosynthetic or respiratory membrane complexes produce ROS when O_2 is more abundant than their "legitimate" electron carriers. ROS buildup from unbalanced metabolism could also explain the toxicity of high-nutrient media to organisms adapted to oligotrophic lifestyles (Del Giorgio and Gasol 2008). ROS accumulation is further observed as a general consequence of diverse stresses, such as temperature, osmotic, and dehydration stress (Franca et al. 2007; Higuchi et al. 2009; Kong et al. 2004; Prasad et al. 1994; Ross and Alstyne 2007; Suggett et al. 2008), and indeed is common in all damaged and dying cells (see above). Importantly, HOOH is essentially as permeable to biological membranes as H_2O (Halliwell and Gutteridge 2007), and therefore, stressed organisms leak HOOH into their environment, potentially leading to problems for their neighbors. Last, HOOH release is not inevitably a result of cellular "accidents" caused by stress, but is also involved in intercellular signaling (Orozco-Cardenas and Ryan 1999; Rhee 2006), cell envelope development and repair (Lagrimini 1991; Ross et al. 2005), iron acquisition (Rose et al. 2008), and allelopathy and competition (Babior 2000; Levine et al. 1994; Oda et al. 1992; Oda et al. 1997; Twiner and Trick 2000; Visick and Ruby 1998).

Whereas biological release must be considered as an intermittently important source, most pelagic HOOH arises through photochemistry. HOOH is formed directly in the water column by the photooxidation of DOM (Cooper and Zika 1983; Cooper et al. 1988; Draper and Crosby 1983; Wilson et al. 2000). HOOH production is stimulated by light in the visual range, but is much faster under ultraviolet irradiation (Gerringa et al. 2004). HOOH also forms in the atmosphere by the direct photolysis of water (Kasting et al. 1985), and rainwater typically contains 100 times as much HOOH as surface seawater (Kieber et al. 2001a). Rainfall events can cause transient, yet dramatic, increases in the HOOH concentration of surface seawater (Avery et al. 2005; Hanson et al. 2001; Kieber et al. 2001a; Kieber et al. 2001b; Willey et al. 2004; Willey et al. 1999; Yuan and Shiller 2000).

In contrast to HOOH formation, microbial degradation is the primary sink for HOOH in the ocean (Cooper and Zepp 1990; Moffett and Zafiriou 1990; Petasne and Zika 1997). This is unsurprising given the toxicity of ROS to microbes. As many (perhaps most) aerobic organisms are able to eliminate HOOH from their environment, the bulk HOOH concentration is drawn down by the collective action of microorganisms, detoxifying their immediate environment. The concentration of HOOH measured at the surface of pelagic waters is remarkably globally consistent (Fig. 3). In a study covering 63 stations in oceanic, coastal, and freshwater environments, with mixed layer depths spanning an order of magnitude, surface concentrations averaged 95.9 ± 29.3 nM (Morris 2011). Given that DOM concentrations, irradiance, and bacterial concentrations are likely highly variable across these sites, it is striking that the steady state HOOH concentration is so consistent. One interpretation is that ~100 nM HOOH represents a "safe" concentration, and selection is strong on the HOOH-degrading abilities of microbial communities to preserve this level in the wake of large fluctuations in rates of HOOH production.

What factors contribute to the rate of HOOH degradation? Certainly, bacterial abundance is likely to play a part, but it is unclear whether all or even most organisms in some communities contribute to HOOH removal. Interestingly, the genomes of some abundant oceanic microbes (e.g., *Prochlorococcus*, *Pelagibacter*) are conspicuously deficient in enzymatic defenses related to HOOH protection. In sterile, filtered seawater exposed to sunlight, HOOH concentrations rise dramatically, achieving levels within 24 hours that are lethal to axenic cultures of *Prochlorococcus* (Morris et al. 2011). More generally, natural bacterial and algal populations appear to be susceptible to HOOH damage at concentrations much lower than those necessary to impair laboratory strains (Drabkova et

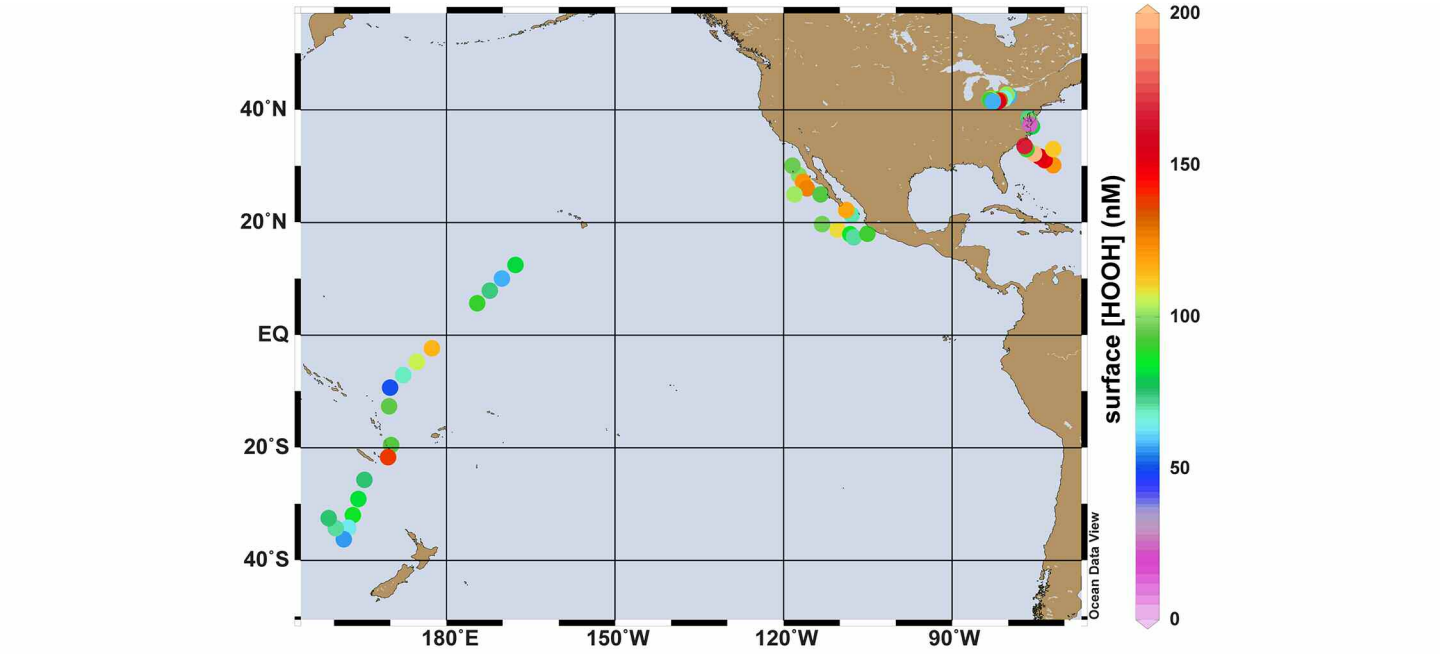

Fig. 3. Surface HOOH concentrations in pelagic waters. Near-surface water was collected and assayed for HOOH concentration by acridinium ester chemiluminescence using either a FeLume flow injection instrument (King et al. 2007) or an Orion-L microplate luminometer (Morris et al. 2011). Station locations and expanded protocols may be found in Morris (2011).

al. 2007a, 2007b; Samuilov et al. 1999; Skurlatov and Ernestova 1998; Tichy and Vermaas 1999; Xenopoulos and Bird 1997). Thus, the toxicity of naturally occurring, low concentrations of HOOH may be underappreciated, and may be a significant source of loss of some important species under certain conditions. Moreover, the susceptibility of dominant microbes like *Prochlorococcus* suggests that a minority of the total microbial community (the so-called "helpers," *sensu* Morris et al. 2011) is responsible for maintaining HOOH near tolerable limits for these sensitive species.

Solar radiation

Microbial populations in the ocean are also at risk of death from solar radiation. Increasing fluxes of photosynthetically active radiation (PAR, i.e., visible light) eventually saturate the ability of phototrophs to harvest photons, and at excessive levels, actually diminish the rate of PP (Falkowski and Raven 2007; Vassiliev et al. 1994). Even under optimal conditions, ROS formed during photosynthesis continually degrade elements of the light-harvesting machinery, particularly the photosystem II reaction center protein D1 (Blankenship 2002). Photoinhibition occurs when this damage exceeds the repair capacity of the cell. In most cases, photoinhibition caused by PAR is assumed to be reversible (i.e., it does not result in mortality), although some phototrophic organisms are known to be vulnerable to killing by PAR at levels commonly experienced in the mixed layer. For instance, the *Prochlorococcus* ecotype eMIT9313 is highly abundant below the thermocline in many waters, but is unable to grow when light is greater than ~10% surface irradiance (Moore and Chisholm 1999). PAR also reduces the viability and metabolic activity of heterotrophic bacteria, albeit with a great deal of variability between taxa (Arana et al. 1992; Helbling et al. 1995; Jeffrey et al. 2000).

In contrast to PAR, ultraviolet radiation (UV) is capable of damaging a much broader range of targets, and is known to negatively impact aquatic microbial communities (Cullen et al. 1992; Häder 2001; Häder et al. 2011; Helbling et al. 1995; Herndl 1997; Karentz et al. 1996; Llabres and Agusti 2006; Muller-Niklas et al. 1995). UV is capable of penetrating to significant depths in the water column, especially in the oligotrophic oceans, where as much as 10% of longer wavelength UVA (320-400 nm) is still present at 20 m depths (Fig. 4B). Despite the higher energy of UVB wavelengths (280-320 nm), many experiments suggest that UVA is responsible for the majority of the effects of UV in nature, largely because much more UVA impacts the Earth's surface (Fig. 4A) (Cullen et al. 1992; Whitehead et al. 2000). Due to anthropogenic ozone depletion, UVB is more abundant at higher latitudes, and may have a stronger impact on communities in these waters (Häder et al. 2011; Whitehead et al. 2000).

Damage from UV is both quantitatively and qualitatively different from PAR photoinhibition. As mentioned earlier, UV can cause indirect damage by accelerating the rate of ROS generation. UV can also directly damage DNA and other macromolecules, because these compounds absorb UVB much more strongly than PAR. UV irradiation of DNA forms cyclobutane dimers between neighboring thymine residues that not only lead to mutagenesis if unrepaired, but also cause RNA polymerase to "stick" to the lesion, causing a short-term inhibition

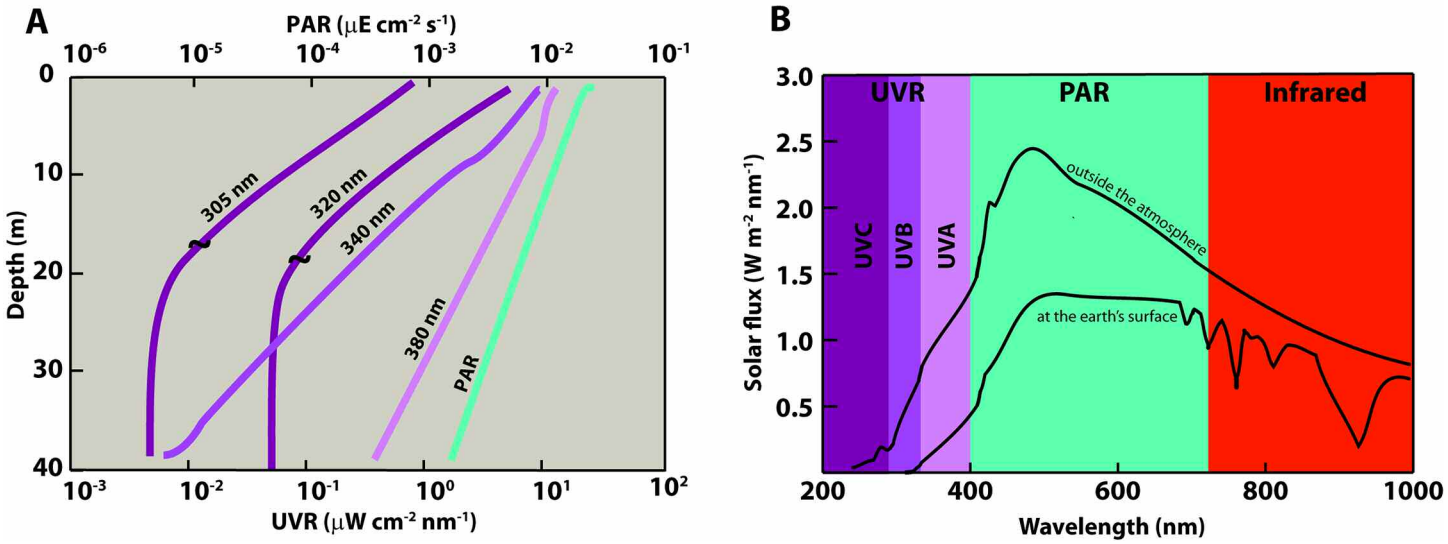

Fig. 4. Solar radiation fluxes. A) Penetration of PAR, UVA (380 nm), and UVB (305-340 nm) radiation into coastal waters near Palmer Station, Antarctica. ~ on 305 and 320 nm traces in panel A, limit of reliable detection. Redrawn from Helbling et al. (1995). B) Solar radiation in and above the Earth's atmosphere. Redrawn from Whitehead et al. (2000).

of transcription that can have wide-ranging and potentially lethal effects (Vincent and Neale 2000). UV also affects proteins, including pigment-bearing complexes (Araoz and Häder 1997; Six et al. 2007; Vernet 2000), rubisco (Vincent and Neale 2000), flagella (Roy 2000; Sommaruga et al. 1996), and catalase (Heck et al. 2003).

Many organisms are strongly protected against UV, with defensive mechanisms including light-activated DNA repair proteins (i.e., photolyases) and sunscreens (e.g., mycosporine-like amino acids and sporopollenin) (Roy 2000). However, other organisms, particularly smaller photoautotrophs, are conspicuously susceptible (Dillon et al. 2003; He and Häder 2002; Karentz et al. 1991; Llabres and Agusti 2006; Llabres et al. 2010; Moharikar et al. 2006; Rai et al. 1996; Rai and Rai 1997; Sobrino and Neale 2007; Song and Qiu 2007), perhaps due to the shorter path length between their cell surface and their DNA, as well as their reliance on UV-labile pigments and proteins for their primary metabolic functions (Vernet 2000). This differential susceptibility is a clear impediment to the exploitation by picophytoplankton of the highest productivity environments nearest the ocean's surface. In some cases, these differences are severe enough to impact the ecological range of a species. For instance, incubations of field populations of *Prochlorococcus*, but not their sister group *Synechococcus*, were extremely vulnerable to UV radiation, experiencing ~100% mortality after less than 1 day's exposure to unfiltered sunlight at surface flux densities (Llabres and Agusti 2006; Llabres et al. 2010). This is strikingly similar to the relative effects of HOOH on these two genera (Morris et al. 2011), and it is difficult to tell how much of this mortality is caused by direct (e.g., genetic) and indirect (e.g., ROS formation) effects of UV.

Evidence of differential susceptibility to UV, particularly in the smallest size classes, warns against the assumption of simple relationships between irradiation and productivity in the mixed layer. This problem is exacerbated by the observation that other agents of phytoplankton mortality—viruses and protistan grazers—are themselves negatively impacted by UV (Jeffrey et al. 2000; Sommaruga et al. 1996). The lethal effects of UV on individual microorganisms, or even functional groups, are poorly studied, as most research has focused on the process level, e.g., primary or secondary production, or enzymatic activity of one kind or another. Attempts to model the impact of UV on plankton use "biological weighting functions" that sum the relative effects of different wavelengths on processes (Boucher and Prezelin 1996; Cullen et al. 1992). Similar techniques should also be applicable for studies of mortality, both in the field and with axenic cultures. Future experiments should focus on developing robust weighting functions for the major important taxa and/or functional groups amongst the oceanic plankton.

Chemical pollution

Both HOOH and UV are natural stressors that have likely been problematic for marine plankton as long as phototrophs have colonized the surface mixed layer (Liang et al. 2006; Mckay and Hartman 1991). Positive feedbacks from anthropogenic activity may increase the danger from these toxins (e.g., by ozone depletion), but these changes are quantitative rather than qualitative, and it may be safely assumed that the tools for surviving these changes are already extant in the planktonic metagenome. The pollution of the environment with exotic compounds produced by human industry, on the other hand, has the potential to create stresses that the majority of the plankton is ill equipped to tolerate. While much

attention has been given to the role of microbes in degrading exotic organic compounds, almost no research exists on what negative effects, if any, these substances have on the extant microbial community.

Most reviews of marine pollution focus specifically on nutrient pollution, largely caused by terrestrial run-off and human agriculture. The eutrophication brought about by these inputs has a number of problematic consequences for marine communities, mostly deriving from the induction of nuisance algal blooms. One major problem is the spread of hypoxic "dead zones" near many coastlines (Diaz and Rosenberg 2008). As oxygen concentrations decrease, multicellular aerobes (e.g., all animals) die off. Whereas one may expect that the microbial community will shift in favor of facultative or even obligate anaerobes, to our knowledge there is no evidence that obligately aerobic microorganisms are killed by the absence of oxygen. In contrast, trace metal loading associated with terrestrial run off is definitely toxic to marine microbes, including algae (Pinto et al. 2003), heterotrophic bacteria, and fungi (Babich and Stotzky 1985). Whereas heavy metals are a primary concern for human-impacted soils and estuaries (Babich and Stotzky 1985), there is early evidence that small elevations in metal concentrations may also impact oligotrophic marine flora adversely. For instance, a number of well-known studies show that addition of iron to oligotrophic seawater can dramatically increase productivity in some waters (e.g., Behrenfeld et al. 1996; Coale et al. 1998). On the other hand, if HOOH is added along with Fe, simulating rainfall deposition, the growth subsidy conferred by Fe is eliminated (Willey et al. 2004; Willey et al. 1999). These authors concluded that the primary effect of HOOH was to oxidize Fe(II) to Fe(III), which is significantly less soluble, and thus less bioavailable; however, an equally plausible explanation is that Fe-catalyzed radical production from HOOH (i.e., the Fenton reaction) killed phytoplankton at a rate sufficient to mask the growth benefits of the Fe. Similarly, the algicidal effects of the Fenton-reactive Cu are well known, and trace concentrations of Cu induce mortality in some ecotypes of *Prochlorococcus* (Mann et al. 2002). Thus, it is reasonable to think of metals as ROS-sensitizers, and to treat most cases of metal toxicity in the ocean as an extension of ROS toxicity.

The effects of other pollutants in the ocean (for instance, petroleum and estrogenic compounds) on microbes are poorly understood, despite large amounts of research into bioremediation of these substances. It is likely that many of these compounds are toxic to certain species but not others, and thus will have important effects on community structure, and perhaps, as a consequence, biogeochemical cycling. Whether they cause mortality or not is completely unknown. Much more research, both qualitative and quantitative, is required before the effects of these compounds can be placed in the larger context of understanding microbial death in the ocean.

Section 5. Interactions between grazing, viruses, and autologous death processes

It should be clear at this stage that much remains to be learned regarding death processes in the ocean. In particular, few studies have considered more than one death process at a time, and almost none have considered autologous death processes. One reason for this is that confounding methodological challenges face the researcher seeking to study these processes in the field. Specifically, several of these mortality sources present similar and interrelated symptoms: for instance, ROS can both cause and be caused by PCD, and can simultaneously, yet differentially, affect multiple trophic levels. Viral infection has been shown to cause HOOH release (Evans et al. 2006), and may trigger apoptotic features in phytoplankton (Lawrence et al. 2001) or co-opt the cell's apoptotic mechanisms during infection and lysis (Bidle et al. 2007). Experimental manipulations have further shown that viral infection of bacteria can increase in the presence of flagellate grazers (Simek et al. 2001; Weinbauer et al. 2007), and grazing of viral-infected cells may be greater than for noninfected cells (Evans and Wilson 2008).

To make matters worse, well-established methods for studying one mortality source are subject to interference by others. We have already mentioned the interference between PCD and necrosis measurements. To present another example, dilution assays are potentially vulnerable to interference by HOOH damage. The principle of the dilution assay (Landry et al. 1995) is that while prey growth rate is unaffected by dilution, the encounter-dependent grazing rate (and hence predation-based mortality) decreases exponentially, and therefore predation rates may be estimated from the difference between the apparent growth rates in diluted and undiluted cultures. However, if the prey organism requires "help" to tolerate an environmental stress such as HOOH (e.g., as in *Prochlorococcus*; Morris et al. 2011), then diluting out the pool of helpers may result in *increased* mortality, affecting the conclusions drawn from such an experiment. Clearly, it is important for future experiments to consider all possible sources of mortality, at a minimum using careful controls to reduce the interference of mortality sources other than the ones under investigation.

Section 6. Biogeochemical consequences of the mode of death

Grazing, viral lysis, PCD, and necrosis each result in different fates for the organic matter and nutrients contained within the microbial cell that is killed. Depending on the method of cell death, the cell's contents can either be incorporated into the biomass of higher trophic levels, or else released to be respired or recycled into biomass by heterotrophic microbes. The method of death also determines whether the cell contents are retained in the upper water column or sink out below the mixed layer. Each of these fates significantly affects the flow of organic matter and individual

nutrients in the open ocean, regulating the role of the oceans in global carbon and nutrient budgets. We can largely separate modes of death into three classes: lytic processes, which result in the release of large amounts of DOM; aggregative processes, which result in the production of bulkier material that sinks at varying rates into the sediments; and incorporation into biomass of higher trophic levels (Fig. 1). In the following sections, we consider how grazers, viruses, and autolysis are involved in these processes.

Grazing and biogeochemistry

Grazers' biogeochemical roles are highly dependent on their efficiencies at both ingesting and assimilating their prey (Fig. 5). A variable fraction of the prey killed by planktonic grazers is actually ingested, with the rest lost to the DOM pool by "sloppy feeding" (Roy et al. 1989). Sloppy feeding has been primarily assessed with particle feeders (e.g., setae filtering copepods), which have been shown to release > 50% of their prey as DOM (Moller et al. 2003), and therefore, stimulate bacterial productivity (Kamjunke and Zehrer 1999; Titelman et al. 2008). While sloppy feeding has not been addressed with other feeding modes, it also likely plays some role with phagotrophs, filter feeders, and raptorial feeders. Whereas separate from true "sloppy feeding," the discarded houses of appendicularians often contain uningested prey. These discarded mucous feeding webs comprise a rapidly sinking, particle-rich class of aggregates that may be responsible for significant export of organic matter to depth (Lombard and Kiorboe 2010; Robison et al. 2005; Vargas and Gonzalez 2004).

Like all organisms, grazer metabolism is not perfectly efficient, and only a portion of the food ingested is used for biomass accumulation or metabolic maintenance. The rest is either egested as fecal pellets (metazoans) or unused vacuole contents (protozoans). Mesozooplankton egestion or assimilation efficiencies (EE and AE, respectively) have been studied for many years, due to both the prevalence of fecal pellets in sediment trap material (Turner 2002) and the potential for assessing in situ ingestion rates from fecal production if AE is known. Mesozooplankton AE typically increases with food quality (Cowie and Hedges 1996; Mitra and Flynn 2007) and is inversely correlated to ingestion rate and prey concentration (Besiktepe and Dam 2002; Cowie and Hedges 1996; Liu et al. 2006; Mitra and Flynn 2007). Whereas it is highly variable (Conover 1966a; Liu et al. 2006), mean AE tends to be near 70% for carbon (Conover 1966a; Cowie and Hedges 1996; Liu et al. 2006; Pagano and Saintjean 1994), though AE's for nitrogen (Checkley and Entzeroth 1985; Cowie and Hedges 1996), phosphorus (Liu et al. 2006), and trace metals (Schmidt et al. 1999) may be different. Mesozooplankton taxa also exhibit differential abilities to digest prey types based on their chemical and structural defense mechanisms (Paffenhofer and Koster 2005). Mesozooplankton grazing and subsequent egestion thus leads to the efficient creation of large, rapidly sinking particles that are often depleted in nitrogen, amino acids, and other high quality food sources. By contrast, protozoan grazers typically do not egest large particles (Mostajir et al. 1998) and hence are generally considered to be part of the microbial loop that recycles, rather than exports, organic matter (Azam et al. 1983). However, the presence of mini-pellets (3-50 μm) in some sediment trap samples (Gowing and Silver 1985), suggests that such a simplistic interpretation of protozoan egestion may not always be applicable.

Mesozooplankton fecal pellets have sinking speeds that can vary from 10 m d^{-1} for small copepods (Turner 2002) to over a km d^{-1} for large salp fecal pellets (Caron et al. 1989; Madin 1982). They are at times a dominant component of shallow

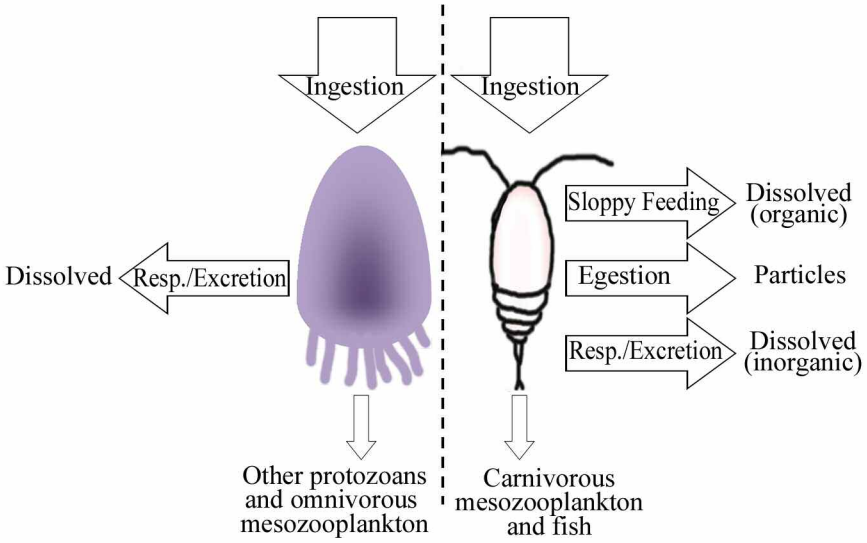

Fig. 5. Biogeochemistry of grazers. The primary biogeochemical role of protozoan (left) and metazoan (right) grazers.

sediment trap samples (Wassmann et al. 2000) and have even been traced from the surface ocean to benthic sediments at a depth of >4500 m (Pfannkuche and Lochte 1993). Despite their high sinking rates, mesozooplankton fecal production often exceeds the total export of particulate organic matter (POM) from the surface ocean (Landry et al. 1994), implying that some microbial degradation occurs before the pellets leave the euphotic zone. Fecal pellets can be lost due to ingestion or disruption by mesozooplankton grazers (Iversen and Poulsen 2007; Poulsen and Kiorboe 2005) and may also serve as hotspots for bacterial activity (Hansen and Bech 1996). Additionally, some phytoplankton taxa can survive ingestion and packaging into fecal pellets intact and remain viable if the fecal pellet is disrupted (Jansen and Bathmann 2007).

Digested prey can either be excreted/respired or incorporated into biomass. As a byproduct of metabolism, grazers excrete ammonium, urea, soluble reactive phosphorus, and DOM and also respire CO_2 (Frangoulis et al. 2005). Whereas the composition of the dissolved material released through egestion and sloppy feeding depends upon the chemical constituents of the prey and can contain refractory material, excretory products are primarily small, highly labile molecules that are rapidly taken up by phytoplankton. The stoichiometry of excretory products varies based on the taxonomic composition of the grazers (Atienza et al. 2006; Strom et al. 1997) and the stoichiometry of their prey (He and Wang 2008; Miller and Roman 2008; Saba et al. 2009) and impacts the relative stimulation of different phytoplankton and heterotrophic bacteria groups.

Metabolic rates (and hence respiration and excretion) are affected by several different environmental and taxonomic variables. Grazer gross growth efficiencies (GGE) on carbon are typically in the range of 20% to 30%, but highly variable. The general trend of similar GGEs across taxa and regions suggests that active metabolic rates (a function of ingestion rate) dominate over basal metabolic rates (a function of biomass). Despite this common trend of ~30% GGEs for carbon, it is important to note that the GGE of organisms depend on the substrate considered, and hence ratios of excretion to respiration can vary significantly (Landry 1993b). Specific metabolic rates also show a strong positive correlation with temperature (Ikeda et al. 2001; Makarieva et al. 2008; Steinberg et al. 2000) and a negative correlation with body size (Ikeda 1985; Ikeda et al. 2001).

Protozoan and metazoan grazers have traditionally been considered to have distinctly different biogeochemical roles, with protozoans contributing to a recycling loop while metazoans contribute to both export and energy transfer to higher trophic levels (Fig. 5; Azam et al. 1983; Ducklow et al. 2001). The dichotomy between microbial loop and 'classical' food chains arises both from the size differences between protozoan and metazoan grazers and the ability of most metazoans to package their egested material into rapidly sinking fecal pellets. Despite these stark differences, recent evidence suggests a gradient of ecological roles for grazers. Copepod fecal pellets are at times almost completely recycled within the euphotic zone (Poulsen and Kiorboe 2006; Viitasalo et al. 1999), whereas transfer of carbon from microzooplankton to metazoans can allow for transfer of carbon from protozoan grazing processes to metazoan fecal pellets (Stukel and Landry 2010; Stukel et al. 2011). When large phytoplankton are not abundant, mesozooplankton may derive most of their energy from protozoans (Fessenden and Cowles 1994), leading to the concept of a 'multivorous' food web (Legendre and Rassoulzadegan 1996) that blends the distinctions between the microbial loop and classical food chain.

Zooplankton affect phytoplankton and bacteria in both direct ways (top-down grazing pressure) and indirect ways (stimulation of production through nutrient regeneration). Top-down pressure by mesozooplankton on phytoplankton biomass has been shown in several regions (Goericke 2002; Landry et al. 2009; Olli et al. 2007), while protozoans may control the growth of picophytoplankton in the open-ocean and hence contribute to the development of high-nutrient, low-chlorophyll regions (Landry et al. 1997). On the other hand, grazers have been suggested to contribute to bottom-up processes that lead to increased production through regeneration of nutrients (Fernandez and Acuna 2003; Nugraha et al. 2010). Grazers can thus exert a negative pressure on prey biomass, while simultaneously increasing turnover times (and hence production) of their prey.

Viruses and biogeochemistry

Viral lysis results in the release of the host cell's contents to the POM and DOM pools, a process termed the 'viral shunt' because it shunts organic matter away from the classical grazing food web that leads to higher trophic levels (Fig. 6; Wilhelm and Suttle 1999). An estimated 6% to 26% of carbon fixed in surface waters flows through this viral shunt where it is then available to heterotrophic bacteria, forming a closed loop with respect to organic matter cycling (Wilhelm and Suttle 1999). Of the carbon released due to viral lysis of cultivated bacteria, 51% to 86% was determined to be dissolved combined amino acids, 2% to 3% was dissolved free amino acids, 2% to 3% was glucosamine, and 4% to 6% was the viruses themselves (Middelboe and Jorgensen 2006). Experiments with cultures (Middelboe et al. 2003) and natural samples (Middelboe and Lyck 2002) have shown that this organic matter released during cellular lysis is quickly metabolized by heterotrophic bacteria, increasing bacterial respiration and growth.

Attempts to directly quantify the fate of lysis products using radiolabeled viral lysates added to natural samples have been complicated by continual degradation and uptake of labeled material during preparation of the labeled lysate (Noble and Fuhrman 1999). While acknowledging that their measured rates may be underestimates, Noble and Fuhrman (1999) determined that lysis products turn over relatively rap-

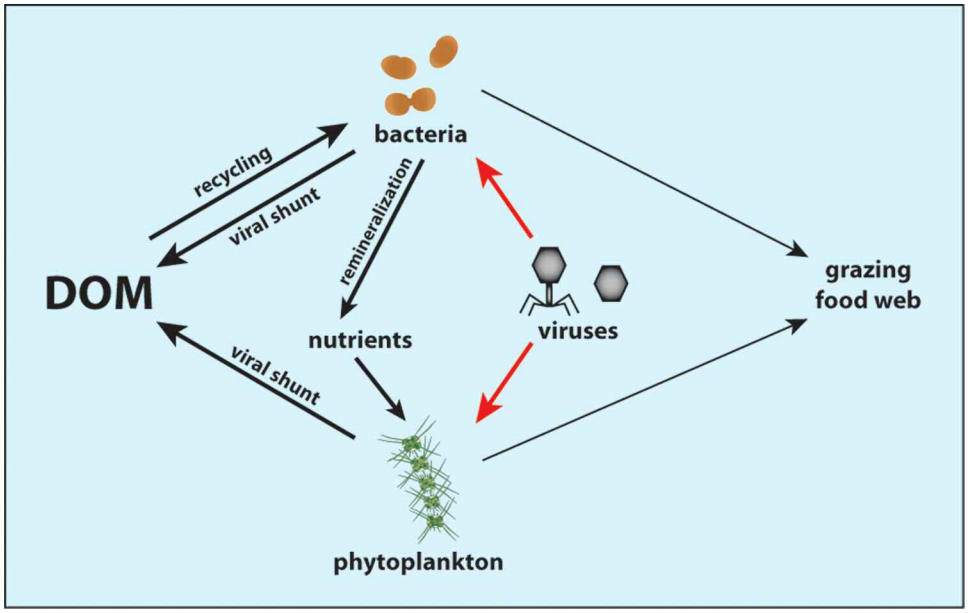

Fig. 6. Effect of viral lysis (in red) on shunting organic matter away from the grazing food web and toward the DOM pool where it can then be recycled by heterotrophic bacteria, resulting in remineralization of nutrients for uptake by phytoplankton.

idly, within 1-4 days. They also found that high molecular weight DOM in lysis products was broken down into low molecular weight DOM more rapidly in waters with greater microbial activity, but that labeled phosphorus lysis material was taken up more rapidly (<7 h) in oligotrophic phosphorus-limited waters. Along these lines, the cellular DNA released through viral lysis is thought to be extremely important to nutrient dynamics because of its relatively high nitrogen and phosphorus content, and has the potential to supply a large portion of microbial phosphorus demand in oligotrophic and phosphorus-limited waters (Brum 2005; Holmfeldt et al. 2010; Riemann et al. 2009).

Investigations have also shown that viruses can have considerable effects on the flow of organic matter and nutrients between bacteria and phytoplankton. Sharp increases in bacterial abundance and production during the decline of phytoplankton blooms have been extensively reported (e.g., Bratbak et al. 1998; Brussaard et al. 1996). Viral lysis of phytoplankton blooms essentially converts algal biomass to DOM that then fuels bacterial production (Bratbak et al. 1998). The nutrients in cellular contents released via lysis can be remineralized by heterotrophic bacteria (Fig. 5) or broken down photochemically, thus relieving nitrogen and phosphorus limitation of phytoplankton (Gobler et al. 1997), and providing highly bioavailable forms of iron for phytoplankton uptake (Poorvin et al. 2004).

The overall effect of viral lysis of bacteria or phytoplankton in the ocean, which has been reviewed several times (e.g., Brussaard 2004; Fuhrman 1992, 2000; Wilhelm and Suttle 1999; Wommack and Colwell 2000), is a conversion of living cells to DOM, followed by bacterial oxidation of the released organic matter and regeneration of inorganic nutrients, which are available for uptake by phytoplankton (Fig. 6). Thus, while viruses cause mortality of bacteria and phytoplankton, they also promote production of non-infected bacteria and phytoplankton, increasing biomass and productivity overall. This conversion of cellular contents to DOM decreases the flow of organic matter to higher trophic levels and importantly has the effect of retaining organic matter and nutrients in the upper water column (Fig. 1), with the caveat that viral lysates may promote formation of aggregates that can sink out of the upper water column (Peduzzi and Weinbauer 1993; Proctor and Fuhrman 1991).

Despite the considerable research devoted to this topic, the fate of cellular contents released as a result of viral lysis still remains an open field of research in oceanography. Topics requiring further study include determining (1) what portion of the organic matter released from viral lysis is bioavailable and what portion is refractory, (2) what portion is broken down by photochemical reactions, (3) what portion is respired by heterotrophic bacteria versus incorporated into bacterial cells, (4) do these various pathways for viral lysates change in different environments and (5) if so, is viral lysis more central to nutrient and organic matter cycling in certain environments? The answers to these questions will go a long way in refining our understanding of the roles and importance of viruses in marine environments. The first step will be to develop reliable methods to track the fate of cellular contents released due to viral lysis.

Another aspect of viral infection that distinguishes it from other forms of mortality is that viral infection can alter the flow of carbon and nutrients before cellular death. The

genomes of marine viruses can contain auxiliary metabolic genes (AMGs; *sensu* Breitbart et al. 2007), sometimes referred to as "host genes," which encode for cellular machinery involved in nutrient cycling, carbon metabolism, and even photosynthesis (Mann et al. 2003, 2005; Millard et al. 2009; Sullivan et al. 2005, 2010; Weigele et al. 2007). These AMGs can be expressed during infection (Clokie et al. 2006; Dammeyer et al. 2008; Lindell et al. 2005, 2007; Sharon et al. 2007), in some instances enabling greater viral production (Bragg and Chisholm 2008; Hellweger 2009). The best-studied AMGs are the core photosynthesis genes, *psbA* and *psbD*, which are widespread within cyanophage lineages and are distinct from the homologues in their cyanobacterial hosts (Sullivan et al. 2006). Approximately 60% of the *psbA* genes in surface waters are of viral origin, suggesting that virus-enhanced photosynthesis may be a significant contributor to oceanic PP (Sharon et al. 2007).

This emerging facet of viral ecology has the potential to substantially alter our view of the effects of viral infection in the cycling of organic matter and nutrients in marine environments. The challenge now is to determine what portion of nutrient uptake and carbon fixation, and metabolism is a result of the expression of viral AMGs during infection. Following the example of AMGs that encode for core photosynthesis genes in cyanophages, this will require (1) extensive evaluation of the types of AMGs present in marine viral genomes using sequenced genomes from cultivated viral isolates (e.g., Sullivan et al. 2005), (2) examination of viral genomes and metagenomes to investigate the range of diversity of these viral AMGs in natural samples (e.g., Sullivan et al. 2006), (3) determination of the portion of the total gene abundance that is of viral origin (e.g., Sharon et al. 2007), (4) examination of transcripts to determine if the viral AMGs are expressed in culture (e.g., Lindell et al. 2005) and in natural samples (e.g., Sharon et al. 2007), and (5) studies of the transcriptome of natural samples to determine the expression of individual viral AMGs relative to the expression of the homologues of these genes in microorganisms, resulting in an estimate of the total portion of the process encoded for by the gene that is a result of viral infection. The presence of viral AMGs may be related to environmental conditions. For example, phosphorus-related AMGs may only be present in viruses from areas with low nutrient concentrations (Sullivan et al. 2010). Therefore, the presence and expression of these viral AMGs will need to be evaluated over space and time to determine their contribution to total oceanic nutrient and organic matter fluxes.

Biogeochemistry of PCD and necrosis

The study of PCD and necrosis in the ocean is in its infancy. While several decades' worth of research has been invested in understanding the impact of UV on marine communities, we still know few specifics of how this stress affects microbial populations. The other sources of autologous mortality that we discussed earlier are even more poorly understood. Still, based on observations of cultures and other aquatic communities (e.g., freshwater), we can make certain predictions about how autologous death processes will differ from viral lysis and grazing in their effects on biogeochemical processes.

Regarding PCD, we must first stress that no unambiguous observations of phytoplankton PCD in the field are known. If, however, PCD occurs in algal blooms, it might have profound consequences for the fate of bloom PP. One characteristic of apoptotic cells as opposed to necrotic ones is that lysis is delayed during PCD, sometimes for days (Bidle and Falkowski 2004). Many bloom-forming species are large, motile, or both, and death without lysis may enhance sinking rates and increase export of bloom PP to deep waters. In contrast, viral bloom termination likely regenerates most of the PP into the microbial loop, minimizing loss. Still, it remains completely unknown what effect PCD might have on the fate of PP, and it is an important topic for future workers in algal PCD to pursue.

UV and HOOH can be treated together, since their biogeochemical effects are similar. Both kill by lysis (see above) and thus, like viruses, contribute to the DOM pool, feeding the microbial loop and regenerating nutrients to productive surface waters (Fig. 1). Also like viruses, not all taxa are equally vulnerable to these stresses, leading to an uneven distribution of mortality that is quite distinct from the typical effects of grazing. However, susceptibility to UV and HOOH is ecologically very different from susceptibility to viruses: rarity is no defense against them, and vulnerability is not subject to predator/prey "arms race" dynamics. To the contrary, vulnerability is often an evolutionary trade-off for other traits that are adaptive (e.g., small cell size, streamlined genomes). For instance, smaller cells are more vulnerable to UV (Vernet 2000), but are also superior competitors for limiting nutrients due to their higher surface area to volume ratio. As long as the growth benefit of small size outweighs the cost of increased vulnerability, then vulnerability is likely to spread in the population (Morris et al. 2012).

In addition to increasing nutrient flow to the DOM pool, UV and HOOH are also potentially capable of affecting the bioavailability of released nutrients. In particular, much attention has been given to the importance of HOOH and UV photochemistry in controlling Fe speciation and degrading DOM. Fe is very scarce in the open ocean and potentially limits productivity in some regions (Behrenfeld et al. 1996). Whereas Fe(II) is much more soluble than Fe(III) at the pH of seawater, it is rapidly oxidized to Fe(III) in the presence of O_2 (Coale et al. 1998). In a dark, oxygenated ocean, all free Fe would probably exist as Fe(III). Fe(III) can be oxidized to Fe(II), however, by photochemical reactions involving DOM (i.e., the photo-Fenton reaction; Zepp et al. 2002), which has the effect of increasing the pool of soluble Fe, with potentially profound implications for PP (Coale et al. 1998).

HOOH and UV may also be important in transformations of DOM. The DOM pool is highly heterogeneous, including

species that are labile due to their predictable structure (e.g., nucleic acids and proteins) or their relative simplicity (e.g., straight-chain polysaccharides, low molecular weight acids), whereas other compounds are extremely resistant to degradation due to their complex and/or energetically stable structures (e.g., aromatic compounds, lignin) (Sulzberger and Durisch-Kaiser 2009). This latter DOM fraction can have residence times in seawater of 10^3 years or more, longer than the mixing time of the ocean (Kirchman 2008). As described above, the interaction of DOM with solar radiation can generate ROS, and these oxidants are theoretically capable of breaking the more recalcitrant components of the DOM pool into smaller, potentially more labile compounds (Mopper et al. 1991).

Thus, mortality induced by HOOH or UV likely not only feeds the DOM pool, but also probably accompanies chemical reactions that improve the bioavailability of that pool. It is possible that these stressors could simultaneously suppress some taxa while stimulating others. Unfortunately, our current understanding of necrotic death in the field is extremely limited, and many technically challenging experiments are necessary before any firm conclusions may be reached regarding the magnitude and sign of the effects of these forces on marine microbes. There is hope that technological and methodological improvements may make such studies more feasible. For instance, single-cell genomics can address the prevalence of genetic lesions in different populations, and the types of lesions may suggest whether damage from ROS or UV are responsible. Increasingly sensitive methods for studying metabolomics in the field should be able to detect telltale signs of oxidative stress, such as carbonylated proteins and lipid peroxides, and more sensitive mass spectrometric methods may be targeted against the oligotrophic DOM pool to assess what, if any, effects oxidants have on its lability. Last, improved methods for obtaining axenic cultures of fastidious, streamlined, oligotrophic organisms (Connon and Giovannoni 2002; Morris et al. 2011; Nichols et al. 2010) should allow novel insights into the vulnerabilities that influence the differential success of these organisms in the field as opposed to the laboratory. The importance of these studies is high in this period of accelerating global change, as anthropogenic effects (e.g., ozone depletion, heavy metal pollution) may exacerbate each of these mortality sources.

Section 7. Modeling death

Ecological and biogeochemical models of pelagic bacteria and phytoplankton are typically some derivative of Nutrient-Phytoplankton-Zooplankton (NPZ) type models (Franks et al. 1986; Riley et al. 1949), with a varying number of state variables. These models are all explicitly concentration-based models; they keep track of the concentration of organisms within a parcel (or parcels) of water. This is an important distinction, as it implies that they need not explicitly model death. They need only estimate the net balance between growth and death. With improved computing power, individual based models are increasingly being used to model mesozooplankton concentrations in particular. The continued evolution and expansion of these models to the microbial realm (e.g., Cianelli et al. 2009; Hense 2010) will require explicit functions for phytoplankton and bacterial death. In this chapter, we will consider model treatment of death in phytoplankton and bacteria, with a particular focus on prevalent NPZ-type models.

The phytoplankton 'mortality' term is a ubiquitous parameter in NPZ models that serves a purely pragmatic role. It is typically parameterized as a specific loss rate that returns phytoplankton biomass to either the particulate detritus or the dissolved phase. In physically coupled models, they are necessary to prevent phytoplankton persistence at low concentrations in deep water where grazers may not be present. Nevertheless, they bear little resemblance to the specific loss mechanisms described above. The parameterization of this term is usually based not on any knowledge of necrosis or PCD rates, but instead is set at levels that maintain a healthy population in the surface ocean. This mortality term has been alternately characterized as true protistan mortality, respiration, or mixing/transport out of the euphotic zone. Whereas not explicitly incorporated in typical NPZ models, the effects of nutrient depletion-induced PCD and UV, PAR, or HOOH-induced necrosis may be implicitly included within the growth parameters that approximate the net growth of the organism (growth minus necrosis). For instance, many models include a photoinhibition term (Franks 2002) based on production versus irradiance measurements that incorporate necrosis due to UV, PAR, and irradiance-generated ROS. The importance of explicit inclusion of necrosis and PCD in ecological and biogeochemical models is still largely unknown due to the paucity of experiments that have shown these processes to differentially affect pelagic communities and the nature of in situ growth rate measurements that implicitly include necrotic effects when measuring net growth.

Zooplankton grazing has been represented in NPZ models in many different ways. At its most basic, grazing is parameterized as a product of grazer concentration, prey concentration, and some functional feeding response (FFR) that is specific to the grazer-type and a function of prey concentration (Gentleman et al. 2003). Typically, a saturating function is used to reflect the handling time limitation of zooplankton at high food concentrations. FFRs sometimes include a feeding threshold, which allows persistence of low populations of phytoplankton. In box and 1-dimensional models, FFRs can determine the stability of the ecosystem (Franks et al. 1986; Murray and Parslow 1999). Early NPZ type models (e.g., Steele and Frost 1977) parameterized their zooplankton grazing terms based on copepods and other metazooplankton, though the discovery that protozoans dominate grazing in the pelagic has led to a shift toward zooplankton with a faster response time and higher maximum specific grazing rate. Many NPZ models, particularly those that

attempt to represent both a blooming and a non-blooming type of phytoplankton, now include two zooplankton state variables (Aumont et al. 2003; Kishi et al. 2007). A protozoan group will grow rapidly and crop bacteria-like phytoplankton, whereas a mesozooplankton group grows more slowly allowing large diatoms to temporarily escape grazing pressure. Allometric NPZ type models now incorporate continuous or semi-continuous size-spectra, thus allowing for a full range of size-classes of zooplankton that graze appropriately sized prey (Poulin and Franks 2010). Some models also use zooplankton FFRs that allow the zooplankton community to switch to preferential grazing on the most abundant prey group, thus simulating a dynamic zooplankton community adapted to local conditions and also allowing increased diversity of the prey community (e.g., Fasham et al. 1990).

In addition to these models that explicitly model the grazer community, some concentration-based models (particularly high spatial resolution global models and long-term climate simulations) use implicit grazing losses for phytoplankton (e.g., Dunne et al. 2005). Such models often assume a covariance of prey and predator and hence model grazing as a product of prey concentration squared and an FFR. They are particularly applicable in situations when community change rates are slow relative to zooplankton turnover times, but are likely inappropriate during bloom situations or when studying systems with slow-growing grazers.

Traditional multi-compartment NPZ type models contain, at most, a few phytoplankton functional groups and typically only a single (if any) heterotrophic bacterial group. Thus these compartments must be treated as highly aggregated groups comprised of many different species. Because viruses are typically believed to play a greater role in maintaining diversity (which is not assessed by these models) than in controlling abundances, they have often been neglected in models. Miki et al. (2008) has produced the most comprehensive model to date regarding the effects of viruses in marine environments, with parameters including mortality of bacteria due to viral lysis and grazing, regeneration of nutrients and DOM as a result of viral lysis, host specificity of viruses infecting different functional groups of bacteria, the effects of lysogeny and the development of viral-resistance in bacteria, and indirect relationships between viruses and protozoans.

This model (Miki et al. 2008) and others designed to explicitly model viral activities (Middelboe et al. 2001; Thingstad 2000) suggest several important avenues of research. For instance, empirical tests of the models should be undertaken using simplified communities of cultivated viruses, bacteria, and protozoans. Models should also be expanded to include phytoplankton, with both the lysis of phytoplankton by viruses and their uptake of inorganic nutrients regenerated from DOM in lysates through bacterial activity. Application of models to field situations where viral lysis is suspected to be important, as has been attempted for *Phaeocystis globosa* bloom dynamics (Ruardij et al. 2005), is also an important step in improving our ability to correctly incorportate viruses into models. While simplified mathematical descriptions of viral effects may be sufficient for traditional NPZ-type models, the inclusion of more accurate viral dynamics will be increasingly important for more complex and inclusive models, such as emergent properties type models (Follows et al. 2007), which can contain from tens to potentially thousands of taxa and hence may be used to generate realistic diversity patterns. Additionally, much work needs to be done to incorporate the newly recognized importance of viral AMGs into the overall flow of carbon and nutrients through food webs.

Thus, while modeling grazing has been an active field for years, there is much room for improvement in the inclusion of necrosis and viral lysis in models. The assumption that non-grazer related mortality can primarily be subsumed into a lower growth rate neglects the role of these processes in generating POM and DOM. Furthermore, each mode of death has a different response form. Viral lysis is likely related to the square of a taxon's abundance, grazing is related to the product of prey and predator abundance, and necrosis is directly related to the taxon's abundance, but may also be related to abiotic factors such as nutrient concentration, [HOOH], or ambient radiation, each of which may themselves vary as a function of biotic or abiotic parameters. Models using these different responses can potentially be combined with mesocosm experiments and detrital live/dead stains (Verity et al. 1996) to tease apart the relative importance of necrosis, grazing, and viral lysis in phytoplankton and bacterial death. Vitally, experiments must be crafted to simultaneously measure multiple sources of mortality.

Section 8. Simultaneous measurements of multiple modes of death

There have been several studies comparing the mortality of bacteria or phytoplankton from grazing and viral lysis in marine environments. These rates are generally quantified using separate methods, although as we mentioned earlier, parallel dilution series with water free of grazers and water free of grazers and viruses have been used to measure both together (Evans et al. 2003). The majority of comparative studies show that grazing mortality exceeds viral lysis (Baudoux et al. 2006, 2008; Bettarel et al. 2002; Boras et al. 2010; Choi et al. 2003; Evans et al. 2003; Kimmance et al. 2007; Umani et al. 2010) except occasionally during the collapse of algal blooms (Baudoux et al. 2006). Exceptions to this rule are presented by Wells and Deming (2006), who found that viral lysis was more important in deep Arctic waters, and Hwang and Cho (2002), who found that the two rates were comparable in the oligotrophic open ocean.

Grazing rates are not only typically more rapid, but they also tend to be more variable than viral lysis (Boras et al. 2009; Choi et al. 2003; Umani et al. 2010). To understand the overall impact of these two factors, long time series studies are necessary. Boras et al. (2009) conducted a comprehensive 2-year

time series in which mortality of bacteria from both grazing and viral lysis was quantified in oligotrophic coastal waters of the Mediterranean Sea. They found that, on average, the amount of bacterial production consumed by grazers exceeded bacterial production lysed by viruses in the first year, but that viral lysis caused more loss of bacterial production than grazing in the second year. Averaged over the 2 y, 3.12 ± 3.35 μg C L^{-1} d^{-1} was transferred to higher trophic levels through grazing of bacteria and 2.5 ± 2.9 μg C L^{-1} d^{-1} was converted to DOM through viral lysis of bacteria. Further studies like this in differing oceanic regions are needed to obtain a clearer view of the overall magnitude and variability of grazing and viral lysis so that food web models can accurately depict the flow of organic matter in marine environments.

Furthermore, mortality due to grazing and viral lysis does not always account for total mortality of bacteria and phytoplankton (e.g., Boras et al. 2009; Guixa-Boixereu et al. 1999; Hwang and Cho 2002). One study quantified grazing, viral lysis, and total cell lysis (measured using the dissolved esterase assay) of *Phaeocystis globosa* during spring blooms, with the interpretation that the difference between viral lysis and total cell lysis could be a result of autolysis (Baudoux et al. 2006). The authors found that viral lysis was the dominant cause of cellular lysis in the alga, but also found that estimates of viral lysis often *exceeded* total estimated cell lysis, a problem that they suggested was caused by comparing results from the two methods. This study highlights the need for rigorous methodological evaluation when interpreting results from experiments that use multiple methods to quantify various forms of mortality, since each method has different limitations and biases.

Incorporating measurements of mortality due to autolysis and necrosis into studies of grazing and viral lysis of bacteria and phytoplankton in marine environments is also necessary to determine what portion of planktonic mortality these account for, under which conditions they are most important, and to balance models of carbon flux through these systems. In particular, these factors are probably important for the picoplanktonic inhabitants of the oligotrophic oceans. Whereas these regions have low productivity per unit area, they are so vast that, collectively, they comprise a sizable fraction of total global PP. Thus, it is crucial to understand how highly abundant species like *Prochlorococcus* live and die in the ocean. We suggest that traditional dilution methods may be modified to incorporate tests of HOOH and UV mortality at the same time they measure rates of grazing and viral lysis. First, the spectrum of solar irradiation impacting dilution bottles may be manipulated based on the material used to make the bottle and/or the incubator: Plexiglas for PAR only, Mylar for PAR + UVA, or UVT plastic for the full solar spectrum (e.g., Gerringa et al. 2004; Llabres et al. 2010). Second, HOOH exposure may be limited either by adding purified catalase to remove HOOH, or by diluting the community in the presence of a constant concentration of cultured, HOOH-scavenging "helper" bacteria (Morris et al. 2008); in this way, direct UV damage may be separated from indirect damage mediated by HOOH and other ROS. Third, samples may be collected for detection of specific biomarkers of UV or ROS damage (e.g., pyrimidine dimers, lipid peroxides, or carbonylated proteins), potentially in a taxon-specific manner (e.g., by coupling FISH probes to other detection methods). It may also be useful to assay for caspase activity, particularly in higher productivity waters or near blooms where PCD might be important.

Section 9. Conclusions

Current estimates show that while grazing generally causes the majority of microbial mortality in the open ocean, viral lysis also results in a significant (and highly variable) fraction. Further, PCD and necrosis, though poorly studied to date, are also likely important in some circumstances. The distinction between these vectors of death is important because each results in a different fate for the cellular material of the dead microorganism. Grazing results in incorporation of that cellular material into higher trophic levels, conversion of the organic matter to DOM as a result of sloppy feeding, and the egestion of fecal pellets that may contribute to the DOM pool or be exported to the deep ocean. In contrast, viral lysis and autologous cell death result in conversion of the cellular biomass to DOM, thereby reducing the amount of organic matter flowing to higher trophic levels through grazing and increasing recycling of organic matter and nutrients within the microbial loop.

As suggested in this chapter, further research is needed to explore each of these mortality factors individually. Also, we suggest that simultaneous estimates of all three processes, under varying conditions and across time and space, are necessary to evaluate their relative magnitude and to improve models of organic matter and nutrient flow in the oceans. We hope this chapter will stimulate careful evaluation of current methodologies used to estimate the causes of mortality, as well as the development of novel methods to investigate them. Furthermore, interactions between each cause of death remain an open field of investigation. Microorganisms are integral to the function of Earth's oceans, and further investigation into the various forms of microbial death is vital to increase our understanding of their various roles in marine environments, as well as refine our knowledge of the broader importance of the oceans in global processes.

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