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VIRUS-BACTERIA INTERACTIONS UNDER DIFFERENT CONDITIONS OF COASTAL ECOSYSTEMS

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“…les rêves peuvent facilement s'envoler mais ce n'est pas pour cela que j'aime la science.”

Louis Pasteur, 1849

INTRODUCTION

Object of the study. The International Committee on Taxonomy of Viruses (ICTV, http://www.ictvonline.org/), established in 1966, define virus species as: "*a polythetic class of viruses that constitutes a replicating lineage and occupies a particular ecological niche*". This definition makes viruses inherent from the surrounding environment and their cellular hosts, as their functional activity is strongly moderated by both factors (Van Regenmortel et al., 1991; Singleton and Sainsbury, 1993).

Viruses by being as ancient as life itself (Hendrix et al., 1999, Koonin et al., 2008) and most abundant biological entities in the hydrosphere (Suttle, 2005) are the oldest predators in the world and create the highest natural selective force on the cellular life (Bamford et al., 2002; Bamford, 2003). Planktonic viruses (also known as virioplankton) – active component of aquatic ecosystem (Weinbauer, 2004), coexisting with their hosts (Van Valen, 1973) and structured by the ambient factors (Womack and Colwell, 2000), is of scientific interest of this dissertation. Virioplankton and its impact on host ecology at the community level under the different environmental conditions is the subject of discussion.

Relevance of the problem. The importance of planktonic viruses in trophic interactions and ecosystem functioning were not recognized until the last decade of the XX century (Bergh et al., 1989; Furhman, 1999). Despite an enormous efforts in aquatic virus ecology made ever since, it is still unclear how and to what extent virus-mediated nanoscale processes such as virus genes expression inside the host (Sullivan et al., 2010), duration of latent period and progeny formation (Smith and Thieme, 2012), switch between lysogeny and lysis (Pradeep-Ram and Sime-Ngando, 2010) are linked to global-scale biodiversity and biogeochemistry (Brussaard et al., 2008). Moreover, viruses and virus-mediated processes are overlooked in most of ecological models dealing with material and energy fluxes as well as they are ignored in climate change predictions. Incredibly little is known how global warming will shape virus-host dynamics and how virus-mediated processes will influence ecosystem feedback on climate change (Danovaro et al., 2011). These questions are of major importance, in particular for semi-enclosed European Seas (e.g. the Baltic Sea) and coastal environments (e.g. the Curonian Lagoon, the Kiel Fjord), where sea surface temperature (SST) is rising more intensively than in other areas (Belkin, 2009). On the other hand, recent metagenomic studies emphasize that trophic interactions (e.g. predation) rather than nutrient limitation is a key factor structuring microbial community (Mou et al., 2008). Thus, the understanding of virus-mediated

quantitative (biomass, size, age, etc.) and qualitative (diversity, productivity, ecotrophic efficiency, etc.) conversions in aquatic habitats is highly relevant to both fundamental and practical issues of ecosystem management, including total productivity and biodiversity as well as ecosystem resilience and conservation aspects. In addition, a plenty of studies based on both isolated phage-host systems and natural communities have generated a number of hypotheses explaining the variability in virus activity (Parada et al., 2006). Still there is a lack of comprehensive experimental validation of these hypotheses, even though that identification of which virus regulation mechanism prevails in the particular habitat is vital for understanding of ecosystem functioning.

Scope of the study. Coastal environments are extremely productive systems of high spatio-temporal complexity, which modulate the biological processes and, thus, habitat and niche partitioning of aquatic organisms. Virus-host interaction models predict that viral predation is a key controlling mechanism for community regulation in highly productive ecosystems (Winter et al., 2010). Field observations and experimental studies reveal high infection rates (Winter et al., 2004) and, therefore, significant virus contribution to bacteria and phytoplankton mortality (Suttle and Chan, 1994; Jacquet et al., 2005; Baudoux et al., 2006), especially in nutrient rich environments (Wilcox and Fuhrman, 1994). The differences in the rates of bacterial mortality and, in general, dynamics and strength of predator-prey interactions limit the community richness and evenness (Jardillier et al., 2005), drives the biogeochemical cycles (Fuhrman, 1999) and regulates the metabolic balance of ecosystem (Wootton and Emmerson, 2005; Allesina and Tang, 2012).

Virioplankton studies in the Baltic Sea indicate the importance of virus lysis at various organizational levels from strain to ecosystem as well as significant differences between habitats within the environment (Weinbauer and Höfle, 1998; Eissler et al., 2003; Weinbauer et al., 2003; Holmfeldt et al., 2010; Anderson et al., 2012). The role of viruses in the coastal lagoons of the Baltic Sea is largely overlooked and lacks a detailed description of their seasonal dynamics and spatial distribution as well as virus-mediated processes such as bacterial mortality and recycling of carbon and nutrients. In addition, none of the studies performed in the Baltic Sea region to date addressed the question of how climate change and in particular global warming might influence virus activity in terms of virus production and virus induced bacterial mortality.

The current research was performed in two different ecosystems and involved observational studies based on field sampling data and experimental studies conducted in the semi-enclosed mesocosms and microcosms, using natural microbial communities (Fig. 1). The investigations performed in the Curonian Lagoon consisted of field sampling surveys (2005–2009) and microcosm experiment (1st STOP, done in 2010) under *in situ* conditions (Fig. 1). In the observational study, the interactions between viruses and various biotic parameters was investigated to clarify the spatio-temporal patterns of virioplankton dynamics in the Curonian Lagoon. The results from these surveys allowed to construct and experimentally test hypotheses on virus-bacteria interactions and viral impact on bacterial community (Fig. 1, 1st STOP experiment).

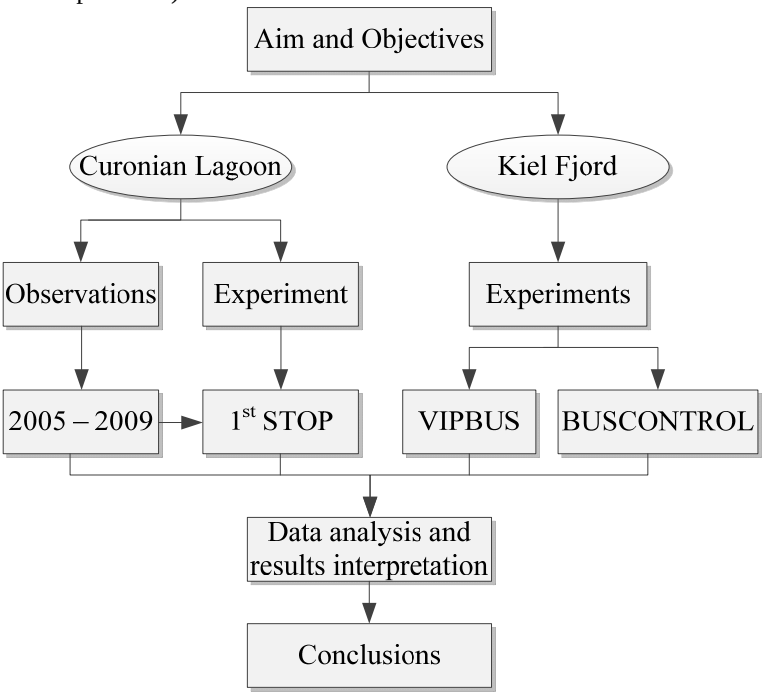

Figure 1. Scheme shows work flow of performed activities during the years of PhD research, integrating the study area (the Curonian Lagoon and the Kiel Fjord) and type of performed investigations (observational or experimental).

Mesocosm experiments in the Kiel Fjord (Fig. 1, VIPBUS and BUSCONTROL) were dedicated to evaluate under what temperature conditions the effect of viruses on bacterial mortality might change significantly and how it might affect trophic interactions within lower part of the pelagic food web. The design of the experiments and working hypotheses were constructed based on the proposed third universal ecological response to global warming model (Daufresne et al., 2009). In both experiments, the natural Fjord plankton community was used and temperature manipulated according to the proposed climate change scenarios for the Baltic Sea region (IPCC, 2007).

This research at least in part fills the gaps of our understanding about the role of viruses in the microbial loop, discuss how well the observed variety of responses of different virioplankton variables on spatio-temporal scale and distinct temperature regimes could be predicted by the biotic factors and how it contributes to the explanation of possible net outcome of the trophic interactions between viruses and their prokaryotic hosts. Further, this dissertation address question how field-based observations and experimental results might explain possible feedback processes of different types of interactions in the context of ecosystem functioning, eutrophication and global warming, and provide insights into how habitat with all its complexity by covering physical, chemical and biological elements rather than individual factor allows better understanding of the ecology of viruses in the coastal environments of the Baltic Sea.

Aim and objectives of the study. The aim of the study was to evaluate the patterns of virus-host interactions based on the assessment of viral impact upon host community structure, abundance and mortality.

To achieve this aim, the following objectives were formulated:

1. to reveal the patterns of virioplankton morphological diversity and seasonal dynamics in the Curonian Lagoon;
2. to determine the interactions between virus abundance and infectivity as well as structure and density of bacterial host community under different conditions of pelagic part of the Curonian Lagoon.
3. to evaluate the effect of viruses on bacterial mortality under different temperature regimes.

Novelty of the study. This study has a contribution to knowledge in the context of both local and global scales. Well-established concepts in aquatic virus ecology were applied and experimentally tested for the first time in the coastal environments of the Baltic Sea, the Curonian Lagoon (south-eastern

part of the Baltic Sea, Lithuania/Russia) and the Kiel Fjord (eastern part of the Baltic Sea, Germany). Evidence for significant virus contribution to prokaryotic community regulation in these ecosystems and dependence of virus-host interactions on environmental conditions was provided. This study also attempts to provide an insight into how climate change will affect biotic interactions and, thus, ecosystem services supply. Two mesocosm experiments conducted during this study were dedicated to highlight the effect of global warming on virus-mediated processes such as bacterial mortality, and revealed the possibly existing temperature threshold significantly modulating virus activity. A detailed study on the response of different virus variables (abundance, burst size and infection rates, production and decay) under various biotic and temperature conditions also provides an new insights into the underlying mechanism of virus-host interactions, and forms basis for further construction of hypotheses and predictions to understand whether viruses will hamper or stimulate ecosystem production under the future climate change (Brussaard et al., 2008; Danovaro et al., 2011). In addition, from the methodological point of view, this work provides with a detailed comparison of methods based on different approaches and dedicated to estimate virus-mediated mortality, thus, also contributes to the practical aspects of virioplankton studies.

Scientific and practical significance of the results. The results of this study revealed spatial complexity of microbial assemblages and different patterns of their temporal dynamics along the gradient of the Curonian Lagoon. Present observational and experimental findings also indicate that viral activity cannot be explained by a single variable (abundance, cell size or growth rate of the host, which refers to host physiological state control hypothesis), and probably is upregulated by the complex of different controlling mechanisms (refers to habitat control hypothesis, Parada et al., 2006). Therefore, it represents high level of complexity of trophodynamicss at the base of the food web and, thus, demands for a comprehensive view on ecosystem management and integration of virus-host trophic interactions into the local ecosystem and biogeochemical models. The magnitude of virus-mediated processes and its dependence on spatio-temporal scale, as shown in this study, largely disclose why the relatively higher levels of nutrients and apparently high plankton productivity in the Baltic Sea coastal environments do not significantly affect bioproductivity of top predators. The results also are highly relevant to the issues of climate change and supplement our understanding on the potential impact of viruses on microbial food web under global warming conditions.

Defensive statements:

1. Virioplankton distribution and patterns of seasonal dynamics depend on variation of distinct host communities in different parts of the Curonian Lagoon.
2. The effect of viruses on the prokaryotic community depends on virus decay rates.
3. Virus burst size is limited by indirect effects of environmental conditions and cannot be explained by the host community structure, abundance and activity.
4. Increased Sea surface temperature will promote lytic virus infections.
5. Virus-mediated bacterial mortality is a threshold temperature dependent process.

Scientific approval. The results and material of this dissertation were presented at seven international conferences and published in two original papers, both in peer-reviewed scientific journals.

Conferences:

1. 4th European Conference on Lagoon Research (4th EUROLAG) "Research and Management for the Conservation of Coastal Lagoon Ecosystems, South–North comparisons", 14–18 December 2009, Montpellier, France. Presentation: „Virioplankton abundance and seasonal development correlation with shifts of different groups of plankton community in the Curonian Lagoon". Authors: Sulcius S., Paskauskas R.

2. 1st International Symposium on "Viruses of Microbes", 21–25 June 2010, Paris, France. Presentation: „Links between Virus–Like Particles, Bacteria and Chlorophyll *a* in Eutrophicated Boreal Lagoon". Authors: Sulcius S., Paskauskas R., Griniene E.

3. 5th European Conference on Lagoon Research (5th EUROLAG) "Coastal Lagoons in a Changing Environment: Understanding, Evaluating and Responding", 25–30 July 2011, Aveiro, Portugal. Presentation: „Time scale and hydrological regime based dynamics of virioplankton in the eutrophicated Curonian Lagoon". Authors: Sulcius S., Paskauskas R.

4. 12th Symposium on Aquatic Microbial Ecology (SAME 12), 28 August–02 September 2011, Rostock/Warnemünde, Germany. Presentation: „Temperature-mediated virus-induced bacterial mortality". Authors: Sulcius S., Paskauskas R.

5. 6th Aquatic Virus Workshop (AVW 6), 30 October – 03 November 2011, Texel, The Netherlands. Presentation: "Warming stimulate frequency

of infected cells and impinge on virus burst size". Authors: Sulcius S., Paskauskas R., Sommer, U.

6. 14th International Symposium on Microbial Ecology (ISME 14), 19 – 24 August 2012, Copenhagen, Denmark. Presentation: "Habitat dependent virus–bacteria interactions". Authors: Sulcius S., Paskauskas R., Javidpoor J., Sommer, U.

7. International Symposium on "Recent achievements and future directions in Aquatic Mesocosm Research", 16–19 October 2012, Heraklion, Crete, Greece. Presentation: "Temperature facilitated virus induced bacterial mortality". Authors: Sulcius S., Paskauskas R., Martinkute E., Javidpoor J., Sommer, U.

Publications:

1. Sulcius, S., Staniulis, J., Paskauskas, R. (2011). Morphology and distribution of phage-like particles in eutrophic boreal Lagoon. Oceanologia, 53(2), 587–603.

2. Sulcius, S., Staniulis, J., Paskauskas, R. (2011). Comparative analysis of methods for quantitative assessment of virus-like particles in eutrophicated aquatic environments. Botanica Lithuanica, 17(2–3), 127–133.

Structure of the dissertation. Dissertation consists of the following chapters: Introduction, Literature review, Materials and methods, Results, Discussion, Conclusions. References include 480 sources. The volume of the dissertation is 122 pages. It contains 6 tables and 21 figures. Supplementary information is 5 pages and contains 5 tables and 3 figures. Dissertation is written in English with Lithuanian summary.

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DEFINITIONS AND ABBREVIATIONS

Definitions, specific to virioplankton ecology:

Bacteriophage (phage) – a virus that infects a bacterial host. Typically consists of a protein capsid that encloses genetic material and tail.

Burst size – yield of viruses released during the lysis of individual bacteria.

Cheshire Cat escape strategy – the mechanism of host resistance to prevent virus infection.

Cyanophage – a phage that infects cyanobacteria.

Kill-the-Winner (KtW) hypothesis – describe the trade-offs between host competitive and defence abilities, where the abundance of the most active bacterial hosts is controlled by phage predation.

Latent period – time at which virus progeny are released into the environment.

Lysis – a process of virus release, which cause the complete destruction of a cell wall or membrane. It is considered as the final step of virus replication cycle.

Lytic infection – lifestyle of viruses always resulting in the release of virus progeny upon host lysis.

Lysogeny – lifestyle of temperate phages, which integrate into the host's genome or are maintained as plasmids until an induction event triggers the lytic cycle.

Polythetic class – number, but not all, of the properties of the set that possess each individual and each property in the set is possessed by most but not all individuals, and no property is possessed by all individuals.

Virus (like) particle – a virion or is hypothesized to be viral but for which an infectious cycle has not been observed.

Virioplankton – in this thesis refers to free floating virus (like) particles in marine or freshwaters.

Viriosphere – the portion of the Earth where viruses interact with their hosts.

Virus decay – loss processes for viruses either by induced particle destruction, physical removal or by loss of infectivity due to damage of viral genetic material or proteins.

Virus turnover time – the time needed for the standing stock of viruses to be replaced as it decays by new viruses.

Virus shunt –virus mediated release of nutrients from organisms to pools of dissolved and non-living particulate organic matter.

Other definitions, used in this work:

Court Jester hyphotesis – state that changes in physical and environment conditions rather than biotic interactions themselves drives evolution (opposite to Red Queen hypothesis).

Grazers – phagotrophic protists, in particular heterotrophic nanoflagellates, feeding on prokaryotes and other protists.

Habitat – a place where the physical, chemical and biological elements of ecosystems provide a suitable environment for organism's existence.

Predator – always results in cell death, including viral lysis.

Red Queen hyphotesis – state that biotic interactions are more important than physical and environmental change in driving evolutionary change (opposite to Court Jester hypothesis).

Metabolic Theory of Ecology – predicts the effects of body size and temperature on metabolism through considerations of vascular distribution networks and biochemical kinetics.

Mesocosm experiment – an experimental system that enables to create close to natural environmental conditions, whilst allowing the manipulation of environmental factors.

Microorganism – non-taxonomic term used for microscopic organisms, in this thesis applied for viruses, prokaryotes, protists and single-celled algae.

Microbial succession – selection and development of sequential microbial populations in natural or disturbed systems.

Abbreviations used in this work:

CR – contact rate.
CHLa – chlorophyll *a* concentration
CS – contact success.
RSG+ – redox sensor green positive cells.
PI+ – propidium iodide positive cells.
BS – burst size.
LR – lysis rate.
VP – virus production.
FIC – frequency of infected cells.
BGR – bacterial growth rate.
BGE – bacterial growth efficiency.
BCS – bacterial community structure.
VMBM – virus mediated bacterial mortality.
DOM – dissolved organic matter.
POM – particulate organic matter.
KtW – "Kill the Winner" hyphotesis
KKW – "Kill the Killer of the Winner" hypothesis
HNF – heterotrophic nanoflagellates.
SST – sea surface temperature.
PBINs – Phage–bacteria infection networks
MTE – metabolic theory of ecology

I. LITERATURE REVIEW: Viriosphere – an environment for viruses to interact with their hosts

Planktonic viruses are dominating aquatic environments and rule over distribution, diversity and evolution of other microorganisms at both single cell and global ocean scales. From the molecular level interactions (Hambly and Suttle, 2005), "hotspots" in water column (Seymour et al., 2006) to the entire ecosystem and whole hydrosphere (Bratbak and Heldal, 2000), the ability of individual organism to survive and, therefore, the flow of energy and matter through the food web are determined by the activity of virioplankton (Furhman, 1999; Rohwer and Thurber, 2009).

Bacteriophages (also phages) – viruses that infect bacteria, were discovered by Frederick W. Twort in 1915 (Duckworth, 1976). Marine virioplankton was found in the mid of the last century (Zobell, 1946) and showed to be numerically abundant in the water column (Safferman and Morris, 1963). However, their importance in ecosystem functioning was not recognized until the 1990's and erupted papers of Bergh et al. (1989) and Heldal and Bratbak (1991). Since then emerging questions and challenges in aquatic virus ecology has been driven by methodological advances. From the quantitative methods to measure the distribution, abundance and productivity of virioplankton (Borsheim et al.; 1990) to single virus metagenomics (Allen et al., 2011), all new approaches yielded greater ecological insight into the structure and functioning of aquatic viral communities.

Viruses became an integrated part of aquatic food web ecology and, thus, necessitated the rearrangement of old and development of new conceptual, theoretical and even statistical paradigms (Bratbak et al., 1992; Thingstad and Lindel, 1997; Furhman, 1999; Thingstad, 2000), which have had to be tested against experimental results, and that changed the understanding of processes of the classical microbial loop (Pomeroy, 1974; Azam et al., 1983). All these and new findings result in more than one hundred original publications per year and exponentially growing cumulative citations in the emerging field of aquatic virus ecology (Middelboe, 2008; Jacquet et al., 2010).

In the following sections of this literature review, available knowledge about the dynamics and distribution of viruses at different space and time scales will be provided. Emphasis will be made on the factors significantly shaping virus-host interactions at the community level.

1. Virus abundance, diversity and spatio-temporal distribution

The free floating virus particles (virioplankton) are the most numeric and of greatest diversity life forms in the hydrosphere (Hendrix, 2002; Suttle, 2007). As for all organisms, there are global factors that influence abundance

and distribution of viruses and local forces that create variability and promote their diversity.

The standing stock of viruses, in general, is related to trophic conditions (Noble and Fuhrman, 2000), with higher values in more productive habitats within ecosystems (Weinbauer et al., 1993) and between different environments (Weinbauer, 2004). Virus density also depends on the type of water (fresh or saline) (Maranger and Bird, 1995), depth (Hara et al., 1996; Steward et al., 1996), distance from the shore (Cochlan et al., 1993), age of water masses and presence of organic and inorganic particles in the water column (Kernegger et al., 2009) as well as on oxygen concentration (Weinbauer, 2004) or system stratification (Weinbauer et al., 1995). The temporal dynamics, from hours to years, of total virus abundance could be explained by host availability and productivity (Colombet et al., 2006), stage of annual communities development (Bratbak et al., 1990), seasonal changes (Lymer et al., 2008b), simultaneous infection and lysis (Bratbak et al., 1996) and/or physical conditions such as temperature (Choi et al., 2009), turbidity or solar radiation (Heldal and Bratbak, 1991). The spatial distribution of viruses tightly depends on the widespread and biogeography of host as well as on virus capability of multiple infection (Wichels et al., 2002). On the other hand, there is some evidence of virus movement between different biomes (Sano et al., 2004), probably at the same time indicating their high global diversity and adaptation capacity.

Virus-to-bacteria ratio (VBR) is considered as important variable indicating the potential of viruses to control bacterial abundance and allows to infer the level of stability between viruses and their hosts, or even allows the comparison between habitats or ecosystems, since it may remain quite consistent when bacteria and virus counts change dramatically (Hara et al., 1991; Choi et al., 2003). Usually higher VBR values are found in freshwaters (mode = 22.5, mean 20.0) and more nutrient-rich environments, than in marine waters (mode = 2.5, mean 10.0) and under oligotrophic conditions (Maranger and Bird, 1995). In general, VBR do not exceed value of 100 (Wommack and Colwell, 2000) and show common trend of increased VBR at low bacterial abundance (Tuomi et al., 1995). Since VBR was suggested to reflect the multiplicity of infection (MOI) (Watanabe and Takesue, 1972), and it was shown that instantaneous lysis occurs when MOI exceeds 125 (Kruegger and Northrop, 1930), it makes VBR good indication of virus pressure on host mortality. The observed extremly low (0.1) and high (126.7) VBR for some polar waters were coincident with high (over 100% of total bacterial production) and low grazing rates (Madan et al., 2005; Laybourn-

Parry et al., 2006), respectively, indicating that VBR may reflect the dominant source of prokaryotic mortality. On the other hand, critical VBR values may indicate capability of viruses to survive in certain environment (Vanucci et al., 2005). That means that VBR also relies on infection rates, virus burst and host cell size (Hara et al., 1991). VBR dynamics is also related to season and spatial scale (Filippini et al., 2008, Payet and Suttle, 2008), suggesting environmental regulation upon virus-host interactions.

Though viruses account for substantial amount of microorganisms (Suttle, 2007) in world's ocean, they also are exceptionally diverse in morphology and genetic organization (Hendrix, 2002). Taxonomic structuring of viruses based on different morphological or genetic properties has been proposed by several authors (Bradley, 1967; Ackermann and Eisenstark, 1974; Chen et al., 1996; Wichels et al., 1998; Rohwer and Edwards, 2002; Dorigo et al., 2004; Djikeng et al., 2008; Culley et al., 2010), and generally the use of both types of descriptors in virus taxonomy has been approved by the International Committee on Taxonomy of Viruses (ICTV) (Van Regenmortel and Fauquet, 2000). However, despite recent methodological achievements, the patterns of viral diversity at both global and local scales are still poorly understood.

More than 6000 bacteriophages, belonging to 10 families and infecting members of 179 bacterial genera, are morphologically defined to date (Ackermann and Prangishvili, 2012). However, this number increases rapidly, since more viruses are discovered and described each year. The diversity of viral morphologies ranges from a variety of icosahedral tailed phages (Moebus, 1991) to long filamentous, spindle or lemon-shaped viruses (Middelboe et al., 2003; Prangishvili et al., 2006; Pina et al., 2011), probably depending on host community structure and type of interactions between virus and host (Miki and Jacquet, 2008), salinity or trophic gradient (Guixa-Boixareu et al., 1996; Bongiorni et al., 2005) as well as on marine or freshwater (Bratbak et al., 1990; Mathias et al., 1995). Usually aquatic phages have isometric head and range from 20 to 200 nm, where size class of 30–60 nm comprise the majority of viruses observed in marine ecosystems (Wommack et al., 1992). Larger size class particles (60 to 90 nm) are prevalent in fluvial and lacustrine ecosystems (Mathias et al., 1995; Drucker and Dutova, 2006). The vast majority of all observed viruses (96%) belong to order *Caudovirales* (Ackermann, 2003; Ackermann and Prangishvili, 2012) and is divided into three families (*Myoviridae*, *Siphoviridae* and *Podoviridae*) according to their relative proportion of the capsid and tail structures (Ackermann, 2007). Among these families *Siphoviridae* or viruses that possess long and noncontractile tails constitute around 2/3 of all tailed viruses

(Ackermann, 2001). Meanwile *Myoviridae* cover the vast majority of viruses that infect cyanobacteria (cyanophages) (Millard et al., 2009). Some phages larger than 300 nm as well as temporal changes in the size structure of free and intracellular phages, or even host independent phenotypic development of virions have been reported in aquatic environments (Bratbak et al., 1992; Hennes and Simon, 1995; Häring et al., 2005). However, high diversity of atypical morphology, including non-tailed or filamentous phages can constitute the largest portion of viruses in some habitats (Pina et al., 1998; Hofer and Sommaruga, 2001; Prangishvili and Garrett, 2004).

Virus genetic diversity is shown to be relatively high at both local and global scales (Hendrix et al., 2000; Breitbart and Rohwer, 2005), even though their genome organization is similar (Paul and Sullivan, 2005). However, the data on their geographical patterns of distribution are more profound and are limited by the lack of universal phylogenetic markers (like the rDNA genes) as well as numerous drawbacks of classical cultivation-based techniques (Weinbauer and Rassoulzadegan, 2004). Some virus groups are extremely diverse, but spatially restricted over and/or among ecosystems (Steward et al., 2000; Prestel et al., 2008). Some phages are unique and habitat specific (Prigent et al., 2005; Desnues et al., 2008). While other groups are widely distributed or exhibit family-specific gradient changes over different spatial scales (Kellogg et al., 1995; Filée et al., 2005; Angly et al., 2006; Comeau et al., 2006; Williamson et al., 2008). In general, viral diversity, including temporal dynamics, is shown to be mostly dependent on the abundance and diversity of the co-occurring potential host populations, while environmental gradient has little direct effect on the structure of viral assemblages (Marston et al., 2003; Winget and Wommack, 2009; Jameson et al., 2011). The enormous diversity of aquatic viruses is maintained and promoted by their genome organization (Lawrence et al., 2002; Weinbauer and Rassoulzadegan, 2004) that allows high frequency virus-mediated gene transfer (Ohnishi et al., 2001) between phages (e.g. through co-infection) (Hambly and Suttle, 2005; Silander et al., 2005) and their hosts (Filée et al., 2002; Canchaya et al., 2003). This also makes viruses' inexhaustible source of diversity for both phage and host, and may lead to better adaptation or niche realization of host population (Lindell et al., 2005). Moreover, the ability of fast gene acquisition and genetic reassortment enables phage movement between biomes (Sano et al., 2004), thus increasing their geographic widespread and relatively lowering their overall diversity (Breitbart and Rohwer, 2005).

2. Virus production, decay and life strategies

Usually the increase in virus abundance is associated with virus production (Hara et al., 1991; Auget et al., 2005), while these rates are changing with water trophic conditions (Rowe et al., 2008) and are higher in more nutrient rich environments (Fischer and Velimirov, 2002; Bongiorni et al., 2005) than in oligotrophic systems (Säwström et al., 2009). Although the overall virus production is stimulated by the resource supply (Weinbauer et al., 2003) and increased temperature (Winter et al., 2005), it is not always associated with higher bacterial production (Lymer and Vrede, 2006), also implying that additional nutrient availability trigger viruses to enter lytic cycle (Tuomi et al., 1995; Bohannan and Lenksi, 1997). Moreover, since virus particle is phosphorus (P) rich, it's thought that virus production is more P limited rather than other nutrients (Wilson et al., 1998; Williamson and Paul, 2004; Lymer and Vrede, 2006). The contribution of virus encoded photosynthesis genes (e.g. *psbA* or *hli*) to virus production and the importance of their expression during latent period to supply additional energy for virus replication is rather ambiguous (Lindell et al., 2005; Hellweger, 2009). However, availability of these internal cell resources is critical for virus assemblage into particle and number of phage progeny (burst size), strongly determining virus production. A number of other studies have found a positive and statistically significant correlations between virus and bacteria production and abundance (Winter et al., 2004; Mei and Danovaro 2004; Boras et al., 2009), with factors explaining this relation primarily driving host rather than virus community (Wommack and Colwell, 2000). Thus, these observations also suggest that the metabolic status of the host, assuming that actively growing bacteria are more susceptible to virus infection, is critical for virus infection and production.

Changes in virus production vary strongly on temporal scale within and between years, months, weeks, days and hours (Bratbak et al., 1996; Larsen et al., 2001; Winget et al., 2011). In the context of different time scale, variation in virus production reflects the direct or indirect impact of different factors upon this parameter. For instance, it was suggested that short term (hours and days) dynamics of virus production is closely related to host daylight (Brussaard et al., 2004; Winget and Wommack, 2009) or a stage of cell cycle (Hwang and Cho, 2002). Weekly dynamics corresponds to the development stages of a particular population (e.g. during bloom periods) or whole community (Baudoux et al., 2006), where both host productivity and encounter rates are increased. Long-term changes in virus production are driven by global ambient factors such as temperature, nutrient loadings and

ecotrophic status of aquatic systems or even evolutionary proceses. It was also shown that lytic virus production dominates over lysogenic in coastal surface waters (Wilcox and Fuhrman, 1994), rather than oligotrophic systems or meso- and bathypelagic zones (Parada et al., 2007).

Decrease in virus numbers is usually referred to virus decay (Noble and Furhman, 2000). Several major sources of virus decay such as UV radiation (Suttle and Chen, 1994; Weinbauer et al., 1999), adsorption to particles (Proctor and Fuhrman, 1991), enzymatic activity (Noble and Fuhrman, 1997) contribute to over 3/4 of total virus loss in aquatic environments. Other sources such as direct grazing on virus particles and predation on already infected cells (Gonzalez and Suttle, 1993), capsid thickness and genome density in the capsid (De Paepe et al., 2006) or ecosystem hydrodynamics conditions (Bettarel et al., 2009) may also play an important role in virus loss. The relative predominance of all of these factors differ among habitats and elucidations of causative agents that mostly contribute to virus loss in a certain habitat and/or under the certain conditions in natural environments still remain very challenging (Bongiorni et al., 2005).

Virus decay caused by solar radiation (in particular UV-B) is a primary source of virus loss (Cottrell and Sutle, 1995) and is associated either with UV-induced particle destruction or loss of infectivity due to damage of viral genetic material (Wommack et al., 1996). The latter proccess might be reversible and depends on virus capacity to restore its infectivity by repairing virus DNA through photoreactivation (Weinbauer et al., 1997; Wilhelm et al., 1998; Kellogg and Paul, 2002). While virus loss due to damage of capsid proteins is irreversible process (Wells and Deming, 2006). However, different viruses exibit different sensitivity to UV radiation (Noble and Fuhrman, 1997; Jacquet and Bratbak, 2003; Linden et al., 2007), implying that effect of sunlight at the community level might depend on the structure of virus assemblages and probably is related to virus adaptability in a particular environment (Linden et al., 2007).

In eutrophic environments adsorbtion to diferent types of particles is major cause of virus loss from water column, including high molecular weight dissolved organic matter (Baudoux et al., 2006) or attachment to non-host cell surface (Brussaard et al., 2005). The formation of colloidal or transparent exopolymeric particles as well as production and release of extracellular proteolytic enzymes associated with increased bacterial activity or final stages of bloom development was suggested as a source of virus removal and degradation rather than inhibiton of their infectivity (Motegi and Nagata, 2007; Corinaldesi et al., 2010). These differences were suggested to

be an important factor in determing virus decay, since loss of infectivity not always leads to loss of detectability of virus particle by the standard epifluorescence microscopy (Wommack et al., 1996). This might lead to underestimation of virus loss as well as to overestimation of infective phages present in environment, when actual proportion of viruses capable for successful propagation is far from the dominant (Waterbury and Valois, 1993; Bongiorni et al., 2005; Corinaldesi et al., 2010).

The abundance of viruses in water sample is the net result of virus production and decay, processes, which are closely related to virus replication strategy. Two types of virus proliferation, lysogeny and lytic infection are common in aquatic environments (Jiang and Paul, 1998). The differences between these replication strategies determine the fate of bacterial production in ecosystem (Miki and Jacquet, 2008). Lytic infection always causes cell lysis, while during lysogenic cycle phage genome replicates with host cell until enters lytic cycle under certain circumstances. Lysogeny is considered as a survival strategy for both viruses and their hosts (through phage regulated host metabolic gene expression) under unfavourable conditions (Ripp and Miller, 1997; Paul, 2008), and is reported for a wide range of bacterial isolates from aquatic environments (Stopar et al., 2004; Leitet et al., 2006). It is also suggested that lysogeny plays an important role in horizontal gene transfer (HGT) and promotes production of gene transfer agents (GTAs) (Miller, 2001; Zhao et al., 2010), thus playing a significant role in bacterial evolution (Canchaya et al., 2004). At least two important factors are responsible for conversion from lysogeny to lytic cycle: 1) multiplicity of infection (MOI) and 2) nutritional conditions (Wilson and Mann, 1997). Concequently, these are affected by environmental factors. Thus, the prevalence of one life strategy over another is changing on temporal and spatial scales (Jiang and Paul, 1994), differs between populations at the same time (Ortmann et al., 2002) and is driven by system trophic conditions (Jiang and Paul, 1998), host density and metabolic status (Weinbauer, 2004) and physical and chemical factors (Jiang and Paul, 1996). The limited knowledge about the regulation mechanisms of the dynamics of virus life strategies prevents our understanding of virus role in the ecosystem.

3. Virus infection rates and trophic interactions in the microbial loop

Classicaly virus infection is considered as density-dependent and species-specific process (Wommack et al., 1999), even thought that some viruses may infect phylogenetically closely related organisms (Sulivan et al., 2003). The infection rates of viruses in natural plankton communities vary within a wide range (Winter et al., 2004). However, at any given moment of time

approximately one-fifth (~17%, vary from 0%–70%) of total bacterial community is infected by phage (Proctor and Fuhrman, 1990; Suttle, 1994; Hennes and Simon, 1995; Wommack and Colwell, 2000; Weinbauer et al., 2003; Jacquet et al., 2005) and more than 100 infections occur every second in each liter of sea water (Suttle, 2007), resulting in 4–13% of infection rate of new hosts on the daily basis (Suttle and Chen, 1992). This correspond to ~25% (vary from < 1% to > 200%) of total virus mediated bacterial mortality (Waterbury and Valois, 1993; Weinbauer and Höfle, 1998; Paul and Kellogg, 2000; Matteson et al., 2012) and removal of 0– >100% (up to 800%) of bacterial production (Binder, 1999; Tuomi and Kuuppo, 1999). The effect of virus infection has much broader ecological implications for ecosystem functioning than just mortality of host organisms (Suttle, 2007; Jiao and Zheng, 2011; Weinbauer et al., 2011). Viral activity promotes the cycling of nutrients within microbial loop (stimulates primary and bacterial production) (Wilhelm and Suttle, 1999; Ory et al., 2010), suppress transfer of dissolved organic matter (DOM) to higher trophic levels (Corinaldesi and Danovaro, 2003), drives particle aggregation and enhance sinking (Mari et al., 2005), determines community structure (Hewson et al., 2003), improves fitness, survival and/or resistance of infected cells and modulates gene transfer via transduction (Jiang and Paul, 1998; Paul et al., 2002; Pedulla et al., 2003; Lindell et al., 2004; Sullivan et al., 2005) as well as regulates population (bloom) dynamics (Brussaard et al., 2007).

Virus infection followed by the cell lysis limits the growth rate of bacteria (Thingstad, 2000), also implying that more active bacterial community may sustain higher viral density (Corinaldesi et al., 2003). In contrast, protist grazing is size selective process (Hahn and Hoefle, 1999) and often affects the total community abundance rather than a single population (Bratbak et al., 1994; Fuhrman and Noble, 1995). The net effect of these two different sources of bacterial mortality is different. Bacteriovory transfers the particulate organic carbon (POC) to higher trophic levels, thus act as “link” between basal production and top predators. Whereas viral-mediated bacterial mortality reduces cell growth efficiency and increase respiration rates of remained community, thus serves as “sink” of organic carbon for higher trophic levels (Middelboe and Lyck, 2002). This also results in the redistribution of organic material consumption in water column (Miki et al., 2008). Upon lysis of susceptible bacteria, carbon and nutrients are released back to the dissolved phase – process termed as “viral shunt” (Wilhelm and Suttle, 1999) and is rapidly recycled by the remaining cells (Middelboe and Jørgensen, 2006). However, the stoichiometry of the released particulate

(POM) and dissolved (DOM) organic material is different from the element composition of the lysed organisms (Suttle, 2007). Virus-mediated DOM is nitrogen rich and comparatively depleted in phosporus, while POM, which is a subject of export to deeper waters (biological carbon pump), is relatively more carbon rich (Suttle, 2007). Another important feature of DOM originated from cell lysis is its bioavailability. Experimental studies demonstrate that lysates of heterotrophic bacteria and cyanobacteria is highly bioavailable in comparison to other sources (e.g. grazing exudates, siderophores, etc.) (Middelboe et al., 1996; Middelboe et al., 2003; Mioni et al., 2005; Poorvin et al., 2011) and might significantly contribute to nutrient regeneration and increased primary production and bacterial metabolism (Poorvin et al., 2004; Holmfeldt et al., 2010; Shelford et al., 2012).

Trophic interactions within microbial loop go beyond the simple direct conections between predators and their prey (Miki et al., 2008; O'Sullivan and Emmerson, 2011; Töpper et al., 2013). Recent mathematical framework proposes at least nine (3×3) possible interaction pathways among bacteria, viruses and heterotrophic nanoflagellates (HNF) (Miki and Jacquet, 2008), pointing out that the prevalence and strength of one pattern over another is based on the trade-offs and trait changes of interacting communities (Miki and Jacquet, 2010). This includes size and spieces selective bacterial mortality and feedback mechanism of prokaryotic community to prevent grazing and virus infection as well as exploitative competition between predators and within each interacting community (Miki and Jacquet, 2008). Miki and Jacquet (2010) suggested a theoretical model, which at once explain the high differences of virus role in trophic interactions observed for natural environments (Steward et al., 1996; Filippini et al., 2006; Danovaro et al., 2008; Matteson et al., 2013) and coexistence of viruses and HNF in the context of intraguild predation (Holt and Huxel, 2007). This model states that virus function depends on 1) direct or 2) indirect density mediated or on 3) trait mediated interactions among bacteria, viruses and HNF. Both density-dependent processes (e.g. higher number of bacteria results in higher number of HNF) have negative effect on viruses, while changes in host growth rates or community structure have indirect positive effect (Miki and Jacquet, 2010). This picture of virus role in trophic interactions is also in agreement with the nature of selective viral predation, which plays an important role in population dynamics (Schroeder et al., 2003; Yoshida et al., 2008; Matteson et al., 2013), in particular preventing dominance of the better competitor for the available resources. Population blooms, which is a sort of disbalance between organisms competing for the same nutrients, offer dense and

monospecific host populations for viral infections (Brussaard et al., 1996). Numerous studies have demonstrated the ability of viruses to terminate population blooms (Castberg et al., 2001) or keep it at non-blooming levels (Larsen et al., 2001; Brussaard et al., 2005). However, some studies also indicate the existence of threshold level for host community, necessary to produce the significant amount of viruses (Suttle and Chan, 1994).

Intra- and interspecific relations between members of microbial loop also have a different impact on various viral variables (Simek et al., 2001; Weinbauer et al., 2003; Evans et al., 2007; Simek et al., 2010) such as infection rates and virus burst size, which in turn define their function and net effect on trophic dynamics. For instance, findings indicate positive relationship between VBR and bacterial production (BP), and dependence of both parameters on dissolved organic carbon (DOC) and total phosphorus (TP) (Lymer et al., 2008a; Lymer et al., 2008b), while ratios of C:P and N:P as well as average cell volume is negatively associated to VBR (Lymer et al., 2008a). High-nutrient status of cell and its size has been shown to increase BS, while virus capsid diameter has opposite effect (Weinbauer and Peduzzi, 1994; Weinbauer and Höfle, 1998; Gons et al., 2006). Frequency of infected cells (FIC) might be positively related to BS and negatively associated with total nitrogen (TN), chlorophyll *a* concentration (CHL*a*) and BP (Lymer et al., 2008a). This is in accordance with observations that nutrient supply favours virus production then BP remains constant (Lymer and Vrede, 2006). Other studies have found a positive and statistically significant correlation between virus-mediated bacterial mortality and bacterial production (Weinbauer et al., 2003; Boras et al., 2009).

These complex interactions led to the development of a number of theories, trying to explain the dynamics and role of viruses in the microbial loop at different organizational levels, from molecular to ecosystem (Corinaldesi et al., 2003). Parada et al. (2006) provided with a several not mutually exclusive hyphoteses, trying to differentiate the factors responsible for different patterns of virus dynamics, including size and morphology of cells, host physiological state and a complex (not necessary additive) effect of habitat type (Parada et al., 2006). While other models, to be discussed in the next section, suggest more holistic view on trophic role of viruses.

4. Concepts and models in virus ecology

The coexistence of enormous diversity of microorganisms competing for a relatively small subset of limiting resources, known as paradox of the plankton (Hutchinson, 1961), is in contradiction with classical theoretical ecology, which usually predicts competitive exclusion of less fit species

(Hardin, 1960). However, this paradox is often explained by so called bottom-up diversity control mechanism, which states that organisms coexist because they have specialized for different substrates (Øvreås et al., 2003). Recent findings based on metagenomic analysis are questioning this notion. It seems that most of the bacteria are generalists and are able to consume a broad spectrum of organic carbon, also implying that trophic interactions such as predation, might be more important factor structuring plankton communities (Mou et al., 2008).

The application of simple predator-prey model based on Lotka-Voltera equations for lytic phages was the first mathematical formalization of viruses as bacterial predators (Nowak and May, 1993). Ever since population dynamics model of phage-bacteria interactions known as Kill the Winner (KtW) model was introduced in the field of marine microbial ecology by Thingstad and Lignell (1997), it has been widely used for predictive purposes in experimental studies and for conceptual understanding of population behavior. As most of ecological models, KtW was created in a simplistic manner and continuously improved (Thingstad, 2000; Miki and Yamamura, 2005; Winter et al., 2010). KtW was proposed as a generic solution for Hutchinson (1961) paradox applicable for different communities and organizational levels (Winter et al., 2010). The model explains how species with different efficiency for nutrient uptake may coexist in the environment were they are competing for the same resources.

The functional traits of species and trade-offs between populations seem to be the most reliable explanation of species coexistence in aquatic environments nowadays (McGill et al., 2006; Litchman and Klausmeier, 2008). In the context of KtW, it means that there is a trade-off between competitive ability and resistance to phage infection (Bohannan et al., 2002; Thomas et al., 2011). Better competitor for limiting resource would be more susceptible to virus infection and lysis than less competitive, but more resistant cells. However, the cost of resistance does not necessarily correlate with the degree of resistance (Winter et al., 2010). KtW model implies at least two important aspects of virus-host ecology: 1) aquatic environments are dominating by resistant hosts, and 2) most of the viruses are produced by the rare, but very actively growing cells. These assumptions are supported by recent observations (Suttle, 2007) and experimental manipulations (Bouvier and Del Giorgio, 2007), and reveal the dominance of low activity clade SAR11 in the oceans and the appearance of previously undetected bacterial species (OUT's) under the reduced viral pressure in the mesocosms. The domination of lysogeny in slow growing species also supports the KtW

model predictions (Hewson and Fuhrman, 2007). From metagenomic data generated so called "Bank" model (Breitbart and Rohwer, 2005) is in agreement with KtW and also predicts the domination of only limited number of virus genotypes in the environment at the particular point of time. In addition, this model explains virus diversity in the context of their dynamics.

KtW appears to be relevant at different organizational scales, from strains to the communities (Thingstad, 2000; Rodriguez-Valera et al., 2009; Rodriguez-Brito et al., 2010; Winter et al. 2010), and even on the evolutionary context (Rohwer and Thurber, 2009). The co-evolution between predator and its prey in general, and virus-host pair in particular, is a key force of natural selection (known as "Red Queen" hypothesis (Van Valen, 1973), opposite to "Court Jester" model (Barnosky, 2001)) and is reflected by "boom and bust" type behaviour of both populations observed in natural environment and experimental studies (Lenski and Levin, 1985; Bratbak et al., 1992; Bidle et al., 2007).

Evolutionary success of both virus (continuing to evolve toward infectious states) and its host (continuing to evolve toward resistant state) is determined by environmental factors, including nutrients availability, host physiology and the ability to produce a sufficient amount of progeny (Chao et al., 1977; Bohannan et al., 2002; Sandaa et al., 2009; Winter et al., 2010; Jacquet et al., 2010). On the other hand, the KtW model explains not only the coexistence of organisms with different metabolic strategies, but also includes phenotypic and life cycle stages trade-offs (Bohannan and Lenksi, 1999; Frada et al., 2008). Such trait based response mechanism known as "Cheshire Cat" escape strategy (Frada et al., 2008) not only prevents host from phage infections, but has a broad implications to the trophic interactions in the food web (e.g. prey availability to grazers) and biogeochemical cycles (e.g. formation of resistant, but not blooming forms of host).

As ecological model, KtW relies on several assumptions, which often deviate from natural conditions and, thus, limits its applicability or predictive power. First of all, KtW relies on the assumption of steady-state conditions and has been criticized for incapability to explain sustained oscilations, which increases chance for extinction. The extinction of both virus and sensitive bacteria populations was observed after emergence of resistant cells in chemostat experiments (Lenski and Levin, 1985). Another major drawback of this model is that KtW takes into account only one limiting resource (Winter et al., 2010). However, most of the environments, in particular coastal zones, are highly heterogenous in terms of nutrients. Moreover, not all resources are equaly available for microorganisms (Jacquet et al., 2010). This limits the

application of this model to very complex ecosystems. The host specificity is a major prerequisite of KtW model. However, the infection of multiple host by the same phage (Sullivan et al., 2003) or host vulnerability to different phages (Holmfeldt et al., 2007) is common in aquatic ecosystems (Wichels et al., 1998), making applicability of KtW rather difficul. Recently proposed phage–bacteria infection networks (PBINs) (Flores et al., 2011; Weitz et al., 2013), which takes into account the cross-infection of multiple phages and multiple bacteria, suggest nested virus-host interaction structure (opposite to KtW) rather than a coupled interactions in isolation as assumed in the original KtW.

KtW is often accused of being oversimplistic, including the lack experimental evidence of KtW mechanism for viruses ("Kill the Killer of the Winner" (KKW) hypothesis), even though intraguild predation or cell resistance is reliable feedback mechanism of KtW (Miki and Yamamura, 2005). However, in this thesis I have leaned on the KtW philosophy to highlight the role of viruses in different environments of the studied aquatic ecosystems. All above mentioned limitations of this predator-prey model have been taken into consideration when results were discussed and interpreted.

5. Global change biology in the context of virus-host interactions

To date three universal ecological response models to global warming, i.e. spieces geographical shift, seasonal changes in life cycle of spieces and reduction of body size, are proposed and aproved by long-term observations and experimental studies (Daufresne et al., 2009 and references therein; McCauley and Mabry, 2011 and references therein). However, the prediction capacity at local scales or forecasting of species traits remains limited (Harley et al., 2006). This is probably because the effect of climate change vary on spatio-temporal scale (Parmesan and Yohe, 2003; Joshi et al., 2011; Philippart et al., 2011). The impact of temperature on predator-prey dynamics is strongly limited by thermal optima, which differs between taxonomic groups of both predator and prey, and also because relation between temperature and aquatic ectotherms is not always linear (Englund et al., 2011) as it has been assumed previously by Metabolic Theory of Ecology (Brown et al., 2004). The effect of temperature and its magnitude seem also to depend on the structure of the aquatic food web itself and the strength of interactions between organisms (Petchey et al., 2010). However, litle is known about how climate change and in particular global warming will act on the life strategy and production of viruses (Danovaro et al., 2011). Experimental studies imply that that global warming may have direct and

indirect (Eriksson Wiklund et al., 2009; Vidussi et al., 2011; Sheridan and Bickford, 2011), positive and negative (Sarmento et al., 2010; Vázquez-Domínguez et al., 2012) as well as additive or magnifying (Zhang et al., 2007; Wohlers et al., 2009) effects on virioplankton-mediated processes. However, viruses are diverse in their response to climate conditions, thus a possible feedback mechanism to global warming may also vary, including different organizational levels.

Several studies have found positive direct effect of increased temperature on virus infections (Wilson et al., 2001) and lytic virus production (Choi et al., 2009; Matteson et al., 2012), and negative on virus decay rates (Mathias et al., 1995; Yuan et al., 2011). Some other studies have not found any relations between virus activity and warming conditions (Vidussi et al., 2011). The effect of distinct climate related factors such as CO_2 induced decrease in pH level of seawater (Larsen et al., 2008) or increased UV radiation (Bouvy et al., 2011) is marginal at the community level, but has a strong selective pressure on different virus types (Wommack et al., 1996; Jacquet and Bratbak, 2003; Weinbauer, 2004; Larsen et al., 2008) or host populations (Krause et al., 2012).

Since viruses are inherent from their hosts, climate change may have indirect effect on viral activity by influencing host physiology and metabolism. It is predicted that temperature will substantially increase growth rate of ectotherms (Bickford et al., 2010; Xu et al., 2010) as well as their respiration (Yvon-Durocher et al., 2010). It is known that increased microbial activity and bacterial production stimulate virus infections. Viral shunt results in net increase of community respiration (Suttle, 2007), thus, may have additive effect to climate change mediated shift toward heterotrophic processes and carbon loss in the ecosystem. Physiological changes, e.g. associated with the reduction of the cell size, might result in decrease of the efficiency of energy transfer to higher trophic levels (Sommer and Lengfellner, 2008). Structural changes in community composition, accompanied by adaptive nature of virus infection mechanism, may favour phages which have a broad range of available hosts (Brussaard et al., 2005; Danovaro et al., 2011). On the other hand, Global Warming is likely to force domination of harmful bloom forming species (Paerl and Huisman, 2009; Liu et al., 2011; Elliott, 2012; Posch et al., 2012), including duration and intensity of blooms (Neumann, 2010; Hordoir and Meier, 2012), which, besides of increased extinction probability for other populations (Moran et al., 2010; Ezard et al., 2011), will favour density-dependent viral infections.

Consequently, this will affect competitive ability of grazers for the same hosts.

Another possibe mechanism of indirect effects of climate change on virus activity is alteration of bottom-up forcing by induced shifts in the redistribution of carbon and nutrient assimilation (Yvon-Durocher et al., 2011; Wohlers-Zöllner et al., 2012) or cascading effect due to changes in predator communities in higher trophic levels (Eriksson Wiklund et al., 2009; Pitois et al., 2012). Temperature induced response in terms of timing and magnitude of population development, both predator and prey, might create a mismatch between grazers and their food requirements (Edwards and Richardson, 2004; Durant et al., 2007), thus potentially releasing viruses from competition for the prey (Pradeep-Ram et al., 2005).

Shallow and semi-enclosed ecosystems such as the Baltic Sea is vulnerable to rising of the Sea surface temperature (SST), which is predicted to increase by 2 to 4 °C at the end of this century (Graham et al., 2008; Moran et al., 2010). The effect of climate change is extremely complex and goes beyond the direct physiological and metabolic responses (Fiorini et al., 2011). Global warming will lead to formation of new habitats (limiting their availability and quality), to which existing species will be challenged to adapt (Prowse et al., 2006; Falkowski and Oliver, 2007; Philippart et al., 2011; Winder et al., 2011; Doney et al., 2012; Pitois et al., 2012). Therefore, it will also demands for regional scale resolution predictive models and integration of coevolutionary history of predator-prey system in a particular habitat. Thus, understanding of the role of viruses in the context of the future climate in these environments is of the greatest importance.

II. MATERIALS AND METHODS

Set of different virus and bacteria variables and physico-chemical parameters were measured during the field surveys and experimental studies, and are presented in Table 1.

Table 1. List of measured variables marked as a response (R) or explanatory (E) according to their treatment in data analysis.

	Curonian Lagoon		**Kiel Fjord**	
	Observations	Experiments		
	2005–2009	**1st STOP**	**VIPBUS**	**BUS CONTROL**
Biotic state variables				
Virus abundance	R	E	E	E
Capsid size	+	–	–	–
Burst size	–	R/E	E	E
Virus morphology	+	–	–	–
Virus to bacteria ratio	R/E	R/E	R/E	R/E
Bacteria abundance	E	R/E	E	R
Bacteria cell volume	–	E	E	–
Bacteria community structure	–	E	–	–
Frequency of infected cells	–	R/E	R	R
Metabolically active cells	–	–	–	R/E
Non-viable cells	–	–	–	R/E
Chlorophyll *a* concentration	E	–	–	–
Abiotic state variables				
NO_x^-, mg/l	–	–	E	E
PO_4^-, mg/l	–	–	E	–
Temperature	E	E	E	E
Salinity	E	–	–	–
O_2, mg/l	E	–	–	–
pH	E	–	–	–
Rates				
Virus production	R	E	R/E	R/E
Bacteria growth rate	–	E	–	–
Contact rate	E	E	E	E
Contact success	–	E	E	E
Virus decay rate	–	E	–	–
Lysis rate	–	R	R	R
Bacterial mortality rate	–	R	R	R

Measured variables were used for data analysis and interpretation as predictors or response variables, depending on the research hypothesis, type of analysis or logical relationship.

All field surveys, experiments, lab preparations, sample analyses and calculations were performed by the author of this thesis. The authority of the design of two mesocosm experiments, where VIPBUS and BUSCONTROL were joint studies, belongs to the division of the Experimental Ecology at Helmholtz Centre for Ocean Research Kiel (GEOMAR, Kiel, Germany). Chlorophyll *a* concentration measurements were conducted with a help of the colleagues at Coastal Research and Planning Institute, Klaipėda University and the Nature Research Centre. Data on nutrient concentration, bacterial abundance and cell size from mesocosm experiments were provided by the division of the Experimental Ecology at GEOMAR.

1. Observational study in the Curonian Lagoon

The Curonian Lagoon (N 55°30′, E 21°15′) is a shallow transitory freshwater basin connected to the Baltic Sea through the narrow strait in its northern part (Fig. 2). It is a temperate and eutrophic water body characterized by high nutrients and chlorophyll *a* concentration and primary production (Gasiūnaitė et al., 2005; Krevš et al., 2007; Gasiūnaitė et al., 2008; Semenova and Aleksandrov, 2009). Plankton communities follow typical seasonal cycle for eutrophic water basins (Olenina, 1998; Gasiūnaitė et al., 2008), while differences in assemblage structure occur along lagoon gradient, mostly as a result of water regime (Gasiūnaitė, 2000; Gasiūnaitė and Razinkovas, 2004; Pilkaitytė and Razinkovas, 2006).

Water regime in the lagoon is strongly influenced by wind direction, discharge of the River Nemunas (~90% of the total runoff) in central part and irregularly recurrent seawater intrusions in the northern part of the lagoon (Ferrarin et al., 2008). These factors determine the distribution and residence time of water masses within the ecosystem. Consequently, three areas important for plankton communities could be distinguished in the lagoon, according to the level of physical forcing (Gasiūnaitė et al., 2008): 1) hydrodynamically active brackish water zone (spatially and temporally unstable salinity gradient); 2) the least hydrodynamically active part (freshwater stagnant zone); 3) the River-lagoon transition (with abrupt changes in current velocity, hydrodynamically active freshwater zone).

A total of three field sampling campaigns were conducted over the period of 2005–2009 in the Lithuanian part of the Curonian Lagoon (Table 2). All samples were taken using a Ruttner collector from the surface water (0.5–1.0 m depth). Samples for morphological description of virus-like particles

were collected during two days cruise in summer (31 July–01 August) 2005. A total of 13 stations for the assessment of virus community structure were sampled along the lagoon gradient (Fig. 2; SI Table 1).

Table 2. General information on field surveys performed during the period of 2005–2009 in the Curonian Lagoon.

Year	Sampling site	Coordinates	Scale of study	Sampling resolution	Sampling events
2005	13	SI Table 1	Spatial		13
2007–2008	S2	55°42'13.60"N 21°06'55.79"E	Temporal	Week*	38
	N1	55°18'08.70"N 21°00'39.80"E	Temporal	Week*	38
2009	S1	55°41'25.38"N 21°07'57.84"E	Temporal	Month	8
	N2	55°17'06.62"N 21°03'16.37"E	Temporal	Month	8
	V3	55°22'11.99"N 21°10'55.87"E	Temporal	Month	8
	R4	55°17'42.35"N 21°23'01.77"E	Temporal	Month	8
Total	**19**				**121**

***sampling during winter of 2007–2008 was performed every two weeks.**

Samples for the assessment of temporal dynamics of virioplankton and associated host communities were collected weekly from March to November 2007 and twice a month from December 2007 to February 2008 at two sampling sites (Fig. 2 S2 and N1). Monthly samples were taken from March to October 2009 at four sampling sites (Fig. 2 S1, N2, V3, R4), which represent hydrodynamically active brackish (oligohaline) water zone (S1, S2), intermediate (V3) and the stagnant central freshwater (N1, N2) parts of the Curonian Lagoon as well as the River Nemunas (R4) (Fig. 2). Overall, 19 stations were sampled in the Lithuanian part of the lagoon (Fig. 2; Table 2) during the period of 2005–2009 and a total of 121 sampling event were performed (Table 2). In every sampling event, the total abundance of viruses and bacteria as well as chlorophyll *a* concentration were measured.

2. Experimental study in the Curonian Lagoon

To estimate virus production and decay as well as the effect of viruses on bacterial community structure, the microcosm experiment and small scale incubations (hereafter referred as 1stSTOP study) were conducted in October 2010, using size fractionation approach and natural communities

from sampling sites S1, N2, V3 and R4 (Fig. 2). Two treatments, one with removed protist grazers (G-), another untreated control (C, containing both grazers and viruses), were created in triplicates and compared after the incubation period. Particle removal was done by pre-filtering water, within 1 h after sampling, through a 56-µm mesh screen to exclude large eukaryotes, followed by filtration through 5-µm pore size filter to remove most of the flagellate protists. The abundance of viruses and bacteria after filtration compared to *in situ* conditions remained unchanged and comprised 80% to 99% and 94% and 97% of natural abundance of virus and bacteria fraction, respectively ($p > 0.05$ for all T test pairs), which implies no manipulation effect on both fractions. The size fractionation reduced the impact of flagellate grazers on both viral and bacterial community and relatively increased the virus pressure on prokaryotes in G– treatment. Such situation is likely to occur as a consequence of suddenly changing hydrodynamics conditions in the limnentic part of the lagoon (Gasiūnaitė and Razinkovas, 2004). Triplicated microcosms were incubated at *in situ* conditions for 24 h in 3 L transparent plastic bags, previously cleaned with acid (1.2 N HCl) and rinsed with 100 kDa filtered lagoon water.

Virus production measurements were done (including mesocosm studies in the Kiel Fjord) using dilution approach (Wilhelm et al., 2002) adapted for tangential flow filtration (Winget et al., 2005). In all occasions 300 ml of 5 µm pre-filtered water sample was recirculated through 300 000 kDa filter by simultaneously adding virus-free water (< 100 000 kDa, 1.2 L). This procedure reduced encounter rate between viruses and their hosts and, thus, minimized new infections. Virus removal rate in virus-free water preparation varied from 52 to 91% and bacterial recovery rate was from 39 to 82% and significantly differed from the *in situ* standing stock (for both removal and recovery rates $p < 0.01$). The significant differences resulting from the cells concentration is the major disadvantage of this approach. However, the total numbers of bacteria in all cases were high enough to produce sufficient results. Triplicates of 50 ml were incubated at *in situ* conditions in the dark for 24 h. Sub-samples for bacteria and virus counts were taken every 3 h.

The decay rates of virioplankton community were assessed after Noble and Fuhrman (1997). Triplicated samples of 50 ml were filtered through 0.2 µm pore size polycarbonate filters and were incubated at *in situ* conditions over 12 h period, with subsampling (2 ml) done every hour.

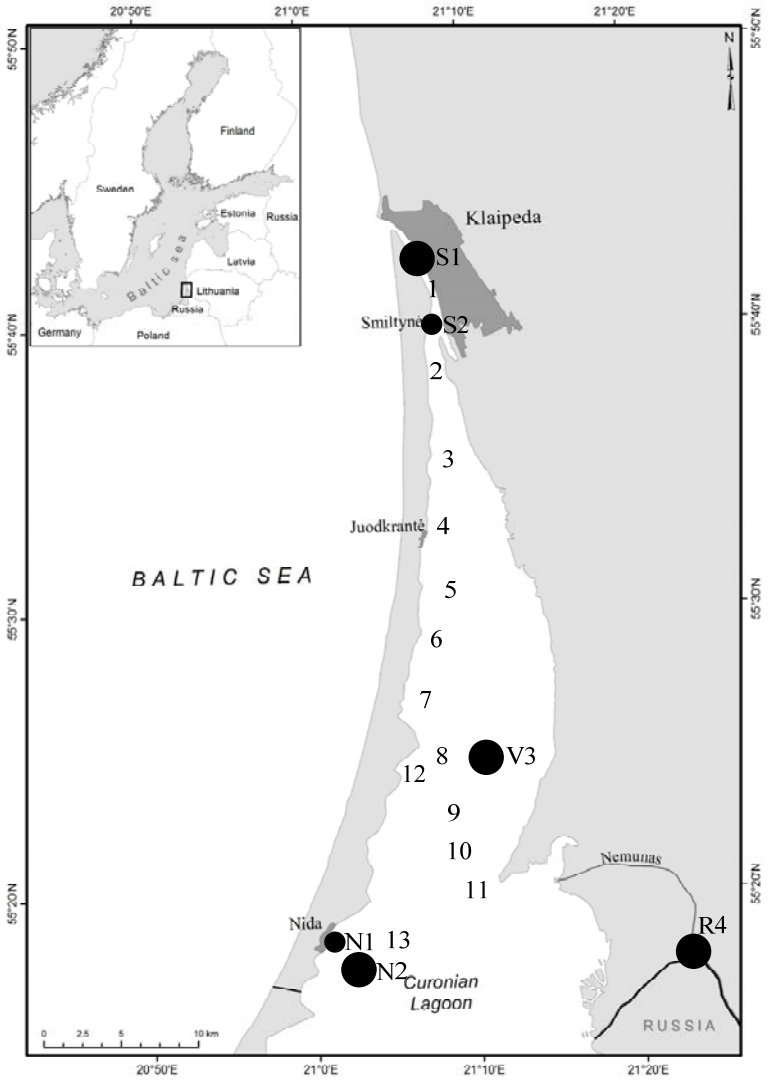


Figure 2. Location of sampling sites in the Curonian Lagoon. Numbers indicate stations for spatial survey in 2005; Large and small circles denote stations for seasonal sampling in 2007 and 2009, respectively.

3. Mesocosm experiments in the Kiel Fjord

Two indoor mesocosm studies were carried out at the Helmholtz Centre for Ocean Research Kiel (GEOMAR) in Kiel, Germany. Both dedicated to investigate the effects of temperature on natural plankton communities collected from approximately 6 m depth in the Kiel Fjord (the Baltic Sea) and placed in triplicates inside the temperature controlled climate chambers. The design of the experiments and working hypotheses were constructed based on the proposed third universal ecological response to global warming model (Daufresne et al., 2009). In both experiments, temperature gradients towards both cooling and warming were created to ensure that the observed effects is a response to temperature and not merely a stress response to a temperature change in either direction.

In the first experiment (VIPBUS, 2010), the temperatures in the three climate chambers were adjusted to 9.5°C (*Δ*-4°C treatment), 13.5°C (*in situ*°C treatment) and 17.5°C (*Δ*+4°C treatment). The temperature of 13.5°C corresponded to *in situ* conditions in the Kiel Fjord at the start of the experiment. The other temperature regimes were equivalent to *in situ* +4°C and *in situ* -4°C, thereby establishing an overall temperature gradient of 8°C. Mesocosms were filled one day before the experiment started as it took~24 h until target temperatures were reached. Light supply, covering the full range of photosynthetically active radiation (PAR: 400–700 nm), during the experiment was provided by a computer-controlled system, generating a light curve with a light/dark cycle of ~17/7 h. To initiate a phytoplankton bloom, inorganic nutrients were added to the mesocosms in Redfield stoichiometry on the first day of the experiment, with concentrations of 16.0 µmol l^{-1} NO_3^- and 1.0 µmol l^{-1} PO_4^{3-}. Mesozooplankton grazers were separately net caught (64 µm mesh size) in the Fjord and later added to the mesocosm at the density of 10 individuals l^{-1}. To minimize settling of particulate organic matter onto the bottom and maintain homogenous water body, the mesocosms were gently mixed by attached propellers. Samples for virus and bacteria abundance were taken daily, while virus production, burst size and frequency of infected cells were measured once a week.

In the second experiment (BUSCONTROL, 2011), the temperature in four climate chambers was adjusted to 8°C (*Δ*-5°C treatment), 10.5°C (*Δ*-2.5°C treatment) and 15.5°C (*Δ*+2.5°C treatment) and 17°C (*Δ*+5°C treatment). The *in situ* temperature in the Kiel Fjord at the start of the experiment was ~13°C. Thus, the adjusted temperatures corresponded to *in situ* -5°C, *in situ* -2.5°C, *in situ* +2.5°C and *in situ* +5°C, thereby establishing

an overall temperature gradient of 10°C. Light conditions, nutrient addition and mesocosm mixing were the same as in VIPBUS study.

The VIPBUS experiment was done in mesocosms with a volume of 1.4 m^3 (water depth: ~100 cm, diameter ~140 cm) and lasted for 29 days. In these enclosures, the experiments of up to 4 weeks can be performed without creating wall growth artefacts (Sommer et al., 2007). The BUSCONTROL study was performed in mesocosms with a total volume of 0.3 m^3 and lasted (12 days) until algae growth on the walls and sedimentated aggregates on the bottom of the enclosures were obvious. However, more detailed evolution of the experiments has been described elsewhere (Taucher et al., 2012), while in this dissertation the focus was done only on the small part of this multidisciplinary study.

4. Transmission Electron Microscopy

Two different techniques of sample preparation for transmission electron microscopy (TEM) were used in the present study. First was committed to identification and structural studies of virus-like particles in the Curonian Lagoon, while the second was devoted to provide information on bacteria containing assembled viruses and rough estimates of burst sizes in individual infected cell during the experimental studies.

The material, for the morphological description of phage-like particles (SI Fig. 1e,f) was taken from the surface layer of the Curonian Lagoon. The samples were collected into 1 L polyethylene bottles and kept in cold (+4°C) until further processing. In the lab, the samples were filtered through 0.45 µm pore-size membrane filter to remove larger particles. Virus-like particles were concentrated 200 times by filtration onto Pragopor 11 nitrocellulose filters (Pragochema) and preserved with glutaraldehyde (EM grade) to final 1% concentration and stored at +4°C until analysis (Sulcius et al., 2011). Three microliters of a high-titer viral stock preparation were applied onto a Formvar-carbon-coated 400-mesh palladium grid and allowed to adsorb to the grid and desiccate. The grid was then stained with 1 or 2 drops of 2% (wt/vol) aqueous uranyl acetate solution for ~30 s. The phages on the grid were counted using JEOL JEM-100S transmission electron microscope at instrumental magnification of ×15.000 and ×25.000, an accelerating voltage of 60 kV (Sulcius et al., 2011). At least 10 randomly taken micrographs (SI Fig. 1c) were analysed for each sample.

To assess the frequency of visibly infected cells (FVIC) and virus burst size in experimental studies, the whole cell approach was used with slight modifications from that described in Weinbauer and Peduzzi (1994). The material was collected into 0.5 L polyethylene bottles and gravity filtered

through 5 μm mesh. The material was concentrated to 2 ml by using tangential flow filtration system (VivaFlow 200, Sartorius) with 300 000 kDa PES filter. The bacterial recovery rate varied between 72 to 88% of the *in situ* standing stock (with no significant differences $p > 0.05$). The cells from the concentrate then were harvested on Formvar-carbon-coated 400-mesh cupper grid (Agar Scientific Ltd.) by centrifuging at 24.000 g for 30 min at 4°C. The grid was stained with a 2% (wt/vol) aqueous uranyl acetate solution for ~20 s and washed with miliQ. At least 200 cells for burst size (SI Fig. 1a,b), frequency of visibly infected cells (FVIC) (SI Fig. 1a,b) and cell size (SI Fig. 1d) were analysed using MORGAGNI 268 (FEI Company) transmission electron microscope at an accelerating voltage of 90 kV and 20–65 000× instrumental magnification. Cells that contained at least 3 clearly maturated phages were considered as FVIC.

Taxonomic and size structuring of phage-like particles is approved by the International Committee on Taxonomy of Viruses (ICTV), and was performed as proposed by Bradley (1967), Ackermann and Eisenstark (1974), Maniloff and Ackermann (1998). All the observed phages were divided in to three families (*Myoviridae*, *Siphoviridae*, *Podoviridae*) and morphological types (A1–3, B1–3, C1–3) as well as placed into 5 size classes (30–60 nm; 60–80 nm; 80–100 nm; 100–120 nm; 120–160 nm). The relative distribution of these classes was examined at all the study sites.

5. Epifluorescence microscopy

Viruses and bacteria were enumerated by epifluorescence microscopy followed by staining with SYBR Green I (SI Fig. 2) as described in Patel et al. (2007) after slight modifications. The material was collected into 50 ml polyethylene bottles and immediately fixed with 0.2 μm pore-size pre-filtered glutaraldehyde (EM grade) to final 2% concentration and left for 30 min in ice. After that samples were kept at -20°C until processing and slide preparation. The samples were processed as soon as possible, but no later than one week after the sampling. In the lab, the samples (*in situ* samples were diluted 1:10) were filtered onto 0.02 μm pore-size Anodisc filters and stained for 15 min in the dark with SYBR Green I working solution (1:400), using 0.1% (vol/vol) p-phenylenediamine as anti-fade mounting medium. The duplicate slides were counted after the preparation or kept at -20°C until analysis. Microscopy was done using Olympus IX70 or Nikon T*i* Eclipse inverted microscope at blue light excitation (488 nm). At least 200 virus-like particles and bacteria were counted by examination of at least 10 randomly selected fields per slide to take a close-up at 1000× magnifications.

Redox sensor green (RSG) staining was performed using the protocol recommended by the manufacturer (Life Technologies Corp.). The samples of 1ml were collected into 1.5 ml Eppendorf tubes and treated with 1 µl of RSG without any prior dilution of the reagent. In parallel, 1 µl of propidium iodide (PI) was added to assess membrane integrity of the cells. Tubes were gently mixed and incubated at *in situ* temperature in the dark for 10 min. After that samples were fixed with 0.2 µm pore-size pre-filtered glutaraldehyde (EM grade) to final 2% concentration and left for 30 min in ice. The samples were kept at -20°C until processing, but no longer than one month. Later samples were filtered onto 0.2 µm pore-size black-stained Nuclepore filters. Filters were then mounted on a slide with a drop of immersion oil and examined by Nikon T*i* Eclipse inverted microscope at blue light excitation (488 nm), using UV and red light excitation filters, respectively for RSG and PI. At least 20 randomly selected fields and 100 RSG-positive (RSG+) cells and PI-positive (PI+) cells were counted per slide to take a close-up at 1000× magnifications.

6. Terminal restriction fragment length polymorphism analysis

Bacterial community structure was determined by terminal restriction fragment length polymorphism (TRFLP) analysis after the descriptions by Paul et al. (2012). Water samples (250 ml) for DNA extraction were pre-filtered through 5 µm plankton mesh and collected onto 0.2 µm polycarbonate filters. DNA extraction was carried out as described by Ghiglione et al. (2005). For T-RFLP analysis, partial 16S rDNA was amplified by PCR using bacterial 16S primers 27F_FAM (AGA GTT TGA TCC TGG CTC AG) and 926R_HEX (CCG TCA ATT CCT TTG AGT) (Pandey et al., 2007). The reaction mixture consisted of 1 µl of non-diluted, half-diluted or 10-1 diluted template DNA, 25 mM dNTPs, 10 µM of each primer, 10× buffer and 0.5 units of KAPA Taq polymerase (KAPA Biosystems) for 25 µl reaction. Restriction of purified PCR products (6 h) was done separately with HhaI and RsaI restriction enzymes. Samples (2–6 µl, depending on the amount of PCR product) were mixed (1:1–1:6, depending on the amount of PCR product) with Hi-Di (Applied Biosystems, Carslbad, CA, USA) solution and 0.01 µl of size-standard 600LIZ (Applied Biosystems, Carslbad, CA, USA) and run on ABI analyser.

Each sample was measured twice and both technical replicates were used for the analysis. The fluorescent peak profiles were analysed with Peak Scanner software (Applied Biosystems). The peak data was processed and the peak profiles constructed using web-based open access programme T-REX (Culman et al., 2009). The programmes default values were used for noise

filtering, peak alignment was done using a clustering threshold of 0.6 and relative peak heights were used for constructing the peak profiles.

7. Calculations and statistical data analysis

7.1. Statistical methods

Statistical analyses were conducted and data visualized using STATISTICA v7 (StatSoft Inc., Tulsa, OK, USA), PRIMER v 6.1.12 (Primer-E Ltd., Plymouth Marine Laboratory, Plymouth, UK) and BRODGAR v 2.7.1 (Highland Statistics Ltd., Newburgh, UK) with R (2.9.1) packages. In most of the analyses differences, were considered significant at a p value of < 0.05.

Differences in mean values of measured variables between different sampling sites or experimental treatments were tested for significance by one-way ANOVA followed by Tukey's honestly significant difference (HSD) or the protected Fisher's least significant difference (LSD) tests. Test for normality (Kolmogorov-Smirnov test) and homogeneity of variance (Levene and Cochran's C tests) was performed, and data transformations (logarithmic or square root) were done (if needed) to meet requirements for parametric statistical tests (for all data compared per test). Linear and multiple (followed by stepwise backward selection) regression analyses were performed after assessment of covariation (variance inflation factor, VIF < 5) between explanatory variables. The adjusted coefficient of determination (R^2) was used as performance measure of the regression analysis.

Non-parametric multivariate analyses available from PRIMER-6 were conducted on untransformed (when the actual values caused the differences) or log (x+1) transformed (e.g. for T-RFLP) data to test for significant differences between sampling stations (ANOSIM) and to identify the contribution of variables to the observed dissimilarity between locations (SIMPER), using Bray-Curtis similarity or Manhattan distance matrices created from pair-wise comparisons of normalized and/or dispersion weighted variables. The community profiles were clustered based on the Bray-Curtis similarity matrix of log (x+1) transformed data. Because of the high number of peaks in each profile and because peak heights were used for calculation of the results, the maximum similarities between any samples were restricted to approximately 97%. The samples with replicates less similar than this were removed from the analysis. The evaluated peak heights reflect the relative amounts of different species in the sample and, thus, give more information about the bacterial community structure than presence/absence data. However, they add to the total complexity of the community profiles, thus reducing the similarity between samples.

Differences between technical replicates were tested using paired t-test. The Shannon-Wiener Index of Diversity (H′) and the Pielou's Evenness Index (J′) were calculated to quantify bacterial community diversity.

Redundancy analysis (RDA) was conducted to relate various biotic variables to the environmental factors from the selected sampling site or treatment, assuming that relation between most of the measured variables is linear. Backward selection procedure was performed to obtain a set of variables that independently and significantly explain the variation of response variables. Co-variation of explanatory variables was tested based on variance inflation factor (VIF value less than 50 was used as recommended in Brodgar software).

7.2. Calculations

The number (per ml of water sample) of viruses (VA) and bacteria (BA) collected on the Anodisc filters were determined as recommended by Noble and Furhman (1998) following corrections by Patel et al. (2007):

$$VA \text{ (or BA)} = RSF \times X \times (100/n)/V_{water}$$

where X is the average number of viruses or prokaryotes counted per field, n is the number of boxes (of 100 total) counted within the 10 ×10 reticle grid per field, RSF is the grid-reticle scaling factor (representing the ratio of the filtered area to the 10 × 10 grid visible in the eyepiece) and V is the volume of water filtered (ml). The RSF value was calculated dividing by filterable area of the Anodisc filter (2.84×10^8 mm^2) by the area of the 10 × 10 square eyepiece grid reticle (8836 mm^2).

The contact rate between viruses and bacteria was calculated using the following equation (Murray and Jackson, 1992) and corrected for bacterial abundance to estimate the number of contacts per cell on a daily basis:

$$CR\ (day^{-1}) = Sh \times 2d \times D_v \times VA \times BA$$

where Sh is the Sherwood number (1.06 for a bacterial community with 10% motile cells; Wilhelm et al. 1998), d is the equivalent spherical cell diameter (0.78–0.92 μm; this study), D_v is the diffusivity of viruses ($2.9–4.6 \times 10^{-12}$ m^2/s; for viral capsid size of 90 nm, Sulcius et al., 2011) and VA and BA are the abundances (ml^{-1}) of viruses and bacteria, respectively. If we assume that every contact resulted in infection, we will get an upper limit to the number of cells that could be infected per day (max contact success) for measured abundances of bacteria and viruses (Suttle and Chan, 1994). However, the more accurate contact success might be calculated from the number of cells lysed per day divided by the number of contacts per bacterium per day times 100 (Wilhelm et al., 2002).

Frequency of infected cells (FIC) was calculated from FVIC as determined by TEM using conversion factor of 7.1 (Weinbauer et al., 2002) with the following equation:

$$FIC = 7.1\ FVIC - 22.5\ FVIC^2$$

and data were expressed as percentages. Virus-mediated bacterial mortality (VMBM) was estimated using the model of Binder (1999):

$$VMBM = (FIC + 0.6\ FIC^2)/(1 - 1.2\ FIC)$$

This equation assumes that infected and uninfected bacteria are grazed at the same rate and that the latent period equals the bacterial generation time (Proctor et al., 1993; Guixa-Boixereu et al., 1996). Also, steady state is assumed in this model.

The volume of the cells were calculated as $V_{cell} = (\pi/4)W_{cell}^2(L_{cell} - W_{cell}/3)$, where L is the length (μm) and W is width (μm) of the cells. This formula applies both to rods and cocci (L = W). The specific bacterial growth rate (μ) was estimated as:

$$\mu(day^{-1}) = ((\log10\ BA_2 - \log10 BA_1) \times 2.303)$$

where BA_1 is the number of bacteria at the beginning and BA_2 at the end of the experiment or incubation. This calculation was applied only for samples with higher cell numbers at the end of incubations or for the periods of bacteria increase during the mesocosm experiments. Then bacterial generation time (g) calculated as:

$$g = \ln(2)/\mu$$

Virus production rates were determined from the slope of abundance vs time for independent replicates (Wilhelm et al., 2002). Then VP rates were determined from first-order and virus decay rate from the log-linear regressions of viral abundance versus time after correcting for the loss of the bacterial host between the experimental samples and the natural lagoon water community. Production was calculated as in Hewson and Fuhrman (2007):

$$VP = b \times (BA_1/BA_2)$$

where b is the slope of the regression line, BA_1 the abundance of bacteria prior dilution and BA_2 the number of bacteria after dilution. The sum of net viral production and viral decay represents gross viral production (Corinaldesi et al., 2010). Viral turnover time was estimated by dividing viral production rates by the viral abundance in samples after correcting for the dilution of bacterial hosts between the samples and the natural lagoon water community. The correction for lost bacteria was done due to necessity to account for the loss of potentially infected cells during the filtration (Wilhelm et al., 2002). The lysis rate of bacteria was calculated by dividing virus production by measured burst size at each zone of the lagoon.

8. Methodological considerations

Methodological constrains, rising from an attempt to simplify environmental complexity and elucidate the target organism, along with sample treatment and lab procedures, inevitably cause the deviations from the natural co-occurrence and inherent patterns of study objects. Some of the most important methodological limits inevitably accompanied this study are presented within the following section.

Diel and daily dynamics of virus production and, thus, their abundance at the particular moment in time, vary within hours and differ between sites (Bratbak et al., 1996; Winget and Wommack, 2009). Therefore, sampling time might impinge on the counts of virus and bacteria abundance, even though most of the sampling was performed within interval of few hours between stations. The different filtration and concentration flow rates and, thus, sample processing time were used depending on presence of organic particles in the water. Extensive sample manipulation may cause the loss of portion of ambient bacterial community, act as a stress agent and/or change their metabolic activity. The induction of prophage during sample manipulation also could not be refused. Although actual cell loss wasn't significant, the effect of sample processing on cell viability or community structure were not evaluated and no effect of sample handling was assumed.

In the microcosm experiment, using size fractionation approach, the effect of viruses on bacterial community was measured indirectly, i.e. by grazers removal, but not by manipulation of viral fraction. Maximum virus production rates in temporal studies were calculated from contact rates between viruses and their host. Despite that this method is easy and useful, in particular for high number of samples, the necessity to use conversion factor and lack of direct observation of changes in virus abundance is a major drawback of this approach (Peduzzi and Luef, 2009). The incubation time and subsampling is also important factor and depends on generation time of predator and prey. Thus, if the effect of viruses on bacterial community might occur in shorter or longer period than incubation time of 24 h, as used in this study, then interpretation of the results might be intricate.

In all calculations, the average virus burst size determined for the Curonian Lagoon and the Kiel Fjord was used. Though this is more relevant than use of average virus burst size of 50, derived from the analysis of marine environments (Heldal and Bratbak, 1991), it is known that BS can vary between different host morphologies or species (Weinbauer and Peduzzi, 1994). In addition, when the decay rate was measured, only the effect of solar radiation and natural destruction was evaluated. Other loss factors such as

attachment to the particles or non-host cell, loss of infectivity was not included in this study. This may limit the calculation of balance between virus production and decay in natural environment. Estimates of bacterial mortality were also based on conversion factors and average values and, thus, may include some deviation from actual mortality rates in natural environment. Also none of the approaches and methods applied in this study were able to discriminate between virus lytic and lysogenic infection and production. Changes in the ratio and magnitude of lytic and lysogenic production may limit the detection of the response variables to manipulated factor in the experiments.

The major limiting factor in the experimental enclosures is volume, closely related to the duration of the experiment. Due to small volume of microcosms (3 litre), the increased experiment duration would possibly create an artificial conditions (nutrient depletion), thus mask the treatment effect. On the other hand, the short-term effect of viruses is not always obvious and possible to determine by the standard methods, both microscopy (EFM, TEM) and molecular fingerprints (TRFLP). The control of environmental factors is another critical aspect of mesocosm experiments. In VIPBUS and BUSCONTROL experiments, temperature deviated between 0.4 and 0.8°C in the different treatments, even though this variability within treatments was much smaller than variability between treatments. Moreover, the lack of UV radiation during indoor mesocosm studies might have positive effect on the accumulation of viral biomass (Yuan et al., 2011), thus increasing contact probability between viruses and their hosts, in turn stimulating infection.

The application of quantitative methods, assuming that different sample type can significantly affect the efficiency and accuracy of counts (Wen et al., 2004), is of the greatest importance. Two microscopy-based methods, transmission electron microscopy (TEM) and epifluorescence microscopy (EFM), were used in this study. It is worth to note, that virus morphological diversity was likely overlooked, since filamentous or pleomorphic viruses are difficult to observe by the traditional TEM technique (Ackermann and Heldal, 2010). Coastal environments such as the Curonian Lagoon, rich in detritus and allochthonous organic substances, are known to interfere with quality of the data produced by both approaches. Detritus and organic matter could strongly and negatively influence TEM counts (Proctor, 1997) or create an unacceptable level of background fluorescence in EFM (Bettarel et al., 2000). However, the choice of appropriate fluorochromes may partly compensate miscounting. The use of SYBR Green I in this study, instead of SYBR Gold and Yo-Pro, was chosen because of short staining procedure

(reduced loss of virus particles) and ability to distinguish between DNA containing particles and humic or detritus material (emit yellow colour instead of green). However, the major drawback of EFM and SYBR Green I is that other organisms and particles containing genetic material could also be stained. Ultramicrobacteria, free mitochondria or ribosomes as well as free oligonucleotides originated from cell lysis or grazing activity are stained by the nucleic acid-specific fluorochromes. Although the first two have a minor effect on total virus counts (Hennes and Suttle, 1995; Weinbauer and Suttle, 1997), the free DNA of non-viral origin can constitute from a few up to 99% for samples from estuaries as well as other environments (Paul et al., 1991). So far there are no data on the concentration of free DNA in the water column of the Curonian Lagoon.

The use of redox dyes such as RSG in the aquatic environmental ecology is still limited (Kalyuzhnaya et al., 2008; Konopka et al., 2011) and possible limitations of this method remain to be revealed. Therefore, use of several independent methods to elucidate virus dynamics and their role in bacterial community regulation is appropriate choice to overcome methodological limitations of particular approaches (Sulcius et al., 2011).

Finally, the research and data analysis in this thesis was based on statistical methods, which do not allow to infer any nonlinear relationship between variables. This may result in overlooked patterns of the response in virus-host dynamics (Weitz and Dushoff, 2008), particularly in cases where no correlation or statistically significant differences were found among variables. Therefore, only significant changes will be discussed in the following chapters of this dissertation. In addition, both studies were limited to community level, therefore, it is very likely that changes in interactions between viruses and bacteria at lower organizational level were overlooked (Nedwell and Rutter, 1994; Ortmann et al., 2002).

III.RESULTS

1. Spatio-temporal dynamics of virioplankton variables in the Curonian Lagoon

1.1 Morphological description of phage-like particles in the Curonian Lagoon

Numerical and relative distribution of virioplankton community along the gradient of the Curonian Lagoon is presented in SI Table 2. All samples contained a mixture of morphologically different phage-like particles (SI Fig. 3). At least 26 forms of phages (morphotypes) could be distinguished by phenotypic criteria based on the relative proportions of phage head and tail (if present). Most of the viruses (SI Fig. 3) had isometric heads and contractile tails and could be assigned to the *Myoviridae* family, and further subdivided into morphotype A1 (icosahedral capsid) and A2 (elongated capsid) according to head shape (SI Fig. 3c and 3a). Morphotype A3 (a relatively more elongated capsid than A2) was absent in all samples. Bacteriophages with isometric heads and short tails were attributed to the *Podoviridae* family (SI Fig. 3y) and constituted the second largest (19%) group of bacteriophages found in the Curonian Lagoon. Only one type (C1, icosahedral capsid) of these subgroups was found at all the stations; phage-like particles belonging to subtypes C2 (elongated capsid) or C3 (a relatively more elongated capsid than C2) were not observed. Phages belonging to the *Siphoviridae* family subgroups B1 (icosahedral capsid; SI Fig. 3r) and B3 (elongated capsid; SI Fig. 1a,d) were also observed.

Multi-dimensional scaling (MDS) analysis revealed that the relative distribution of different families was dependent on their location (Fig. 3). Significant differences were observed between all the groups located relatively closer to populated areas and the groups relatively distant from these areas in the lagoon (ANOSIM $p < 0.05$). Moreover, stations located at different points of the lagoon showed a different relation to the proportional distribution of families (SI Table 2). The dominance of *Myoviridae* (no less than 65%) was evident at the study sites relatively closer to densely populated areas (Klaipėda, Juodkrantė, Nida; SI Table 1). Analysis of family contributions (SIMPER) to the differences between stations (Fig. 3) located relatively closer to populated areas (stations 1 and 2; 4 and 5; 12 and 13) and stations relatively distant from these areas (stations 3; 6–11) showed that the differences between the stations in groups 1 and 2 (Fig. 3) could be attributed to *Siphoviridae* (46.9%), those between the stations of groups 2 and 3 to *Myoviridae* (46.5%) and those between the stations of groups 2 and 4 to *Podoviridae* (48.2%). The differences between the stations located closer to

populated areas were related mainly to the distribution of two families in a sample. *Podoviridae* (47.7%) and *Myoviridae* (37.9%) contributed mostly to the differences between group 1 and 3, *Siphoviridae* (46.4%) and *Podoviridae* (43.3%) to the differences between group 1 and 4 and *Siphoviridae* (46.2%) to the differences between group 3 and 4.

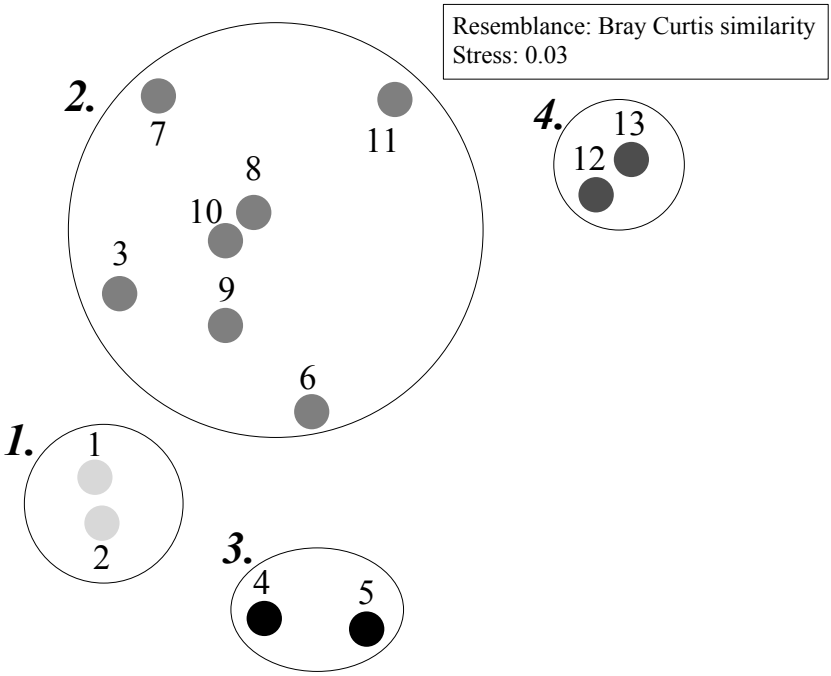


Figure 3. Multidimensional scaling (MDS) plot of variation in assemblages of virioplankton between sampling stations in the Curonian Lagoon. Circles include stations grouped based on ANOSIM analysis.

In general, tailed phages made up to more than 97% of the total number of phages detected and long-tail phages were dominant, with tail lengths from 20 nm to 630 nm (SI Table 2). Phages with isometric heads were more frequent than prolate phages, and phages with contractile tails were more frequent than phages with non-contractile tails. In terms of abundance, *Myoviridae* were dominant at all the study sites (SI Table 2). The intrusion of sea water and, thus, salinity was observed in the northern part of the lagoon at stations 1–4 (Fig. 2). However, the numerical distribution of *Myoviridae* as well as of *Podoviridae* and non-tailed phages between the freshwater and

oligohaline parts of the lagoon was insignificant ($p > 0.05$). However, the distribution of *Siphoviridae* differed significantly (T-test $p < 0.01$) between the oligohaline and freshwater parts of the lagoon.

Positive interactions ($r = 0.89$; $p < 0.01$) between the abundance of myoviruses and chlorophyll *a* (CHL*a*) concentration was observed, while podoviruses were correlated ($r = 0.57$; $p = 0.05$) with virus-to-bacteria ratio (VBR). In addition, VBR negatively correlated with the total number of bacteria ($r = -0.60$; $p < 0.05$). Total number of phages was associated neither with CHL*a* nor total bacteria density. Also, a negative correlation between *Siphoviridae* and *Podoviridae* families was found ($r = -0.65$, $p < 0.05$) along an estuarine gradient.

1.2 Size distribution of virus-like particles

Cluster analysis (based on Bray-Curtis similarity matrix) followed by SIMPROF test revealed that all the study sites in the Curonian Lagoon could be divided into three different groups corresponding to size classes (Fig. 4) or three zones corresponding to geographical distribution.

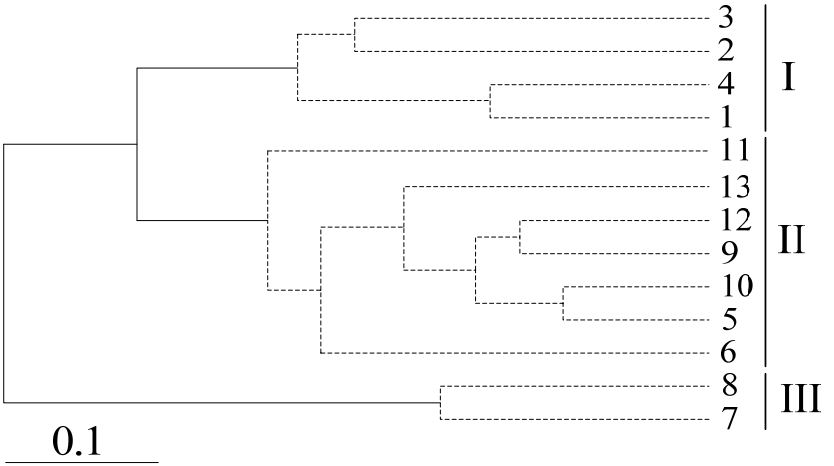


Figure 4. Dendogram of cluster analysis of variation in virioplankton capsid size classes between sampling stations in the Curonian Lagoon. Scale bar represent 10% similarity.

Group I was dominated by the 30–60 nm and 60–80 nm size fractions covered 4 stations (Fig. 4) with elevated water salinity recorded at the time of the study. Groups II and III represented the distribution of capsid sizes in the freshwater part of the lagoon. Group III covered two stations located in the

open part of the lagoon and were dominated by 30–60 nm size fractions (up to 48%). In group II, 30–60 nm size fraction did not exceed 10%; the group was dominated by 80–100 nm and 100–120 nm capsid size phages. Both the latter size fractions constituted from 48% to 70% per station, respectively.
Analysis of size class contributions (SIMPER) to the differences between groups (I, II and III in Fig. 4) revealed that group I (oligohaline zone) differed from group II (freshwater) mainly in 30–60 nm capsid size fraction (57.2%). Differences between the conditionally marine group I and the freshwater group III were due to 80–100 nm (34.9%) capsid phages. The difference between the two freshwater groups was due to the much higher relative abundance of 30–60 nm size fraction phages in group III (52%). Analysis of similarity (ANOSIM) revealed significant differences between groups I and III and between groups II and III ($p < 0.05$), whereas the differences between groups I and II were not so obvious ($p = 0.07$).

1.3 Temporal dynamics of virus abundance in the Curonian Lagoon

Biological and physicochemical variables observed during field campaigns in 2007 and 2009 at different sampling stations are provided in Table 3. Virus abundance (VA) at station S2 (2007) varied by an order of magnitude and constituted between 0.1×10^8 and 1.6×10^8 particles ml^{-1}. Spring virioplankton peak (1.2×10^8 particles ml^{-1}) was reached on 26 April and coincided with maximum bacteria numbers (4.0×10^6 cells ml^{-1}), while summer peak (1.6×10^8 particles ml^{-1}) on 12 July matched the highest CHL*a* concentration (48.6 µg l^{-1}). Maximum virus abundance (1.6×10^8 particles ml^{-1}) in autumn season (25 October) was associated neither with bacteria maximum numbers nor CHL*a* concentration. The latter two parameters reached their highest values of autumn season two weeks earlier (10 October) than virioplankton peak was observed, and constituted 4.2×10^6 cells ml^{-1} and 36.1 µg l^{-1}, respectively. The minimum spring virioplankton abundance (1.0×10^7 particles ml^{-1}) was registered on 19 May, one week after the decline of both bacteria numbers and CHL*a* concentration (Fig. 5a). Whereas summer decline in virus abundance (1.4×10^7 particles ml^{-1}) was not clearly associated with lower bacteria values (3.7×10^6 cells ml^{-1}) and high CHL*a* concentration. However, both minimums were observed after a drastic salinity changes at this station. One week in spring and three weeks in summer before minimum abundance of virus particles were registered, salinity higher than 5 PSU (6.9 PSU on 12 May and 5.3 PSU on 26 July) was recorded at this station, even though that overall virus density dynamics at this station was not related to salinity ($r = -0.08$, $p > 0.05$). The common

trend of the decline of virus and cell numbers and CHL*a* concentration were obvious in late autumn (Fig. 5a).

During the spring bloom the highest CHL*a* concentration reached 44.8 μg l^{-1}, while in summer, two CHL*a* peaks were recorded (Fig. 5a) with values up to 48.6 μg l^{-1}. Absolutely all minimum values and sharp changes in CHL*a* concentration were associated with increased seawater inflow in the lagoon, and salinity higher than 5 PSU had a significant effect on CHL *a* (ANOVA, $F = 2.93$, $p < 0.02$). After the decline of phytoplankton spring bloom (at the end of May) at station S2, bacterial cell numbers started to increase significantly until they reached an annual maximum of 5.4×10^6 cells ml^{-1} in June and didn't get below (with one exception on July 26 and bacteria numbers of 1.6×10^6 cells ml^{-1}) 2 million cells ml^{-1} until late autumn. Minimum values during the period of active vegetation (April–October) were always recorded at high salinity concentration as well (> 4 PSU).

Virus to bacteria ratio (VBR) varied from 3.4 to 69.5 (average 37.8 ± 13.8). An increase of VBR was associated with lower numbers of host cells, while minimum VBR values were led by the decline in virus abundance. There was positive relation between virus and bacteria abundance (BA) ($r = 0.59$, $p < 0.01$) and both variables were stimulated by increased temperature ($r = 0.45$, $p < 0.01$ for viruses, and $r = 0.70$, $p < 0.01$ for bacteria). The significant and negative correlation ($r = -0.53$, $p < 0.01$) was observed between cell numbers and VBR. The CHL*a* concentration did not show any linear relation to virus abundance ($r = 0.28$, $p > 0.05$), while bacteria positively responded to increased CHL *a* concentration ($r = 0.61$, $p < 0.01$). Both, bacteria (-0.33, $p < 0.01$) and chlorophyll *a* concentration (-0.55, $p < 0.01$) was negatively associated with salinity.

There were no essential differences between stations S2 and N1 in terms of absolute values or variation ranges of selected variables (Table 3), with one exception for CHL*a* concentration (T-test $p < 0.01$). The latter variable was significantly higher at N1 than at S2 station. The similar seasonal development patterns of different communities were observed at both stations. However, some differences in timing and interactions between communities at these sampling areas were observed.

At station N1 spring virioplankton peak (1.6×10^8 particles ml^{-1}) was two weeks later than at S2 and reached its maximum on 12 May. This peak matched spring highest CHL*a* concentration (60.1 μg l^{-1}), while bacteria maximum numbers (4.0×10^6 cells ml^{-1}) were observed one week later (on 19 May). There was also delay (comparatively to station S2) in summer CHL *a* maximum (73.1 μg l^{-1}), with only one pronounced peak (vs 2 peaks at

station S2) observed on 3 September (Fig. 5b). This CHL*a* peak was also coincident with relative increase in virus abundance (1.7×10^8 particles ml^{-1}).

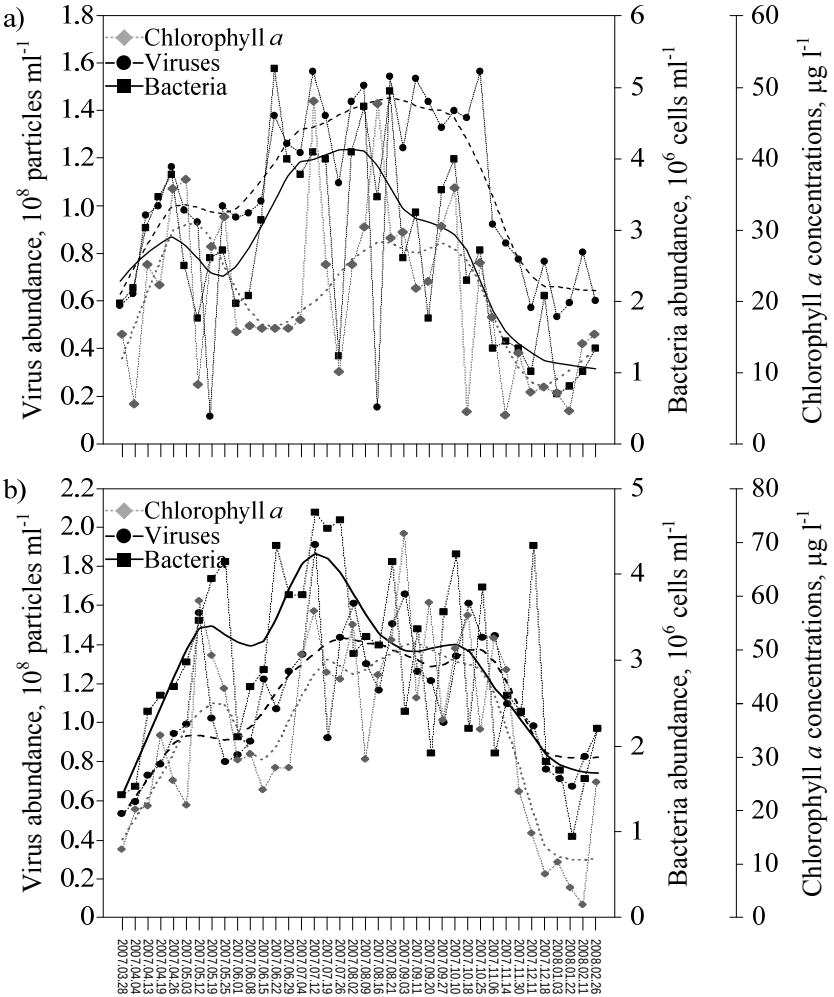


Figure 5. Seasonal dynamicss of virus and bacteria abundance and chlorophyll *a* concentration at a) oligohaline (S2) and b) freshwater (N1) parts of the Curonian Lagoon in 2007. Curves represent weighted linear least squares fit of the data points.

Table 3. Ranges of quantitative biotic variables measured in 2007 and 2009 at different parts of the Curonian Lagoon.

Parameter	Sampling stations						ANOVA
	S1	**S2**	**N1**	**N2**	**V3**	**R4**	
Year	2009**	2007*	2007*	2009**	2009**	2009**	*p* value
VA	0.3–0.6	0.1 – 1.6	0.5 1.9	0.3–0.9	0.2–0.8	0.2–0.6	0.17*, 0.83**
BA	1.0–4.4	1.1 – 5.4	0.9–4.8	0.8–5.6	0.4–6.2	0.5–4.7	0.45*, 0.99**
VBR	10.5–42.0	3.4 – 69.5	18.8–76.8	8.6–37.9	8.4–60.0	9.5–44.0	0.21*, 0.74**
CHL *a*	8.9–71.7	3.3 – 48.6	1.4–73.1	9.1–82.9	6.7–119.4	3.2–61.3	**0.00***, 0.63**
VP	0.2–2.1	0.2 – 7.8	0.3–6.3	0.1–2.3	0.05–3.6	0.05–2.3	0.09*, 0.97**
TEMP	1.2–22.7	0.4 – 22.3	0.1–22.7	1.0–21.1	1.2–22.2	0.9–22.7	0.4.7*, 0.97**
SAL	0.0–6.5	0.0 – 6.9	0.0– 0.0	0.0– 0.0	0.0– 0.0	0.0– 0.0	–

VA – virus abundance (× 10^8 ml^{-1}), BA – bacterial abundance (× 10^6 ml^{-1}), VBR – virus to bacteria ratio (VBR), CHL*a* – chlorophyll *a* (µg l^{-1}), VP – virus production (×10^8 ml^{-1} h^{-1}), TEMP – temperature (°C), SAL – Salinity (PSU).
Significant differences are marked in bold; *p value for 2007, ** p value for 2009.

The highest annual bacterial abundance (4.8×10^6 cells ml^{-1}) was observed together with the overall highest number of viruses (1.9×10^8 particles ml^{-1}) on 12 July. The minimum virioplankton abundance was also associated with the lower values of CHL*a* concentration and/or bacteria numbers (Fig. 5b). Virus-to-bacteria ratio varied from 18.8 to 76.8 and on average was 40.4 ± 14.5. As at S2 station VBR was strongly and negatively correlated with total bacterial abundance ($r = -0.61$, $p < 0.01$) and had a weak associations with VA ($r = 0.31$, $p = 0.06$). The latter parameter at this station was linearly related to both potential host communities (for bacteria $r = 0.48$, $p < 0.05$ and for CHL*a* $r = 0.78$, $p < 0.01$). And all biotic variables were positively corresponded to temperature changes ($p < 0.01$ for all variables). Bacteria numbers increased with CHL*a* concentration ($r = 0.44$, $p < 0.01$), indicating tight coupling between two communities (same as in S2 station).

Stepwise (backward selection) regression analysis showed the different relationship between virus abundance and potential host communities between the two stations (Fig. 6). Bacteria was the only significant factor ($p < 0.01$) explaining more than 1/3 of the total VA variability at S2 station (adjusted $R^2 = 33.27\%$). The equation of the fitted regression model for S2 station is: $VA = 20.15 \times BA + 4.42$. While at station N1, the only significant factor was CHL*a* concentration ($p < 0.01$), explaining almost 2/3 of the total VA variability at this station (adjusted $R^2 = 59.20\%$). The equation of the fitted regression model for N1 station is: $VA = 0.15 \times CHLa + 5.90$.

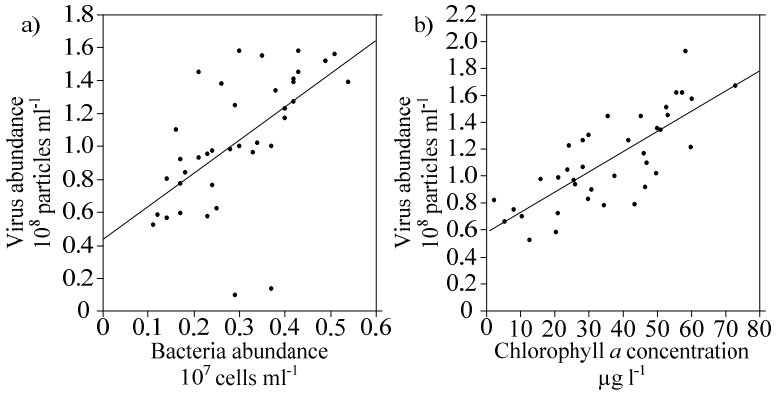


Figure 6. Relationship (Pearson's correlation) between virus density and bacteria abundance (a) and chlorophyll *a* concentration (b) at sampling stations S2 and N1, respectively.

In 2009, overall virus abundance varied from 0.2×10^8 particles ml^{-1} to 0.9×10^8 particles ml^{-1} and was relatively lower than in 2007 (Table 3). CHL*a* concentration changed from few micrograms per litre in spring and autumn to more than 100 $\mu g\ l^{-1}$ during the summer season (Fig. 7). ANOVA revealed no statistically significant differences between sampling stations in virus and bacteria numbers or CHL*a* concentration, suggesting high homogeneity within ecosystem in terms of microbial abundance. However, all biotic variables of interest showed different patterns in terms of timing and magnitude (Fig 7) as well as different interactions between communities within and between stations were observed.

At all freshwater sites (N2, V3 and R4), spring peaks for virus abundance were observed in April. While at sampling station S1 virus abundance reached its maximum values two times, in March and May (0.4×10^8 particles ml^{-1} and 0.5×10^8 particles ml^{-1}, respectively). The highest virus abundance in spring was recorded at site R4 (0.6×10^8 particles ml^{-1}) and remained stable at this sampling station until July, then it dropped down almost by factor of 2 and peaked again in August and October (Fig. 7).

Most pronounced summer peak was observed at station N2 (0.9×10^8 particles ml^{-1}). It also was the highest virus density on the annual scale observed in that year. At station V3 two maximum peaks were recorded, with virus numbers of 0.7×10^8 particles ml^{-1} and 0.8×10^8 particles ml^{-1}, reached in early (June) and late (September) summer, respectively. There was no pronounced summer peak for station R4. Summer maximum values (0.5×10^8 particles ml^{-1}) at S1 station were detected in August, with lower overall quantities of viruses compared to all of the freshwater sites (Fig. 7).

At a particular sampling site, virus abundance did not correlate with cell numbers and CHL*a* concentration over the annual scale. One exception at station R4 was observed. Virus density at this site was related with bacteria ($r = 0.65$, $p = 0.08$), which was a significant factor ($p = 0.08$) explaining ~29% of total virus variability. The equation of the fitted regression (backward selection) model for R4 station is: $VA = 6.02 \times BA + 2.85$. There were also no relations between virus abundance between different sampling sites.

In 2009, bacterial numbers showed quite consistent increase from ~0.5–1.0×10^6 cells ml^{-1} in early spring to 5–6×10^6 cells ml^{-1} in mid-summer (Fig. 7). At all stations there was one pronounced summer peak. Bacteria abundance reached their maximum values in June at stations N2 and R4 (5.6×10^6 cells ml^{-1} and 4.7×10^6 cells ml^{-1}, respectively) and in July at stations S1 and V3 (4.4×10^6 cells ml^{-1} and 6.2×10^6 cells ml^{-1}, respectively). There were no coincidence between bacterial and viral or CHL*a* peaks (except

station N2 for bacteria and CHL*a*). Bacteria numbers did not correlate with any parameter within, but followed the same trend between different sampling sites. Cell numbers at station S1 was strongly and positively ($r = 0.96$, $p < 0.01$) associated with bacteria density at station V3. Cell counts at station R4 were correlated with bacteria abundance at N2 ($r = 0.80$, $p < 0.05$) and V3 ($r = 0.64$, $p = 0.09$) stations.

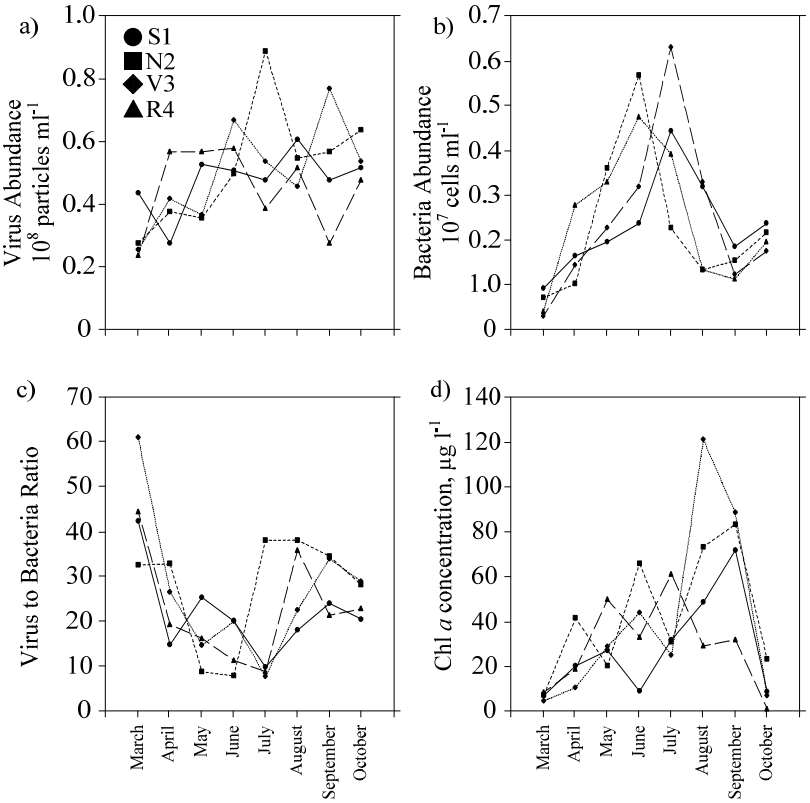


Figure 7. Seasonal dynamicss of virus (a) and bacteria (b) abundance, virus to bacteria ratio (c) and chlorophyll *a* concentration (d) at different sampling stations in the Curonian Lagoon in 2009.

VBR varied from less than 10 to more than 60 (Fig. 7). A general decrease from early spring to mid-summer was seen at all stations, with minimum values (except N2) being reached at the same time (July). This

continuous decrease in VBR was associated with constant increase in cell abundance, but its timing (VBR maximums were observed in different months, with some delay in stations S1 and V3) and magnitude was not associated with actual bacterial numbers. VBR was strongly and negatively associated with bacteria abundance at all sampling stations and even different years of study (SI Table 3). There were also positive relations between different sites in the Curonian Lagoon. VBR at station S1 correlated with V3 ($r = 0.86$, $p < 0.01$) and R4 ($r = 0.71$, $p < 0.05$) stations, and both of the latter stations correlated with each other ($r = 0.81$, $p < 0.05$). The VBR dynamics at station N2 did not follow common trend observed for other stations.

Three CHL*a* concentration peaks were observed at station N2 and two at station R4. At all stations phytoplankton blooms and the highest CHL*a* concentration were recorded in late summer. Analysis of the linear relationship showed that CHL*a* did not correlate with any other parameter in the particular sampling site, with one exception in R4 station. At this station, CHL*a* strongly and positively ($r = 0.83$, $p < 0.05$) corresponded to temperature conditions. However, similar trends in CHL*a* concentration dynamics were found between sampling stations S1 and N2 ($r = 0.70$, $p = 0.05$) and V3 ($r = 0.79$, $p < 0.05$) as well as N2 andV3 ($r = 0.84$, $p < 0.01$).

1.4 Maximal potential virus production and its dynamics

Maximum potential virus production calculated from contact rates and given burst size for each sampling site varied by factor of 72 displaying high seasonal variability and constituted between 0.05×10^8 and 7.8×10^8 viruses ml^{-1} h^{-1}. The Lowess (locally weighted least squares) analysis technique applied for time series data from stations S2 and N1 provided with an underlying pattern in both sampled areas with a general increase from spring to summer and slight decrease toward autumn and winter months (Fig. 8a). However, different timing and magnitude of VP over the annual cycle was observed between sampling stations (Fig 8a). The main difference in terms of phage production was the emerging split in the magnitude of total virus production between two environments from June to September, with significantly ($F = 5.53$, $p < 0.05$) higher values at station S2 (Fig. 8a).

There were also differences in timing and manner of community development. Spring VP peak (1.2×10^8 particles ml^{-1} h^{-1}) at station S2 (26 April) was reached two weeks earlier than at N1 (1.1×10^8 particles ml^{-1} h^{-1}) (12 May), and both peaks were not coincided with maximum bacteria numbers and CHL*a* concentration. However, at S2 station max VP values was in a good agreement with sudden seawater intrusion into the northern part of the lagoon and rapidly increased salinity (6.9 PSU).

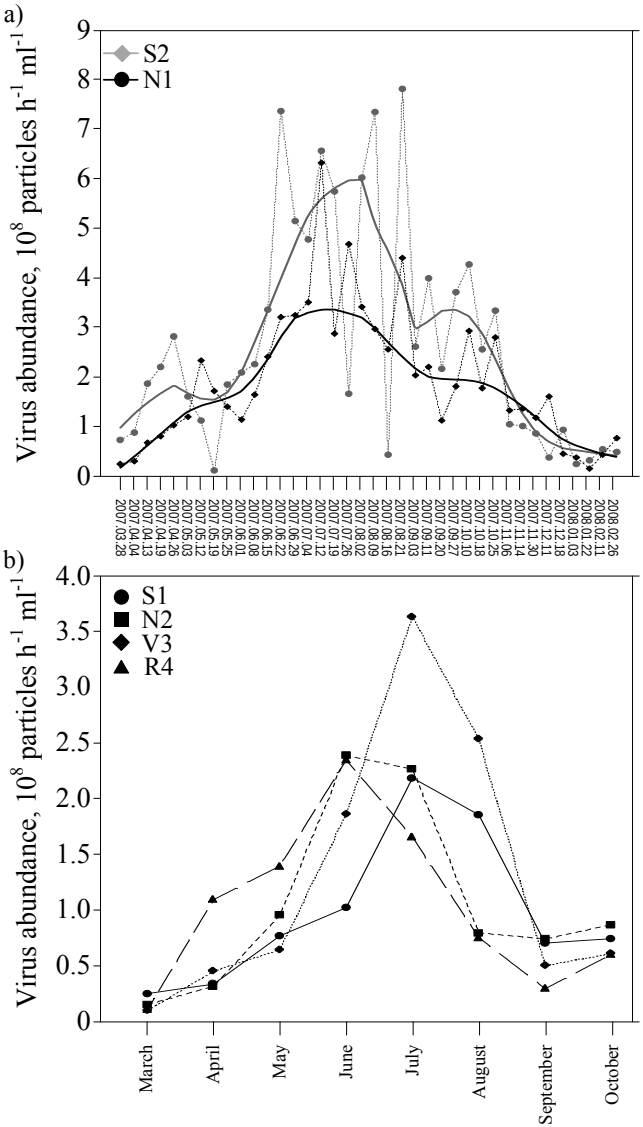


Figure 8. Seasonal dynamics of virus production in 2007 (a) and 2009 (b) at sampling stations in the Curonian Lagoon.

Over the annual scale VP was only moderately higher (ANOVA $F = 2.96$, $p = 0.09$) at S2 station than at N1 (Fig. 8a, Table 3), with more pronounced boom and bust type of community behaviour. Four drastic increases in VP at S2 station, comparatively to one in N1, were observed during the summer season in the Curonian Lagoon. The overall magnitude of virus activity was lower at N1, at least in terms of potential bacteriophage production.

Strong and positive correlations were observed between VP at stations S2 and N1 ($r = 0.73$, $p < 0.01$) in 2007 as well as between stations S1 and N2 ($r = 0.64$, $p = 0.09$), S1 and V3 ($r = 0.97$, $p < 0.01$), N2 and V3 ($r = 0.74$, $p < 0.05$) and N2 and R4 ($r = 0.84$, $p < 0.01$) in 2009, representing similar development patterns of virioplankton activity in different areas. There was also a statistically significant relation between temperature and VP in all stations in both study years, with one exception for station R4 (SI Table 4).

Virus production in 2009 also differed in its timing and magnitude (Fig. 8b). The maximum VP values for stations N2 (2.3×10^8 particles ml^{-1} h^{-1}) and R4 (2.3×10^8 particles ml^{-1} h^{-1}) were observed in June, while for stations S1 (2.1×10^8 particles ml^{-1} h^{-1}) and V3 (3.6×10^8 particles ml^{-1} h^{-1}) in July (Fig. 8b). The precocity of VP at stations N2 and R4 led to an earlier decrease during the later months of its development.

Overall virioplankton community in the Curonian Lagoon is morphologically diverse and dominated by myoviruses (~60% of all observed viruses). Virus density ranges from 0.1 to 1.6×10^8 particles ml^{-1} over annual scale and has distinct patterns in terms of timing, magnitude and interrelation to host communities. These differences are not reflected in actual abundance of planktonic viruses between sampling areas (ANOVA $p > 0.05$ for all stations over all study years) in the Curonian Lagoon.

2 Virus control upon bacterial community in the Curonian Lagoon

2.1 Distribution of virus and bacteria density-related parameters

In 1st STOP experiment, the abundance of viruses and bacteria varied from 1.62×10^7 to 2.02×10^7 particles ml^{-1} and from 1.22×10^6 to 1.81×10^6 cells, respectively. The highest number of viruses was observed in hydrodynamically active part of the lagoon (station V3, avg. $1.92 \pm 1.33 \times 10^7$ particles ml^{-1}), while bacteria were most abundant in the stagnant zone (station N2, avg. $1.73 \pm 0.09 \times 10^6$ cells ml^{-1}). The increase of bacteria density was observed along the River Nemunas–the Curonian Lagoon inlet transition (Table 4). VBR ranged from 9.91 to 16.54, with higher values in hydrodynamically active zone (Table 4). Contact rate varied from 1.08 to 1.42 h^{-1} along lagoon gradient, with success of 0.5%–1.4% per contact (Table

4). However, there were no significant differences between sampling stations in terms of virus and bacteria density-related parameters (Table 4).

2.2 Distribution of virus and bacteria trait-related parameters

Virus burst size ranged from 37 to 93 between sampling stations and was significantly (ANOVA $F = 13.91$, $p < 0.001$) higher in stagnant (station N2, avg. 79.3 ± 13.1) than in more hydrodynamically active parts of the lagoon (total avg. 51.11 ± 18.14) (Fig. 9a). Frequency of infected cells varied from 10.1% to 38.5%, with the lowest values in stagnant part (station N2, avg. 14.2 ± 3.6%). However, FIC didn't statistically differ between sampling stations (ANOVA $F = 1.43$, $p > 0.05$) (Table 4). There was also no correlation between FIC and BS in the Curonian lagoon ($r = -0.11$, $p = 0.79$) (Table 5).

Table 4. Virus and bacterial parameters (represented as mean ± standard deviation) observed in 1st STOP study. Significant differences are marked with an asterisk.

Parameter	Zone of the Lagoon				ANOVA	
					F statistic	*p* value
	S1	**N2**	**V3**	**R4**		
VA	1.76±0.84	1.79±1.49	1.92±1.33	1.77±1.79	0.83	0.51
VP	1.24±0.53	1.37±0.31	1.17±0.40	0.58±0.13	2.68	0.12
DR	3.26±0.47	0.55±0.08	3.58±1.17	2.98±0.21	4.46	**0.04***
VP/DR	2.24±1.11	14.41±3.38	2.11±1.08	1.11±1.023	34.44	**<0.00***
VT	1.67±0.71	1.87±0.55	1.49±0.62	0.80±0.24	2.08	0.18
FIC	22.45±5.13	14.20±3.61	26.67±10.48	21.62±8.79	1.43	0.31
BS	57.00±18.04	81.23±14.79	53.07±18.95	43.09±14.81	13.91	**<0.00***
VBR	12.01±1.58	10.37±0.56	13.43±2.99	12.79±2.90	1.04	0.43
CR	1.15±0.05	1.25±0.10	1.18±0.08	1.28±0.13	1.32	0.33
CS	0.99±0.45	0.69±0.16	1.01±0.27	0.62±0.14	1.59	0.27
BA	1.48±0.14	1.73±0.09	1.46±0.23	1.42±0.25	1.63	0.26
CV	0.79±0.60	0.87±0.40	0.80±0.75	0.88±0.44	0.58	0.63
LR	2.26±1.15	1.79±0.13	2.46±0.48	1.30±0.13	1.51	0.28
VMBM	35.73±12.66	18.84±5.85	50.24±33.23	35.45±21.75	1.12	0.40

VA – virus abundance (10^7 particles ml^{-1}), VP – virus production (10^6 particles ml^{-1} h^{-1}), DR – decay rate (10^{-2} d^{-1}), VP/DR – VP/DR ratio, VT – virus turnover (d^{-1}), , FIC – frequency of infected cells, BS – burst size, VBR – virus to bacteria ratio, CR – contact rate (h^{-1}), CS – contact success (%), BA – bacteria abundance (10^6 cells ml^{-1}), CS - cell volume, μm^{-3}, LR – lysis rate, 10^4 ml^{-1} h^{-1}VMBM – virus mediated bacteria mortality (%).

Bacteria size ranged from 0.03 μm^3 to 4.67 μm^3 (avg. 0.84 ± 0.57 μm^3), with a slight decrease (from 0.88 to 0.79 μm^3) in averaged cell volume along the river–inlet transition. Specific bacterial growth rate varied from 0.1 d^{-1} to 0.3 d^{-1} (avg. 0.17 ± 0.07 d^{-1}) corresponding to bacteria generation time between 2.4 and 8.7 days (avg. 5.20 ± 2.12 days). There were no differences

in cell size and growth rate between different sampling stations (Fig. 9b, Table 4). Neither BS nor FIC correlated with the cell size (Table 5).

2.3 Bacterial community composition and diversity

Gradient changes in bacterial community composition from the River Nemunas to the Curonian Lagoon inlet were observed (Global R = 0.25, p = 0.002) (Fig. 10). There was no treatment (removal of protist grazing, G–) effect (Global R = -0.003, p = 0.443) on the changes in community structure over 24 h incubation. ANOSIM revealed significant differences in community structure between S1 (hydrodynamically active brackish water part of the lagoon) and N2 (stagnant freshwater zone) (R = 0.70, p = 0.002) and R4 (hydrodynamically active freshwater part of the lagoon) (R = 0.30, p = 0.038) sampling stations. There were also differences in the structure of assemblages between the stagnant lagoon zone and the river sampling station (R = 0.62, p = 0.002). Intermediate zone (V3) between brackish and fresh water parts of the lagoon did not statistically differ from S1, R4 and N2.

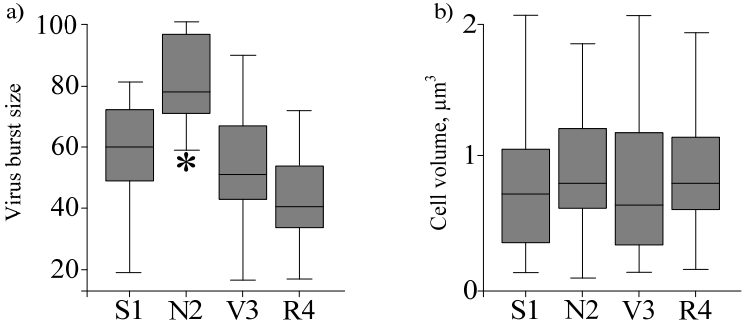


Figure 9. Box plots of virus burst size (a) and cell volume (b) at 4 sampling stations in the Curonian Lagoon. Box boundaries represent standard error, whiskers show standard deviation and lines within boxes mark the mean. Significant differences are marked with an asterisk.

Communities were different in their richness (ANOVA F = 71.83, p < 0.001) and evenness (ANOVA F = 9.53, p = 0.027) (Fig. 11). The Shannon index was the highest in hydrodynamically active brackish water part of the lagoon (S1, H' = 4.02 ± 0.05) and significantly different from all other sampling areas (HSD p<0.01 for all comparisons) (Fig. 11b). Species richness in the intermediate part of the lagoon (V3, H' = 2.88 ± 0.14) was higher than in stagnant (HSD p = 0.018) and riverine (p=0.027, H' = 2.13 ± 0.09) parts, but lower than in brackish water zone (S1, HSD p = 0.006).

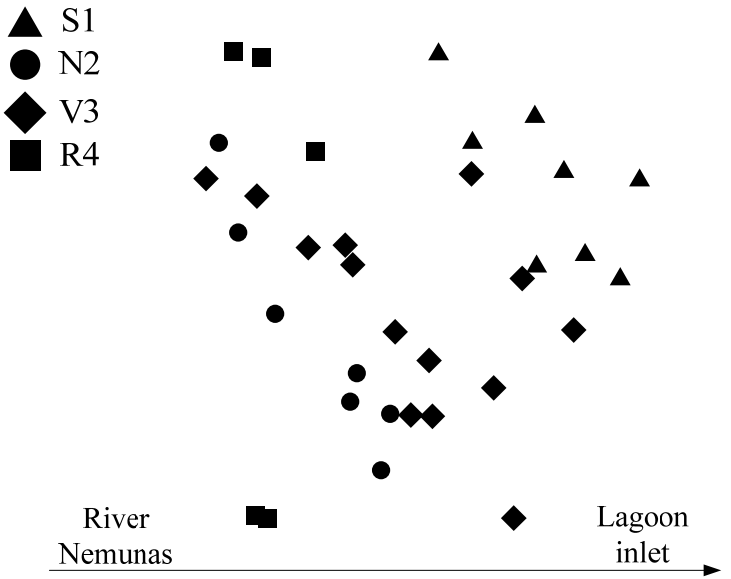


Figure 10. Multidimensional scaling (MDS) plot of the bacterial community composition as determined from TRFLP profiles at 4 sampling stations in the Curonian Lagoon. Stress value: 0.15.

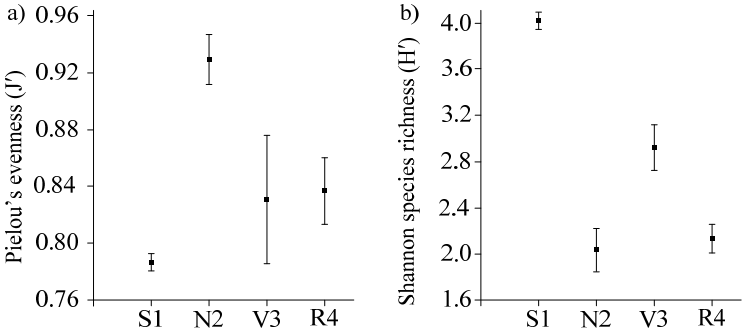


Figure 11. Box plots of the bacterial community diversity determined from TRFLP profiles represented as Pielou's evenness (J′) (a) and Shannon species richness (H′) (B) at 4 sampling stations in the Curonian Lagoon. Boxes represent mean and whiskers show standard deviation.

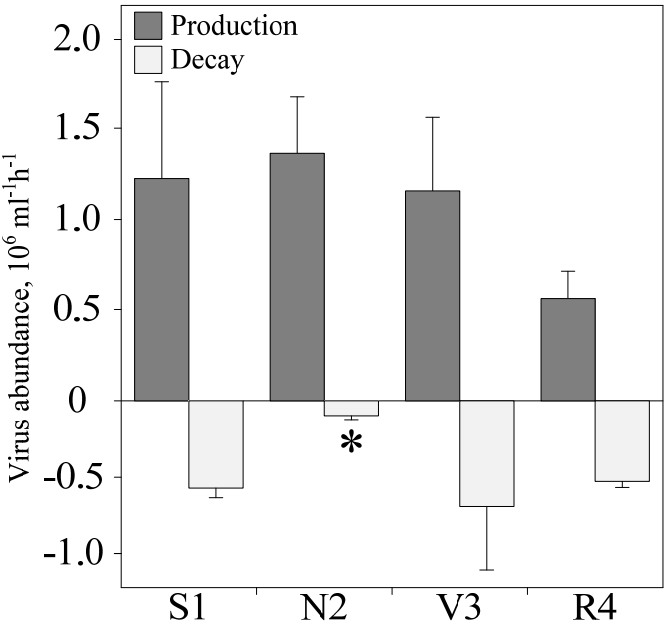


Figure 12. Column plots of virus production and decay as determined from incubation based approaches at 4 sampling stations in the Curonian Lagoon. Column represent mean and whiskers show standard deviation. Significant differences are marked with an asterisk.

The lowest diversity was observed in stagnant part of the lagoon (H = 2.04 ± 0.13), while species evenness was the highest at this sampling station (Fig. 11a) and significantly different from hydrodynamically active zones (ANOVA F = 9.53, p = 0.027). No statistically significant differences were observed between hydrodynamically active parts along the lagoon gradient in terms of species evenness (ANOVA F = 1.68, p = 0.325).

2.4 Virus-mediated bacterial mortality

Lysis rate varied from 1.05 to 3.43 × 10^4 cells ml^{-1} h^{-1} (avg. 1.95 ± 0.75 cells × 106 ml^{-1} h^{-1}), corresponding to total bacterial mortality of 12.11–88.27% in the Curonian lagoon. In general, higher values of VMBM were observed in hydrodynamically active part of the lagoon (Fig. 13). However, there were no differences in total virus mediated bacterial mortality and in the rate of cell lysis between four tested zones of the lagoon (Table 4).

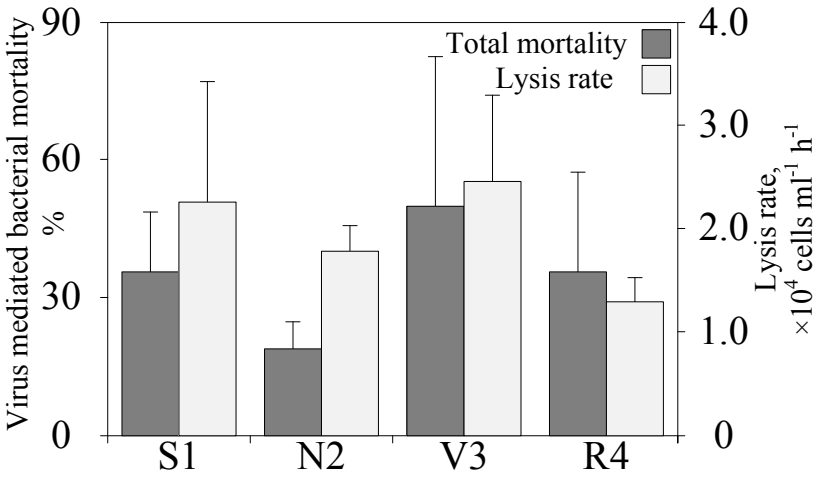


Figure 13. Column plots of total virus-mediated bacterial mortality (calculated from FIC) and lysis rate (calculated from VP and BS) at 4 sampling stations in the Curonian Lagoon. Columns represent mean and whiskers show standard deviation.

Table 5. Pearson's correlation coefficients (r) of the relationship between virus and bacteria variables in the Curonian Lagoon. Significant correlations (at $p < 0.05$) are marked in bold.

	VA	VP	VT	DR	FIC	BS	VBR	CR	CS	BA
VA	–									
VP	-0.2	–								
VT	-0.3	×	–							
DR	0.2	-0.3	-0.3	–						
FIC	0.2	-0.3	-0.3	**0.8**	–					
BS	-0.2	0.5	0.6	-0.4	-0.3	–				
VBR	**0.7**	-0.4	-0.5	**0.6**	0.3	-0.3	–			
CR	**0.6**	-0.5	-0.5	-0.2	-0.2	-0.0	0.4	–		
CS	-0.1	**0.7**	**0.7**	0.2	-0.1	-0.1	-0.2	**-0.6**	–	
BA	-0.3	0.5	0.5	**-0.7**	-0.3	0.2	**-0.9**	-0.2	0.1	–
VMBM	0.2	-0.3	-0.3	**0.8**	×	-0.2	0.4	-0.1	-0.1	-0.3

VA – virus abundance (10^7 particles ml^{-1}), VP – virus production (10^6 particles ml^{-1} h^{-1}), VT – virus turnover (d^{-1}), DR – decay rate (10^{-2} d^{-1}), FIC – frequency of infected cells, BS – burst size, VBR – virus to bacteria ratio, CR – contact rate (h^{-1}), CS – contact success (%), BA – bacteria abundance (10^6 cells ml^{-1}), VMBM – virus-mediated bacteria mortality (%).

In backward selection regression model (VMBM = 0.79 × DR + 7.98, F = 17.25, p = 0.002) virus decay rate was the only significant (t = 4.15, p = 0.002) factor explaining almost 60% (adjusted R^2 = 0.59) of total bacterial mortality.

Overall, this experimental study revealed differences in virus burst size and decay rates between different pelagic parts of the Curonian Lagoon. Virus decay was significant predictor of bacterial mortality in the lagoon. Other density and trait-related variables of both virus and bacteria communities didn't change significantly with sampling site (with exception for bacterial community structure). However, some of them were correlated (Table 5). No effect of viruses on bacterial community structure was found on the short term scale incubation experiment.

3 Virus–bacteria interactions under different temperature conditions

Results from two (VIPBUS and BUSCONTROL) indoor mesocosm experiments conducted in the southern part of the Baltic Sea (Kiel Fjord, Kiel, Germany) will be presented in the following sections of this chapter.

3.1 Response of abundance of viruses and bacteria to temperature manipulations

The abundance of viruses was an order of magnitude higher than of bacteria cells in both experiments. However, the actual numbers and its dynamics were different. In VIPBUS experiment (2010), the overall virus density varied from 1.01 to 9.92 × 10^6 particles ml^{-1}, with differences (HSD p < 0.05) found between control treatment (*in situ*°C) and treatment with reduced temperature (*Δ*-4°C) (Fig. 14a). There were no differences in virus density between cooled treatment and treatment with increased temperature conditions. In the BUSCONTROL experiment (2011) viruses constituted from 1.03 to 3.83 × 10^7 particles ml^{-1} and were an order of magnitude higher than in VIPBUS. There were no pronounced differences between treatments (Fig 14b). In both studies no overall co-variation between virus and bacteria numbers was observed.

Bacteria abundance in VIPBUS varied by a factor of 30 and constituted from 0.03 to 0.93 × 10^6 cells ml^{-1}. ANOVA indicated significant differences between treatments (F = 5.38, p < 0.01, Fig. 14a). Protected LSD test showed substantially lower amount of bacteria under the elevated temperature conditions compared to *in situ* (p < 0.05) and cooled (p < 0.01) treatments. There were no differences (LSD p > 0.05) in bacteria abundance between *Δ*-4°C and *in situ*°C treatments. In addition, cell volume in *Δ*+4°C treatment was on average (Vol_{Cell} = 0.21 μm^3 ± 0.42 μm^3) higher than in *Δ*-4°C (Vol_{Cell} = 0.05 μm^3 ± 0.01 μm^3) and *in situ*°C (Vol_{Cell} = 0.06 μm^3 ± 0.05 μm^3)

treatments. In BUSCONTROL experiment bacteria varied by a factor of 26 and constituted from 0.1 to 2.65 × 10^6 cells ml^{-1}. No significant differences between treatments were observed (Fig. 14b).

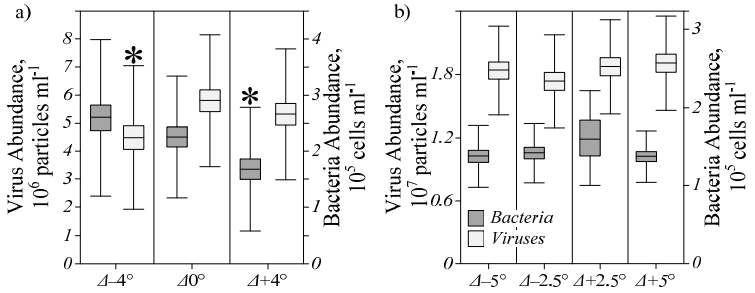


Figure 14. Box plots of virus and bacteria abundance between different temperature treatments in a) VIPBUS and b) BUSCONTROL experiments. Box boundaries represent standard error, whiskers show standard deviation and lines within boxes mark the mean. Significant differences are marked with an asterisk.

In both experiments, the overall VBR dynamics was positively (VIPBUS $r = 0.54$, $p < 0.01$; BUSCONTROL $r = 0.42$, $p < 0.01$) related to virus density and negatively (VIPBUS $r = -0.62$, $p < 0.01$; BUSCONTROL $r = -0.35$, $p < 0.01$) to bacteria abundance. However, some differences between replicates were observed (SI Table 5). There were observed an obvious difference in averaged values of VBR between two experiments (Fig. 15), with higher values in VIPBUS (overall avg. 33.98 ± 28.71). In BUSCONTROL study VBR changed from 1.51 to 128.87 (avg. 14.77 ± 10.63) and varied within, but not between treatments (ANOVA $F = 0.05$, $p = 0.98$). More pronounced differences in VBR were observed between treatments in VIPBUS experiment. In this study, VBR varied from 3.58 to 204.54 (avg. 26.76 ± 34.22) in *Δ*-4°C treatment, from 7.13 to 96.49 (avg. 34.76 ± 24.45) under *in situ* conditions and from 5.50 to 99.57 (avg. 40.99 ± 25.38) in *Δ*+4°C treatment. Unprotected LSD test ($F = 2.45$, $p = 0.09$) showed significant differences between *Δ*+4°C and *Δ*-4°C ($p < 0.05$) treatments, but not between *Δ*+4°C and *in situ*°C ($p > 0.05$) (Fig. 15). There was also no difference between *Δ*-4°C and *in situ*°C treatments. No significant correlations ($p > 0.05$ for all possible combinations) between treatments in terms of VBR dynamics were observed in two experiments, suggesting different traits of virus-host interactions.

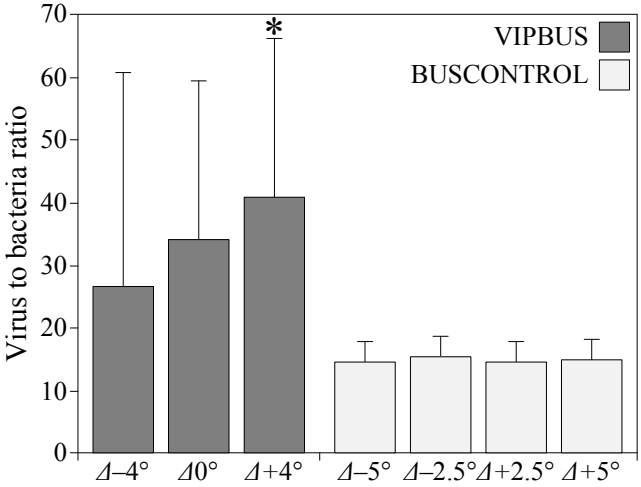


Figure 15. Column plots of virus-to-bacteria ratio at different temperature regimes in mesocosm experiments. *Δ* refers to temperature difference from the *in situ* conditions at the beginning of the experiment. Columns represent mean and whiskers show standard deviation. Significant differences are marked with an asterisk.

In addition, the number of metabolically active (RSG+) and nonviable cells (PI+) was measured in BUSCONTROL experiment. Both RSG+ and PI+ were present at the relatively low proportion of total counts (from 0.64% to 24.48% (avg. 4.63% ± 3.78%) for RSG+ and from 0.17% to 11.56% (avg. 2.45% ± 1.28%) for PI+ of total abundance) and constituted from 0.78 to 32.06 × 10^4 cells ml^{-1} (avg. 6.18 ± 4.71 × 10^4 cells ml^{-1}) and from 0.16 to 6.35 × 10^4 cells ml^{-1} (avg. 3.22 ± 0.68 × 10^4 cells ml^{-1}) for RSG+ and PI+ respectively. There were no statistically significant ($p > 0.05$) differences between temperature treatments in RSG+ and PI+ cells. Weak, but significant interactions were observed between the total number of prokaryotic cells and RGS+ ($r = 0.18$, $p < 0.05$) as well as nonviable cells ($r = -0.40$, $p < 0.01$). There were no associations between RSG+ and PI+ cells ($p > 0.05$). However, PI+ was also co-varied positively with total virus abundance ($r = 0.75$, $p < 0.01$) and negatively with virus production ($r = -0.21$, $p < 0.01$).

Distance-weighted least squares smoothing revealed different patterns in the development of each treatment over the course of the experiments (Fig. 16). In VIPBUS study, the timing of the prokaryotic cell peak showed

dependence on temperature. In warmer treatment (*Δ*+4°C) bacteria reached their maximum values earlier than in *in situ*°C and *Δ*-4°C treatments (Fig. 16c). More pronounced pattern of cell density decline and delayed recovery was observed in *Δ*-4°C treatment, suggesting that temperature decrease might have stronger impact on bacteria abundance than temperature elevation.

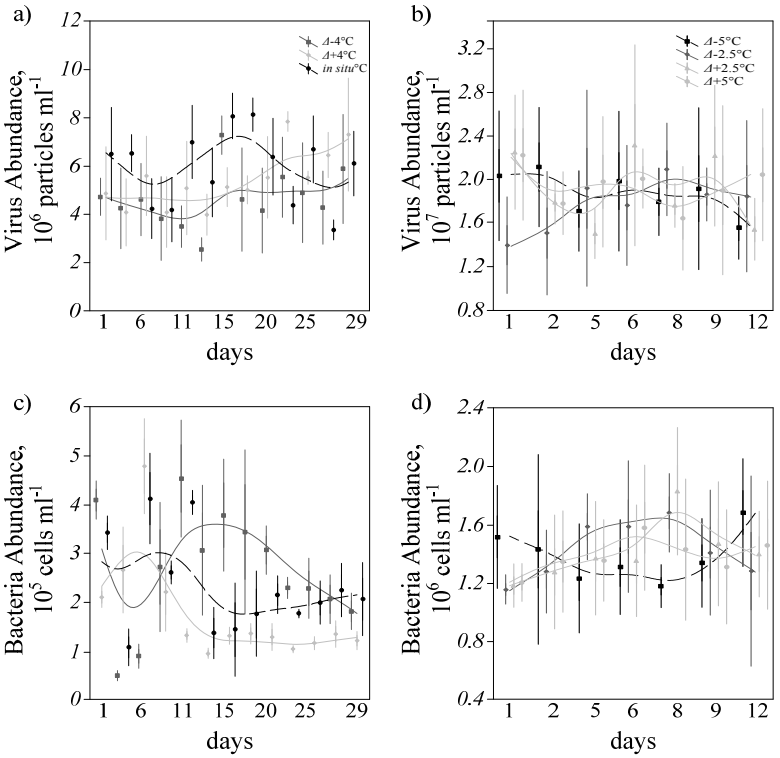


Figure 16. Dynamicss of virus (a – VIPBUS, b – BUSCONTROL) and bacteria (c – VIPBUS, d – BUSCONTROL) abundance over the course of experiment. Whiskers show standard deviation. Curves represent weighted linear least squares fit of the data points.

The development patterns of bacteria in BUSCONTROL experiment were rather ambiguous (Fig. 16d). Both treatments with elevated temperature conditions showed similar development patterns. The maximum cell density in *Δ*+5°C treatment was reached earlier than in *Δ*+2.5°C treatment; while its

magnitude was lower (Fig. 16d). The opposite development trends between two cooled treatments (*Δ*-2.5°C and *Δ*-5°C) were observed, with initial decline and elongated stagnation in terms of cell number in *Δ*-5°C treatment. Development of prokaryotic community in *Δ*-2.5°C treatment was more similar to warmer treatments.

In VIPBUS experiment, a slight decline followed by a pronounced peak in terms of virus abundance was observed in *in situ*°C treatment (Fig. 16a) and coincided with bacteria decrease (Fig. 16c). In *Δ*+4°C treatment a slight increase in virus density was observed over the period of incubation. Meanwhile, in *Δ*-4°C treatment, the shift of virus abundance had step-like manner rather than was consistently increasing (e.g. as in *Δ*+4°C treatment) (Fig. 16a) and was also coincident with bacterial decline. Over the course of BUSCONTROL experiment there were no obvious differences in development trends of virus abundance (Fig. 16b). However, more pronounced boom and bust type of community behaviour were seen compared to VIPBUS. There also were no common patterns between virus and bacteria dynamics within a particular treatment.

3.2 Virus burst size and frequency of infected cells under different temperature conditions

Virus burst size (BS) observed in VIPBUS experiment (37.63 ± 16.99) was as twice higher as in BUSCONTROL study (21.24 ± 10.93) (Fig. 17).

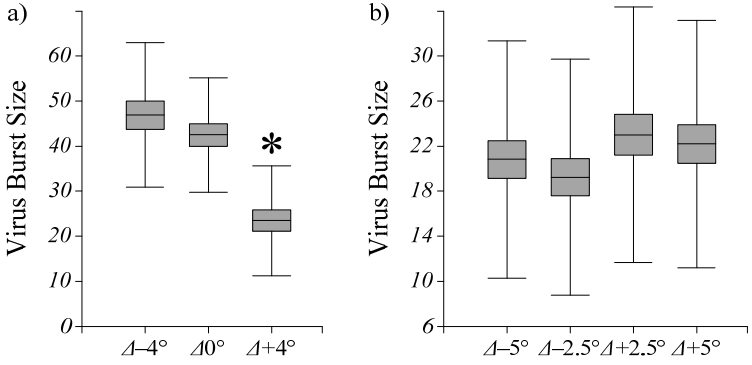


Figure 17. Box plots of virus burst size between different temperature treatments in a) VIPBUS and b) BUSCONTROL experiments. Box boundaries represent standard error, whiskers show standard deviation and lines within boxes mark the mean. Significant differences are marked with an asterisk.

Response of BS to the temperature treatment was also different between two experiments and treatments. In VIPBUS study, significant differences (ANOVA F = 22.14, p < 0.000) were observed between *Δ*+4°C and *Δ*-4°C (HSD p < 0.01) and *in situ*°C (HSD p < 0.01) treatments (Fig. 17a). BS in *Δ*+4°C was on average (23.50 ± 12.17) two times lower than in *Δ*-4°C (46.93 ± 16.01) and 1.8 times lower than under *in situ*°C (42.47 ± 12.72) conditions. The latter two treatments didn't differ between each other. Burst size was negatively (r = -0.51, p < 0.01) associated with VBR, while positively with total abundance of prokaryotes (r = 0.50, p < 0.01). Burst size co-varied with frequency of infected cells, showing negative relation between these two parameters (r = -0.92, p < 0.01). The differences in virus burst size in VIPBUS study was observed on the 20th day of the experiment (2nd sampling occasion out of 3 in total) and remained low until the end of the experiment.

In BUSCONTROL experiment, virus burst size varied within, but not between treatments (Fig. 17b). However, there were no statistically significant changes between temperature treatments (ANOVA F = 0.99, p = 0.401). Overall, burst size increased with increasing virus production (r = 0.66, p < 0.01). The BS relation to frequency of infected cell was not clearly evident (r = -0.15, p = 0.06) as for VIPBUS study. No changes were visible in BS over 12 days of BUSCONTROL study (ANOVA F = 0.21–0.80, p > 0.05 for all treatments).

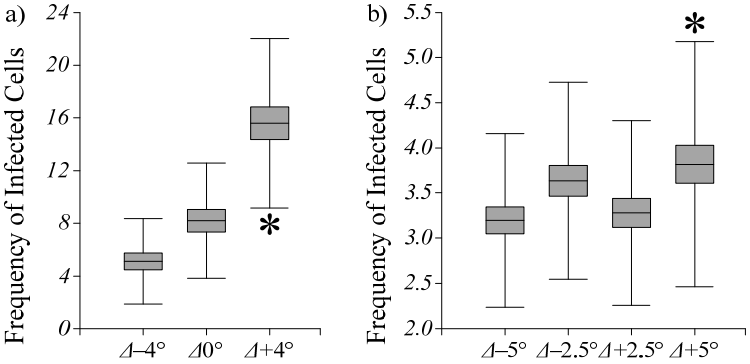


Figure 18. Box plots of frequency of infected cells (FIC) between different temperature treatments in a) VIPBUS and b) BUSCONTROL experiments. Box boundaries represent standard error, whiskers show standard deviation and lines within boxes mark the mean. Significant differences are marked with an asterisk.

Along with changes in BS there was also observed a significant difference (ANOVA $F = 28.17$, $p < 0.000$) in the fraction of virus infected cells between treatments in VIPBUS experiment (Fig. 18a). In this study, the frequency of infected cells (FIC) varied from 0.14% in the *in situ*°C treatment (avg. 8.21% ± 4.37%) to 29.24% of total prokaryotic community in *Δ*+4°C treatment (15.60% ± 6.42%). The smallest fraction of infected cells was observed in the cooled treatment and constituted on average 5.15% ± 3.25% and didn't differ significantly from the *in situ*°C treatment. Meanwhile, in *Δ*+4°C treatment, the averaged FIC values were significantly higher (HSD $p < 0.01$) than in other treatments (Fig. 18a). In VIPBUS experiment, FIC was negatively related to total abundance of bacteria ($r = -0.56$, $p < 0.01$), and positively to VBR ($r = 0.48$, $p < 0.05$). There were no associations between infection and virus density and production. The significant changes in FIC in *Δ*+4°C treatment were observed at the same time as for BS. FIC increased over two times during the course of the experiment. In *Δ*-4°C treatment, FIC showed decreasing trend from the beginning to the end of the experiment even though these changes were insignificant.

As for BS, fraction of infected cells detected in BUSCONTROL study was much lower than in VIPBUS (Fig. 18b) and constituted from 1.88% to 7.32% (avg. 3.50% ± 1.13%) of total bacteria numbers. Significant differences (ANOVA $F = 2.90$, $p = 0.037$) were observed between *Δ*+5°C and *Δ*+2.5°C (LSD $p < 0.05$) and between *Δ*+5°C and *Δ*-5°C (LSD $p < 0.05$) treatments (Fig. 18b). However, the averaged values were consistent over the course of the experiment and didn't change significantly. The positive co-variation ($r = 0.38$, $p < 0.01$) between virus infection and production were observed. FIC did not correlate with total abundance of prokaryotic cells, RSG+ and PI+ counts.

3.3 Temperature-facilitated virus production and virus-mediated bacterial mortality

Virus production (VP) varied from 2.21 to 4.95×10^5 particles ml^{-1} h^{-1} (avg. $3.25 \pm 0.57 \times 10^5$ particles ml^{-1} h^{-1}) and from 0.48 to 46.94×10^5 particles ml^{-1} h^{-1} (avg. $10.19 \pm 0.67 \times 10^5$ particles ml^{-1} h^{-1}) in VIPBUS and BUSCONTROL experiments, respectively. On average, VP in VIPBUS study was almost three times lower than in BUSCONTROL (Fig. 19).

In both experiments, the highest virus production was observed in most warmed treatments (*Δ*+4°C and *Δ*+5°C). However, there were no differences between cooled and warmed treatments in terms of VP. Only in VIPBUS study significantly lower VP was observed in *in situ*°C treatment (Fig. 19a). In VIPBUS study, VP was negatively correlated with cell density ($r = -0.45$,

$p < 0.05$), while in BUSCONTROL experiment positive interactions between VP and prokaryotic abundance were observed ($r = 0.35$, $p < 0.01$). There were also positive associations between virus production and burst size ($r = 0.66$, $p < 0.01$) and the number of infected cells ($r = 0.37$, $p < 0.01$).

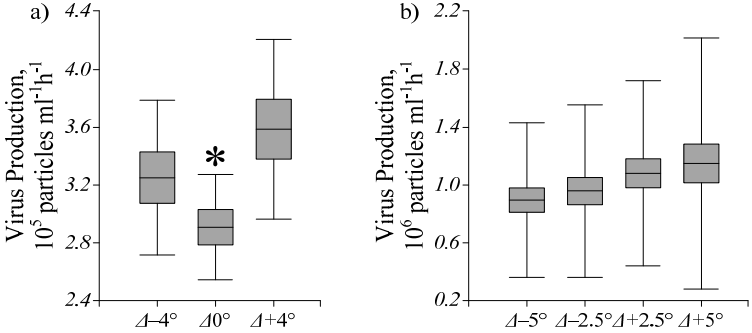


Figure 19. Box plots of virus production between different temperature treatments in a) VIPBUS and b) BUSCONTROL experiments. Box boundaries represent standard error, whiskers shows standard deviation and lines within boxes mark the mean. Significant differences are marked with an asterisk.

Significant shift in terms of virus-mediated bacterial mortality (VMBM) was observed in both experiments. In VIPBUS, VMBM varied from 3.31% to 31.15% (avg. 12.76% ± 8.76%) between treatments. Significantly (ANOVA $F = 23.87$, $p < 0.000$) higher bacterial mortality was observed in *Δ*+4°C treatment (avg. 22.38% ± 8.79%), while there was no difference between *Δ*-4°C and *in situ*°C treatments (avg. 10.03% ± 1.60% and 5.88% ± 1.85%, respectively). In BUSCONTROL study, the overall bacterial mortality rates varied within similar range (from 4.11% to 43.31%, avg. 12.23% ± 7.88%) as in VIPBUS. The differences in VMBM between treatments were less pronounced (ANOVA $F = 2.50$, $p = 0.061$). However, the unprotected LSD test showed significant differences ($p < 0.05$) in mortality rates between *Δ*+5°C (avg. 14.54% ± 9.71%) and *Δ*-5°C (avg. 10.42% ± 6.46%) and *Δ*+2.5°C (avg. 10.95% ± 6.99%) treatments.

In summary, temperature-induced significant changes in virus and bacteria densities (VIPBUS experiment) resulted in substantial changes of VBR between treatments (Fig. 15). In both experiments significant increase of frequency of infected cells was observed at temperatures elevated by 4 and 5°C from the *in situ* conditions.

IV. DISCUSSION

1. Virioplankton ecology in the Curonian Lagoon

1.1. Snapshot of virus spatial diversity and distribution patterns

Phenotypic investigations on virioplankton range from description of new phages to illustrations of biodiversity (Ackermann, 2001; Castberg et al., 2002) and remain a prime characteristics of viruses in most of unexplored aquatic ecosystems (Drucker and Dutova 2006; Jakubowska-Deredas et al., 2012). Twentysix different phages described on the basis of morphological properties, revealed pronounced differences in the phenotypic composition along the environmental gradient of the Curonian Lagoon. In general, the lagoon was dominated by the members of *Myoviridae*, which at the moment of sampling was most diverse group compared to *Siphoviridae* and *Podoviridae*. Description of 39 bacteriophages from freshwater (Demuth et al., 1993), 22 phage-host systems from the North Sea (Wichels et al., 1998), and 75 cyanophage isolates from the inshore and offshore waters of West Atlantic (Waterbury and Valois, 1993) reported similar community composition, even though these studies have not found any differences in spatial distribution of virus morphotypes. While Cochlan et al. (1993) reported that most of phages found in the northern part of the Baltic Sea (brackish waters of the Gulf of Botnia) displayed podovirus morphology and varied at both horizontal and vertical scales. This is not contradicting with results from this study, which demonstrates relative increase of podoviruses in the brackish part of the lagoon influenced by the Baltic seawater intrusions, even though the differeces between freshwater and oligohaline zones were not significant (Sulcius et al., 2011). Analysis of more than 6000 morphologically described prokaryotic viruses has shown the domination of members of the *Siphoviridae* (characterized by long and noncontractile tails), which constitutes up to 60% of the total order *Caudovirales* (Ackermann and Prangishvili, 2012). Observations from most of freshwater and estuarine environments in general report similar proportion or even the domination of siphoviruses in pelagic samples (Wichels et al., 2002; Auguet et al., 2005; Drucker and Dutova, 2006). However, some other studies from coastal ecosystems are in disagreement with this pattern (Bettarel et al., 2011; Huang et al., 2012). For instance, analysis of virioplankton assemblage from the largest estuary in the United States of America (Chesapeake Bay) have shown that even though myoviruses were more diverse than other groups in this ecosystem, the community was dominated by the members of *Podoviridae* (Bench et al., 2007; Chen et al., 2009).

It has been shown that the members of *Myoviridae* and *Podoviridae*, unlike siphoviruses, have established clear spatial distribution (Williamson et al., 2008). In the case of the Curonian Lagoon, only slight decrease in myoviruses toward central part of the lagoon was observed. However, some studies also indicate that myoviruses may be more diverse on temporal rather than on spatial scale (Boehme et al., 1993; Wang and Chen, 2004). Changes in podo- and siphoviruses were relatively more pronounced than myoviruses. The siphoviruses demonstrate a clear geographical pattern and separation between brackish and freshwater habitats ($p < 0.001$). The members of the *Siphoviridae* family strongly decreased from freshwater to oligohaline part of the Curonian Lagoon. Tailless virus-like particles were evenly distributed along the estuarine gradient, while podoviruses, similar to observations in Bach Dang estuary along salinity gradient (Bettarel et al., 2011), were more abundant in the oligohaline part of the lagoon. Together these results suggest that viruses of different morphology have diverse sensibility to the environmental gradients of the lagoon and, therefore, may have different impact on host population.

Morphology of phages was also shown to be related to host density and activity (Murray and Jackson, 1992), but not necessarily to trophic status of water body (Weinbauer et al., 1993). Host community structure may explain a significant portion of the variance in virus phenotypic diversity (Mathias et al., 1995; Bettarel et al., 2011). Most of the phages observed in the Curonian Lagoon possessed tails, which suggests that they are not viruses of eukaryotes. Cyanobacteria *Aphanizomenon flos-aquae* and *Microcystis aeruginosa* make a significant contribution to phytoplankton structure in the Curonian Lagoon (Gasiūnaitė et al., 2005), and both colony forming species were present at high densities during the study period (Olenina, 2006). According to Safferman et al. (1983), cyanophages range in size between 50 and 100 nm and most of viruses isolated in river estuaries (up to 80%) belong to the *Myoviridae* family (Lu et al., 2001). Myoviruses were the only group positively and strongly correlated with CHL*a* concentration ($r = 0.89$, $p < 0.001$), with an average capsid size of 79 nm in the freshwater part of the Curonian Lagoon. The phenotypic description of *A. flos-aquae* virus in freshwaters and the data on its role in population dynamics are very limited (Granhall, 1972; Coulombe and Robinson, 1981). However, the electron micrograph analysis of samples taken from the eutrophic lake in Sweden during the period of active vegetation of this cyanobacteria showed that *A. flos-aquae* virus has 50–60 nm capsid size and 20–30 nm contractile tail

(Granhall, 1972), thus corresponds to the *Podoviridae* family. On the other hand, Yoshida and colleagues (2006) have reported that myoviruses were extremely abundant during the bloom period of the *M. aeruginosa* in Lake Mi-kata (Japan). It was also shown that *Microcystis* isolates can produce both morphological forms of viruses (myo- and podoviruses), probably depending on the clonal composition of population (Deng and Hayes, 2008). Moreover, Holmfeldt et al. (2007) have found that viruses representing all three different families (*Myoviridae*, *Siphoviridae* and *Podoviridae*) are capable to infect same host, but success of infection was different between morphotypes. Although it is difficult to identify existing virus-host system solely from virus morphology (Lawrence et al., 2002), shift in phenotypic diversity represent differences in interactions between viruses and their hosts (Mann, 2003).

During the bloom periods an increase in density of a particular virus type also corresponds to its predatory nature in the context of "Kill the Winner" (KtW) model (Tarutani et al., 2000; Shapiro et al., 2010). That means if season related changes favour the domination of a certain host (such as *A. flos-aquae* or *M. aeruginosa* in the Curonian Lagoon), a decrease in morphological diversity can be expected. This in part explains the overall predominance of myoviruses and a positive correlation with CHL*a*, which was mostly determined by cyanobacteria (Olenina, 2006) as well as the deficiency of associations with the total number of viruses, which represent highly diverse virioplankton community. However, the quantitative evaluation of cyanophages impact on phytoplankton community structure and activity as well as on mass cyanobacteria development needs to be determined in further investigations of the Curonian Lagoon.

Finally, the results discussed in this chapter represent only the snapshot of virus diversity, which is known to change over the annual scale (Goddard et al., 2005). Moreover, between year comparisons of virus phenotypic diversity in the same ecosystem and its relation to various variables may also be different. For instance, the studies in Mono Lake (USA) showed no differences in virus diversity between habitats in one year (Brum et al., 2005), but revealed pronounced separation in next year (Brum and Steward, 2010). In addition, the lag between host and viral population dynamics or virus cross infectivity makes detection of relationships between different morphological forms and host community rather difficult (Jameson et al., 2011). At the moment, there are no data on pelagic virus diversity from the other Baltic Sea lagoons, making inter-ecosystem comparison at regional scale impossible as well.

1.2. Virioplankton – dynamic component of the microbial food web

Previous reports indicate the influence of lagoon hydrodynamic conditions, including water residence time, river discharge and wind induced water mixing on population dynamics (Pilkaityte and Razinkovas, 2006; Lesutienė et al., 2012), community structure and development patterns (Gasiūnaitė, 2000; Gasiūnaitė and Razinkovas, 2004; Kasperoviciene and Vaikutiene, 2007), metabolic and prey consumption rates (Krevs et al., 2007; Griniene et al., unpublish.) as well as spatio-temporal interdependence in terms of trophic interactions (Lesutienė et al., 2008). Important to note that these differences are not necessarily accompanied by changes in the abundance of a certain microbial assemblage (Gasiūnaitė et al., 2012), suggesting distinct habitat effect on different properties of each community (Shade et al., 2008). Most pronounced differences in virioplankton seasonal dynamics occurred between the stagnant freshwater part and the transitory oligohaline zone of the lagoon.

Assuming that viruses can originate only from the host cell through the infection process, a number of studies have argued that linear correlation between virus density and potential host community is a good predictor of virus role in trophic interactions (Proctor and Furhman, 1990; Wommack et al., 1992; Wommack and Colwell, 2000 and references therein; Parsons et al., 2012). Therefore, most important finding of the present observational study is different covariation between virus abundance, prokaryotic counts and chlorophyll *a* concentration in the stagnant (and freshwater) and the transitory (and oligohaline) parts of the Curonian Lagoon, suggesting the existence of different virus-host systems over annual scale. Such spatial differentiation of virus-hosts interactions, driven by direct or indirect impacts of hydrodynamic conditions, is common for estuarine ecosystems with pronounced environmental gradients (Vanucci et al., 2005; Pan et al., 2007; Bettarel et al., 2011). For instance, the average water residence time in the stagnant part of the lagoon (>100 days, sampling stations N1 and N2) is substantialy higher than in the transitory zone (< 20 days, sampling stations S1 and S2) (Ferrarin et al., 2008). Some studies indicate that longer residence time stimulates accumulation of viral biomass (Mari et al., 2007; Weinbauer et al., 2009). The effect of water movement on spatial distribution of virioplankton is rather ambiguous (Auguet et al., 2005; Barros et al., 2010), even though it might be predictable (Schernewski and Jülich, 2001). Corinaldesi et al. (2003) suggested that hydrodynamic conditions have indirect effect on viral distribution by influencing bacterial activity and host

cell abundance. It is also known that increased water residence time has a positive effect on CHL*a* and algal biomass accumulation irrespective to nutrient concentration (Arin et al., 2002; Istvánovics and Honti, 2012), while higher river discharge and short flushing time act negatively on phytoplankton community in estuarine ecosystems (Pinckney et al., 1998). Thus this may explain the significant differences in CHL*a* concentration between two distinct zones in 2007. On the other hand, this indicates that hydrodynamic conditions act as a selective force for determining the abundance of host community (Colombet et al., 2006; Lopes et al., 2010), which is critical for density dependent nature of virus-host interactions and phage lytic production (McDaniel and Paul, 2005).

There were no differences between freshwater and oligohaline parts of the Curonian Lagoon in terms of total bacterial abundance, while communities were distinct in their structure (as shown in 1st STOP study). The estuarine gradients are known to have a considerable effect on both virus and bacterial community assemblages (Wommack et al., 1999; Troussellier et al., 2002; Crump et al., 2004) and suggest different co-occurring virus-host systems (Frederickson et al., 2003), which, in turn, may explain the distinct co-variation between viruses and bacteria between habitats (Hewson et al., 2006). However, no repeatable patterns of virus-host dynamic were found between two sampling years. For instance, in the oligohaline part of the lagoon, bacterial density was a good predictor of VA in 2007, but not in 2009. Such inter-annual changes in co-varying factors within aquatic habitat are explained by complex indirect environmental effects on both predator and host communities (Lymer et al., 2008b; Berdjeb et al., 2011). It may also represent differences in life strategy of viruses between years (Boras et al., 2009). In most cases, good correlation between viruses and host community appear when lytic cycle is dominating over lysogenic. On the other hand, some studies have suggested that lack of co-variation between virus and host communities is limited by spatial and temporal scales, over which data set was collected (Jiang et al., 2004; Ghiglione et al., 2005), and which might result in overlooked patterns of community dynamics (Long and Azam, 2001; Seymour et al., 2006; Watson et al., 2011). Assuming that viral and bacterial activity is changing within hours (Winget and Wommack, 2009), it is not possible to detect short-term variability at low sampling resolution. This may result in observation of different interactions between viruses and their hosts on temporal scale (Tijdens et al., 2008). Thus, different sampling resolution (1 weak in 2007 and 1 month in 2009) could explain observed different

interactions between variables in study years, while differences in spatial scale are probably determined by the relative domination of distinct host groups (phyto- vs bacterioplankton) and their structures, which in turn depends on movement of water masses (Martínez Martínez et al., 2012).

Differences in timing and magnitude of virus and potential host communities' development, in addition, support the habitat control hypothesis typical for many estuarine ecosystems. Due to relatively small size of the Curonian Lagoon, global forces of microbial succession such as light regime and temperature (Rivkin and Legendre, 2001) suppose to have uniform effect on microbial assemblages. However, persisting communites at the certain habitat corresponded differently in both study years. Such differences could be explained by the distinct relation to a specific host groups (Payet and Suttle, 2008; Filippini et al., 2008; Pereira et al., 2013) as well as type of trophic interactions in general (Hewson et al., 2006) and in the Curonian Lagoon in particular (Griniene et al., unpublish.). In addition, a cross system comparison revealed that there are different drivers of viral abundance in marine and freshwater environments (Clasen et al., 2008). Thus, taken together river discharge, seawater intrusion and water residence time, these factors might significantly influence microbial community and determine different virus-host systems, which in turn diversely correspond to the environmental conditions in terms of timing and magnitude.

Another important observation of the present study in the Curonian Lagoon is emerging differences in virus production between the stagnant (N1) and the more hydrodynamically active zone (S2). The season related changes in temperature regime determine both virus and bacterial activity (Glud and Middelboe, 2004). Above the threshold temperature level trophic interactions prevail over environmental limitations (Gurung and Urabe, 1999; Demoling et al., 2007), therefore, emerging differences in microbial activity may represent distinct types of predator-prey relations (Maurice et al., 2010). Assuming that temperature limitation may occur during the late autumn-winter-early spring period and removing these data points from the analysis, much more significant differences between two sites were observed ($F = 5.53$, $p < 0.05$) in terms of VP. The production rates at S2 ($4.07 \pm 2.10 \times 10^8$ particles ml^{-1}) was twice as high as at N1 ($2.2 \pm 1.25 \times 10^8$ particles ml^{-1}) during the period of active vegation of plankton communites, confirming that season related changes have different effect on VP in different habitats. These observations are also in agreement with the regresion analysis, which showed that cell abundance in the transitory part of the lagoon, in opposite to

the stagnant zone, is important factor explaining VA dynamics, thus implies that bacteria contribute more to VP rates in this aquatic area. In addition, changes in temperature conditions or salinity are also known to induce shift from lysogenic to lytic production (Shkilnyj and Koudelka, 2007; Säwström et al., 2007). Intensive mixing of fresh and seawater creates non steady state conditions, and in turn may stimulate virus production in oligohaline part of the lagoon (Wilhelm et al., 2002; Cissoko et al., 2008).

Distinct trophic interactions in the lower part of the food web suggest another explanation for differences in VP rates (Ory et al., 2010) observed between oligohaline and freshwater parts. The experimental studies showed that nano-fraction sized organisms are preferentialy grazed by microzooplankton in oligohaline part of the lagoon (Griniene, 2012). This may significantly reduce HNF abundance and thus bacterivory rates in this area. In turn, it will also release viruses from HNF pressure and stimulate virus infection and production (Miki and Yamamura, 2005). On the other hand, in the freshwater part of the lagoon, microzooplankton exhibits higher grazing pressure on organisms from pico-size fraction (Griniene, 2012), thus suggesting that viruses and grazers are competing for the same host. Consequently, it may lower overall virus production. It is also worth to note that approach used to infer virus production clearly overestimates VP rates, even thougth allows rapid and easy calculations (Peduzzi and Luef, 2009). However, it provides a potential carrying capacity of host (Deng et al., 2012) and is probably more important in revealing viral activity patterns rather than giving the actual VP values. In addition, modelling studies on virus production have indicated that virus and bacteria abundance were best predictors of VP (Winter et al., 2005), proving that use of contact rate (R) to infer VP is reasonable. Other techniques used to estimate VP also rely on a number of different assumptions (Winget et al., 2005) and have specific drawbacks, therefore, some reports indicate that calculation of VP rates is methodology sensitive (Guixa-Boixareu et al., 1999). Moreover, several of these techniques are limited in field application (Steward et al., 1992a, 1992b) or for large amount of samples (Wommack and Colwell, 2000).

1.3. Virus role in trophic interactions within the microbial loop

The overall impact of viruses on the bacterial mortality is attributed to their 1) abundance, 2) infectivity and 3) phage loss rates, while interplay of these parameters allows coexistence of virus–bacteria system in the natural environments (Heilmann et al., 2010). The only parameters that showed pronounced differences between the stagnant and the transitory zones in

1st STOP study were virus burst size (BS) and virus decay rates (DR), respectively corresponding to virus infectivity and virus loss categories.

Virus decay rate was the only significant factor explaining bacterial mortality in the Curonian Lagoon, and, therefore, indicate the importance of virus loss processes in trophic interactions. Solar radiation is a primary source of virus loss in photic zone of the pelagic ecosystem (Cottrell and Suttle, 1995). However, different viruses display different sensitivity to UV radiation (Jacquet and Bratbak, 2003), implying that effect of sunlight at the community level might depend on the structure of virus assemblages and probably is related to virus adaptability in a particular environment (Linden et al., 2007). On the other hand, differences in water turbidity may mitigate virus loss to UV radiation as well (Clokie and Mann, 2006; Bettarel et al., 2009). It is also known, that bacterial diversity has negative effect on contact success, thus increase virus removal rate through virus attachment on the wrong host (Brussaard et al., 2005). Therefore, distinct rates of virus decay among different areas in the lagoon might be a result of different structure of virus (Sulcius et al., 2011) and host assemblage (1stSTOP study) or changes in turbidity along the lagoon gradient (Krevš et al., 2007).

Decay rates of viruses in the Curonian Lagoon were within the range reported for other coastal environments (Heldal and Bratbak, 1991; Noble and Furhman, 1997). However, the calculated decay rate is likely underestimating total viral loss in the lagoon because of methodological limitations and environmental constrains. The approach used in this study besides cell removal also involve the exclusion of large amount of various types of particles and colloidal substances known as a major source of virus loss, particularly in the eutrophic environments (Proctor and Fuhrman, 1991; Bongiorni et al., 2005). In the Curonian Lagoon, as in many other, supply of these particles as well as their sedimentation rates are strongly influenced by the riverin discharge (Voss et al., 2000; Cetkauskaite et al., 2001; Trimonis et al., 2010), suggesting likely higher virus removal from the pelagic part in the hydrodynamically more active zones. In addition, loss rate of viruses indirectly depends on hydrodynamic conditions and water mixing (Bettarel et al., 2009), in particular through the host diffusivity (Gourley and Kuang, 2004) and nutrient supply (Thingstad, 2000; Motegi and Nagata, 2007) and requires much higher values of decay rate in well mixed environment to ensure stable virus-host coexistence in the habitat (Heilmann et al., 2010).

In fact, virus has two types of "habitats" to interact with. One habitat is the natural environment outside the cell, with net effect on virus loss

(represented by virus decay rates). The second "habitat" is inside the cell, with net effect of virus production (represented by virus burst size). These two "habitats" are linked by the probability of the successful infection (FIC), which depends on the community structure and host growth rate. Differences in virus burst size might be explained by fluctuations in host growth rate (referred as cell physiological state control hypothesis) more than in prey availability (Middelboe, 2000; Young et al., 2000). Neither community structure nor bacterial growth rate or abundance were able to explain the differences in BS between sampling sites of the Curonian Lagoon, thus do not supporting this scenario. In addition, low growth rate and long doubling time may also explain lack of differences between experimental treatments. Since BS is cell limited (total volume of viruses cannot exceed total volume of cell) some studies have found good correlation between cell size, virus capsid diameter and the number of internal viruses (Weinbauer and Peduzzi, 1994; Weinbauer and Höfle 1998; Brum et al., 2005). However, it doesn't seem that cell size limited BS hypothesis (Parada et al., 2006) may explain differences observed in the Curonian Lagoon, since no changes in cell volume were found. It was not possible to test the effect of virus latent period on BS within experimental design used in this study. However, it is known that BS increase with latent period (Abedon et al., 2001) and both optima depend on host quality in terms of internal resources (Bouvier and Maurice, 2011). On the other hand, viruses displaying shorter latent period may have competitive advantage for the same host (Abedon et al., 2003; Shao and Wang, 2008), even though it may result in lower BS. It is also worth to note the existence of optimal latent period, which results in the highest possible BS within the shortest possible time (Wang, 2006). Optimal latent period is changing with host growth rate (Hadas et al., 1997) and not always depends on environmental conditions (Brussaard et al., 2004), even though water mixing might reduce BS (Dennehy et al., 2007). High BS together with high FIC and their strong positive relation probably would suggest existence of superinfection, which is destabilizing factor of population ecology (Gons et al., 2006). However, such situation is expected during the bloom periods (Thingstad and Lindell, 1997) and under non resource limited conditions (Weinbauer et al., 1993), thus not applicable for the conducted study.

Habitat control of burst size hypothesis covers complex interaction of trophic structure, host productivity and environmental gradients, including hydrodynamic-mediated water mixing and nutrient supply (Parada et al., 2006 and references wherein). The lack of association between BS and host

physiology and activity as well as virus capsid size and abundance distribution suggest that interactions between virus and bacteria can not be directly attributed to a single parameter and most likely is a result of uniqe habitat properties, including water residence time, sediment types and water-sediment interactions, which differ between studied areas in the Curonian Lagoon (Ferrarin et al., 2008; Žilius et al., 2012).

However, differeces in virus burst size do not imply the differences in carbon flow within microbial food web, particularly when host diversity is high. This is because irrespect of BS only infected cell is to be lysed, and if FIC is equal between environments, potentialy the same amount of nutrients will be released to water column. Higher BS ensure higher VP per cell, but due to limited supply of new hosts in stagnant environments, resistance may occur, consequently lowering rate of successful infections (Thyrhaug et al., 2003; Brockhurst et al., 2006). Therefore, higher BS in this case might be a strategy of viruses to ensure high VP rates in a community dominated by resistant hosts (Levin et al., 1973; Gons et al., 2006). Possibly higher grazing rates on pico fraction organisms in the stagnant part of the lagoon (Griniene, 2012) may also induce higher BS as well as mask the differences in FIC, due to higher proportion of infected cells lost to protists grazing (Miki and Yamamura, 2005). On the other hand, in well mixed environments viruses with smaller burst sizes might infect more successfully due to continuous supply of new hosts, thus higher BS may also reflect limited host availability (Kerr et al., 2006). Finally, this illustrates the differences of virus activity between organizational levels (Staniewski et al., 2012), e.g. increased virus efficiency per infected cell in the stagnant zone of the lagoon (higher BS), but equal between habitats at ecosystem level (no differences in FIC).

2. Additive effect of viruses on heterotrophic processes in the microbial loop under the future climate

Even though the ecological response to global warming is evident (Walther et al., 2002; Sheridan and Bickford, 2011), it is still difficult to predict net outcome of virus-mediated processes on the ecosystem functioning in the future climate (Danovaro et al., 2011). However, the results of the present study suggest possible additive effect of viruses on increased carbon loss within microbial loop under global warming conditions (Kritzberg et al., 2010; Faithfull et al., 2011).

2.1. Trait- and density-dependent response of virioplankton to global warming

The predicted shifts in water acidity and temperature due to climate change (IPCC, 2007) seems will have only moderatly direct effect on virioplankton infectivity (Feng et al., 2003; Bertrand et al., 2012). However, the rising sea surface temperature (SST) will change trophic structure of the food webs (Shackell et al., 2010; Yvon-Durocher et al., 2011), primarily by affecting top predators (Kordas et al., 2011) or by uncoupling predator-prey interactions (Durant et al., 2007). It will also result in increased productivity (O'Connor et al., 2009) and reduced size and biomass of single-cell organisms (Daufresne et al., 2009; Boyce et al., 2010; Sjöstedt et al., 2012). Consequently, this will create trophic cascade (Baum and Worm, 2009; Casini et al., 2009), which alters the metabolic balance of ecosystem toward heterotrophic processes in the lower part of the food web and, therefore, will lower system ecotrophic efficiency (Berglund et al., 2007; Yvon-Durocher et al., 2010). As Metabolic Theory of Ecology (MTE) predicts (Brown et al., 2004) and as a number of studies have proved, rising temperature will have direct effect on the bacterial growth efficiency (BGE), which consequently will induce changes in community structure and prokaryotic abundance. Since the relation between viruses and their hosts is changing in parallel with host dependence on temperature regime (Pradeep-Ram et al., 2005; Pradeep-Ram et al., 2010), it is likely that virus response to global warming will be driven by alterations in virus-host density- and trait-mediated processes (Bouvy et al., 2011; Fuhrman et al., 2011; Degerman et al., 2013).

On the one hand, host availability benefits infections and lytic phage production irrespect to environmental factors (Colombet et al., 2006), while on the other, increased virus pressure relies on host activity, in turn strongly determined by ambient conditions (Middelboe, 2000), and isn't necessarily reflecting in changes of overall abundance of predator and prey (Bonilla-Findji et al., 2008). This indicates that factors limiting density- and trait-mediated processes of both virus and host communities are different (Bouvy et al., 2011; Winder and Sommer, 2012). Therefore, virioplankton response to global warming will also depend on which process will be affected more by the changing temperature in the certain habitat.

It is also possible that changes in prey or predator densities will be balanced by trait-mediated effects (Kunert and Weisser, 2003; Werner and Peacor, 2003) as a result of complex interplay of factors contributing to global change (Lang et al., 2012). Therefore, net outcome of viral activity

might be restrained at the community level. However, results from this study revealed similar trends in virus infection rates in the presence of different host communities between two mesocosm experiments (Taucher et al., 2012) indicating that warming in general will benefit the efficiency of viral shunt (Winter et al., 2008), thus consequently resulting in increased community respiration rates (Middelboe and Lyck, 2002) and enhanced loss of energy and carbon within microbial loop (López-Urrutia et al., 2006).

Taken together the results suggest possible common patterns of direct and indirect as well as positive and negative response of virus-host interactions to changing temperature conditions in terms of their abundance, infection and interrelation (Table 6). Taken separately, dynamics of viral and bacterial variables within particular treatment (Table 6) reveals possible traid-offs between density- and trait-mediated effects (Chao et al., 1977, Messenger et al., 1999; Bohannan and Lenski, 2000; Kerr et al., 2006) as a function of temperature regime. Finally, assuming that virus-mediated processes will increase carbon flow within microbial loop stimulate resulting in higer respiration rates (Suttle, 2007), increased bacterial mortality due to viral lysis will have additive effect on the reduction of bacterial growth efficiency and positive feedback between rising SST and CO_2 production (Vázquez-Domínguez et al., 2007; Kritzberg et al., 2010).

2.2. Common patterns of temperature mediated virus-host interactions

Temperature has a substantial effect on the ectotherms, limiting their metabolism and reproduction rates (Woodward et al., 2010; Arendt, 2011; Hjernquist et al., 2012). Since prokaryotic activity such as growth rate and substrate assimilation is a threshold temperature dependent process (Shiah and Ducklow, 1997), different patterns of host dynamics (Pomeroy and Wiebe, 2001; Søndergaard and Danielsen, 2001) and viral activity (Wilson et al., 2001; Jiménez-Mercado et al., 2007), might be expected under cooling and warming regimes.

Microbial activity and community development change dramatically under and above temperature threshold (Nedwell et al., 1999; Vrede, 2005). While threshold level itself differs between environments and populations (Simon and Wünsch, 1998; Apple et al., 2006; Hall and Cotner, 2007; Kirchman et al., 2009). In both mesocosm experiments, the initial temperature matched to the decadal mean (1993–2002) of the SST in the Kiel Fjord and constituted 13–13.5°C, suggesting that existing communities were under the temperature threshold (14°C, as proposed for temperate regions) limiting their activity (Ochs et al., 1995; Felip et al., 1996). The following

temperature manipulations (in both directions) were done according to the proposed future climate scenarios for the Baltic Sea (Neumann, 2010). Therefore, to reveal common pattern of virioplankton response as well as to evaluate to what extent nutrients contribute to variation of virus and bacteria variables under different temperature conditions, the data from two experiments were divided into two groups (under and above) corresponding to the temperature threshold of 13.5°C (consistent to *in situ*°C treatment in VIPBUS study, which was not included into any of two groups).

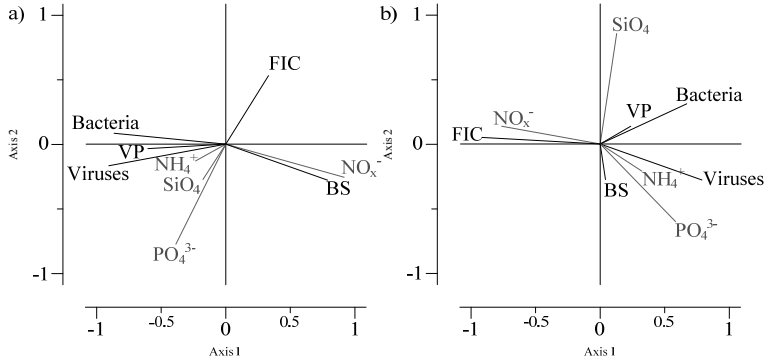


Figure 20. RDA analysis biplot shows interactions between virus and bacteria densities, burst size, FIC, virus production (treated as response variables) and nutrient concentrations (used as explanatory variables) under (a) and above (b) temperature threshold of 13.5°C degrees. Nutrients explained 26.68% (significant factor NO_x^- ($F = 14.18$, $p < 0.05$)) and 23.99% (significant factors NO_x^- ($F = 14.18$, $p < 0.05$), PO_4^{3-} ($F = 2.98$, $p < 0.05$), SiO_4, ($F = 2.97$, $p < 0.05$) of total variation in response variables, respectively.

Most interesting finding revealed by the redundancy analysis (RDA) is trait dependent reaction of viral community to changes in temperature with proposed threshold level (Fig. 20). Under low temperature regime inorganic nitrogen (NO_x^-) in forms of nitrates (NO_3^-)/nitrites (NO_2^-) was the only important predictor, tightly associated with virus burst size ($r = 0.55$, $p < 0.01$). However, this relation has disappeared under increased temperature, and switched to strong correlation ($r = 0.69$, $r < 0.01$) between NO_x^- and frequency of infected cells (Fig. 20). This conversion might have important implications in terms of temperature driven bottom up regulation of virus-host interactions (Sandaa et al., 2009; Shurin et al., 2012) and clearly

indicates changes in nutrient-mediated virus proliferation strategy (Bohannan and Lenski, 1999; Hewson and Fuhrman, 2008). Availability of inorganic nitrogen to prokaryotic community, in particular NO_3^- and NO_2^-, is determined by temperature optima (Reay et al., 1999). In addition, at low temperature conditions, the bacteria nutrient demand is higher (Wiebe et al., 1993), which consequently reduce bacterial growth rate and, thus, limits host availability for virus infections. Therefore, relative increase in virus burst size rather than infection rates (VIPBUS study) below temperature threshold might reflect changes in virus strategy to coupe with its host (Levin, 1977; Massenger et al., 1999). While temperature facilitated changes in assimilation rates of NO_x^- within bacterial assemblage might indirectly promote virus infections or induce lytic cycle of virus production (Lymer and Vrede, 2006). Consequently, dependence of BS and FIC upon nutrient concentration is different under and above temperature threshold.

At lower temperature conditions, BS was also negatively related with cell abundance ($r = -0.49$, $p < 0.01$), while no correlation was observed at increased temperature. This suggests that warming might disturb linkage between trait- and density-mediated processes. On the other hand, the relation between the number of viruses and bacteria remained strong below ($r = 0.56$, $p < 0.001$) and above ($r = 0.43$, $p < 0.01$) proposed temperature threshold. Temperature shift has also no effect in association between BA and virus production, showing host density dependent response of virus activity irrespect of temperature conditions (Colombet et al., 2006).

The overall virus-mediated bacterial mortality was also different between cooling (avg. VMBM = 11.15% ± 6.99%) and warming (avg. VMBM = 13.68% ± 9.04%) impacts (Student's t-test = 2.14, $p < 0.05$), with slightly higher bacteria loss to virus predation under elevated temperature conditions (Fig. 21). It is important to note that dependence of virus-mediated bacterial mortality was not a linear function of temperature (VMBM = 0.91 + 0.88 × *temperature*; adjusted $R^2 = 42.29$, $F = 3.66$, $p = 0.11$), but rather has regime shift response, since significantly higher proportion of infected cells and, therefore, increased virus-mediated bacterial mortality (Fig. 21) were observed in treatments with temperature elevated by more than 4°C.

Since prokaryotic assemblages are extremely sensitive to even small changes in nutrient supply or trophic structure within microbial loop, and bioavailability of nutrients released by virus lysis is high, the observed shift to higher VMBM might significantly contribute to the regulation of community composition and may change species competitive ability.

Moreover, the relative importance of these changes might be more pronounced in oligotrophic ecosystems, where microbial communities are nutrient depleted. However, if FIC and, therefore, VMBM, will not be associated with increased host growth rate and shorter laten period it might increase virus loss to grazing (Miki and Yomamura, 2005). Therefore, trait-mediated changes in virioplankton response to global warming will depend on both virus and prokaryotic communites, thus very likely will significantly differ across environments and spatio-temporal scales (Rowe et al., 2012).

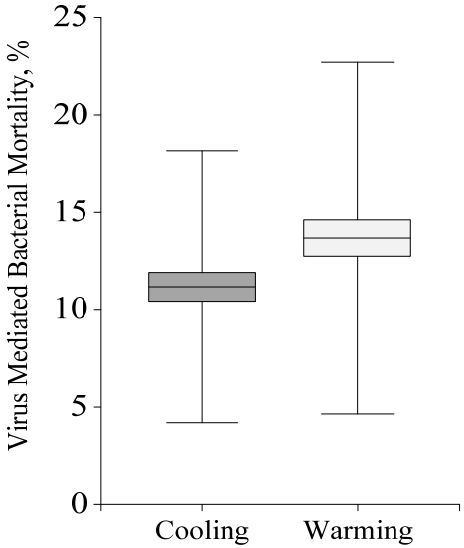


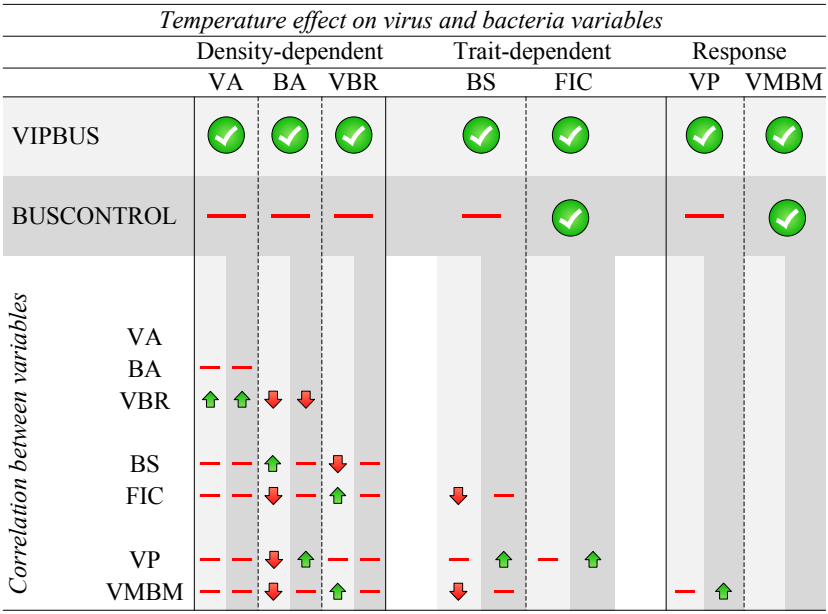
Figure 21. Virus-mediated bacteria mortality under (cooling) and above (warming) proposed temperature threshold of 13.5°C.

2.3. Response-associated variability of viral impact on bacteria community

Community response to temperature changes depends on its structure (Petchey et al., 2010), which is not constant from year to year, in particular during the seasons of active vegetation (Yannarell et al., 2003). Therefore, different response of quantitative virus and bacteria variables observed between two mesocosm experiments (Table 6) might be attributed to trait-mediated processes such as distinct plankton community structure (Taucher et al., 2012) or strength of viral infection itself (Singh et al., 2004).

There are no data on changes in metabolic activity or structure of bacterial assemblages during the course of the experiments, limiting the interpretation of trait-mediated interactions between viruses and their hosts (Marston and Sallee, 2003; Sandaa and Larsen, 2006; Bench et al., 2007). However, changes in cell volume, differences in timing and magnitude of bacterioplankton development between treatments indicate that temperature had a substantial effect on host community (Andersson et al., 2010). Moreover, these changes are not necessary have to be related with increased cell densities or actual bacterial production between treatments (Middelboe et al., 2001; Hoppe et al., 2008).

Table 6. Summary of temperature-induced responces in virus trait- and density-associated parameters in two mesocosm experiments. Green circles indicate response of selected variable to temperature manipulation. Significant and positive correlations between virus and bacteria variables are presented as green arrow, while negative as red arrow. Minus sign indicate no response or correlation between variables.



Limitations associated with application of distinct mesocosm designs between two studies could also contribute to the observed differences (Petersen et al., 2003). Even thought interrelation between viral and bacterial parameters as well as their covariance with environmental factors is temperature sensitive, it strongly depends on the ability of viruses and bacteria to adapt to changing conditions (Bettarel et al., 2009). Since adaptation and, thus, response to certain temperature regime requires some time, which differs between communities (Degerman et al., 2013), this suggests that temporal and size scale dependent variation has considerable impact on virus–hosts interactions (Martínez Martínez et al., 2012).

Changing temperature restructure natural habitats (Sommer et al., 2007; Rüger and Sommer, 2012) and significantly alter trophic interactions between organisms (Matteson et al., 2012). This was mostly evident in VIPBUS study, where both viruses and bacteria responded differently to temperature manipulations. Under warming regime (Δ+4 °C treatment) prokaryotic density declined, while no changes in virus abundance were found (compare to *in situ*°C treatment). An opposite situation was observed in cooling treatment (Δ-4 °C treatment), where the number of bacteria remained constant compared to ambient conditions, while virus density was significantly reduced. This mitgh indicate different temperature effect on standing stock of predator and its prey irrespective of their interrelationship as well as changes in virus and bacteria loss processes with warming (Mathias et al., 1995; Noble and Fuhrman, 1997). For example, it is known that increased temperature has positive effect on bacterivorous protists (Weitere et al., 2008). Therefore, reduction of cell numbers might be associated with increased grazing pressure rather than virus activity. In addition, shift toward larger cell size to relieve of grazing (Simek et al., 2001; Sommer and Lengfellner, 2008) in opposite to virus induced conversion to smaller cells (Weinbauer and Höfle, 1998; Miller, 2001; Simek et al., 2010) may support this scenario in Δ+4°C treatment of VIPBUS study.

However, these changes in host and virus densities had considerable impact on VBR dynamics (Fig. 15), implying different types of relation between predator and its prey. For instance, under *in situ* conditions, VBR was related to bacteria abundance ($r = -0.69$, $p < 0.05$), suggesting that viruses have a minor impact on bacterial dynamics. However, this relation has changed under warming conditions (Δ+4 °C treatment). Covariation between cell numbers and VBR disappeared, while standing stock of viruses was strongly correlated with VBR ($r = 0.70$, $p < 0.05$). These changes

indicate significant shift in density dependent virus-host interations as a response to temperature manipulation. Neither BA nor VA correlated with VBR under reduced temperature, while both communities were tightly related with each other ($r = 0.79$, $p < 0.05$), suggesting that under less favourable conditions, the standing stock of bacteria strongly depends on their predators. In addition, infection was promoted by higher ratio between viruses and bacteria, resulting in overall negative effect on virus burst size and density of host community study, suggesting the existence of interplay between density- and trait-mediated effects in predator-prey interactions (Levin, 1977; Gons et al., 2006; Sandaa et al., 2009; Deng et al., 2012).

Virus activity is not a simple function of host physiology and metabolism (Bouvier and Maurice, 2011). The lack of relation between host size and virus burst size as well as lower BS in Δ+4 °C treatment encompassed by larger cells imply that BS was not limited by host physiology (Brown et al., 2006; Parada et al., 2006). In VIPBUS study, the overall virus burst size was inversely related to frequency of infected cells ($r = -0.92$, $p < 0.01$). Field observations (Brum et al., 2005), theoretical predictions and experimental studies (Levin, 1977) suggest that BS may balance FIC (and vice versa) to ensure coexistence between predator and prey (Lenski and Levin, 1985). And in the context of these experiments, it also indicates that virus infectivity (including FIC) was upregulated by elevated temperature (Kimes et al., 2012). On the other hand, it seems that community composition might determine the magnitude of the viral infection (Winter et al., 2008), where FIC is mostly controlled by the dynamics of subpopulations of sensitive and resistant cells (Thyrhaug et al., 2003; Brockhurst et al., 2006). Since virus burst size also depends on bacterial activity (Winter et al., 2004; Gons et al., 2006), this may explain the absence of differences in virus burst size under reduced temperature conditions, assuming that in both experiments the actual temperature in cooled treatments were below proposed temperature threshold level for active bacterial metabolism (Felip et al., 1996; Gurung and Urabe, 1999; Demoling et al., 2007).

CONCLUSIONS

1. Overall, 26 morphologically different forms of planktonic viruses were found in the Curonian Lagoon. Members of the *Myoviridae* family dominated along the gradient of the pelagic part of the lagoon. Observed correlations between myoviruses and chlorophyll *a* concentration, and podoviruses and virus-to-bacteria ratio suggest that different members of virioplankton assemblage reflect the dynamics of distinct host group.
2. Total virioplankton abundance range from 0.1 to 1.6×10^8 particles ml^{-1} over annual scale and show similar pattern of dynamic in different parts of the Curonian Lagoon.
3. Variation in virioplankton abundance in the hydrodynamically active part of the lagoon is best predicted by changes in bacterial abundance, while in the stagnant zone by changes in chlorophyll *a* concentration. Maximal virioplankton production in the hydrodynamically active part of the lagoon is twice as high as in the stagnant zone.
4. Virus-mediated bacterial mortality depends on virus decay rates in the pelagic part of the lagoon regardless their abundance and infectivity.
5. Virus burst size in the stagnant zone of the lagoon is on average twice higher than in the hydrodynamically active parts. Virus burst size depends on indirect effect of environmental conditions and could not be explained by bacterial community structure and activity.
6. Changing temperature regime has effect on virus-to-bacteria ratio rather than on actual densities of viruses and bacteria in the pelagic part of the ecosystem.
7. Rising sea surface temperature has positive effect on virus-mediated bacteria mortality by enhancing viral infection rate.
8. Virus-mediated bacterial mortality (VMBM) is a threshold temperature -dependent process. Irrespective of host community structure, VMBM significantly increased at temperatures elevated by 4°C above *in situ* conditions.

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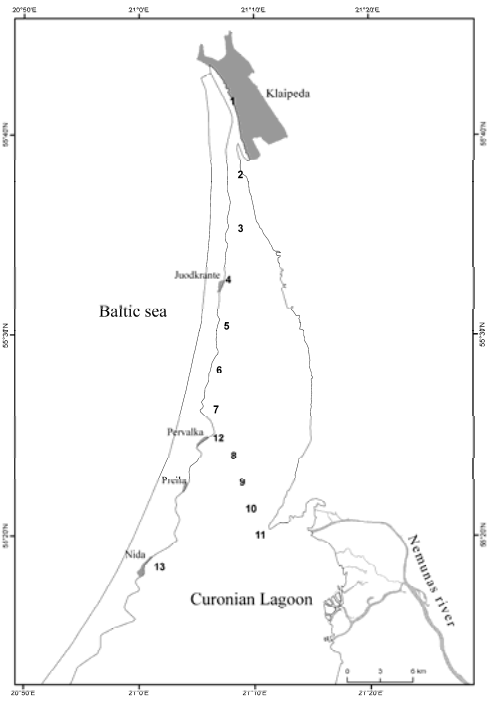
SUPPLEMENTARY INFORMATION

1. Materials and methods

Table 1. Sampling sites of field survey in 2005.

Sampling station	Coordinates	Location on the map
1.	55^0 41,200' N 21^0 07,400' E	
2.	55^0 37,571' N 21^0 08,867' E	
3.	55^0 34,886' N 21^0 08,867' E	
4.	55^0 32,367' N 21^0 07,393' E	
5.	55^0 30,041' N 21^0 07,643' E	
6.	55^0 27,857' N 21^0 06,995' E	
7.	55^0 25,898' N 21^0 06,714' E	
8.	55^0 23,571' N 21^0 08,200' E	
9.	55^0 22,249' N 21^0 08,933' E	
10.	55^0 20,939' N 21^0 09,692' E	
11.	55^0 19,620' N 21^0 10,500' E	
12.	55^0 24,735' N 21^0 06,002' E	
13	55^0 18,097' N 21^0 01,002' E	

Field survey for characterization of virioplankton morphology was carried out at 13 sites in the Lithuanian part of the Curonian Lagoon during a two-day cruise in 2005. Samples from stations 1–5 were collected on 31 July and from stations 6–13 on 1 August from the surface water (0.5–1 m depth).

Figure 1. Transmission Electron Microscopy (TEM) micrographs of water samples. Frequency of visibly infected cells and virus burst size (a–b) as well as analysis on morphology and size of both virus and bacterial community (c–f) could be performed using TEM technique.

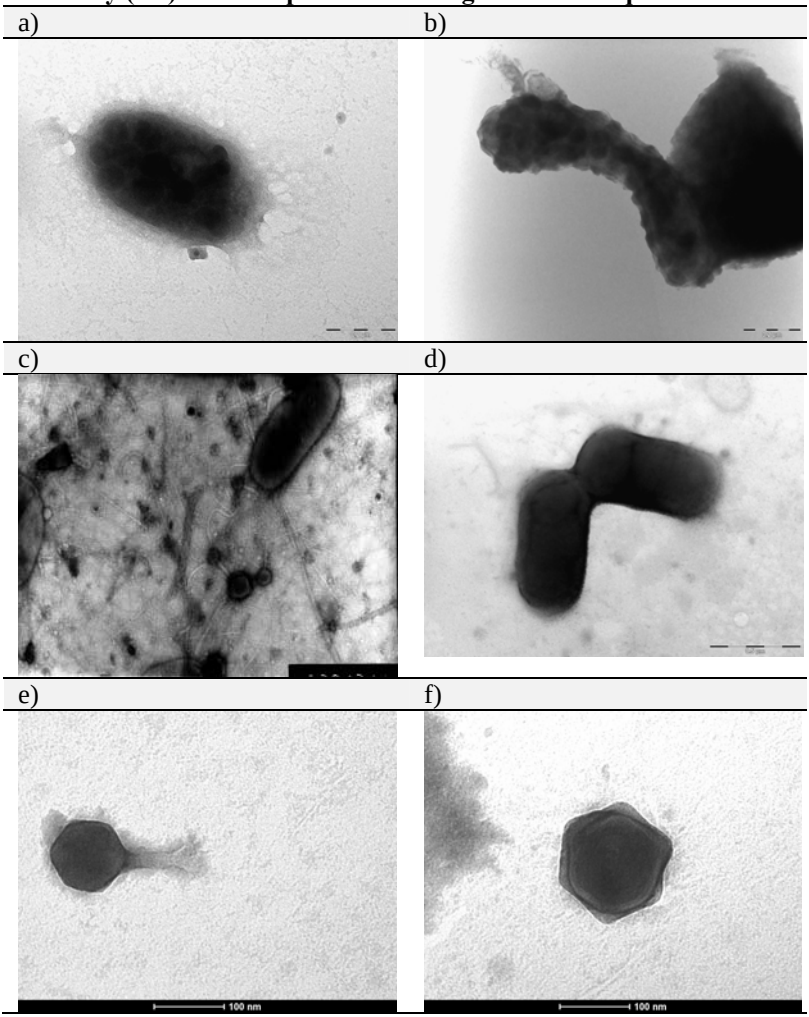
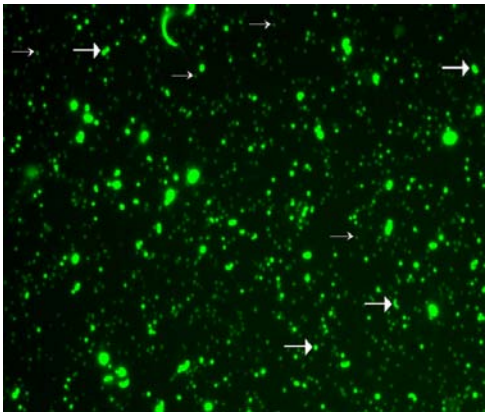


Figure 2. Epifluorescence Microscopy (EFM) images of water samples. EFM enables simultaneous observations of total abundance of both virus and bacteria, after staining with SYBR Green I. Arrows show virus-like particles, arrows in bold –cells.



2. Results

Table 2. Numerical (mean ± standard deviation) and relative (%) distribution of virioplankton community along the Lithuanian part of the Curonian Lagoon.

St.	VA	CS	TL	BA	VBR	Myo	Sipho	Podo	NT
		nm	nm			%	%	%	%
1	2.4	86.3±27.4	107.4±90.7	0.9	27	66.9	2.8	27.6	2.7
2	2.0	71.5±22.6	114.9±76.4	0.9	22	69.8	2.4	25.7	2.1
3	2.6	81.0±22.5	137.7±91.0	1.5	23	62.0	10.5	26.4	1.1
4	1.9	95.7±27.1	231.9 ±199.1	–	–	76.3	3.8	18.4	1.4
5	2.9	96.1±22.6	205.0±64.6	0.9	31	76.9	5.5	14.0	3.6
6	5.1	91.3±19.8	224.9±86.1	1.9	27	67.9	10.0	18.2	3.9
7	3.6	70.8±27.2	136.9±119.7	1.0	38	55.5	17.7	24.7	2.0
8	3.8	68.8±20.5	138.1±96.4	0.8	49	61.9	16.5	20.5	1.0
9	2.6	97.7±20.3	116.7±97.9	1.7	16	66.0	12.3	21.7	0.0
10	3.0	92.1±23.0	175.3±121.8	0.6	48	61.4	12.8	21.8	4.1
11	2.0	103.1±27.4	122.3±70.5	0.9	22	58.6	21.8	15.7	3.9
12	2.3	100.2±18.9	209.7±140.3	1.2	19	65.3	23.4	6.5	4.6
13	1.9	89.9±18.6	241.5±81.4	1.1	18	65.1	25.8	5.3	3.7

St – sampling station, VA – virus abundance (10^7 particles ml^{-1}), CS – capsid size (± standard deviation), TL – tail length (± standard deviation), BA – bacteria abundance (10^6 cells ml^{-1}), VBR – virus-to-bacteria ratio, Myo – Myoviridae, Sipho – Siphoviridae, Podo – Podoviridae, NT – tailless phages.

Figure 3. Morphological diversity of phage-like particles (VLP) observed in the the eutrophic Curonian Lagoon: (a–n; p, ag, ai) different types of *Myoviridae*; (o, q–w, ae–af, ah, ad) – *Siphoviridae*; (x–ac) – *Podoviridae*; (aa) – tailless VLP of diameter 200nm. Scale bar 100 nm.

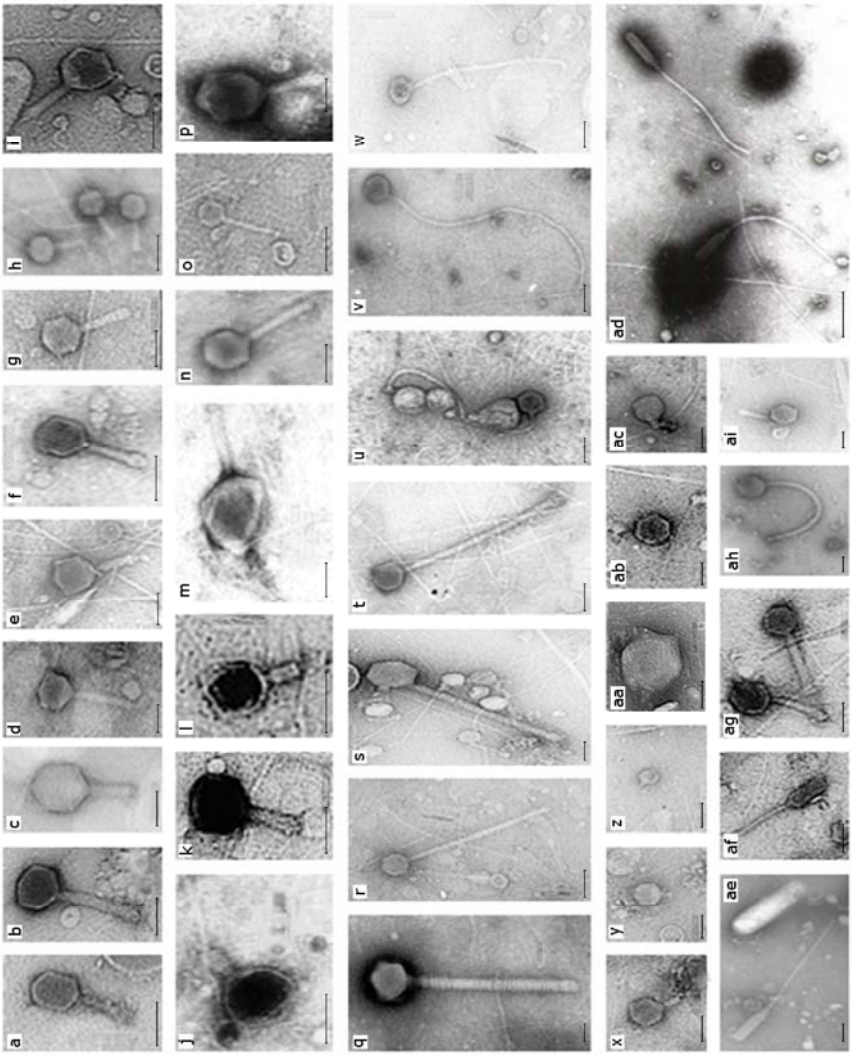


Table 3. Pearson's correlation (r) coefficients between bacteria abundance and virus-to-bacteria ratio observed at different sampling sites in the Curonian Lagoon.

Years	S1	S2	N1	N2	V3	R4
2007		-0.53**	-0.61**			
2009	-0.76*			-0.86**	-0.79*	-0.87**

***p* values *< 0.05; ** < 0.01.**

Table 4. Pearson's correlation (r) coefficients between virus production and water temperature observed at different sampling sites in the Curonian Lagoon.

Years	S1	S2	N1	N2	V3	R4
2007		0.66***	0.63***			
2009	0.85***			0.64*	0.78**	0.58

***p* values *< 0.05; ** < 0.01.**

Table 5. Pearson's correlation (r) coefficients between virus-to-bacteria ratio and virus and bacteria densities observed in different replicates (R1–R2) of temperature treatments in VIPBUS experiment.

	Temperature treatment								
	Δ–4 °C			*in situ* °C			*Δ*+4 °C		
	R1	**R2**	**R3**	**R1**	**R2**	**R3**	**R1**	**R2**	**R3**
VA	0.40	0.54	0.16	0.53*	0.73**	-0.31	0.82**	0.78**	0.78**
BA	-0.45	-0.73**	-0.70**	-0.52	-0.81**	-0.80**	-0.58*	-0.64*	-0.59*

p values *< 0.1; ** < 0.05.
VA – virus abundance (10^7 particles ml^{-1}), BA – bacteria abundance (10^6 cells ml^{-1}).

The virus-to-bacteria ratio (VBR) is used to study the relationship between virioplankton and bacterioplankton and is an indicator of the potential importance of viruses to control bacterial abundance (Wommack and Colwell, 2000). VBR dynamics is independent of actual numbers and might change even if density of one interacting community remains stable. This in turn, results in absence of any correlation between VBR and unchanged abundance of corresponding community. Therefore, correlation between VBR and viruses or bacteria indicates the interrelation between the production and loss of viruses and bacteria in aquatic environments.