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Indirect interactions in the microbial world: specificities and similarities to plant–insect systems

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Abstract Trophic interactions between bacteria, viruses, and protozoan predators play crucial roles in structuring aquatic microbial communities and regulating microbe-mediated ecosystem functions (biogeochemical processes). In this microbial food web, protozoan predators and viruses share bacteria as a common resource, and protozoan predators can kill viruses [intraguild predation (IGP)] and vice versa, even though these latter processes are probably of less importance. However, protozoan predators (IG predator) and viruses (IG prey) generally occur together in various environments, and this cannot be fully explained by the classic IGP models. In addition, controlled experiments have often demonstrated that protozoan predators have apparently positive effects on viral activity. These surprising patterns can be explained by indirect interactions between them via induced trait changes in bacterial assemblages, which can be compared with trait-mediated indirect interactions (TMIIs) in terrestrial plant–insect systems. Here, we review some trait changes in bacterial assemblages that may positively affect the activities and abundance of viruses. It has been suggested that in bacterial assemblages, protozoan predation may enhance growth conditions for individual bacteria and induce both phenotypic trait changes at the individual (e.g., filament-forming bacteria) and group level as a result of changes in bacterial community composition (e.g., species dominance). We discuss the specificities of aquatic microbial systems and attempt find functional similarities between aquatic microbial systems and terrestrial plant–insect systems with regard to TMII function.

Keywords Aquatic microbial community · Bacteria · Bacteriophage · Trait-mediated indirect interactions · Predator–parasite links

Introduction

Microorganisms play essential roles in the structure, maintenance, and overall functioning of all life and ecosystems on Earth. For many years, general ecology has not focused on microbial systems, except when using them as model systems to test some general theories in population dynamics, evolutionary dynamics, and pattern formation. On the other hand, attempts have been made to apply classic theories from general ecology (e.g., theories about competition and prey–predator dynamics) to microbial ecology to improve our understanding of microbial systems. Application of the latest theories from general ecology, e.g., indirect interactions (Ohgushi 2007), metacommunity (Leibold et al. 2004), and network perspective (Solé and Bascompte 2006) to microbial systems is also expected to improve our understanding of microbial interactions. Advances in environmental microbiology and microbial ecology can, in turn, be expected to enable ecologists to revise general ecological theories. So, feedback between these two fields holds the promise of considerable advances in our understanding both of the macroscopic worlds that we can see and the microbial worlds that we cannot see.

In this paper, we focus on microbial interactions in aquatic systems (lakes and oceans), especially among

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bacteria, viruses, and protozoan predators, as they are the dominant groups in terms of abundance and biomass (Suttle 2007). Typically, bacterial abundance in the ocean is of the order of 10^6–10^7 individuals/ml, and the relative abundance of viruses, bacteria, and heterotrophic nanoflagellates [(HNF), the dominant group of protozoan predators of bacteria] is $10{:}1{:}10^{-3}$ (Noble and Fuhrman 1998; Tanaka and Rassoulzadegan 2002). This implies that frequent interactions occur between organic and inorganic substrates, bacteria, viruses, and protozoan predators, which act as strong trophic links connecting phytoplankton-derived organic resources to the higher trophic levels via heterotrophic bacteria, and form the basis for the important biogeochemical roles played by microbial food webs in aquatic systems (referred to as the microbial loop by Azam et al. 1983, and the viral loop by Bratbak et al. 1992). In addition, viral lysis and protozoan predation are two major sources of bacterial mortality (Fuhrman and Noble 1995; Fuhrman 1999; Strom 2000; Wommack and Colwell 2000), although their relative contribution to bacterial mortality may vary greatly among and within ecosystems (Weinbauer and Höfle 1998; Colombet et al. 2006; Lymer et al. 2008) or seasons (Jacquet et al. 2005; Pradeep Ram et al. 2005). Thus, viruses and protozoan predators would exert strong selection pressures on bacteria and lead to the counter-evolution of viruses and protozoan predators. Interactions between microbes contribute to the evolution and maintenance of microbial diversity, and in turn, diversified interactions could determine the structure of microbial communities and regulate microbe-mediated biogeochemical processes (ecosystem functions). This is why it is crucial to understand the complex interactions between microbes in natural ecosystems.

We first briefly summarize the direct trophic interactions between these 3 major components. We next review density-mediated indirect interactions (DMIIs) and demonstrate the importance of trait-mediated indirect interactions (TMIIs) in explaining some "surprising" phenomena that cannot be explained from the standpoint of DMII. We then discuss the similarities and dissimilarities of TMIIs in the microbial world compared with plant-based TMIIs in terrestrial systems. We hope that the TMII concept will shed new light on the microbial world and that newly discovered aspects of microbial TMIIs will also contribute to the development of new theories of TMII in general ecology.

Direct trophic interactions

Bacterivory by HNF and lysis by viruses are 2 major causes of bacterial mortality in aquatic systems (Wommack and Colwell 2000; Weinbauer 2004). Many studies suggest that grazing loss is roughly equal to bacterial growth in oligotrophic (low-productive) systems and that HNF predation alone cannot explain the balance between bacterial growth and mortality in more eutrophic (productive) systems (Strom 2000; Domaizon et al. 2003). It is suggested that both predation and viral lysis make major contributions to bacterial mortality in such productive systems (Lymer et al. 2008). These two processes are, in fact, only part of the direct trophic interactions that occur between bacteria, viruses, and HNF.

As viral infection is responsible for severe bacterial mortality, it is not surprising to find that bacteria have evolved various strategies to resist viral attack (reviewed by Weinbauer 2004). In addition to strategies for escaping from recognition by viruses (Bohannan and Lenski 2000) and immune-like responses after infection (Lenski 1988; Weinbauer 2004), the constitutive or induced production of ectoenzymes is known to destroy the capsid proteins of free-living viral particles (Noble and Fuhrman 1997). Although it is not clear whether ectoenzymes are produced to resist viruses or whether this is just a side effect of ectoenzymes produced to use other organic particles such as growth resources in the surrounding environment, they do contribute to eliminating viruses (Weinbauer 2004; Middelboe 2008). HNF predation is also known to be one of the processes behind viral loss. Although the direct predation rate of HNF on free-living viral particles accounts for <10% of HNF predation on bacteria (i.e., Suttle and Cheng 1992; Gonzáles and Suttle 1993; Bettarel et al. 2005), it could have nonnegligible effects on the size distribution of viral assemblages (Demuth et al. 1993; Weinbauer 2004). In addition, it is also worth noting that viruses within bacterial host cells are killed indirectly by HNF predation of infected bacterial cells (e.g., Pinheiro et al. 2007), and this effect has been incorporated into theoretical models (Binder 1999; Miki and Yamamura 2005). It is also interesting to note that infected bacterial cells seem to be more vulnerable to predation than uninfected cells (Evans and Wilson 2008).

There is also a little evidence suggesting that other types of direct trophic interactions may also occur. Only one study so far reported isolation and characterization of a double-stranded DNA (dsDNA) virus infecting an HNF (*Bodo* sp.) from a natural marine system (Garza and Suttle 1995), but virus-like particles similar to this dsDNA virus were recently observed in the cell of an HNF species, *Cafeteria roenbergensis* (Massana et al. 2007). Bacteria that predate other bacteria, such as *Bdellovibrio* spp., are also known to exist (Martin 2002; Yair et al. 2003) and have been shown to be highly diverse (Snyder et al. 2002) and widely distributed in soil and aquatic environments (Yair et al. 2003). La Scola et al. (2008) discovered a hyperparasitic virus whose host is not a bacterium but a virus infecting an amoeba (*Acanthamoeba castellanii*).

This small virus (50 nm), called Sputnik, does not multiply when inoculated into *A. castellanii*, but does grow in *A. castellanii* that has already been infected with another virus called *A. polyphaga* mimivirus or mamavirus (APMV). Coinfection with Sputnik results in a significant decrease in the production of infective new particles of APMV, which implies that Sputnik is a parasite of APMV and has therefore been described as the first virophage. Another important point to note is that protozoa within the HNF size range (5–20 μm) are unlikely to always be bacterivorous. Recent molecular analyses revealed the functional diversity of HNF-sized protozoa, which include parasites on many types of hosts (Lefèvre et al. 2007; Lepère et al. 2008; Gleason et al. 2008).

Density-mediated effects

Unexpected diversities in the direct interactions among microbes imply the existence of complex interaction networks involving density-mediated indirect interactions. Here, we focus on three trophic interactions that probably occur most often, which consist of one of the typical trophic modules, intraguild predation (IGP), between bacteria, viruses, and HNF (Fig. 1a). HNF and viruses compete for bacteria as a common resource, and HNF predate viruses both directly by predation on free-living viruses (omnivorous IGP), and indirectly via predation on infected bacteria (coincidental IGP). Ecological theory predicts that the coexistence of IG predators and IG prey species will be achieved only under limited conditions (Holt and Polis 1997), which implies that the coexistence of HNF and viruses is not self-evident. In addition, increased productivity and an increased growth rate of the common resource (in this case, bacteria) are predicted to have a negative effect on IG prey (viruses) via DMII. In our case, bacteria, HNF, and viruses act as initiators, transmitters, and receivers of DMII, respectively (Fig. 1b). It is predicted that any increase in transmitter density (HNF) will reduce the density of the receiver (viruses). However, in aquatic systems, except for extreme environments (e.g., anoxic environments, Weinbauer and Höfle 1998; Colombet et al. 2006), the coexistence of HNF and viruses is so widespread that no microbial ecologists have taken any interest in the mechanism of their coexistence. In addition, some studies have suggested that the increased productivity results in higher contribution of viruses to bacterial mortality in marine (Steward et al. 1996) and freshwater (Weinbauer et al. 2003) systems, whereas other studies suggest the opposite trends (Lymer et al. 2008) or no clear correlation (Bettarel et al. 2004), typically in freshwater systems. Although it is proposed that the latent period of viruses, which determines the strength of coincidental IGP, is one of the key factors in determining the shift in the contribution of viruses (IG prey) along the trophic gradient (Miki and Yamamura 2005), this IGP model cannot explain the differences between the above results. This makes it reasonable to suggest that some additional factors must also be regulating the behavior of this IGP module and that they have not been taken into consideration in module-based models of IGP. A recent theory in ecology suggests that the interaction network outside this IGP module (e.g., higher trophic levels and alternative resources for IG prey) contributes to the persistence of the focal IGP module (Holt and Huxel 2007; Namba et al. 2008), and just such an external stabilizing structure is found in food webs in marine systems (Kondoh 2008). However, even if the microbial IGP module is isolated from external networks in

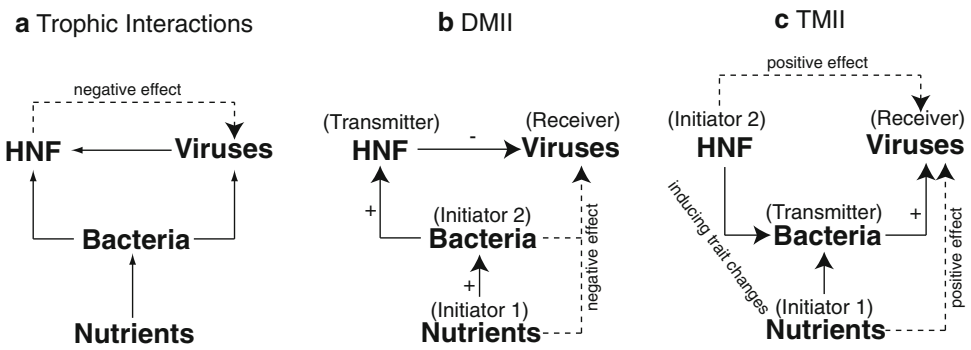

Fig. 1 Direct and indirect density-mediated and trait-mediated interactions among bacteria, viruses, and heterotrophic nanoflagellates (HNF). **a** Major trophic interactions among three functional groups: due to density-mediated effects (exploitative competition and predation), HNF can be expected to have a negative effect on viruses. **b** Density-mediated indirect interactions (DMIIs): the intraguild predation (IGP) module has 3 components, and ecological theory predicts that the nutrient level, which corresponds to the productivity of the system, and the basal resource, bacteria (initiators), have indirect negative effects on viruses (receiver) via an increase in the abundance of HNF (transmitter). **c** Trait-mediated indirect interactions (TMIIs): Enhanced growth of bacteria as a result of increased nutrients, eutrophication, or HNF predation (initiators), and shifts in composition in the bacterial assemblage, cause trait-changes in bacteria (transmitter) at the individual and group levels. In turn, these changes have a positive effect on the viruses (receiver)

an experimental setting, its response still deviates from the theoretical prediction. Several experiments have found that adding an IG predator (HNF) has a positive impact on IG prey (viruses) (see review by Miki and Jacquet 2008). This implies the existence of internal mechanisms that have not been taken into consideration in classic IGP models, resulting in the apparently positive interactions between IG predator (HNF) and IG prey (viruses).

Trait changes in bacterial assemblage and their consequences on viruses

The apparently positive effects of increased bacterial productivity on viruses within the IGP module could be related to trait changes in bacterial assemblage induced by enhanced bacterial growth, which in turn positively affect the strength of trophic interactions between viruses and bacteria. Many experiments using systems involving both bacteria and viruses have shown that adding inorganic and organic nutrients enhances cell-specific activity (biomass growth rate and replication rate) and induces the trait changes in individual bacteria. Enhanced cell-specific activity is expected to result in an increase in both production and infection rates of the viruses. First, enhanced cell-specific activity may lead to a shorter latent period (the period of viral infection prior to cell lysis) and an increase in the burst size (the number of new viral particles produced and released per cell lysis) of lytic viruses (Middelboe 2000; Weinbauer 2004; Lymer et al. 2008). In addition, enhanced growth rate of the host cells could induce a shift of viral lifecycle strategies from the lysogenic mode, in which the virus does not kill the host cell but is replicated as a result of host replication, to the lytic mode, in which viruses destroy the host cell membrane in order to release new viral particles (Williamson et al. 2002; Paul 2008). Second, enhanced cell-specific activity also leads to an increase in the number of outer membrane nutrient transporters, which could also be recognized as receptors for viruses. It is argued that an increase in the number of receptors would enhance the attachment of viruses to hosts and thus the infection rate (Lenski 1988). These mechanisms could explain the experiments in which adding nutrients to systems containing bacteria and viruses enhances viral production in various environments (Hewson et al. 2001; Williamson et al. 2002; Weinbauer et al. 2003; Motegi and Nagata 2007), even though a negative relationship between nutrient conditions and viral activity has also been reported (Lymer et al. 2008). In addition to the physiological responses of hosts and viruses to growth conditions, it is speculated that local adaptation of viruses to different trophic status may also be important. Within the IGP module, higher parasite virulence is predicted to evolve with an increase in host density (Choo et al. 2003). However, the consequences of the counter-evolution of the host species are unclear. The combined effects of short-term physiological responses of bacteria and viruses and their coevolutionary outcomes make it difficult to predict the general patterns of the impact of system productivity on the viral contribution to bacterial mortality.

Enhanced bacterial growth and induced bacterial trait changes could also explain the many experimental findings showing that the presence of HNF, i.e., the trophic interaction between HNF and bacteria, leads to an increase in viral production rate, frequency of infected bacterial cells, and viral abundance (Šimek et al. 2001; Weinbauer et al. 2003, 2007; Sime-Ngando and Pradeep Ram 2005; Jacquet et al. 2007; Pradeep Ram and Sime-Ngando 2008). Although HNF have lethal effects on individual bacteria, HNF predation is known to stimulate the growth rate of the surviving individuals, i.e., both the biomass growth rate and the replication rate, by changing the resource conditions and weakening competition for resources. Resource competition is reduced as a result of: (1) lower bacterial density, and (2) regeneration of inorganic and organic substrates released from the cells of killed individuals. Therefore, via the various mechanisms discussed above, HNF-predation-induced changes in individual bacteria could lead to increased viral production and abundance.

There is another potential mechanism by which HNF could have positive effects on viruses. In addition to inducing changes in the physiological traits of surviving individuals, which can be defined as individual-level trait changes, HNF predation can cause population-level or community-level trait changes by modifying the structure of the bacterial assemblage. For bacterial assemblages that generally consist of distinct, asexually replicating, genetic units, it would not be helpful to attempt to distinguish between intraspecific and interspecific genetic diversity. So, hereafter, we simply use the term "group-level" traits rather than population- or community-level traits. Group-level traits can be defined as the individual traits averaged over all individuals with different traits in the bacterial assemblage, such as average growth rate and average cell size. Emergent traits, such as genetic diversity (phylogenetic diversity), are also group-level traits. Temporal changes in group-level traits are generally driven by evolutionary dynamics (natural selection) and/or community dynamics (species sorting, Leibold et al. 2004), but for bacterial assemblages, we cannot always distinguish between them.

The HNF predation rate depends on bacteria cell size, and this size-selective mortality induces shifts in size distribution of bacterial assemblages. HNF preferentially graze on medium-sized bacteria, resulting in a bimodal size distribution within the bacterial assemblage (Hahn and

Höfle 1999; Pernthaler et al. 2001), and a dominance of filamentous bacteria is often observed (Hahn and Höfle 1999; Šimek et al. 2001, 2007). The shift in size distribution is achieved by both phenotypic plasticity (Corno and Jürgens 2006) and shifts in the genotypic structure of the bacterial assemblage (Jüergens and Matz 2002; Corno 2006). By way of comparison, host specificity is generally high in viruses (i.e., bacteriophages), probably due to rapid coevolution, implying that mortality due to viruses is phylogenetic or taxonomy selective. Because of differences in the selectivity of mortality in HNF and viruses, and it has been argued that they have differing effects on bacterial diversity (Thingstad and Lignell 1997; Thingstad 2000; Miki and Yamamura 2005), and indeed, heavy HNF predation can actually reduce phylogenetic diversity (Weinbauer et al. 2007). It is also speculated that reduced bacterial diversity as a result of high HNF predation pressure could be related to higher susceptibility to viral infection at the group level (Šimek et al. 2001). In general, when the diversity of the host is reduced and the total population size remains constant, the successful attack rate will increase, because each type of parasite can attack a larger fraction of host groups in a context of high host specificity (Keesing et al. 2006).

In most cases in microbial systems, the observed values of parameters such as growth rate, production rate, and cell size are average values for all bacterial individuals, i.e., group-level traits. This is because bacterial assemblage is inevitably treated as a "black box" due to methodological limitations. From a theoretical point of view, it is important to interpret changes induced in these group-level traits as the consequences of both individual-level changes (including phenotypic plasticity) and changes in group-level averages through shifts in the structure of the bacterial assemblage, even if it is difficult to distinguish between them. For example, an increase in the number of viral receptors could also occur at the group level as a result of shifts in bacterial composition toward a dominance of bacterial groups with higher growth rates and with a larger number of receptors, as well as phenotypic plasticity at the individual level.

The positive effects of HNF predation on viruses as a result of individual- and group-level trait changes in bacteria can be interpreted as a consequence of TMII. In this scenario, HNF, bacteria, and viruses are the initiators, transmitters, and receivers of TMII, respectively (Fig. 1c). This scenario is essential to understand the synergistic effects between HNF and viruses (Šimek et al. 2001; Weinbauer et al. 2003, 2007; Sime-Ngando and Pradeep Ram 2005). Interestingly, however, several studies in similar experimental settings have also shown that HNF predation can reduce viral activity and abundance (Maranger et al. 2002; Bongiorni et al. 2005; Horňák et al. 2005; Jardillier et al. 2005). However, as we have shown elsewhere (Jacquet et al. 2007), the type of the indirect effect of HNF on viruses may strongly depend on the season. The sign of the effect of HNF on viruses could be determined by the relative strength of the positive effects via TMII (Fig. 1c) and the negative effects via density-mediated effects (competition and predation) (Fig. 1a). Interestingly, in contrast to the short-term microcosm experiments (less than 1 week), the patterns observed over large spatial or longer time scales, which reflect long-term averaged effects, do not reveal any positive effects of HNF predation on viruses. Vertical distribution patterns in lakes reveal a negative correlation between the abundance and activities of HNF and those of viruses (Weinbauer and Höfle 1998; Colombet et al. 2006). Medium-term incubation (15 days) of sediment systems revealed a negative correlation between protozoan abundance and the virus-to-bacterium ratio and infection frequency (Fischer et al. 2006). The temporal dynamics at the seasonal scale do not display any significant correlation between HNF and viral activities (Bettarel et al. 2003, 2004).

Even though the net effect of positive TMII and negative density-mediated effects over large spatial and time scales are always negative, we hypothesize that a positive indirect TMII could weaken the negative effects of competition and IGP and so contribute to the coexistence of viruses and HNF in both less productive and highly productive systems. Bacterial trait changes induced by environmental changes could also weaken the negative DMII. For example, it is speculated that enhanced bacterial growth during eutrophication leads to a shorter latent period of viruses, improving the competitive ability of IG prey (viruses) for the common resource (bacteria) and so weakening the strength of coincidental IGP (Miki and Yamamura 2005). Unfortunately, it is difficult to test this hypothesis because of the difficulty of experimentally identifying positive and negative effects.

Comparison with plant–insect systems

These indirect interactions between HNF and viruses via trait changes in the bacterial assemblage are comparable with one of the major TMIIs that have been widely observed in terrestrial plant–insect systems (Ohgushi 2005, 2007; Utsumi et al. 2010). In terrestrial systems, herbivory by insects often induces trait changes in plant tissues, which indirectly affect the growth or abundance of other consumers of the same individual plants. These indirect effects can be negative, as a result of induced resistance to herbivory (e.g., Faeth 1986; Karban and Myers 1989), but can also be positive as a result of compensatory regrowth of plant tissue (e.g., Nakamura et al. 2003) or of induced

resistance that is effective against a certain consumer species yet actually benefits other consumers (Martinsen et al. 1998). Although there are apparently many differences, we looked for functional similarities between TMIIs in aquatic microbial systems and those in terrestrial plant–insect systems.

The crucial factor for TMII function is the induction of phenotype changes at the individual level of the transmitter organisms. This is a clear similarity between the two systems from the standpoint of the food web structure. In both systems, TMII occurs among organisms that share a common resource organism via induced phenotypic changes in the resource organism, i.e., bacterivory-induced trait changes in bacteria or herbivory-induced trait changes in plants. Interestingly, however, details of the mechanism by which phenotypic changes are induced differ in bacteria and plants. In plant–insect systems, the physical size of the resource (plant) is much greater than that of consumers (insects). Therefore, in general, herbivory by insects does not result in the death of the plant and so induces phenotypic changes in individual plants that have been subjected to herbivory. On the other hand, in microbial systems, despite the lethal effects of bacterivory, the positive effects of HNF predation on bacterial growth are shared by all surviving individuals in the assemblage, probably as a result of the rapid diffusion of regenerated nutrients. As a result, individual phenotypic changes due to enhanced growth occur in all individuals in the assemblage and may persist over the generation time of the bacteria as long as improved nutrient conditions persist. The corresponding feature in microbial systems is induced by shifts in the genetic composition of the bacterial assemblage and leads to phenotypic changes at the group level. The corresponding processes are less likely in plant–insect systems because plants have a much longer generation time than consumer insects. One distinctive feature of microbial systems is that multiple processes can contribute to phenotypic changes at group level, mediating indirect interactions between consumers.

Although we can view these features as dissimilarities between the two systems, it is worth trying to find functional similarities in TMIIs by changing our angle. Consider an herbivorous arthropod, which is smaller than a single leaf of the plant. Herbivory by this arthropod can induce trait changes of leaves within the same individual plant that has not actually been subjected to herbivory, known as induced systemic resistance (e.g., Agrawal et al. 1999; see also Orians 2005 for review). Insects consuming the stem tissue can also induce a change in the availability of different types of resource, e.g., leaves, and thus affect other types of consumers (Nakamura et al. 2003). In a broader sense, each leaf or each module (e.g., shoot) within an individual plant can be viewed as corresponding to an individual or clonal subgroup within the bacterial assemblage and different types of resources within a given individual plant (e.g., leaves, stems, and roots) as corresponding to distinct clonal subgroups in bacterial assemblages. This means that we can interpret the systemic and diverse responses of individual plants and the diverse group-level responses of bacteria as having comparative TMII functions.

The concept of TMII helps to better define the indirect interactions between HNF and viruses in aquatic systems, and the comparison with terrestrial plant–insects systems may contribute to the generalization of TMII. However, this theory still has limitations for explaining microbial interactions. For example, from the point of TMII theory, viruses can potentially be the initiator that induces trait changes in bacteria, and in turn, HNF can be the receiver of induced trait changes, as it has also been suggested that viral infections affect bacterial growth and composition through a variety of processes, including nutrient regeneration, host-specific infection, occurrence of some resistant types, and killing the winner or looser (Thingstad and Lignell 1997; Fuhrman 1999; Thingstad 2000; Bouvier and del Giorgio 2007; Šimek et al. 2007; Suttle 2007; Miki et al. 2008). However, to the best of our knowledge, there are no reports suggesting positive or negative indirect effects of viruses on HNF (Šimek et al. 2007; Weinbauer et al. 2007; Zhang et al. 2007). Therefore, it would be unlikely that viral-lysis-induced changes in bacterial assemblage can contribute to the coexistence of HNF and viruses. Development of general theory of TMII by mathematical modeling would be necessary to explain such an apparent asymmetric relationship between two consumers of bacteria. The applicability of the TMII theory, which has been developed to describe species-level interaction networks, to microbial interactions, which are described at the functional group level, should also be carefully considered; initiators and receivers of microbial TMII are assemblages of multiple groups of HNFs and viruses, respectively, as well as transmitters of multiple types of bacteria.

Closing remarks

Complex interactions among bacteria, viruses, and protozoan predators affect the basic level of the microbial food web in aquatic ecosystems (Miki and Jacquet 2008). Even within this simple module, TMII probably acts as an important determinant in population and community dynamics. However, it is still not clear whether TMII has any significant effects on microbial-mediated biogeochemical processes in aquatic ecosystems. This is also true of terrestrial plant–insect systems, and TMIIs between microbes in soil systems have not yet been explored. The

lower levels of diffusion of nutrients and organisms in soil would result in different patterns from those observed in aquatic systems. Future research could involve exploring TMIIs induced by the group-level induction of trait changes in terrestrial plant–insect systems, prey–predator systems, and aquatic phytoplankton-based systems.

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