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Bacteriophage Ecology Group (BEG) News

Dedicated to the ecology and evolutionary biology of the parasites of unicellular organisms (UOPs)

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January 1, 2002 issue (volume 11)

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Editorial

Editorials should be written on subjects relevant to The Bacteriophage Ecology Group as an organization, to *BEG News* (either the concept or a given issue of *BEG News*), or the science of Bacteriophage Ecology. While my assumption is that I will be writing the bulk of these editorials, I wish to encourage as many people as possible to seek to relieve me of this duty, as often as possible. Additionally, I welcome suggestions of topics that may be addressed. Please address all correspondences to abedon.1@osu.edu or to "Editorials," *Bacteriophage Ecology Group News*, care of Stephen T. Abedon, Department of Microbiology, The Ohio State University, 1680 University Dr., Mansfield, Ohio 44906. Please send all submissions as Microsoft Word documents, if possible (I'll let you know if I have trouble converting other document formats), and in English.

Mathematics for Microbiologists

Microbiologists are not a terribly mathematically inclined bunch. If we were, we probably wouldn't be microbiologists. Indeed, microbiology is difficult enough without introducing math into one's research equation. Nevertheless, for complex systems mathematical analysis can be simplifying, at least in terms of the synthesis of multiple ideas and data, and, ideally, can even be predictive! This is particularly so for microbial ecology, where one must worry about population-level effects on top of biochemical, morphological, physiological, and genetical complexity. So, consequently, we're stuck with math. Does that mean that microbiology that's mathematically based must be completely opaque to the non-mathematician? Nooooo! Here, then, is a cued guide for the mathematically oriented on how to bring one's math down to a level that a *mere* microbiologist might understand:

Microbiology:	Keep the microbiology in the fore, not the math.
Continuity:	Strive for continuity with previous mathematical analyses of the same or similar phenomena.
Latin:	When starting analyses from scratch, try to avoid assigning Greek letters as variable names; e.g., to the not Greek or not mathematically inclined, *B* is preferable to β.
Expansion:	Outside of equations don't use variable names as abbreviations for phenomena; e.g., don't write simply *B* if what your really mean is *burst size*.
Redundance:	Strive for redundancy by redefining your variables throughout your text.
Comprehension:	Assume minimal mathematical knowledge among your readers. Don't omit crucial logical steps in the development of your math. Mathematicians may scoff at excess detail, but microbiologists, in its absence, will simply become confused.
Interpretation:	You are writing microbiology for microbiologists so try explaining what each equation means in *microbiological* terms.
Justification:	Explain why you are using a particular mathematical technique, just as you should always strive to justify your utilization of an unusual experimental technique.
Supervision:	In multi-authored studies, don't leave the mathematician alone to write the theory section!
Closure:	Eventually return your study to the microbiology.

When written well a study that considers the mathematics of a microbiological phenomenon ultimately should lead the reader to an intuitive understanding of the phenomenon. Barring that, the reader should gain an understanding of why the phenomenon cannot be understood intuitively (usually meaning that it is either too complex or too poorly understood to be sufficiently mechanistically developed). A study that fails to develop significant biological understanding cannot lead its readers to intuitive knowledge, and chances are your more microbiologically minded colleagues will ignore it. Finally, this is a call for conscientious improvement rather than perfection. No doubt my own published math, simplistic as it is, may be criticized as insufficiently transparent for the casual reader.

MicroDude, a.k.a., **Stephen T. Abedon**
is the Developer and Editor of The Bacteriophage Ecology Group web site which is dedicated to the ecology and evolutionary biology of the parasites of unicellular organisms (UOPs)

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New BEG Members

The BEG members page can be found at www.phage.org/beg_members.htm. There are two ways of "joining" BEG. One, the "traditional" way, is to have your name listed on the web page and on the list server. The second, the "non-traditional" way, is to have your name only listed on the list server. The latter I refer to as "non-members" on that list. Members, e.g., individuals listed on the BEG members list page, should be limited to individuals who are actively involved in science (research, instruction, outreach, industry) and who can serve as a phage ecology resource to interested individuals. If you have an interest in phage ecology but no real expertise in the area, then you should join as a non-member. To join as a member, please contact BEG using the following link: abedon.1@osu.edu. Include:

- your name
- your e-mail address
- your snail-mail address
- the URL of your home page (if you have one)
- a statement of whether or not you are the principal investigator
- a statement of your research interests (or phage ecology interests)
- a list of your phage ecology references, if any

Note that it is preferable that you include the full reference, including the abstract, if the reference is not already present in the BEG bibliography. Responsibility of members includes keeping the information listed on the BEG members page up to date including supplying on a reasonably timely basis the full references of your new phage ecology publications. Reprints can also be sent to *The Bacteriophage Ecology Group*, care of Stephen Abedon, Department of Microbiology, The Ohio State University, 1680 University Dr., Mansfield, Ohio 44906. To join BEG as a non-member, please contact BEG using the following link: abedon.1@osu.edu and minimally include your name and e-mail address.

Please welcome our newest members

name (home page links)	status	e-mail	address
Faith Burden	---	fburden @bio.warwick.ac.uk	Biological Sciences Dept., University of Warwick, Coventry, UK CV4 7AL
	interests:	Ecology of temperate bacteriophage of *Staphylococcus aureus*. The use of bacteriophage therapy against MRSA. (contents | BEG members | top of page)	
Lin Tao	PI	ltao @uic.edu	Associate Professor, Department of Oral Biology, College of Dentistry, M/C 690, University of Illinois at Chicago, 801 S. Paulina Street, Chicago, IL 60612, USA
	interests:	Phage-lactobacillus interaction, lactobacillus phage taxonomy and classification, symbiosis and coevolution among women, their vaginal lactobacilli and phages, and the role of sexually transmissible phages in the health and diseases of women. (contents | BEG members | top of page)	
Yanhui Yang	---	yanhui @jingxian.xmu.edu.cn	Center for Marine Environmental Studies, Xiamen University, Xiamen, 361005, P. R. China
	interests:	Relationship between virus and prokaryotic picoplankton in estuary, coastal zone, shelf sea and open waters. Changes in diversity and community structure of viral and picoplankton community along eco-type gradients. (contents | BEG members | top of page)	

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New Links

Links relevant to The Bacteriophage Ecology Group fall into a number of categories (e.g., see Bacteriophage Ecology Links at www.phage.org/beg_links.htm). Listed below are new links found on that page. If you know of a link that should be included on this page, or the whereabouts of a now-dead link, please let me know.

No Entry.

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New Features

In this section I highlight new or updated features of the BEG site. If you have any ideas of how either the BEG site or *BEG News* might be improved, please let me know.

The BEG Meetings page has been updated for the 2002 year.

The look of the site has been updated. See www.phage.org (below) and enjoy!

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Meetings

The BEG Meetings link will continue. Reminders of upcoming meetings will be placed in this section of *BEG News*. If you know of any meetings that might be of interest to BEG members, or would like to recap a meeting that you've attended, then please send this information for posting to abedon.1@osu.edu or to "BEG Meetings," *Bacteriophage Ecology Group News*, care of Stephen T. Abedon, Department of Microbiology, The Ohio State University, 1680 University Dr., Mansfield, Ohio 44906.

Please send photos, etc. from meetings for inclusion in this section.

This is a list of some of what appears to be going on in 2002:

See the BEG Meetings Page for a Calendar and a Meetings overview.
Click on links for more detail.

1. Society of Integrative and Comparative Biology Meeting (January-annual)
2. ASM General Meeting (May-annual)
3. American Society for Virology Meeting (July-annual)
4. Molecular Genetics of Bacteria & Phages (mid-August-annual)
5. International Congress of Virology (late August-biennial?)
6. European Marine Microbiology Symposium (October-?)

This is an initial list of what may or may not be going on in 2003:

1. Society of Integrative and Comparative Biology Meeting (January-annual)
2. ASM General Meeting (May-annual)
3. American Society for Virology Meeting (July-annual)
4. International Phage Meeting (June, July, August?-biennial, even years; now odd? now annual?)
5. Microbial Population Biology Gordon Conference (July, August-biennial, odd years)
6. Molecular Genetics of Bacteria & Phages (mid-August?-annual)
7. ???International Congress of Virology (late August?-biennial?)
8. International Society Microbial Ecology (late August?-biennial, odd years)
9. ???European Marine Microbiology Symposium (October-?)

Below is a scene from the opening-day picnic at Evergreen, 2001
(that's MicroDude wearing the funny glasses):

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Jobs

Looking for job? Looking to fill a position? Please send advertisement and information to abedon.1@osu.edu or to "Jobs", Bacteriophage Ecology Group News, care of Stephen T. Abedon, Department of Microbiology, The Ohio State University, 1680 University Dr., Mansfield, Ohio 44906. Please send all information as text (e.g., as an e-mail) or as Microsoft Word documents, if possible (I'll let you know if I have trouble converting any other document formats), and in English. I will update this section as I receive material, regardless of what date this issue of BEG News goes live.

Click here for International Society for Microbial Ecology Employment Listings.

Click here for American Association for the Advancement of Science Employment Listings.

Click here for AAAS "Microbial Ecology" Search.

Click here for AAAS Ecology *and* Microbiology.

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Submissions

Submissions are non-editorial items describing or highlighting some aspect of bacteriophage ecology including news pieces, historical pieces, reviews, and write-ups of research. Peer review of submissions is possible and a desire for peer review should be indicated. Send all submissions to abedon.1@osu.edu or to "Submissions", Bacteriophage Ecology Group News, care of Stephen T. Abedon, Department of Microbiology, The Ohio State University, 1680 University Dr., Mansfield, Ohio 44906. Please send all submissions as Microsoft Word documents, if possible (I'll let you know if I have trouble converting any other document formats), and in English.

An Expanded Overview of Phage Ecology

by **Stephen T. Abedon**

(for a more easily printed version, click here)

[Note: **Do not reference this manuscript.** Instead, please see the **Phage Ecology** chapter by **Stephen T. Abedon** as it is to appear in ***The Bacteriophages*, Edition 2**, Oxford University Press. The manuscript submission deadline for that publication is February 1, 2002. This manuscript, as presented, is **not** in final submission form. **Any comments and criticisms on this manuscript sent prior to the submission deadline would be *greatly* appreciated by the author**. Please send by e-mail to abedon.1@osu.edu. Thanks.]

Contents

Introduction

Phage ecology is the study of the real-time interactions between phages and environments. These interactions are ecologically important, particularly to the extent that they affect bacteria populations. Here - keeping phages in the fore rather than bacteria or ecosystem functioning - I consider phage organismal, population, community, and ecosystem ecology (Table 1). For complementary approaches to the review of phage ecology see (6,7,11,18,23,39,47,51), plus various recent reviews of aquatic and ecosystem phage ecology (38,53,57,63,64,69,73).

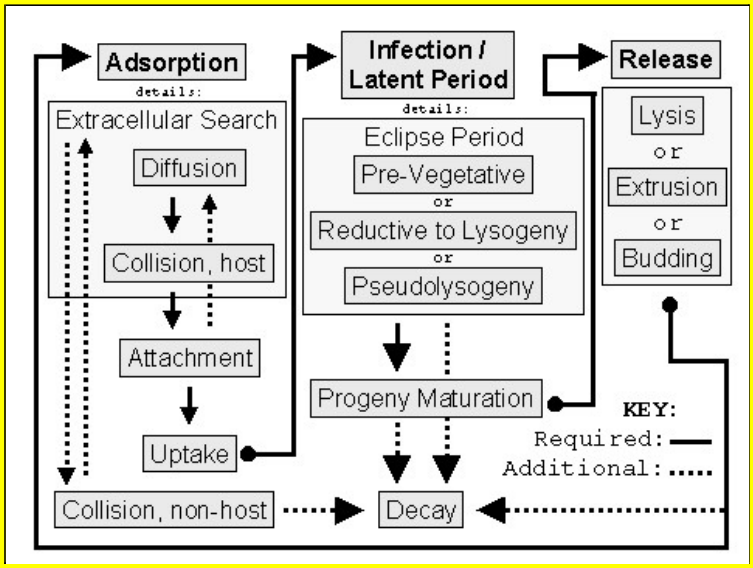
Table 1: Defining Phage Ecology

Ecology	A bacterium is...	Considerations	Experiments
Organismal	...a target, or an entity that impacts on the phage phenotype	Phage anatomy, physiology, and behavior characterized from Darwinian perspective; virion stability, survival, and adsorption; eclipse period, latent period, and burst size; adaptations overcoming barriers to transmission	Single-step growth; adsorption curves; kinetics of phage decay
Population	...an environmental resource	Phage population growth and density; liquid versus spatially structured environments (broth growth versus plaque growth); low versus high phage multiplicity	Single-step growth; adsorption curves; kinetics of phage decay
Community	...a partner in coevolution	Phage-host coevolution; inverse relationship between phage and uninfected-host density; community stability; host resistance; phage host-range breadth and variation; transduction and phage (lysogenic) conversion; competition among different phage species	Phage-host continuous-culture or serial-transfer experiments; *in situ* observation and experiment
Ecosystem	...a lower trophic level	Phage impact on ecosystem nutrient cycling and energy flow; short circuiting of microbial loop	*In situ* observation and experiment

Phage Organismal Ecology

The basic phage life cycle. In the hierarchy of ecological disciplines, phage organismal ecology is most closely allied with the molecular (plus physiological and genetic) characterization of phages. While underlain by copious variety and details, the general phage life cycle (Figure 1) basically involves adsorption, infection, and release, plus considerations of phage decay. The study of these processes - especially from the perspective of *in situ* costs, benefits, expression, and per-infection productivity - is the province of the phage organismal ecologist. More broadly, one can view virus organismal ecology as the study of the adaptations viruses employ to overcome physical, chemical, or biological barriers to their transmission between hosts (44).

Figure 1: General Phage Life Cycle (below).



Phage adsorption. Phage adsorption begins after phage release from infected cells and ends with the uptake of phage genomes into the cytoplasm of adsorption-sensitive hosts. The more rapidly a phage adsorbs a permissive host cell, the greater its likelihood that it will avoid decay (e.g., 19,36,56) and the shorter its overall life cycle (5). Nevertheless, phage mutants displaying faster adsorption than wild type have been isolated from laboratory cultures (30). In addition, by requiring specific adsorption cofactors, some phages, such as phage T4, may be adsorption competent within environments in which healthy hosts are likely (e.g., colons) but adsorption incompetent (or less competent) in environments where healthy hosts are less likely (e.g., sewage) (28,47).

Infection (the latent period). The phage latent period begins with the eclipse, a period during which the artificial lysis of an infected host will not release infective phage particles. Post eclipse of a typical infection I refer to as a period of phage-progeny maturation. During this latter period either infected bacteria release mature phages without lysis (46) or artificial lysis results in the release of phage progeny (35). Maturation, for highly virulent phages, mostly occurs at a constant, linear rate rather than exponentially because the rate of synthesis of virion components is limited by some aspect of the host's anatomy or physiology (40,68). Things are complicated, however, if cells are able to continue to grow and divide during a phage infection since cell growth can increase in number whatever cell components are limiting. At the same time, the synthesis of phage components can have negative effects on host division. These negative effects range from a slowing of host population growth as seen with filamentous phages (46) to a complete cessation of host division as seen with highly virulent phages such as phage T4 (e.g., 2).

A further complication is the *length* of the period of progeny maturation. With lytic phages the timing of host lysis controls the length of this period, with the timing of lysis, in turn, under the control of phage genes and proteins (e.g., holins; 67). Host nutrition status (40,64,68), temperature (32), and physiological state vis-à-vis the standard bacterial growth curve or chemostat doubling times (7,55,61) can also impact on this timing as can phage-controlled processes such as lysis inhibition and lysis from without (1,2,3,4). In addition, among even synchronously infected cells, it has long been known that the overall duration of the phage latent period can vary within a single culture (13). Consequently, most phages during single-step growth display a non-instantaneous rise, which is the time over which a population of synchronously infected hosts display phage-induced lyses (32,61). Progeny maturation and rise periods associated with chronic (a.k.a., continuous or persistent) infections can be particularly long (46).

Phage-progeny release. So long as a virus particle remains inside an infected bacterium, then it is not free to acquire a new host. An infected host may display significant productivity in terms of the intracellular maturation of progeny virions, but such productivity can pale in comparison with the growth rates that phage populations may achieve via the exponential growth that phage-progeny release makes possible. For most phages the release of progeny phages coincides with the destruction of the parental infected cell (lysis; 67). For filamentous phages, that extrude their phage progeny across the host cell envelope, release does not necessarily result in host-cell death (46).

The phage progeny released from an individual bacterium are collectively referred to as a burst. For phage populations one typically determines a parameter know as *burst size* that is equal to the total number of phages produced by a single round of phage infection of host cells, divided by the total number of cells that had been infected prior to phage-progeny release. Measured burst size can vary considerably between individual infected cells (33) and even over the course of released-phage storage (20). Burst-size determination is complicated if hosts aggregate or fail to fully separate in culture (12) or if phage release occurs via a mechanism other than host lysis (7).

Phage decay. If one is willing to accept that phages are alive (e.g., 24), then phage decay is equivalent to virion death (a.k.a., "inactivation" or "loss of titer"; 36) or to a lack of infected-cell productivity without reduction to lysogeny. Phage decay (recently reviewed by 64,73) likely limits the impact of phages on bacteria (65) plus imposes important constraints on the evolution of non-temperate phages since it implies that virion populations cannot survive indefinitely in the absence of sufficient densities of susceptible bacteria (e.g., 27,71). Similarly, the evolution of lysogeny must be dependent at least in part on the relative importance of virion decay versus phage and prophage replication rates as, for example, Stewart and Levin (62) suggest with their "hard times" hypothesis.

Phage Population Ecology

Phage population growth. While phage organismal ecology emphasizes per-infection-productivity and phage community ecology has a host-population-dynamics emphasis, the emphasis of phage population ecology (Table 1) is on phage population growth either within bacteria cultures (5) or within individual infected bacteria (25). Like any organism living within a suitable environment possessing sufficient resources, a phage population will increase in number exponentially over time. Phage exponential growth is especially tractable during phage growth within liquid culture (2,21,32). Phage populations that increase in size most quickly should acquire host cells most rapidly. The acquisition (exploitation) of one bacterium by one phage means that one-less unit of bacteria resource is available for exploitation by a second phage. Over the short term, in relatively simple environments, selection within phage populations therefore should be for both more-rapid population growth and more-rapid host-cell acquisition.

Phage latent-period evolution. Certain phage characteristics should contribute to faster phage population growth (e.g., 31). For instance, we should expect evolution to favor decay-resistant virions, rapid adsorption (though, during plaque growth, not necessarily; see below), short eclipse periods (except given selection for pseudolysogeny or true lysogeny), high rates of progeny maturation (balanced, in some cases, e.g., with filamentous phages, against damage to the host resource), and, once initiated, rapid progeny release (ditto). With or without caveats, conspicuously absent from this list is the duration of phage latent periods and periods of progeny maturation, with the length of both a function of lysis timing. Here I consider forces that impact the evolutionary optimization of the duration of phage latent periods.

From an ecological perspective we can distinguish the members of populations into three groups: prereproductive, reproduction, and postreproductive. Postreproductive phages, variously defined, are irrelevant to this discussion. Prereproductive phages are those engaged in either adsorption (including the extracellular search for susceptible bacteria) or the eclipse, since during these periods the phage is not generating mature phage progeny. Reproductive phages are those infecting bacteria during the phage period of progeny maturation. For phages that must lyse their host bacteria to disseminate phage progeny, we may describe a period of progeny maturation as optimal in duration should the latent period giving rise to it result in maximized phage-population growth rates. Too-short latent periods result in insufficient burst sizes to sustain maximal phage population growth while too-long latent periods slow phage population growth by delaying phage-progeny acquisition of new host bacteria.

When prereproductive periods are short, this means that free phages can rapidly find uninfected cells and then rapidly gear up for intracellular progeny maturation. Such conditions should select for rapid infection turnover (short latent periods) such that phage progeny acquire uninfected hosts before those cells are obtained by competing phages. In general then, high host densities and short phage eclipse periods should select for shorter phage latent periods (5). When prereproductive periods are long, by contrast, the reproductive period, once begun, is more valuable thereby resulting in selection for increased per-infection productivity. Thus, low host densities or long phage eclipse periods should select for larger phage burst sizes even at the expense of longer phage latent periods (5).

Contribution of early adsorbers. The impact of changes in host density on phage population growth and

latent-period evolution are not as straightforward as one might expect given that a phage cohort's mean time until bacteria adsorption varies directly with host density (Figure 2B). The reason for this complication is a consequence of phage adsorption occurring essentially as an exponential decay in free-phage density (Figure 2A). For any phage cohort released at a given moment into a population of hosts, phage adsorption occurs such that some constant fraction of remaining free phages will adsorb over any given interval. As a consequence, more phages from a given cohort will adsorb during a sooner interval compared with some later interval.

Figure 2A: Exponential phage adsorption and phage population growth (below). Free-phage adsorption (e.g., 5) with log(*N* = per ml host density) indicated for different curves and *k* (the phage adsorption constant) = 2.5 x 10^{-9} ml/min. Adsorption curves cross the horizontal line at the average phage adsorption time (mean free time) = 1/*kN*. [Goto 2A, 2B, 2C, 2D, or all four]

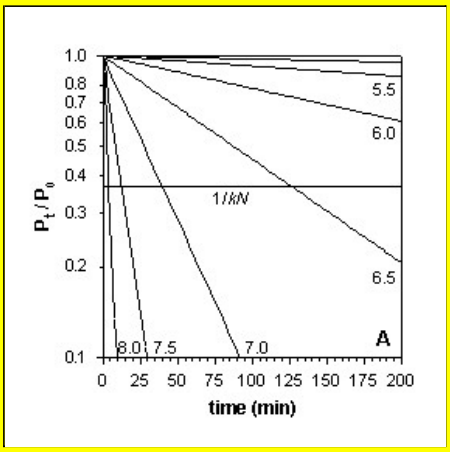
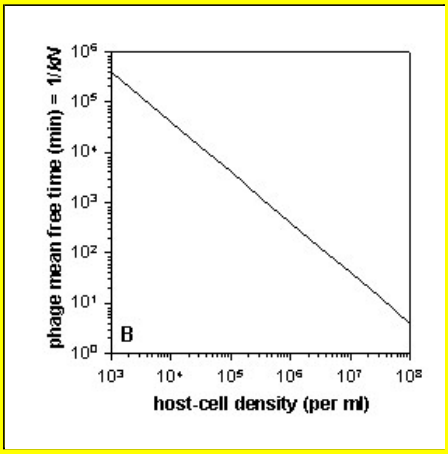


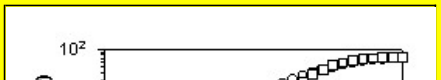
Figure 2B: Exponential phage adsorption and phage population growth (below). Mean free time graphed as a function of bacteria density. [Goto 2A, 2B, 2C, 2D, or all four]



If by chance a phage adsorbs to a host earlier rather than later, then the duration that this phage is prereproductive will be shorter and therefore the total duration of that phage's life cycle will also be shorter. The rate of phage population growth is a function of the duration of the phage life cycle, as well as the per-host burst size. Furthermore, earlier-adsorbing phages are potentially greater in number due to the exponential kinetics of phage adsorption plus spend less time susceptible to non-adsorption-related virion decay (above). Consequently, it stands to reason that earlier-adsorbing members of phage-adsorption cohorts will contribute more to the exponential growth of a phage population than later-adsorbing members.

At greater host densities all phages adsorb relatively rapidly such that the variance in phage pre-reproduction duration is not large. However, at lower host densities the timing of the adsorption of the majority of a phage cohort is spread over much longer intervals (Figure 2A), and the contribution of those phages that by chance adsorb hosts earlier becomes increasingly large and important to overall phage population growth. Thus, the average timing of phage adsorption (the phage mean free time; see 5) may very well decline as a direct function of host density (Figure 2B), but phage population growth as a function of host density does not decline as quickly (Figure 2C). This means that while evolution ought to favor phages with longer periods of progeny maturation as host densities decline, the phage latent period that is optimal for phage-population exponential growth should not increase as fast as host densities decline (5; Figure 2D).

Figure 2C: Exponential phage adsorption and phage population growth (below). Log phage density following 1000-min of phage growth as simulated or calculated at different host densities (log-scale and log-transformation are both intentional as presented). A latent period of 25 min, burst size of 75 phages/cell, and adsorption constant as above were used. Simulations assumed *exponential phage adsorption* (circles) which is equivalent to the adsorption curves in panel A. For calculations it was assumed that individual free phages adsorb after an extracellular search of 1/kN min (squares) as calculated as a function of host density in Figure 2B. [Goto 2A, 2B, 2C, 2D, or all four]



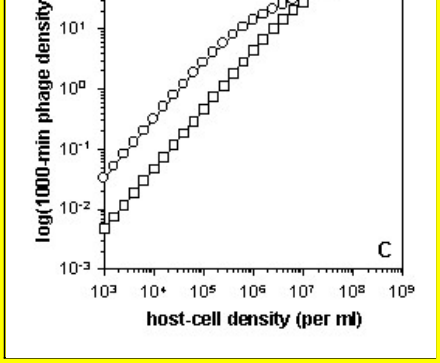
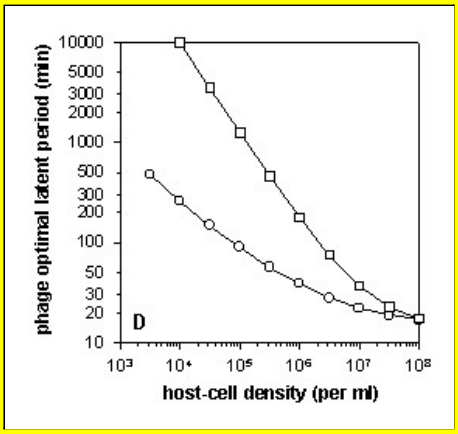


Figure 2D: Exponential phage adsorption and phage population growth (below). Phage latent period that gives rise to maximal phage population growth determined using simulations (adsorption via exponential free-phage decline; circles) and calculations (adsorption for all of free-phage cohort is mean free time in duration; squares) as described for panel C (graph used with permission from ASM). See (5) for discussion of methods. [Goto 2A, 2B, 2C, 2D, or all four]



We would expect similar compromises to hold for phages that release their progeny via extrusion. For such phages, however, the important balance should be between (i) the kinetics of phage maturation and release, (ii) the impact of greater rates of phage release on infected-host replication, and (iii) the overall latent-period duration. For experiments addressing these issues for filamentous phages see (46) and for lytic phages see Abedon (in preparation).

Phage plaque growth. Phage growth may be observed within a simple, spatially structured environment as plaques punctuating an otherwise opaque bacteria lawn embedded within a soft-agar overlay. Phage growth in plaques may be considered to occur in four stages (45): (i) initial adsorption of seeded phages, (ii) initial round of infection, (iii) an "enlargement phase" which involves multiple rounds of adsorption, infection, and release, and (iv) the end of the enlargement phase which typically is associated with physiological changes in the bacteria lawn. Differences between phage growth in plaques versus broth occur throughout the enlargement phase during which the physical structure of solid media (i) slows both phage and host diffusion, (ii) prevents gross environmental mixing, and (iii) probably gives rise to local phage multiplicities that are much higher than one observes over the majority of phage growth in broth. Phage growth within plaques additionally introduces plaque size as a means by which issues of phage fitness may be addressed (e.g., 49,50).

We can imagine at least five selective pressures that act on phages during plaque growth: (i) At the periphery of plaques there should be selection for more-rapid exponential growth, e.g., short phage latent periods when host densities are high (above); (ii) regardless of location within a plaque, during the plaque enlargement phase there should be selection for fast diffusion away from the plaque center such that uninfected hosts surrounding the plaque may be obtained and exploited (essentially the same argument as many suggested explained the classic observation that smaller phages should make larger plaques than larger phages; e.g., 72); (iii) towards the center of plaques - where there is a low prevalence of uninfected hosts - there should also exist a countering selection for greater burst sizes even at the expense of longer latent periods; (iv) throughout the plaque there should be selection exerted by the tendency of phages to decay (48) including by processes of adsorption to cell debris or adsorption to infected cells (the latter due to superinfection exclusion; 3); and (v) there can be selection for maintenance of phage growth despite the physiological aging of the bacterial lawn (e.g., phage T7; 50). Given this myriad complexity, *how*, *where*, and *when* one determines phage fitness during plaque growth is extremely important since different plaque regions may be under different selective pressures that can vary over the course of plaque development.

As a further complication, plaque size does not necessarily correlate with per-infection productivity. It has been hypothesized, for instance, that phages displaying shorter latent periods, even given smaller burst sizes, could display larger plaques (45,74). Longer latent periods resulting in smaller plaque sizes are most commonly (and classically) observed among T-even phages where lysis-inhibition defective (*r*) mutants display larger plaques and conditionally shorter latent periods than lysis-inhibition competent wild-type phages (34,43). I have also observed larger plaques with phage RB69 (also T-even-like; 8) that appear to be a consequence of reductions in phage latent periods (and burst size) rather than due to changes in phage adsorption rates or other increases in per-infection productivity (Abedon, in preparation). It has additionally been hypothesized (45,74) that reducing host-attachment efficiency given phage-host collision can increase rates of plaque enlargement since with slower adsorption phages might spend less time infecting cells and more time diffusing towards the periphery of plaques. Indeed, one explanation for why phage λ lost its tail fiber upon domestication (42) is that

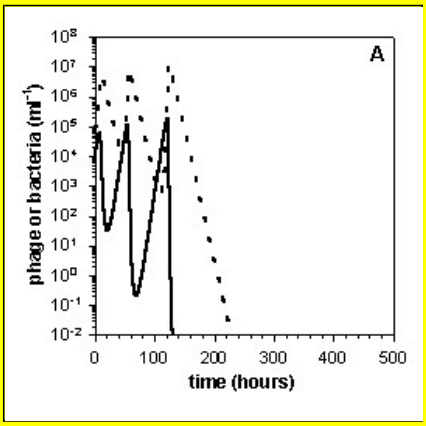
reduced adsorption efficiency resulted in the formation of inescapably selectable larger plaques. Sarma and Kuar (60) observed perhaps similar results with cyanophage N-1.

Phage community ecology

Community stability. Phage community ecology (Table 1) emphasizes the bacteria host, e.g., the impact of phages on bacteria densities and the evolution of phage resistance (10,16). Phage community ecology also considers phage-host coevolution, such as the propensity for phages to evolve strategies that counter mechanisms of host resistance. Bacteria evolution of phage resistance can contribute to the stability of phage-containing communities by impeding bacteria extinction. Stability additionally refers to the range in densities of host and phage populations as they oscillate over time, with greater oscillation amplitude (density variance) corresponding to lower community stability.

Phage community stability in the laboratory typically is studied within continuous phage-bacteria cocultures that are commonly, though when phages are present not necessarily correctly (58) referred to as chemostats. A chemostat possesses a reservoir containing sterile media connected to a well-mixed growth vessel containing microorganisms. Flow from reservoir to growth vessel may be controlled via the use of a peristaltic pump, with outflow from the growth vessel occurring at the same rate as inflow. Phage-host communities within chemostats often are more stable than may be accounted for by phage community ecology theory (16,61). In Figure 3A I present a simulation of a relatively unstable chemostat. Note that phages have driven phage-sensitive bacteria to extinction (<10^{-2}/ml) after about 110 hours of chemostat progression and that, due to outflow from the chemostat growth chamber, phages then decline to extinction about 100 hours later.

> **Figure 3A: Computer-simulated chemostats (below).** Chemostats were simulated employing the method and parameter values of Bohannan and Lenski (14). Time steps here are 1 min rather than 3 min; the initial host and phage densities are 10^4/ml and 10^5/ml, respectively; and unless otherwise noted (in subsequent simulations) the limiting nutrient is glucose which is found in the chemostat reservoir at a density of 0.5 mg/liter. Bacteria are presented as solid lines and phages as dotted lines. Phage and bacteria densities during simulations were sampled for inclusion in graphs once every 30 min. These simulated chemostats contain no phage-resistant bacteria or other bacteria refuges from phage attack. Extinction is assumed to occur at or below densities of 10^{-2}/ml. Bacteria are presented as solid lines and phages as dotted lines. [Goto 3A, 3B, 3C, 3D, or all four]



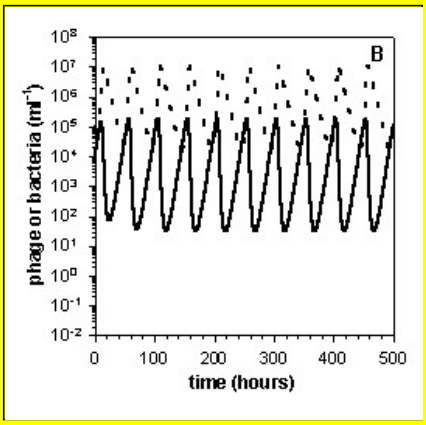
Refuges. Levin *et al*. (52) speculated that refuges for sensitive bacteria away from phage attack could increase the stability of phage-host communities, as subsequent experiments have corroborated (61). In such a scheme the extinction of sensitive bacteria is prevented by their hiding, for example, within chemostat wall populations. Following phage-induced lysis of host populations, presumably only those sensitive hosts survive that remain in hiding. Through cell division, these hosts can supply sensitive hosts to the liquid (unrefuged) phase of the chemostat. Once phage populations have declined, due to their outflow from the chemostat, the liquid-phase host populations can grow back to higher population densities.

Slowed adsorption. Bohannan and Lenski (15) describe bacteria that have entered a "genetic" refuge (phage-resistant mutants) as "invulnerable prey". However, since unless a phage's collision with a bacterium results in some degree of phage-host attachment, then a resistant bacterium is not potential prey but instead some relatively inert component of the environment off of which phages "bounce." Wilkinson (70), on the other hand, has suggested a model in which completely resistant bacteria really are invulnerable *prey*. Here the assumption is that the "predator" species (in this case a *Bdellovibrio*) may reversible interact with non-prey bacteria by pausing following collision. This delay in detachment extends the bdellovibrio's extracellular search. From the perspective of susceptible bacteria, this delay is equivalent to a reduction in the effective predator density. Wilkinson's conclusion upon modeling such a system is that the presence of non-prey bacteria, even in the absence of metabolic competition with prey bacteria, will result in a stabilization of sensitive-bacteria population densities.

Reductions in phage adsorption rates could similarly result in increased community stability. A partial reduction in host reception to phage adsorption (e.g., T2-partially resistant bacteria, a.k.a.,"less vulnerable" bacteria), for instance, should contribute to an increase in community stability by delaying overall of phage-population attachment to sensitive bacteria (17). Consistently, Figure 3B presents a simulated chemostat for which the phage rate of adsorption has been reduced by one half, and bacteria and phage extinctions are thereby avoided. Again with T2 phages, there apparently is a tendency for these phages to be temporarily adsorption-inhibited (up to weeks at room temperature) following release from host cells (59). This phenomenon could also serve to increase community stability by delaying phage adsorption. Indeed, any host refuge from phage adsorption should reduce phage population productivity by reducing the effective host density (a "numerical"

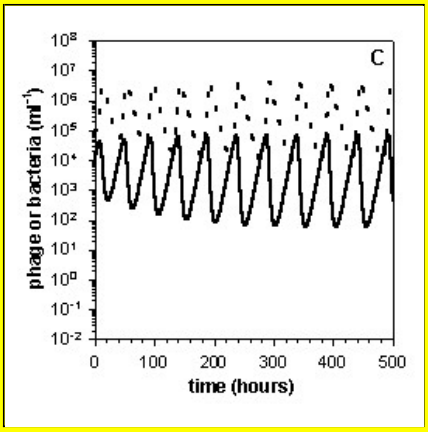
host refuge; 26,61), even if direct physical interactions between refuged (but otherwise phage-sensitive) hosts and free phages are nonexistant. By extension, mechanisms of phage decay, including outflow from chemostat growth chambers, should have the effect of reducing phage number, thereby increasing community stability. Furthermore, phage evolution that counters mechanisms that interfere with phage adsorption or decay should result in a decrease in community stability

Figure 3B: Computer-simulated chemostats (below). Figure shows the same chemostat as Figure 3A though with the phage adsorption constant reduced by one half. [Goto 3A, 3B, 3C, 3D, or all four]



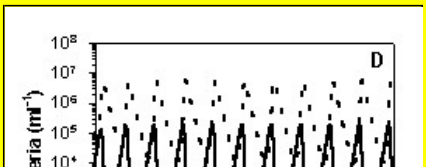
Reduced phage productivity. Host density impacts on community stability by affecting the peak phage densities that follow community-wide host lysis. With more phages than hosts within a batch-culture system, eventually all sensitive bacteria may become adsorbed and lysed (11,26). However, in continuous culture there will be decay in free-phage densities due to outflow from the growth vessel. Consequently, since the rate that host cells are found by free phages is a function of free-phage density (1), there is a race between phage sensitive bacteria survival and free-phage outflow. The lower the peak phage density, the less the bacteria population will be reduced in size due to phage adsorption, and the greater the likelihood that phage adsorption will not reduce the bacteria population to the point of extinction (compare, for example, the peak phage densities in Figure 3A versus Figures 3C and 3D). The smaller the bacteria population available for infection within a chemostat, in turn, the lower the peak phage density (compare Figures 3A and 3C). Bohannan and Lenski (14) demonstrate this point by reducing the bacteria growth potential, through restrictions in the density of a limiting nutrient (glucose) within the nutrient reservoir, and then observing an increase in phage-bacteria community stability. See Figure 3C for a simulated chemostat in which the nutrient density in the chemostat nutrient reservoir has been reduced by one half and note again that the extinction of bacteria and phages is avoided.

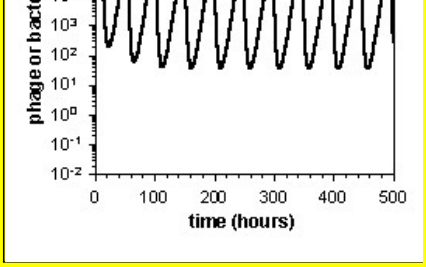
Figure 3C: Computer-simulated chemostats (below). Figure shows the same chemostat as Figure 3A but with one-half as much limiting glucose. [Goto 3A, 3B, 3C, 3D, or all four]



The productivity of phage infections, along with their density, together determines peak phage densities. It is well known that phage growth parameters, such as burst size, can vary as a function of host physiology (discussed above). If the stability of chemostats is an inverse function of peak phage density (i.e., more phages = less stability), then reduced infection productivity given reduced nutrient availability should contribute to an increase in community stability (as I will demonstrate elsewhere with chemostat simulations; Abedon, in preparation). Similarly, we might expect that the T-even-phage lysis-inhibition phenotype (1,3) would be destabilizing since it contributes, particularly at higher host densities, to a larger phage burst size. In Figure 3D the impact on community stability of reducing the phage burst size by one-half is explored, with bacteria and phage extinction yet again avoided.

Figure 3D: Computer-simulated chemostats (below). Figure shows the same chemostat as Figure 3A except with the phage burst size reduced by one half. [Goto 3A, 3B, 3C, 3D, or all four]





Synthesis. It is highly likely that phage-host community stability arises from two relatively simple forces: (i) If sensitive hosts cannot be driven to extinction by even excess phage densities, e.g., as is at least approximated with host refuges from phage attack, then sensitive hosts simply will not be driven to extinction by phages. (ii) If sensitive hosts can be driven to extinction given sufficient phage densities, then hosts will be driven to extinction only if sufficient phage densities are present within an ecosystem. There are two corollaries to the second point: (a) At peak phage density, the fewer phages found within an ecosystem, the smaller the negative impact those phages will have on phage-susceptible bacteria populations and the more stable the system (compare Figure 3A with Figures 3C and 3D). (b) Mechanisms that interfere with a phage's attainment of higher peak densities (or with phage impact on individual bacteria) - e.g., more phage decay, more-rapid outflow, partial inhibition of phage adsorption, reduced phage burst sizes - may lead to an increase in the stability of a phage-bacteria community (ditto, plus Figure 3B). Indeed, as noted by E.S. Anderson in 1957 (p. 205) (11), "It is evident that suboptimal conditions for growth of the host cells may restrict phage multiplication in any environment, even when contacts between the virus and its host occur... Anything which restricts the phage titre limits the selective action of phage."

Phage ecosystem ecology

Phage ecosystem ecology (Table 1) encompasses the biotic as well as the abiotic world, in particular the biogeochemical cycling of nutrients and the flow of energy between and through ecosystems, usually with an aquatic emphasis (for recent reviews see 38,53,57,63,64,69,73). Bacteria consume, produce, and store nutrients and energy plus contribute to the decomposition of other organisms. Phage infections contribute to a solubilization of bacteria cells, whether following host-cell lysis or via the conversion of host components into virion particles. Solubilized bacteria, in addition to no longer functioning as consumers, producers, or decomposers, are also less available as food to bacteria grazers (protists or animals) that obtain their nutrients and energy through the ingestion or engulfment of intact bacteria. Since bacteria are the chief consumers of the water-solubilized components of especially aquatic ecosystems, a major consequence of the phage-induced lysis of bacteria is not just a reduction in the productivity of bacteria populations but also a delay in the movement up food chains of bacteria-contained nutrients and energy. Suttle (64) additionally argues that aspects of virus-induced cell lysis in aquatic environments can have significant positive and negative impacts on the abundance of various greenhouse gasses found within Earth's atmosphere.

In their requirement for intact bacteria, phages in a sense are competitors of the bacteria grazers. Due to the host-range constraints observed among all parasites, individual phages also tend to be more specialized than most grazers in terms of what bacteria within a community they may affect (e.g., 37,66). In addition, phages can make direct, positive contributions to the fitness of bacteria hosts through phage conversion or via the transduction of genes from other bacteria. Phage DNA and protein coats, following abortive infection, could even serve as a bacteria nutrient (38).

Phage ecosystem ecology also represents an elaboration on the various issues of organismal, population, and community ecology already discussed. It follows, therefore, that many or all of the complications, caveats, and considerations discussed throughout this review also affect our understanding of the phage impact on ecosystem nutrient cycling and energy flow. In addition, much of the impact of phages on ecosystems has been discerned from the study of aquatic phage biology. However, aquatic systems - since for the most part they are liquid rather than solid, can be moderately well mixed, and also can be quite large - are among the very simplest phage-containing ecosystems. We might expect that other ecosystems, for example soils (22) or biofilms (41), would display greater complexity in terms of the phage impact on nutrient cycling and energy flow. Thus, both literally and figuratively, our understanding of the impact of phages on real ecosystems has barely scratched the surface of phage ecosystem ecology's ultimate goal: Quantifying the impact of phages on nutrient cycling and energy flow throughout the biosphere.

References

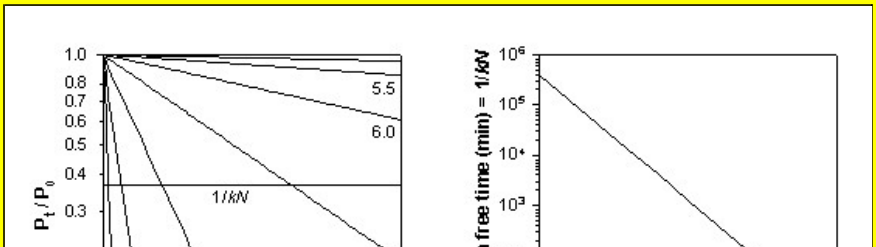
1. **Abedon, S.T.** 1990. Selection for lysis inhibition in bacteriophage. *Journal of Theoretical Biology* **146**:501-511.
2. **Abedon, S.T.** 1992. Lysis of lysis inhibited bacteriophage T4 infected cells. *Journal of Bacteriology* **174**:8073-8080.
3. **Abedon, S.T.** 1994. Lysis and the interaction between free phages and infected cells, p. 397-405. In J.D. Karam (ed.), *The Molecular Biology of Bacteriophage T4*. ASM Press, Washington, DC.
4. **Abedon, S.T.** 1999. Bacteriophage T4 resistance to lysis-inhibition collapse. *Genetical Research* **74**:1-11.
5. **Abedon, S.T., T.D. Herschler, and D. Stopar.** 2001. Bacteriophage latent-period evolution as a response to resource availability. *Applied and Environmental Microbiology* **67**:4233-4241.
6. **Ackermann, H.-W.** 1997. Bacteriophage ecology, p. 335-339. In M.T. Martins, M.I.Z. Sato, J.M. Tiedje, L.C.N. Hagler, J. D□bereiner, and P.S. Sanchez (eds.), *Progress in Microbial Ecology* (Proceedings of Seventh International Symposium on Microbial Ecology). Brazilian Society for Microbiology,
7. **Ackermann, H.-W. and M.S. DuBow .** 1987. *Viruses of Prokaryotes*. CRC Press, Boca Raton, Florida.
8. **Ackermann, H.-W. and H.M. Krisch.** 1997. A catalogue of T4-type bacteriophages. *Archives of Virology* **142**:2329-2345.

9. **Adams, M.H. and F. Wassermann.** 1956. Frequency distribution of phage release in the one-step growth experiment. *Virology* **2**:96-108.
10. **Allison, G.E. and T.R. Klaenhammer.** 1998. Phage resistance mechanisms in lactic acid bacteria. *International Dairy Journa*l **8**:207-226.
11. **Anderson, E.S.** 1957. The relations of bacteriophages to bacterial ecology, p. 189-217. In R.E.O. Williams and C.C. Spicer (eds.), *Microbial Ecology*. Cambridge University Press, London.
12. **Barron, B.A., V.A. Fischetti, and J.B. Zabriskie.** 1970. Studies of the bacteriophage kinetics of multicellular systems: a statistical model for the estimation of burst size per cell in streptococci. *Journal of Applied Bacteriology* **33**:436-442.
13. **Bentzon, M.W., O. Maaloe, and G. Rasch.** 1952. An analysis of the mode of increase in number of intracellular phage particles at different temperatures. *Acta Pathologica et Microbiologica Scandinavica - Section B, Microbiology and Immunology* **30**:243-270.
14. **Bohannan, B.J.M. and R.E. Lenski.** 1997. Effect of resource enrichment on a chemostat community of bacteria and bacteriophage. *Ecology* **78**:2303-2315.
15. **Bohannan, B.J.M. and R.E. Lenski.** 1999. Effect of prey heterogeneity on the response of a food chain to resource enrichment. *American Naturalist* **153**:73-82. 16
16. **Bohannan, B.J.M. and R.E. Lenski.** 2000a. Linking genetic change to community evolution: insights from studies of bacteria and bacteriophage. *Ecological Letters* **3**:362-377.
17. **Bohannan, B.J.M. and R.E. Lenski.** 2000b. The relative importance of competition and predation varies with productivity in a model community. *American Naturalist* **156**:329-340.
18. **Boucher, I. and S. Moineau.** 2001. Phages of Lactococcus lactis: an ecological and economical equilibrium. *Recent Research Developments in Virology* **3**:243-256.
19. **Bremermann, H.-J.** 1983. Parasites at the origin of life. *Journal of Mathematical Biology* **16**:165-180.
20. **Bullas, L.R., A.J. Zuccarelli, and R.L. Nutter.** 1967. Aging effect on phage particles leading to an increase in burst size. *Nature* **216**:1308
21. **Burnet, F.M.** 1934. The bacteriophages. *Biological Reviews of the Cambridge Philosophical Society* **9**:332-350.
22. **Burroughs, N.J., P. Marsh, and E.M.H. Wellington.** 2000. Mathematical analysis of growth and interaction dynamics of streptomycetes and a bacteriophage in soil. *Applied and Environmental Microbiology* **66**:3868-3877.
23. **Campbell, A.** 1961. Conditions for the existence of bacteriophages. *Evolution* **15**:153-165.
24. **Chao, L.** 2000. The meaning of life. *BioScience* **50**:245-250.
25. **Chao, L., K.A. Hanley, C.L. Burch, C. Dahlberg, and P.E. Turner.** 2000. Evolution of virulence in parasites: making hard and soft choices. *Quarterly Review of Biology* **75**:261-275.
26. **Chao, L., B.R. Levin, and F.M. Stewart.** 1977. A complex community in a simple habitat: An experimental study with bacteria and phage. *Ecology* **58**:369-378.
27. **Chopin, A., A. Bolotin, A. Sorokin, S.D. Ehrlich, and M.C. Chopin.** 2001. Analysis of six prophages in Lactococcus lactis IL1403: Different genetic structure of temperate and virulent phage populations. *Nucleic Acids Research* **29**:644-651.
28. **Conley, M.P. and W.B. Wood.** 1975. Bacteriophage T4 whiskers: A rudimentary environment-sensing device. *Proceedings of the National Academy of Sciences, U.S.A.* **72**:3701-3705.
29. **Corbin, B.D., G.M. Aron, and R.J.C. McLeon.** 2001. Bacteriophage T4 multiplication in an Escherichia coli biofilm. *Canadian Journal of Microbiology* **47**:680-684.
30. **Daniels, L.L. and A.C. Wais.** 1998. Virulence of phage populations infecting Halobacterium cutirubrum. *FEMS Microbiol Ecology*. **25**:129-134.
31. **DeFilippis, V.R. and L.P. Villarreal.** 2000. An introduction to the evolutionary ecology of viruses, p. 125-208. In C.J. Hurst (ed.), *Viral Ecology*. Academic Press, San Diego.
32. **Delbrück, M.** 1942. Bacterial viruses (bacteriophages). *Advances in Enzymology* **2**:1-32.
33. **Delbrück, M.** 1945. The burst size distribution in the growth of bacterial viruses (bacteriophages). *Journal of Bacteriology* **50**:131-135.
34. **Doermann, A.H.** 1948. Lysis and lysis inhibition with Escherichia coli bacteriophage. *Journal of Bacteriology* **55**:257-275.
35. **Doermann, A.H.** 1952. The intracellular growth of bacteriophages. I. Liberation of intracellular bacteriophage T4 by premature lysis with another phage or with cyanide. *Journal of General Physiology* **35**:645-656.
36. **Douglas, J.** 1975. *Bacteriophages*. p.77-133. Chapman and Hall, London.
37. **Frank, S.A.** 1994. Polymorphism of bacterial restriction-modification systems: The advantage of diversity. *Evolution* **48**:1470-1477.
38. **Fuhrman, J.A.** 1999. Marine viruses and their biogeochemical and ecological effects. *Nature* **399**:541-548.
39. **Goyal, S.M., C.P. Gerba, and G. Bitton.** 1987. *Phage Ecology*. CRC Press, Boca Raton, Florida.
40. **Hadas, H., M. Einav, I. Fishov, and A. Zaritsky.** 1997. Bacteriophage T4 development depends on the physiology of its host Escherichia coli. *Microbiology* **143**:179-185.
41. **Hanlon, G.W., S.P. Denyer, C.J. Olliff, and L.J. Ibrahim.** 2001. Reduction in exopolysaccharide viscosity as an aid to bacteriophage penetration through Pseudomonas aeruginosa biofilms. *Applied and Environmental Microbiology* **67**:2746-2753.
42. **Hendrix, R.W. and R.L. Duda.** 1992. Bacteriophage lamda PaPa: not the mother of all lambda phages. *Science* **258**:1145-1148.
43. **Hershey, A.D.** 1946. Mutation of bacteriophage with respect to type of plaque. *Genetics* **31**:620-640.
44. **Hurst, C.J. and H.D.A. Lindquist.** 2000. Defining the ecology of viruses, p. 3-40. In C.J. Hurst (ed.), *Viral Ecology*. Academic Press, San Diego.
45. **Koch, A.L.** 1964. The growth of viral plaques during the enlargement phase. *Journal of Theoretical Biology* **6**:413-431.
46. **Kuo, M.Y., M.K. Yang, W.P. Chen, and T.T. Kuo.** 2000. High-frequency interconversion of turbid and

clear plaque strains of bacteriophage f1 and associated host cell death. *Canadian Journal of Microbiology* **46**:841-847.

47. **Kutter, E., E. Kellenberger, K. Carlson, S. Eddy, J. Neitzel, L. Messinger, J. North, and B. Guttman.** 1994. Effects of bacterial growth conditions and physiology on T4 infection, p. 406-418. In J.D. Karam (ed.), *The Molecular Biology of Bacteriophage T4*. ASM Press, Washington, DC.
48. **Lee, Y., S.D. Eisner, and J. Yin.** 1997. Antiserum inhibition of propagating viruses. *Biotechnology and Bioengineering* **55**:542-546.
49. **Lee, Y. and J. Yin.** 1996b. Detection of evolving viruses. *Nature Biotechnology* **14**:491-493.
50. **Lee, Y. and J. Yin.** 1996a. Imaging the propagation of viruses. *Biotechnology and Bioengineering* **52**:438-442.
51. **Lenski, R.E.** 1988. Dynamics of interactions between bacteria and virulent bacteriophage. *Advances in Microbial Ecology* **10**:1-44.
52. **Levin, B.R., F.M. Stewart, and L. Chao.** 1977. Resource limited growth, competition, and predation: A model and experimental studies with bacteria and bacteriophage. *American Naturalist* **111**:3-24.
53. **Martin, E.L. and T.A. Kokjohn.** 1999. Cyanophages, p. 324-332. In A. Granoff and R.G. Webster (eds.), *Encyclopedia of Virology* second edition. Academic Press, San Diego.
54. **McLean, R.J., B.D. Corbin, G.J. Balzer, and G.M. Aron.** 2001. Phenotype characterization of genetically defined microorganisms and growth of bacteriophage in biofilms. *Methods in Enzymology* **336**:163-174.
55. **Middelboe, M.** 2000. Bacterial growth rate and marine virus-host dynamics. *Microbial Ecology* **40**:114-124.
56. **Murray, A.G. and G.A. Jackson.** 1992. Viral dynamics: A model of the effects of size, shape, motion, and abundance of single-celled planktonic organisms and other particles. *Marine Ecology Progress Series* **89**:103-116.
57. **Paul, J.H. and C.A. Kellogg.** 2000. Ecology of bacteriophages in nature, p. 211-246. In C.J. Hurst (ed.), *Viral Ecology*. Academic Press, San Diego.
58. **Paynter, M.J.B. and H.R. Bungay III.** 1971. Characterization of virulent bacteriophage infections of Escherichia coli in continuous culture. *Science* **172**:405-405.
59. **Sagik, B.P.** 1954. A specific reversible inhibition of bacteriophage T2. *Journal of Bacteriology* **68**:430-436.
60. **Sarma, T.A. and B. Kaur.** 1997. Characterization of host-range mutants of cyanophage N-1. *Acta Virology* **41**:245-250.
61. **Schrag, S. and J.E. Mittler.** 1996. Host-parasite persistence: the role of spatial refuges in stabilizing bacteria-phage interactions. *American Naturalist* **148**:348-347.
62. **Stewart, F.M. and B.R. Levin.** 1984. The population biology of bacterial viruses: Why be temperate. *Theoretical Population Biology* **26**:93-117.
63. **Suttle, C.A.** 2000a. Cyanophages and their role in the ecology of cyanobacteria, p. 563-589. In B.A. Whitton and M. Potts (eds.), *The Ecology of Cyanobacteria: Their Diversity in Time and Space*. Kluwer Academic Publishers, Boston.
64. **Suttle, C.A.** 2000b. The ecology, evolutionary and geochemical consequences of viral infection of cyanobacteria and eukaryotic algae, p. 248-286. In C.J. Hurst (ed.), *Viral Ecology*. Academic Press, New York.
65. **Suttle, C.A. and F. Chen.** 1992. Mechanisms and rates of decay of marine viruses in seawater. *Applied and Environmental Microbiology* **58**:3721-3729.
66. **Thingstad, T.F.** 2000. Elements of a theory for the mechanisms controlling abundance, diversity, and biogeochemical role of lytic bacterial viruses in aquatic systems. *Limnology and Oceanography* **45**:1320-1328.
67. **Wang, I.-N., D.L. Smith, and R. Young.** 2000. Holins: The protein clocks of bacteriophage infections. *Annual Review of Microbiology* **54**:799-825.
68. **Webb, V., E. Leduc, and G.B. Spiegelman.** 1982. Burst size of bacteriophage SP82 as a function of growth rate of its host Bacillus subtilis. *Canadian Journal of Microbiology* **28**:1277-1280.
69. **Wilhelm, S.W. and C.A. Suttle.** 2000. Viruses as regulators of nutrient cycles in the sea, p. 551-556. In *Proceedings of the 8th International Symposium of Microbial Ecology*.
70. **Wilkinson, M.H.F.** 2001. Predation in the presence of decoys: An inhibitory factor on pathogen control by bacteriophages or bdellovibrios in dense and diverse ecosystems. *Journal of Theoretical Biology* **208**:27-36.
71. **Williams, S.T., A.M. Mortimer, and L. Manchester.** 1987. Ecology of soil bacteriophages, p. 157-179. In S.M. Goyal, C.P. Gerba, and G. Bitton (eds.), *Phage Ecology*. John Wiley & Sons, New York.
72. **Wilson, G.S. and A.A. Miles.** 1946. The bacteriophage, p. 325-350. In *Topley and Wilson's Principles of Bacteriology and Immunity*. Williams and Wilkins, Baltimore.
73. **Wommack, K.E. and R.R. Colwell .** 2000. Virioplankton: viruses in aquatic ecosystems. *Microbiology and Molecular Biology Reviews* **64**:69-114.
74. **Yin, J. and J.S. McCaskill.** 1992. Replication of viruses in a growing plaque: A reaction-diffusion model. *Biophysical Journal* **61**:1540-1549.

Figure 2: Exponential phage adsorption and phage population growth (below).



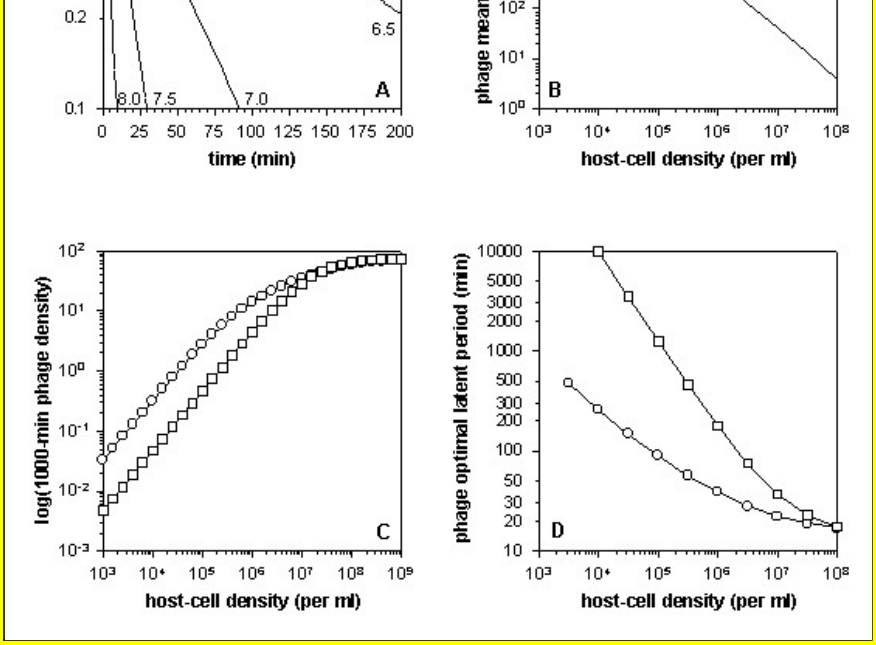
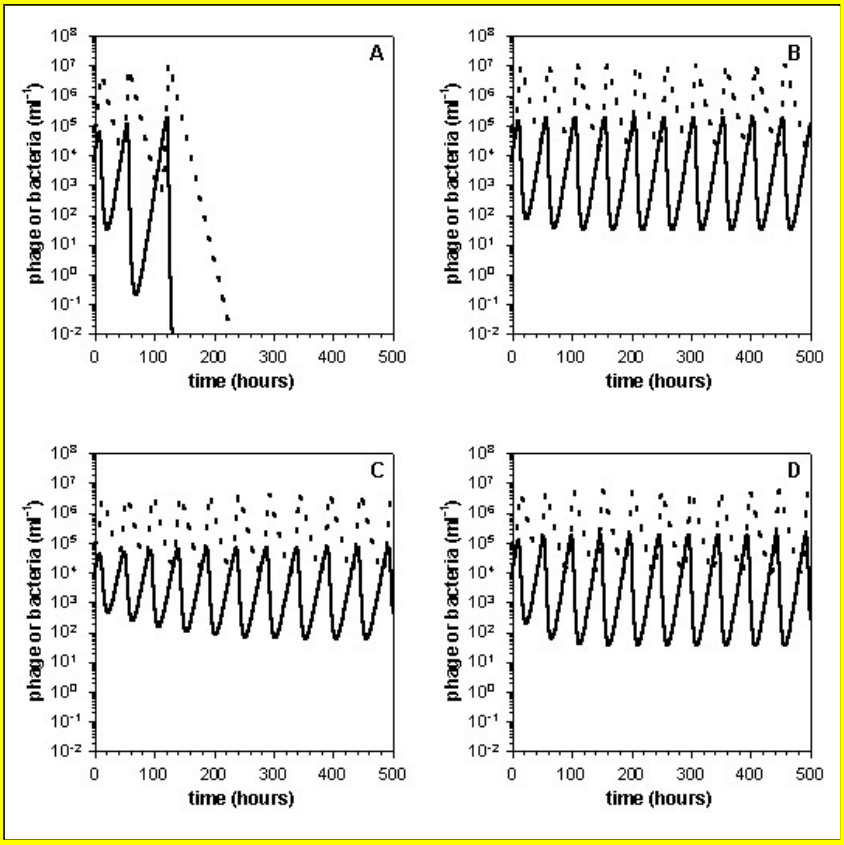


Figure 3: Computer-simulated chemostats (below).



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Letters & Questions

Letters should consist of comments, short statements, or personal editorials. Send all letters to abedon.1@osu.edu or to "Letters", Bacteriophage Ecology Group News, care of Stephen T. Abedon, Department of Microbiology, The Ohio State

University, 1680 University Dr., Mansfield, Ohio 44906. Please send all letters in English and all mailed or attached letters as Microsoft Word documents, if possible (I'll let you know if I have trouble converting any other document formats). In addition, to standard letters, BEG receives questions on a regular basis that may be addressed by BEG members. These questions are listed below. Anybody interested in answering these questions through *BEG News*, e-mail me at the following address: abedon.1@osu.edu. Alternatively, answer by clicking the authors name. Please note that these questions have not been edited for grammar, spelling, or clarity.

No entry.

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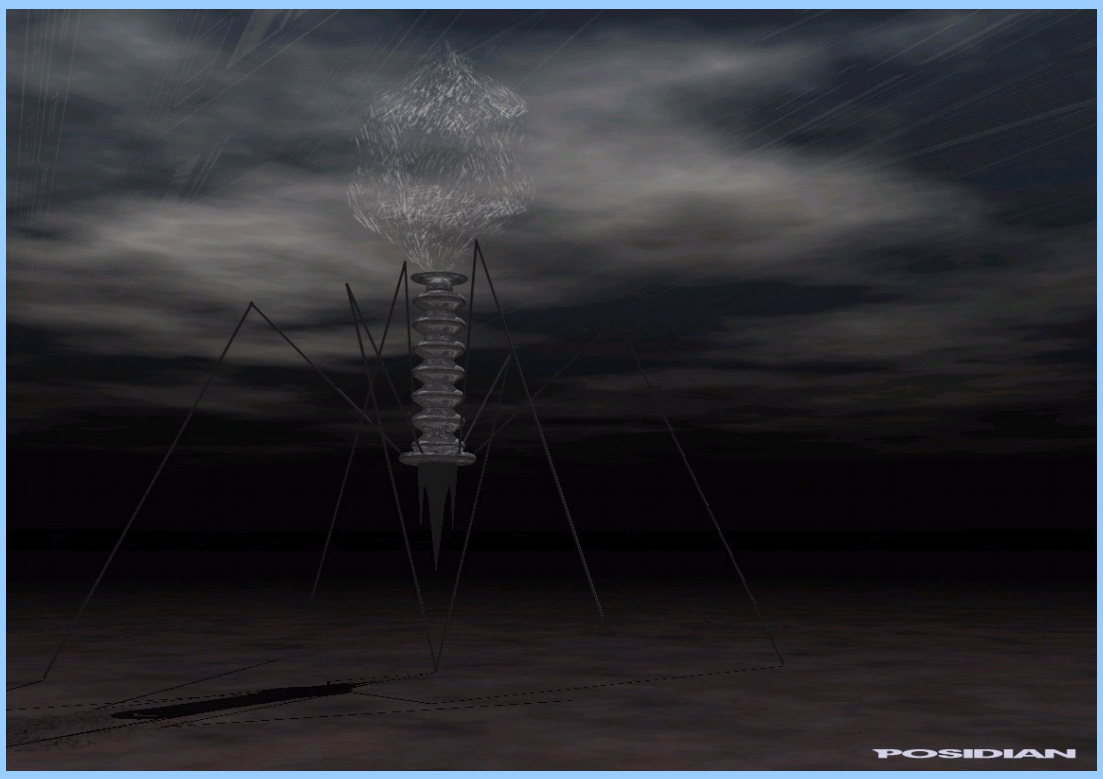
Phage Images

Please send any phage images that you would like to present in this section to "Phage Images," *The Bacteriophage Ecology Group*, care of Stephen T. Abedon, Department of Microbiology, The Ohio State University, 1680 University Dr., Mansfield, Ohio 44906. Alternatively, you may scan the images yourself and send them as an attachment to abedon.1@osu.edu. Please save all scans in gif or jpg formats and preferably with an image size (in terms of width, height, and kbytes) that will readily fit on a standard web page. No copyrighted material without permission, please!



"T4 phage v1 by ~posidian", a.k.a., Joshua Orvis, Associate Director of Bioinformatics, University of Oklahoma Health Sciences Center, joshua-orvis@ouhsc.edu.

Phage Image Archive

- BEG Phage Images Page
- The Face of the Phage
- Bacteriophage T2 by H.-W. Ackermann
- SSV1-Type Phage
- Saline Lake Bacteriophage - David Bird
- Coliphage LG1 - Larry Goodridge
- Bacteriophage HK97 - Bob Duda
- Phage T4 (art) - Francis S. Lin
- Phage T4 on the pedestal outside of Barker Hall at Berkeley
- Electron micrograph of phage P22
- Thin section of T4 phages hitting a microcolony of E. coli K-12 - John Wertz
- T4 phage v1 - Posidian

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New Publications

New bacteriophage publications are listed below. Each quarter not-yet-listed publications from the previous two years will be presented along with their abstracts. The indicator "???" denotes, of course, that specific information is not yet in the BEG Bibliography. Please help in the compilation of the BEG Bibliography by supplying any updated information, correcting any mistakes, and, of course, sending the references to your bacteriophage ecology publications, as well as the

references to any bacteriophage ecology publications that you know of but which are not yet in the bibliography (send to abedon.1@osu.edu or to "BEG Bibliography," *Bacteriophage Ecology Group News*, care of Stephen T. Abedon, Department of Microbiology, The Ohio State University, 1680 University Dr., Mansfield, Ohio 44906). Also, be sure to indicate any listed publications that you feel should not be presented in the BEG Bibliography. This list is also present with available abstracts at the end of *BEG News*.

1. Distribution of virus-like particles in an oligotrophic marine environment (Alboran Sea, Western Mediterranean). Alonso, M. C., Jimenez-Gomez, F., Rodriguez, J., Borrego, J. J. (2001). *Microbial Ecology* 42:407-415. [PRESS FOR ABSTRACT]

2. The bacteriophages of ruminal prevotellas. Ambrozic, J., Ferme, D., Grabnar, M., Ravnikar, M., Avgustin, G. (2001). *Folia Microbiologica* 46:37-39. [PRESS FOR ABSTRACT]

3. Isolation and characterization of bacteriophage-resistant mutants of *Vibrio cholerae* O139. Attridge, S. R., Fazeli, A., Manning, P. A., Stroeher, U. H. (2001). *Microbial Pathogenesis* 30:237-246. [PRESS FOR ABSTRACT]

4. Bacteriophage-bacteriophage interactions in the evolution of pathogenic bacteria. Boyd, E. F., Davis, B. M., Hochhut, B. (2001). *Trends in Microbiology* 9:137-144. [PRESS FOR ABSTRACT]

5. Chemical and microbial characterization of household graywater. Casanova, L. M., Gerba, C. P., Karpiscak, M. (2001). *J Environ Sci Health Part A Tox Hazard Subst Environ Eng* 36:395-401. [PRESS FOR ABSTRACT]

6. Microbial population dynamics and diversity during a bloom of the marine coccolithophorid *Emiliania huxleyi* (Haptophyta). Castberg, T., Larsen, A., Sandaa, R. A., Brussaard, C. P. D., Egge, J. K., Heldal, M., Thyrhaug, R., van Hannen, E. J., Bratbak, G. (2001). *Marine Ecology Progress Series* 221:39-46. [PRESS FOR ABSTRACT]

7. Nucleotide sequence of coliphage HK620 and the evolution of lambdoid phages. Clark, A. J., Inwood, W., Cloutier, T., Dhillon, T. S. (2001). *Journal of Molecular Biology* 311:657-679. [PRESS FOR ABSTRACT]

8. Bacteriophage T4 multiplication in an *Escherichia coli* biofilm. Corbin, B. D., Aron, G. M., McLeon, R. J. C. (2001). *Canadian Journal of Microbiology* 47:680-684. [PRESS FOR ABSTRACT]

9. Progeny of the phage school. Dixon, B. (2001). *ASM News* 69:432-433. [PRESS FOR ABSTRACT]

10. Direct and quantitative detection of bacteriophage by "hearing" surface detachment using a quartz crystal microbalance. Dultsev, F. N., Speight, R. E., Fiorini, M. T., Blackburn, J. M., Abell, C., Ostanin, V. P., Klenerman, D. (2001). *Analytical Chemistry* 73:3935-3939. [PRESS FOR ABSTRACT]

11. Diminished diarrheal response to *Vibrio cholerae* strains carrying the replicative form of the CTXf genome instead of CTXf lysogens in adult rabbits. Faruque, S. M., Rahman, M. M., Hasan, A. K., Nair, G. B., Mekalanos, J. J., Sack, D. A. (2001). *Infection and Immunity* 69:6084-6090. [PRESS FOR ABSTRACT]

12. Phage antibacterials make a comeback. Fischetti, V. A. (2001). *Nature Biotechnology* 19:734-735. [NO ABSTRACT]

13. A conserved genetic module that encodes the major virion components in both the coliphage T4 and the marine cyanophage S-PM2. Hambly, E., Tétart, F., Desplats, C., Wilson, H., Krisch, H. M., Mann, N. H. (2001). *Proceedings of the National Academy of Sciences, USA* 98:11411-11416. [PRESS FOR ABSTRACT]

14. Isolation and characterization of a temperature-sensitive generalized transducing bacteriophage for *Vibrio cholerae*. Hava, D. L., Camilli, A. (2001). *J Microbiol Methods* 46:217-225. [PRESS FOR ABSTRACT]

15. Effects of concentrated viral communities on photosynthesis and community composition of co-occurring benthic microalgae and phytoplankton. Hewson, I., O'Neil, J. M., Heil, C. A., Bratbak, G., Dennison, W. C. (2001). *Aquatic Microbial Ecology* 25:1-10. [PRESS FOR ABSTRACT]

16. Mosaic structure of shiga-toxin-2-encoding phages isolated from *Escherichia coli* O157:H7 indicates frequent gene exchange between lambdoid phage genomes. Johansen, B. K., Wasteson, Y., Granum, P. E., Brynestad, S. (2001). *Microbiology* 147:1929-1936. [NO ABSTRACT]

17. Elimination of fecal coliforms and F-specific RNA coliphage from oysters (*Crassostrea virginica*) relaid in floating containers. Kator, H., Rhodes, M. (2001). *Journal of Food Protection* 64:796-801. [PRESS FOR ABSTRACT]

18. Octamer-based genome scanning distinguishes a unique subpopulation of *Escherichia coli* O157:H7 strains in cattle. Kim, J., Nietfeldy, J., Benson, A. K. (2001). *Proceedings of the National Academy of Sciences, USA* 96:13288-13293. [PRESS FOR ABSTRACT]

19. Antacid increases survival of *Vibrio vulnificus* and *Vibrio vulnificus* phage in a gastrointestinal model. Koo, J., Marshall, D. L., Depaola, A. (2001). *Applied and Environmental Microbiology* 67:2895-2902. [PRESS FOR ABSTRACT]

20. [*Vibrio cholerae* O139 bacteriophages]. Kudriakova, T. A., Makedonova, L. D., Kachkina, G. V., Saiamov, S. R. (2001). *Zhurnal Mikrobiologii, Epidemiologii i Immunobiologii* 28-30. [PRESS FOR ABSTRACT]

21. Population dynamics and diversity of phytoplankton, bacteria and viruses in a seawater enclosure. Larsen, A., Castberg, T., Sandaa, R. A., Brussaard, C. P. D., Egge, J. K., Heldal, M., Paulino, A., Thyrhaug, R., van Hannen, E. J., Bratbak, G. (2001). *Marine Ecology Progress Series* 221:47-57. [PRESS FOR ABSTRACT]

22. Viruses in the plankton of freshwater and saline Antarctic lakes. Laybourn-Parry, J., Hofer, J. S., Sommaruga, R. (2001). *Freshwater Biology* 46:1279-1287. [PRESS FOR ABSTRACT]

23. Examination of bacteriophage as a biocontrol method for salmonella on fresh-cut fruit: a model study. Leverentz, B., Conway, W. S., Alavidze, Z., Janisiewicz, W. J., Fuchs, Y., Camp, M. J., Chighladze, E., Sulakvelidze, A. (2001). *Journal of Food Protection* 64:1116-1121. [PRESS FOR ABSTRACT]

24. Colloidal interactions in suspensions of rods. Lin, K., Crocker, J. C., Zeri, A. C., Yodh, A. G. (2001). *Phys Rev Lett* 87:088301. [PRESS FOR ABSTRACT]

25. Depolymerization of the capsular polysaccharide from *Vibrio cholerae* O139 by a lyase associated with the bacteriophage JA1. Linnerborg, M., Weintraub, A., Albert, M. J., Widmalm, G. (2001). *Carbohydrate Research* 333:263-269. [PRESS FOR ABSTRACT]

26. Physiological function of exopolysaccharides produced by *Lactococcus lactis*. Looijesteijn, P. J., Trapet, L., de, Vries E., Abee, T., Hugenholtz, J. (2001). *International Journal of Food Microbiology* 64:71-80. [PRESS FOR ABSTRACT]

27. Distribution, isolation, host specificity, and diversity of cyanophages infecting marine *Synechococcus* spp. in river estuaries. Lu, J., Chen, F., Hodson, R. E. (2001). *Applied and Environmental Microbiology* 67:3285-3290. [PRESS FOR ABSTRACT]

28. Elution, detection, and quantification of polio I, bacteriophages, *Salmonella montevideo*, and *Escherichia coli* O157:H7 from seeded strawberries and tomatoes. Lukasik, J., Bradley, M. L., Scott, T. M., Hsu, W. Y., Farrah, S. R., Tamplin, M. L. (2001). *Journal of Food Protection* 64:292-297. [PRESS FOR ABSTRACT]

29. The genome of archaeal prophage PsiM100 encodes the lytic enzyme responsible for autolysis of *Methanothermobacter wolfeii*. Luo, Y., Pfister, P., Leisinger, T., Wasserfallen, A. (2001). *Journal of Bacteriology* 183:5788-5792. [PRESS FOR ABSTRACT]

30. Sequence analysis and molecular characterization of the *Lactococcus lactis* temperate bacteriophage BK5-T. Mahanivong, C., Boyce, J. D., Davidson, B. E., Hillier, A. J. (2001). *Applied and Environmental Microbiology* 67:3564-3576. [PRESS FOR ABSTRACT]

31. Growth and survival of clinical vs. environmental species of *Aeromonas* in tap water. Mary, P., Buchet, G., Defives, C., Hornez, J. P. (2001). *International Journal of Food Microbiology* 69:191-198. [PRESS FOR ABSTRACT]

32. Livestock deaths associated with *Clavibacter toxicus*/*Anguina* sp. infection in seedheads of *Agrostis avenacea* and *Polypogon monspeliensis*. McKay, A. C., Ophel, K. M., Reardon, T. B., Gooden, J. M. (2001). *Plant Disease* 77:635-641. [PRESS FOR ABSTRACT]

33. Characterization of two novel *Rhizobium leguminosarum* bacteriophages from a field release site of genetically-modified rhizobia. Mendum, T. A., Clark, I. M., Hirsch, P. R. (2001). *Antonie van Leeuwenhoek* 79:189-197.

34. Effect of phage on survival of *Salmonella enteritidis* during manufacture and storage of cheddar cheese made from raw and pasteurized milk. Modi, R., Hirvi, Y., Hill, A., Griffiths, M. W. (2001). *Journal of Food Protection* 64:927-933. [PRESS FOR ABSTRACT]

35. Phage conversion of Panton-Valentine leukocidin in *Staphylococcus aureus*: molecular analysis of a PVL-converting phage, phiSLT. Narita, S., Kaneko, J., Chiba, J., Piemont, Y., Jarraud, S., Etienne, J., Kamio, Y. (2001). *Gene* 268:195-206. [PRESS FOR ABSTRACT]

36. Increased mutation rate of E. coli K12 lambda cultures maintained in continuous logarithmic growth. Northrop, J. H. (2001). *Journal of General Physiology* 50:369-377. [PRESS FOR ABSTRACT]

37. Pathogenicity and resistance islands of staphylococci. Novick, R. P., Schlievert, P., Ruzin, A. (2001). *Microbes Infect* 3:585-594. [PRESS FOR ABSTRACT]

38. Diversification of *Escherichia coli* genomes: are bacteriophages the major contributors? Ohnishi, M., Kurokawa, K., Hayashi, T. (2001). *Trends in Microbiology* 9:481-485. [PRESS FOR ABSTRACT]

39. Bacteriophage P4282, a parasite of *Ralstonia solanacearum*, encodes a bacteriolytic protein important for lytic infection of its host. Ozawa, H., TANAKA, H., Ichinose, Y., Shiraishi, T., Yamada, T. (2001). *MGG Molecular Genetics and Genomics* 265:95-101.

40. Survival of bacteriophages of Lactococcus lactis in sodium hypochlorite and during storage. Parada, J. L., De Fabrizio, S. V. (2001). *Revista Argentina de Microbiologia* 33:89-95.

41. Comparative study of nine *Lactobacillus fermentum* bacteriophages. Picozzi, C., Galli, A. (2001). *Journal of Applied Microbiology* 91:394-403.

42. Evolutionary role of restriction/modification systems as revealed by comparative genome analysis. Rocha, E. P., Danchin, A., Viari, A. (2001). *Genome Research* 11:946-958. [PRESS FOR ABSTRACT]

43. Changes in bacterial community composition and dynamics and viral mortality rates associated with enhanced flagellate grazing in a mesoeutrophic reservoir. Simek, K., Weinbauer, M. G., Hornak, K., Dolan, J. R., Nedoma, J., Masin, M., Amann, R. (2001). *Applied and Environmental Microbiology* 67:2723-2733. [NO ABSTRACT]

44. Quorum sensing is a global regulatory mechanism in enterohemorrhagic *Escherichia coli* O157:H7. Sperandio, V, Torres, A. G., Giron, J. A., Kaper, J. B. (2001). *Journal of Bacteriology* 183:5187-5197. [PRESS FOR ABSTRACT]

45. Application of *Streptococcus thermophilus* DPC1842 as an adjunct to counteract bacteriophage disruption in a predominantly

lactococcal Cheddar cheese starter: use in bulk starter culture systems. Stokes, D., Ross, R. P., Fitzgerald, G. F., Coffey, A. (2001). *Lait* 81:327-334. [PRESS FOR ABSTRACT]

46. Therapy of infections in cancer patients with bacteriophages. Weber-Dabrowska, B., Mulczyk, M., Górski, A. (2001). *CLIN APPL IMMUNOL REV* 1:131-134. [PRESS FOR ABSTRACT]

47. Interaction of the PhiHSIC virus with its host: lysogeny or pseudolysogeny? Williamson, S. J., McLaughlin, M. R., Paul, J. H. (2001). *Applied and Environmental Microbiology* 67:1682-1688. [PRESS FOR ABSTRACT]

48. Integrated management of bacterial leaf spot of mungbean with bacteriophages of Xav and chemicals. Borah, P. K., Jindal, J. K., Verma, J. P. (2000). *Journal of Mycology and Plant Pathology* 30:19-21.

49. Viruses of fungi and protozoans: Is everyone sick? Bruenn, J. A. (2000). pp. 297-317 in Hurst, C. J. (ed.) *Viral Ecology*. Academic Press, San Diego. [NO ABSTRACT]

50. Lateral gene transfer in prokaryotes. Campbell, A. M. (2000). *Theoretical Population Biology* 57:71-77. [PRESS FOR ABSTRACT]

51. An introduction to the evolutionary ecology of viruses. DeFilippis, V. R., Villarreal, L. P. (2000). pp. 125-208 in Hurst, C. J. (ed.) *Viral Ecology*. Academic Press, San Diego. [NO ABSTRACT]

52. Microvirus of *Chlamydia psittaci* strain Guinea pig inclusion conjunctivitis: Isolation and molecular characterization. Hsia, R. C., Ting, L. M., Bavoil, P. M. (2000). *Microbiology (Reading)* 146:1651-1660.

53. Genomic sequences of bacteriophages HK97 and HK022: Pervasive genetic mosaicism in the lambdoid bacteriophages. Juhala, R. J., Ford, M. E., Duda, R. L., Youlton, A., Hatfull, G. F., Hendrix, R. W. (2000). *Journal of Molecular Biology* 299:27-51.

54. Ecology of bacteriophages in nature. Paul, J. H., Kellogg, C. A. (2000). pp. 211-246 in Hurst, C. J. (ed.) *Viral Ecology*. Academic Press, San Diego. [NO ABSTRACT]

55. Genomic sequence and analysis of the atypical temperate bacteriophage N15. Ravin, V, Ravin, N., Casjens, S., Ford, M. E., Hatfull, G. F., Hendrix, R. W. (2000). *Journal of Molecular Biology* 299:53-73.

56. Comparative analysis of Chlamydia bacteriophages reveals variation localized to a putative receptor binding domain. Read, T. D., Fraser, C. M., Hsia, R. C., Bavoil, P. M. (2000). *Microbial and Comparative Genomics* 5:223-231.

57. The passage and propagation of fecal indicator phages in birds. Ricca, D. M., Cooney, J. J. (2000). *Journal of Industrial Microbiology & Biotechnology.* 24:127-131.

58. Screening environmental samples for source-specific bacteriophage hosts using a method for the simultaneous pouring of 12 petri plates. Ricca, D. M., Cooney, J. J. (2000). *Journal of Industrial Microbiology & Biotechnology.* 24:124-126.

59. The genome sequence of the plant pathogen Xylella fastidiosa. Simpson, A. J. G., Reinach, F. C., Arruda, P., Abreu, F. A., Acencio, M., Alvarenga, R., Alves, L. M. C., Araya, J. E., Baia, G. S., Baptista, C. S., Barros, M. H., Bonaccorsi, E. D., Bordin, S., Bove, J. M., Briones, M. R. S., Bueno, M. R. P., Camargo, A. A., Camargo, L. E. A., Carraro, D. M., Carrer, H., Colauto, N. B., Colombo, C., Costa, F. F., Costa, M. C. R., Costa-Neto, C. M., Coutinho, L. L., Cristofani, M., Dias-Neto, E., Docena, C., El-Dorry, H., Facincani, A. P., Ferreira, A. J. S., Ferreira, V. C. A., Ferro, J. A., Fraga, J. S., Franca, S. C., Franco, M. C., Frohme, M., Furlan, L. R., Garnier, M., Goldman, G. H., Goldman, M. H. S., Gomes, S. L., Gruber, A., Ho, P. L., Hoheisel, J. D., Junqueira, M. L., Kemper, E. L., Kitajima, J. P., Krieger, J. E., Kuramae, E. E., Laigret, F., Lambais, M. R., Leite, L. C. C., Lemos, E. G. M., Lemos, M. V. F., Lopes, S. A., Lopes, C. R., Machado, J. A., Machado, M. A., Madeira, A. M. B. N., Madeira, H. M. F., Marino, C. L., Marques, M. V., Martins, E. A. L., Martins, E. M. F., Matsukuma, A. Y., Menck, C. F. M., Miracca, E. C., Miyaki, C. Y., Monteiro-Vitorello, C. B., Moon, D. H., Nagai, M. A., Nascimento, A. L. T. O., Netto, L. E. S., Nhani, A., Jr., Nobrega, F. G., Nunes, L. R., Oliveira, M. A., de Oliveira, M. C., de Oliveira, R. C., Palmieri, D. A., Paris, A., Peixoto, B. R., Pereira, G. A. G., Pereira, H. A., Jr., Pesquero, J. B., Quaggio, R. B., Roberto, P. G., Rodrigues, V, de, M. R., de Rosa, V. E., Jr., de Sa, R. G., Santelli, R. V., Sawasaki, H. E., da Silva, A. C. R., da Silva, A. M., da Silva, F. R., Silva, W. A., Jr., da Silveira, J. F. (2000). *Nature (London)* 406:151-157.

60. Prophage, phiPV83-pro, carrying panton-valentine leukocidin genes, on the *Staphylococcus aureus* P83 chromosome: comparative analysis of the genome structures of phiPV83-pro, phiPVL, phi11, and other phages. Zou, D., Kaneko, J., Narita, S., Kamio, Y. (2000). *Bioscience, Biotechnology, and Biochemistry* 64:2631-2643. [PRESS FOR ABSTRACT]

61. Flow cytometric analyses of virus infection in two marine phytoplankton species, *Micromonas pusilla* (Prasinophyceae) and *Phaeocystis pouchetii* (Prymnesiophyceae). Brussaard, C. P. D., Thyrhaug, R., Marie, D., Bratbak, G. (1999). *Journal of Phycology* 35:941-948. [PRESS FOR ABSTRACT]

62. Biocontrol of *Erwinia amylovora* using bacteriophage. Gill, J. J., Svircev, A. M., Myers, A. L., Castle, A. J. (1999). *Phytopathology* 89:S27. [NO ABSTRACT]

63. Cyanophages. Martin, E. L., Kokjohn, T. A. (1999). pp. 324-332 in Granoff, A., Webster, R. G. (eds.) *Encyclopedia of Virology second edition*. Academic Press, San Diego. [NO ABSTRACT]

64. Bacteriophage therapy of *Clostridium difficile*-associated intestinal disease in a hamster model. Rdamesh, V., Fralick, J. A., Rolfe, R. D. (1999). *Miroecol. Anarobes[sic?]* 5:69-??? [NO ABSTRACT]

65. Dissolved esterase activity as a tracer of physoplankton lysis: Evidence of high phytoplankton lysis rates in the northwestern Mediteranean. Agustí, S., Satta, M. P., Mura, M. P., Benavent, E. (1998). *Limnology and Oceanography* 43:1836-1849. [PRESS FOR ABSTRACT]

66. Polyvirulent rhizobiophage from a soybean rhizosphere soil. Ali, F. S., Hammand, A. M. M., Loynachan, T. E. (1998). *Soil Biology and Biochemistry* 30:2171-2175. [NO ABSTRACT]

67. Viral lysis of *Phaeocystis pouchetii* and bacterial secondary production. Bratbak, G., Jacobsen, A., Heldal, M. (1998). *Aquatic Microbial Ecology* 16:11-16. [PRESS FOR ABSTRACT]

68. Virus production in *Phaeocystis pouchetii* and its relation to host cell growth and nutrition. Bratbak, G., Jacobsen, A., Heldal, M., Nagasaki, K., Thingstad, T. F. (1998). *Aquatic Microbial Ecology* 16:1-9. [NO ABSTRACT]

69. Ultrastructural analysis of viral invection in the brown-tide alga, *Aureococcus anophagefferens* (Pelagophyceae). Gastrich, M. D., Anderson, O. R., Benmayor, S. S., Cosper, E. M. (1998). *Phycologia* 37:300-306. [PRESS FOR ABSTRACT]

70. Biological control of bacterial blight of geranium with h-mutant bacteriophages. Harbaugh, B. K., Jones, J. B., Jackson, L. E., Somodi, G., Flaherty, J. E. (1998). *95th Annual International Conference of the American Society for Horticultural Science* 33:519. [NO ABSTRACT]

71. Effect of temperature on the algicidal activity and the satability of HaV (*Heterosigma akashiwo* virus). Nagasaki, K., Yamaguchi, M. (1998). *Aquatic Microbial Ecology* 15:211-216. [PRESS FOR ABSTRACT]

72. Lysogeny of *Oenococcus oeni* (syn. *Leuconostoc oenos*) and study of their induced bacteriophages. Poblet-Icart, M., Bordons, A., Lonvaud-Funel, A. (1998). *Current Microbiology* 36:365-369.

73. Comparative analysis of the effect of energy process inhibitors on the efficacy of phage infection in staphylococci. Polishko, T. N. (1998). *Mikrobiolohichnyi Zhurnal* 60:36-42.

74. Seasonal abundance in Skagerrak-Kattegat coastal waters and host specificity of viruses infecting the marine photosynthetic flagellate *Micromonas pusilla*. Sahlsten, E. (1998). *Aquatic Microbial Ecology* 16:103-108. [PRESS FOR ABSTRACT]

75. Vertical distribution of virus-like particles (VLP) and viruses infecting *Micromonas pusilla* during late summer in the southeastern Skagerrak. Sahlsten, E., Karlson, B. (1998). *J. Plankton Res.* 20:2207-2212. [PRESS FOR ABSTRACT]

76. Morphology and abundance of free and temperate viruses in Lake Superior. Tapper, M. A., Hicks, R. E. (1998). *Limnology and Oceanography* 43:95-103. [PRESS FOR ABSTRACT]

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New Publications with Abstracts

For your convenience, a list of new publications without associated abstracts (but with links to abstracts) is found above. The list presented below is identical to the above list except that abstracts are included.

1. **Distribution of virus-like particles in an oligotrophic marine environment (Alboran Sea, Western Mediterranean). Alonso, M. C., Jimenez-Gomez, F., Rodriguez, J., Borrego, J. J. (2001). *Microbial Ecology* 42:407-415.** Viruses are abundant in a variety of aquatic environments, often exceeding bacterial abundance by one order of magnitude. In the present study, the spatial distribution of viruses in offshore waters of the Alboran Sea (Western Mediterranean) have been studied to determine the relationships between viruses and host communities in this oligotrophic marine environment. Viral abundance was determined using two methods: (i) epifluorescence light microscopy using the dsDNA binding fluorochrome DAPI, and (ii) direct counts by transmission electron microscopy (TEM). The results obtained were significantly different; the highest viral counts were obtained by mean of TEM analyses. In all the samples tested the number of viruses was exceeded by the bacterial concentrations, with a ratio between viral and bacterial titers varying between 1.4 and 20. VLP (virus-like particle) counts were not significantly correlated ($p>0.001$) with chlorophyll a concentration or the abundance of cyanobacteria. However, there was a positive and significant correlation with bacterial abundance ($p<0.001$). The analysis of size and morphology of viral particles by TEM and the correlation obtained between the numbers of VLP and bacteria suggest that the majority of the viral particles in the Alboran Sea are bacteriophages. None of the indirect evidence suggested that eukaryotic algae or cyanobacteria were important host organisms in these waters.

2. **The bacteriophages of ruminal prevotellas. Ambrozic, J., Ferme, D., Grabnar, M., Ravnikar, M., Avgustin, G. (2001). *Folia Microbiologica* 46:37-39.** Rumen bacteriophage-lyzed bacterial strains of the genus *Prevotella* were isolated and preliminarily characterized. The strain TCI-1 the species *P. bryantii* was the only prevotella strain successfully infected with filter sterilized rumen fluid from a black-and-white Holstein cow. Two types of plaques were observed, both rather small and turbid. Preliminary electron microscopy observation showed that several morphologically different bacteriophages were present in these plaques. The plaque eluates were further used for the infection of other prevotella strains. The plaques produced by the bacteriophages were observed with two strains, i.e. *P. bryantii* B(1)4 and *P. brevis* GA33. The bacteriophages from both strains were examined by transmission electron microscopy and several morphologically different bacteriophages were observed, among others also a large virion with an icosahedral head with the diameter of approximately 120 nm. The bacteriophage was identified in plaques of bacterial cells of the strain GA33 and has an approximately 800 nm long helical tail, which places it among the largest ruminal bacteriophages described to date. Other bacteriophages from the same indicator strain as well as from *P. bryantii* B(1)4 strain were smaller and tail structures were not observed in all of them

3. **Isolation and characterization of bacteriophage-resistant mutants of *Vibrio cholerae* O139. Attridge, S. R., Fazeli, A., Manning, P. A., Stroeher, U. H. (2001). *Microbial Pathogenesis* 30:237-246.** *Vibrio cholerae* O139 strains produce a capsule which is associated with complement resistance and is used as a receptor by bacteriophage JA1. Spontaneous JA1-resistant mutants were found to have several phenotypes, with loss of capsule and/or O-antigen from the cell surface. Determination of the residual complement resistance and infant mouse colonization potential of each mutant suggested that production of O-antigen is of much greater significance than the presence of capsular material for both of these properties. Two

different in vitro assays of complement resistance were compared and the results of one shown to closely reflect the comparative recoveries of bacteria from the colonization experiments. Preliminary complementation studies implicated two rfb region genes, wzz and wbfP, as being essential for the biosynthesis of capsule but not O-antigen

4. **Bacteriophage-bacteriophage interactions in the evolution of pathogenic bacteria. Boyd, E. F., Davis, B. M., Hochhut, B. (2001). *Trends in Microbiology* 9:137-144.** Many bacteriophages carry virulence genes encoding proteins that play a major role in bacterial pathogenesis. Recently, investigators have identified bacteriophage-bacteriophage interactions in the bacterial host cell that also contribute significantly to the virulence of bacterial pathogens. The relationships between the bacteriophages pertain to one bacteriophage providing a helper function for another, unrelated bacteriophage in the host cell. Accordingly, these interactions can involve the mobilization of bacteriophage DNA by another bacteriophage, for example in *Escherichia coli*, *Vibrio coli* and *Staphylococcus aureus*; the host receptor for one bacteriophage being encoded by another, as found in *V. cholerae*; and the presence of one bacteriophage potentiating the virulence properties of another bacteriophage, as found in *V. cholerae* and *Salmonella enterica*

5. **Chemical and microbial characterization of household graywater. Casanova, L. M., Gerba, C. P., Karpiscak, M. (2001). *J Environ Sci Health Part A Tox Hazard Subst Environ Eng* 36:395-401.** In arid areas, the search for efficient methods to conserve water is of paramount importance. One of the methods of water conservation available today is graywater recycling--the reuse of water from the sinks, showers, washing machine, and dishwasher in a home. The purpose of this project was to characterize the chemical and microbial quality of graywater from a single-family home with two adults. Water samples from a graywater holding tank were analyzed over a seven-month period for total coliforms, fecal coliforms, fecal streptococci, *Staphylococcus aureus* (*S. aureus*), *Pseudomonas aeruginosa* (*P. aeruginosa*), and coliphages. The pH, turbidity, biological oxygen demand (BOD), suspended solids (SS), electrical conductivity (EC), sulfates (SO4), and chlorides (Cl) were also measured. The mean numbers of total coliforms, fecal coliforms, fecal streptococci, and *P. aeruginosa* were 8.03 x 10^7, 5.63 x 10^5, 2.38 x 10^2, and 1.99 x 10^4 CFU/100 mL, respectively. *S. aureus* and coliphages were not detected. In the chemical analysis, mean values of 7.47 for pH, 43 nephelometric turbidity units (NTU) for turbidity, 64.85 mg/L for BOD, 35.09 mg/L for SS, 0.43 mS/cm for EC, 59.59 mg/L for SO4, and 20.54 mg/L for Cl were measured. These data were compared to data taken in 1986 and 1987, when two adults and one child lived in the household. Analysis showed no statistically significant difference in levels of total coliforms and suspended solids between the two data sets. There were statistically significant differences in levels of fecal coliforms, pH, turbidity, chlorides, sulfates, and BOD between the two households. Fecal coliforms, turbidity, and BOD were higher in the household with two adults and one child. Levels of Cl, SO4, and pH were higher in the household with two adults

6. **Microbial population dynamics and diversity during a bloom of the marine coccolithophorid *Emiliania huxleyi* (Haptophyta). Castberg, T., Larsen, A., Sandaa, R. A., Brussaard, C. P. D., Egge, J. K., Heldal, M., Thyrhaug, R., van Hannen, E. J., Bratbak, G. (2001). *Marine Ecology Progress Series* 221:39-46.** Several previous studies have shown that *Emiliania huxleyi* blooms and terminations have been succeeded by an increase in large virus-like particles (LVLP), strongly suggesting the bloom collapse was caused by viral lysis. However, due to methodological limitations, knowledge of how such blooms affect the rest of the microbial community is limited. In the current study we induced a bloom of *E. huxleyi* in seawater enclosures and applied methods enabling us to describe the algae, bacteria and virus communities with greater resolution than has been done previously, The development of the dominating algal, viral and bacterial populations in the nutrient-amended seawater enclosures was followed by flow cytometry (FCM). Light microscopy (LM), PCR-denaturing gradient gel electrophoresis (PCR-DGGE) and pulsed-field gel electrophoresis (PFGE) were used to describe the changes in community composition in greater detail. The algal community was dominated by *E. huxleyi* until termination of the bloom by viral lysis, After bloom termination the additional algal populations present in the enclosures increased in abundance. A marked increase in viruses other than the one infecting *E. huxleyi* was also observed. Total bacterial number and community composition were also greatly influenced by the bloom and its collapse.

7. **Nucleotide sequence of coliphage HK620 and the evolution of lambdoid phages. Clark, A. J., Inwood, W., Cloutier, T., Dhillon, T. S. (2001). *Journal of Molecular Biology* 311:657-679.** HK620 is a temperate lambdoid bacteriophage that adsorbs to the O-antigen of its host, *Escherichia coli* H. The genome of a temperature-sensitive clear-plaque mutant consists of 38,297 nucleotides in which we recognize 60 open reading frames (orfs). Eighteen of these lie in a region of the genome that we call the virion structure domain. The other 42 orfs lie in what we call the metabolic domain.Virions of HK620 resemble those of phage P22. The virion structural orfs encode three kinds of putative proteins relative to the virion proteins of P22: (1) those that are nearly (about 90 %) identical; (2) those that are weakly (about 30 %) identical; and (3) those composed of nearly and weakly identical segments. We hypothesize that these composite proteins form bridges between the virion proteins of the other two kinds.Three of the putative virion proteins that are only weakly identical to P22 proteins are 71, 60 and 79 % identical to proteins encoded by the phage APSE-1, whose virions also resemble those of P22. Because the hosts of APSE-1 and HK620 have been separated from each other by an estimated 200 My, we propose using the amino acid differences that have accumulated in these proteins to estimate a biological clock for temperate lambdoid phages.The putative transcriptional regulatory gene circuitry of HK620 seems to resemble that of phage lambda. Integration, on the other hand, resembles that of satellite phage P4 in that the attP sequence lies between the leftward promoter and int rather than downstream of int.Comparing the metabolic domains of several lambdoid phage genomes reveals seven short conserved sequences roughly defining boundaries of functional modules. We propose that these boundary sequences are foci of genetic recombination that serve to assort the modules and make the metabolic domain highly mosaic genetically

8. **Bacteriophage T4 multiplication in an *Escherichia coli* biofilm. Corbin, B. D., Aron, G. M., McLeon, R. J. C. (2001). *Canadian Journal of Microbiology* 47:680-684.** An *Escherichia coli* K-12 biofilm was grown at a dilution rate of 0.028 h^{-1} for 48 h in a glucose-limited chemostat coupled to a modified Robbins' device to determine its susceptibility to infection by bacteriophage T4. Bacteriophage T4 at a multiplicity of infection (MOI) of 10 caused a log reduction in biofilm density (expressed as colony forming units (CFU) per cm(2)) at 90 min postinfection. After 6 h, a net decrease and equilibrium in viral titer was seen. When biofilms were exposed to T4 phage at a MOI of 100, viral titer doubled after 90 min. After 6 h, viral titers (expressed as plaque forming units (PFU) per cm(2)) stabilized at levels approximately one order of magnitude higher than seen at a MOI of 10. Scanning confocal laser microscopy images also indicated disruption of biofilm morphology following T4 infection with the effects being more pronounced at a MOI of 100 than at a MOI of 10. These results imply that biofilms under carbon limitation can act as natural reservoirs for bacteriophage and that bacteriophage can have some influence on biofilm morphology.

9. **Progeny of the phage school. Dixon, B. (2001). *ASM News* 69:432-433.** Frederick Twort, the eccentric polymath who discovered bacterial viruses, would have robustly welcomed the applications of bacteriophages now emergy, from therapeutics

to environmental protection.

10. **Direct and quantitative detection of bacteriophage by "hearing" surface detachment using a quartz crystal microbalance. Dultsev, F. N., Speight, R. E., Fiorini, M. T., Blackburn, J. M., Abell, C., Ostanin, V. P., Klenerman, D. (2001). *Analytical Chemistry* 73:3935-3939.** We show that it is possible to detect specifically adsorbed bacteriophage directly by breaking the interactions between proteins displayed on the phage coat and ligands immobilized on the surface of a quartz crystal microbalance (QCM). This is achieved through increasing the amplitude of oscillation of the QCM surface and sensitively detecting the acoustic emission produced when the bacteriophage detaches from the surface. There is no interference from nonspecifically adsorbed phage. The detection is quantitative over at least 5 orders of magnitude and is sensitive enough to detect as few as 20 phage. The method has potential as a sensitive and low-cost method for virus detection

11. **Diminished diarrheal response to *Vibrio cholerae* strains carrying the replicative form of the CTXf genome instead of CTXf lysogens in adult rabbits. Faruque, S. M., Rahman, M. M., Hasan, A. K., Nair, G. B., Mekalanos, J. J., Sack, D. A. (2001). *Infection and Immunity* 69:6084-6090.** Toxigenic *Vibrio cholerae* strains are lysogens of CTXf, a filamentous bacteriophage which encodes cholera toxin (CT). Following infection of recipient *V. cholerae* cells by CTX(Phi), the phage genome either integrates into the host chromosome at a specific attachment site (attRS) or exists as a replicative-form (RF) plasmid. We infected naturally occurring attRS-negative nontoxigenic *V. cholerae* or attenuated (CTX(-) attRS negative) derivatives of wild-type toxigenic strains with CTX(Phi) and examined the diarrheagenic potential of the strains carrying the RF of the CTXf genome using the adult rabbit diarrhea model. Under laboratory conditions, strains carrying the RF of CTX(Phi) produced more CT than corresponding lysogens as assayed by a G(M1)-based enzyme-linked immunosorbent assay and by fluid accumulation in ligated ileal loops of rabbits. However, when tested for diarrhea in rabbits, the attRS-negative strains (which carried the CTXf genome as the RF) were either negative or produced mild diarrhea, whereas the attRS-positive strains with integrated CTXf produced severe fatal diarrhea. Analysis of the strains after intestinal passage showed that the attRS-negative strains lost the phage genome at approximately a fivefold higher frequency than under in vitro conditions, and 75 to 90% of cells recovered from challenged rabbits after 24 h were CT negative. These results suggested that strains carrying the RF of CTXf are unable to cause severe disease due to rapid loss of the phage in vivo, and the gastrointestinal environment thus provides selection of toxigenic strains with an integrated CTXf genome. These results may have implications for the development of live *V. cholerae* vaccine candidates impaired in chromosomal integration of CTX(Phi). These findings may also contribute to understanding of the etiology of diarrhea occasionally associated with nontoxigenic *V. cholerae* strains

12. **Phage antibacterials make a comeback. Fischetti, V. A. (2001). *Nature Biotechnology* 19:734-735.**

13. **A conserved genetic module that encodes the major virion components in both the coliphage T4 and the marine cyanophage S-PM2. Hambly, E., Tétart, F., Desplats, C., Wilson, H., Krisch, H. M., Mann, N. H. (2001). *Proceedings of the National Academy of Sciences, USA* 98:11411-11416.** Sequence analysis of a 10-kb region of the genome of the marine cyanomyovirus S-PM2 reveals a homology to coliphage T4 that extends as a contiguous block from gene (g)18 to g23. The order of the S-PM2 genes in this region is similar to that of T4, but there are insertions and deletions of small ORFs of unknown function. In T4, g18 codes for the tail sheath, g19, the tail tube, g20, the head portal protein, g21, the prohead core protein, g22, a scaffolding protein, and g23, the major capsid protein. Thus, the entire module that determines the structural components of the phage head and contractile tail is conserved between T4 and this cyanophage. The significant differences in the morphology of these phages must reflect the considerable divergence of the amino acid sequence of their homologous virion proteins, which uniformly exceeds 50%. We suggest that their enormous diversity in the sea could be a result of genetic shuffling between disparate phages mediated by such commonly shared modules. These conserved sequences could facilitate genetic exchange by providing partially homologous substrates for recombination between otherwise divergent phage genomes. Such a mechanism would thus expand the pool of phage genes accessible by recombination to all those phages that share common modules.

14. **Isolation and characterization of a temperature-sensitive generalized transducing bacteriophage for *Vibrio cholerae*. Hava, D. L., Camilli, A. (2001). *J Microbiol Methods* 46:217-225.** CP-T1 is the only described generalized transducing bacteriophage for the intestinal pathogen *Vibrio cholerae*, yet many of its basic biological parameters remain unknown. Due to low frequencies of transduction and pseudolysogen formation, CP-T1 has not been widely used as a genetic tool. To overcome these limitations, we have isolated a conditional mutant of CP-T1 that exhibits temperature-sensitive plaque formation. Several biological properties of CP-T1ts were determined, including its restrictive temperature, adsorbance profile to host cells, burst time, and burst size. Based on these properties, an optimized transduction protocol was designed which resulted in several fold higher transduction frequencies for a variety of genetic markers from a number of chromosomal loci. Generalized transduction was also demonstrated between classical and E1 Tor biotype strains of *V. cholerae*.

15. **Effects of concentrated viral communities on photosynthesis and community composition of co-occurring benthic microalgae and phytoplankton. Hewson, I., O'Neil, J. M., Heil, C. A., Bratbak, G., Dennison, W. C. (2001). *Aquatic Microbial Ecology* 25:1-10.** Marine viruses have been shown to affect phytoplankton productivity; however, there are no reports on the effect of viruses on benthic microalgae (microphytobenthos). Hence, this study investigated the effects of elevated concentrations of virus-like particles on the photosynthetic physiology and community composition of benthic microalgae and phytoplankton. Virus populations were collected near the sediment surface and concentrated by tangential flow ultrafiltration, and the concentrate was added to benthic and water column samples that were obtained along a eutrophication gradient in the Brisbane River/Moreton Bay estuary, Australia. Photosynthetic and community responses of benthic microalgae, phytoplankton and bacteria were monitored over 7 d in aquaria and in situ. Benthic microalgal communities responded to viral enrichment in both eutrophic and oligotrophic sediments. In eutrophic sediments, Euglenophytes (Euglena sp.) and bacteria decreased in abundance by 20 to 60 and 26 to 66%, respectively, from seawater controls. In oligotrophic sediments, bacteria decreased in abundance by 30 to 42% from seawater controls but the dinoflagellate Gymnodinium sp. increased in abundance by 270 to 3600% from seawater controls, The increased abundance of Gymnodinium sp. may be related to increased availability of dissolved organic matter released from lysed bacteria. Increased (140 to 190% from seawater controls) initial chlorophyll a fluorescence measured with a pulse-amplitude modulated fluorometer was observed in eutrophic benthic microalgal incubations following virus enrichment, consistent with photosystem II damage. Virus enrichment in oligotrophic water significantly stimulated carbon fixation rates, perhaps due to increased nutrient availability by bacterial lysis. The interpretation of data from virus amendment experiments is difficult due to potential interaction with unidentified bioactive compounds within seawater concentrates. However, these results show that viruses are capable of influencing microbial dynamics in sediments.

16. **Mosaic structure of shiga-toxin-2-encoding phages isolated from *Escherichia coli* O157:H7 indicates frequent gene**

exchange between lambdoid phage genomes. Johansen, B. K., Wasteson, Y., Granum, P. E., Brynestad, S. (2001). *Microbiology* 147:1929-1936. Shiga-toxin-2 *(stx* 2)-encoding bacteriophages were isolated from Norwegian *Escherichia coli* O157: H7 isolates of cattle and human origin. The phages were characterized by restriction enzyme analysis, hybridization with probes for toxin genes and selected phage DNA such as the *P* gene, integrase gene and I*S120*3, and by PCR studies and partial sequencing of selected DNA regions in the integrase to *stx* 2 region of the phages. The *stx* 2 -phage-containing bacteria from which inducible phages were detected produced similar amounts of toxin, as shown by a Vero cell assay. The results indicate that the population of *stx* 2 -carrying phages is heterogeneous, although the phages from epidemiologically linked *E. coli* O157: H7 isolates were similar. There appears to have been frequent recombination of *stx* 2 phages with other lambdoid phages.

17. **Elimination of fecal coliforms and F-specific RNA coliphage from oysters (*Crassostrea virginica*) relaid in floating containers. Kator, H., Rhodes, M. (2001). *Journal of Food Protection* 64:796-801.** Declining oyster (*Crassostrea virginica*) production in the Chesapeake Bay has stimulated aquaculture based on floats for off-bottom culture. While advantages of off-bottom culture are significant, the increased use of floating containers raises public health and microbiological concerns, because oysters in floats may be more susceptible to fecal contamination from storm runoff compared to those cultured on-bottom. We conducted four commercial-scale studies with market-size oysters naturally contaminated with fecal coliforms (FC) and a candidate viral indicator, F-specific RNA (FRNA) coliphage. To facilitate sampling and to test for location effects, 12 replicate subsamples, each consisting of 15 to 20 randomly selected oysters in plastic mesh bags, were placed at four characteristic locations within a 0.6- by 3.0-m "Taylor" float, and the remaining oysters were added to a depth not exceeding 15.2 cm. The float containing approximately 3,000 oysters was relaid in the York River, Virginia, for 14 days. During relay, increases in shellfish FC densities followed rain events such that final mean levels exceeded initial levels or did not meet an arbitrary product end point of 50 FC/100 ml. FRNA coliphage densities decreased to undetectable levels within 14 days (16 to 28 degrees C) in all but the last experiment, when temperatures fell between 12 and 16 degrees C. Friedman (nonparametric analysis of variance) tests performed on FC/Escherichia coli and FRNA densities indicated no differences in counts as a function of location within the float. The public health consequences of these observations are discussed, and future research and educational needs are identified

18. **Octamer-based genome scanning distinguishes a unique subpopulation of *Escherichia coli* O157:H7 strains in cattle. Kim, J., Nietfeldy, J., Benson, A. K. (2001). *Proceedings of the National Academy of Sciences, USA* 96:13288-13293.** Multilocus-genotyping methods have shown that *Escherichia coli* O157:H7 is a geographically disseminated clone. However, high-resolution methods such as pulse-field gel electrophoresis demon-strate significant genomic diversity among different isolates. To assess the genetic relationship of human and bovine isolates of *E. coli* O157:H7 in detail, we have developed an octamer-based genome-scanning methodology, which compares the distance be-tween over-represented, strand-biased octamers that occur in the genome. Comparison of octamer-based genome-scanning prod-ucts derived from >1 megabase of the genome demonstrated the existence of two distinct lineages of *E. coli* O157:H7 that are disseminated within the United States. Human and bovine isolates are nonrandomly distributed among the lineages, suggesting that one of these lineages may be less virulent for humans or may not be efficiently transmitted to humans from bovine sources. Restric-tion fragment length polymorphism analysis with lambdoid phage genomes indicates that phage-mediated events are associated with divergence of the lineages, thereby providing one explana-tion for the degree of diversity that is observed among *E. coli* O157:H7 by other molecular-fingerprinting methods.

19. **Antacid increases survival of *Vibrio vulnificus* and *Vibrio vulnificus* phage in a gastrointestinal model. Koo, J., Marshall, D. L., Depaola, A. (2001). *Applied and Environmental Microbiology* 67:2895-2902.** Viable counts of three strains of *Vibrio vulnificus* and its phage were determined during exposure to a mechanical gastrointestinal model with or without antacid for 9 h at 37 degrees C. *V. vulnificus* was eliminated (>4-log reduction) within 30 min in the gastric compartment (pH decline from 5.0 to 3.5). Viable *V. vulnificus* cells delivered from the gastric compartment during the first 30 min of exposure reached 10^6 to 10^8 CFU/ml in the intestinal compartment after 9 h (pH 7.0). Phages were eliminated within 45 min in the gastric compartment (pH decline from 5.1 to 2.5). Less than a 2-log reduction of phage was observed in the intestinal compartment after 9 h (pH 7.0). When the gastric compartment contained antacid *V. vulnificus* counts decreased slightly (<2 log) during 2 h of exposure (pH decline from 7.7 to 6.0), while counts in the intestinal compartment (pH 7.5) reached 10^7 to 10^9 CFU/ml. Phage numbers decreased 1 log after 2 h in the gastric compartment (pH decline from 7.7 to 5.7) containing antacid and decreased 1 log in the intestinal compartment (pH 7.6) after 9 h. Presence of antacid in the gastric compartment of the model greatly increased the ability of both *V. vulnificus* and its phage to survive simulated gastrointestinal transit and may be a factor involved with oyster-associated illness

20. **[*Vibrio cholerae* O139 bacteriophages]. Kudriakova, T. A., Makedonova, L. D., Kachkina, G. V., Saiamov, S. R. (2001). *Zhurnal Mikrobiologii, Epidemiologii i Immunobiologii* 28-30.** Cholera bacteriophages have been isolated from 27 lysogenic cultures of *V. cholerae* O139. As shown the pages under study belong to two morphological groups A1 and F1 and serological types II and XII. The use of prophage typing and the sensitivity test to specific phage made it possible to differentiate *V. cholerae* strains, serogroup O139

21. **Population dynamics and diversity of phytoplankton, bacteria and viruses in a seawater enclosure. Larsen, A., Castberg, T., Sandaa, R. A., Brussaard, C. P. D., Egge, J. K., Heldal, M., Paulino, A., Thyrhaug, R., van Hannen, E. J., Bratbak, G. (2001). *Marine Ecology Progress Series* 221:47-57.** We now know that the abundance of free viruses in most marine environments is high. There is still, however, a lack of understanding of their occurrence and distribution and of in situ relationships between viral and host communities in natural environments. This may be partly due to methodological limitations. Our main aim was therefore to perform a case study in which a variety of methods were applied in order to give an improved, high-resolution description of the microbial communities in a natural environment, In order to do this we combined light microscopy (LM), transmission electron microscopy (TEM), flow cytometry (FCM), PCR denaturing gradient gel electrophoresis (PCR-DGGE) and pulsed-field gel electrophoresis (PFGE) and studied the diversity and succession of algae, bacteria and viruses in a nutrient enriched seawater enclosure. In the enclosure we experienced a situation where the development of the dominating algal population, which consisted of several flagellate species, was followed by proliferation of several different size-classes of viruses. The total bacterial number decreased markedly during the flagellate bloom but the community composition was maintained and the diversity remained high. Our results indicate a close linkage between various algal, bacterial and viral populations and show that virioplankton do not necessarily terminate algal and bacterial blooms but that they keep the host populations at non-blooming levels.

22. **Viruses in the plankton of freshwater and saline Antarctic lakes. Laybourn-Parry, J., Hofer, J. S., Sommaruga, R. (2001). *Freshwater Biology* 46:1279-1287.** 1. Virus-like particle (VLP) abundances in nine freshwater to saline lakes in the Vestfold Hills, Eastern Antarctica (68degree S) were determined in December 1999. In the ultra-oligotrophic to oligotrophic freshwater lakes, VLP abundances ranged from 1.01 to 3.28 X 106 mL-1 in the top 6 m of the water column. In the saline lakes the range was between 6.76 and 36.5 X 10^6 mL-1. The lowest value was found in meromictic Ace Lake and the highest value in hypersaline Lake Williams. Virus to bacteria ratios (VBR) were lowest in the freshwater lakes and highest in the saline lakes, with a maximum of 23.4 in the former and 50.3 in the latter. 2. A range of morphologies among VLP was observed, including phages with short (Podoviridae) and long tails, icosahedric viruses of up to 300 nm and star-like particles of about 80 nm diameter. 3. In these microbially dominated ecosystems there was no correlation between VLP and either bacterial numbers of chlorophyll a. There was a significant correlation between VLP abundances and dissolved organic carbon concentration ($r = 0.845$, $P < 0.01$). 4. The data suggested that viruses probably attack a spectrum of bacteria and protozoan species. Virus-like particle numbers in the freshwater lakes were lower than values reported for lower latitude systems. Those in the saline lakes were comparable with abundances reported from other Antarctic lakes, and were higher than most values published for lower latitude lakes and many marine systems. Across the salinity spectrum from freshwater through brackish to hypersaline, VLP concentrations increased roughly in relation to increasing trophy. 5. Given that Antarctic lakes have a plankton almost entirely made up of bacteria and protists, and that VLP abundances are high, it is likely that viruses play a pivotal role in carbon cycling in these extreme ecosystems.

23. **Examination of bacteriophage as a biocontrol method for salmonella on fresh-cut fruit: a model study. Leverentz, B., Conway, W. S., Alavidze, Z., Janisiewicz, W. J., Fuchs, Y., Camp, M. J., Chighladze, E., Sulakvelidze, A. (2001). *Journal of Food Protection* 64:1116-1121.** The preparation and distribution of fresh-cut produce is a rapidly developing industry that provides the consumer with convenient and nutritious food. However, fresh-cut fruits and vegetables may represent an increased food safety concern because of the absence or damage of peel and rind, which normally help reduce colonization of uncut produce with pathogenic bacteria. In this study, we found that *Salmonella enteritidis* populations can (i) survive on fresh-cut melons and apples stored at 5 degrees C, (ii) increase up to 2 log units on fresh-cut fruits stored at 10 degrees C, and (iii) increase up to 5 log units at 20 degrees C during a storage period of 168 h. In addition, we examined the effect of lytic, Salmonella-specific phages on reducing *Salmonella* numbers in experimentally contaminated fresh-cut melons and apples stored at various temperatures. We found that the phage mixture reduced Salmonella populations by approximately 3.5 logs on honeydew melon slices stored at 5 and 10 degrees C and by approximately 2.5 logs on slices stored at 20 degrees C, which is greater than the maximal amount achieved using chemical sanitizers. However, the phages did not significantly reduce *Salmonella* populations on the apple slices at any of the three temperatures. The titer of the phage preparation remained relatively stable on melon slices, whereas on apple slices the titer decreased to nondetectable levels in 48 h at all temperatures tested. Inactivation of phages, possibly by the acidic pH of apple slices (pH 4.2 versus pH 5.8 for melon slices), may have contributed to their inability to reduce *Salmonella* contamination in the apple slices. Higher phage concentrations and/or the use of low-pH-tolerant phage mutants may be required to increase the efficacy of the phage treatment in reducing *Salmonella* contamination of fresh-cut produce with a low pH

24. **Colloidal interactions in suspensions of rods. Lin, K., Crocker, J. C., Zeri, A. C., Yodh, A. G. (2001). *Phys Rev Lett* 87:088301.** We report direct measurements of entropic interactions of colloidal spheres in suspensions of rodlike fd bacteriophage. We investigate the influence of sphere size, rod concentration, and ionic strength on these interactions. Although the results compare favorably with a recent calculation, small discrepancies reveal entropic effects due to rod flexibility. At high salt concentrations, the potential turns repulsive as a result of viral adsorption on the spheres and viral bridging between the spheres

25. **Depolymerization of the capsular polysaccharide from *Vibrio cholerae* O139 by a lyase associated with the bacteriophage JA1. Linnerborg, M., Weintraub, A., Albert, M. J., Widmalm, G. (2001). *Carbohydrate Research* 333:263-269.** We have studied the interaction between the *Vibrio cholerae* O139 specific phage JA1, belonging to the Podoviridae family, and the capsular polysaccharide (CPS) of the parent strain from which the phage was isolated. Upon incubation of the JA1 phage with the CPS, oligosaccharides were isolated and purified. The oligosaccharides derived from one (shown below) and two repeating units of the CPS were characterized using NMR spectroscopy, mass spectrometry and sugar analysis (structure: see text). The cleavage was found to occur by beta-elimination at the 4-substituted alpha-linked galacturonic acid, which results in a 4-deoxy-beta-L-threo-hex-4-enopyranosyl uronic acid group (Sug). The enzyme associated with the JA1 phage responsible for the depolymerization of the *V. cholerae* O139 CPS is thus a lyase

26. **Physiological function of exopolysaccharides produced by *Lactococcus lactis*. Looijesteijn, P. J., Trapet, L., de, Vries E., Abee, T., Hugenholtz, J. (2001). *International Journal of Food Microbiology* 64:71-80.** The physiological function of EPS produced by *Lactococcus lactis* was studied by comparing the tolerance of the non-EPS producing strain *L. lactis* ssp. *cremoris* MG1614 and an EPS producing isogenic variant of this strain to several anti-microbial factors. There was no difference in the sensitivity of the strains to increased temperatures, freezing or freeze-drying and the antibiotics, penicillin and vancomycin. A model system showed that EPS production did not affect the survival of *L. lactis* during passage through the gastrointestinal tract although the EPS itself was not degraded during this passage. The presence of cell associated EPS and EPS in suspension resulted in an increased tolerance to copper and nisin. Furthermore, cell associated EPS also protected the bacteria against bacteriophages and the cell wall degrading enzyme lysozyme. However, it has not been possible, so far, to increase EPS production using the presence of copper, nisin, lysozyme or bacteriophages as inducing factors

27. **Distribution, isolation, host specificity, and diversity of cyanophages infecting marine *Synechococcus* spp. in river estuaries. Lu, J., Chen, F., Hodson, R. E. (2001). *Applied and Environmental Microbiology* 67:3285-3290.** The abundance of cyanophages infecting marine *Synechococcus* spp. increased with increasing salinity in three Georgia coastal rivers. About 80% of the cyanophage isolates were cyanomyoviruses. High cross-infectivity was found among the cyanophages infecting phycoerythrin-containing *Synechococcus* strains. Cyanophages in the river estuaries were diverse in terms of their morphotypes and genotypes

28. **Elution, detection, and quantification of polio I, bacteriophages, *Salmonella montevideo*, and *Escherichia coli* O157:H7 from seeded strawberries and tomatoes. Lukasik, J., Bradley, M. L., Scott, T. M., Hsu, W. Y., Farrah, S. R., Tamplin, M. L. (2001). *Journal of Food Protection* 64:292-297.** This study compared the effect of different physical and chemical treatments of strawberries and tomatoes to determine their ability to recover seeded viral and bacterial pathogens from produce surfaces. Solutions of salts, amino acids, complex media, and detergents were compared as eluants. Phosphate-buffered saline (PBS) containing 0.1% Tween 80 eluted the highest number of seeded microorganisms. Elution with this defined

solution was then compared under different conditions of physical agitation. Rotary shaking for 20 min at 36 degrees C eluted higher numbers of viruses and bacteria than did low- or high-speed stomaching. Commercially available and laboratory prepared bacteriological differential media were compared for their ability to recover and distinguish eluted *Salmonella montevideo* and *Escherichia coli* O157:H7 strains from seeded produce. The recovery of seeded bacterial pathogens was low when differential media containing selective ingredients were used (MacConkey sorbitol agar, XLD agar, MacConkey agar). Highest recoveries were obtained on a medium consisting of tryptic soy agar supplemented with sodium thiosulfate and ferric ammonium citrate compared with selective media that inhibited up to 50% of the growth of the eluted microorganisms

29. **The genome of archaeal prophage PsiM100 encodes the lytic enzyme responsible for autolysis of *Methanothermobacter wolfeii*. Luo, Y., Pfister, P., Leisinger, T., Wasserfallen, A. (2001). *Journal of Bacteriology* 183:5788-5792.** *Methanothermobacter wolfeii* (formerly *Methanobacterium wolfei*), a thermophilic methanoarchaeon whose cultures lyse upon hydrogen starvation, carries a defective prophage called PsiM100 on its chromosome. The nucleotide sequence of PsiM100 and its flanking regions was established and compared to that of the previously sequenced phage PsiM2 of *Methanothermobacter marburgensis* (formerly *Methanobacterium thermoautotrophicum* Marburg). The PsiM100 genome extends over 28,798 bp, and its borders are defined by flanking 21-bp direct repeats of a pure-AT sequence, which very likely forms the core of the putative attachment site where the crossing over occurred during integration. A large fragment of 2,793 bp, IFa, apparently inserted into PsiM100 but is absent in the genome of PsiM2. The remaining part of the PsiM100 genome showed 70.8% nucleotide sequence identity to the whole genome of PsiM2. Thirty-four open reading frames (ORFs) on the forward strand and one ORF on the reverse strand were identified in the PsiM100 genome. Comparison of PsiM100-encoded ORFs to those encoded by phage PsiM2 and to other known protein sequences permitted the assignment of putative functions to some ORFs. The ORF28 protein of PsiM100 was identified as the previously known autolytic enzyme pseudomurein endoisopeptidase PeiW produced by M. wolfeii

30. **Sequence analysis and molecular characterization of the *Lactococcus lactis* temperate bacteriophage BK5-T. Mahanivong, C., Boyce, J. D., Davidson, B. E., Hillier, A. J. (2001). *Applied and Environmental Microbiology* 67:3564-3576.** The *Lactococcus lactis* temperate bacteriophage BK5-T is one of twelve type phages that define *L. lactis* phage species. This paper describes the nucleotide sequence and analysis of a 21-kbp region of the BK5-T genome and completes the nucleotide sequence of the genome of this phage. The 40,003-nucleotide linear genome encodes 63 open reading frames. Sequence runoff experiments showed that the cohesive ends of the BK5-T genome contained a 12-bp 3' single-stranded overhang with the sequence 5'-CACACACATAGG-3'. Two major BK5-T structural proteins, of approximately 30 and 20 kDa, were identified, and N-terminal sequence analysis determined that they were encoded by orf7 and orf12, respectively. A 169-bp fragment containing a 37-bp direct repeat and several smaller repeat sequences conferred resistance to BK5-T infection when introduced in trans to the host cell and is likely a part of the BK5-T origin of replication (ori)

31. **Growth and survival of clinical vs. environmental species of *Aeromonas* in tap water. Mary, P., Buchet, G., Defives, C., Hornez, J. P. (2001). *International Journal of Food Microbiology* 69:191-198.** The ability of four species of *Aeromonas* (two of clinical and two of environmental origin) to survive and/or grow in tap water microcosms supplemented with sodium thiosulphate was tested. After bottling, the autochthonous microflora reached 6 x 10^5 cfu ml^{-1} after a 5-day incubation period in tap water unfiltered and which was non-autoclaved. In filtered tap water, "ultramicrocells" were detected and final populations of ca. 10^6 cfu ml^{-1} after 7 days were obtained. *Aeromonas* was inoculated at an initial cell concentration of ca. 10^4 cfu ml^{-1}. All strains were able to grow in tap water samples, which were filtered and autoclaved, and a final concentration of 10^5-10^6 cfu ml^{-1} was observed. Any inherent capability of *Aeromonas* to grow in tap water was eliminated by the presence of autochthonous microflora and "ultramicrocells" bacteria. Survival rates were strain- and microcosm-dependent. In unfiltered-non-autoclaved water, viable counts declined to below the detection limit (i.e. 1 log cfu ml^{-1}) in 1.5 to 20 days. The declines in viable counts were even more pronounced in the filtered microcosm. Although inoculation ratios (100/1 in unfiltered-non-autoclaved and 1,000/1 in filtered microcosms) were favourable for aeromonads, at least for I to 3 days, the organisms disappeared in these microcosms. Thus, competition for nutrients was an unlikely cause of the limitation of aeromonads. The bacteriolytic effect of enzymes released by membrane vesicles from the autochthonous microflora and of "tail phage-like particles" bacteriocins were suggested as an in situ control of aeromonad populations. The present study showed that environmental strains of Aeromonas had no ecological advantage over clinical isolates. Thus, waterborne infections and contaminations of foods by pathogenic *Aeromonas* species could not be discounted

32. **Livestock deaths associated with *Clavibacter toxicus*/*Anguina* sp. infection in seedheads of *Agrostis avenacea* and *Polypogon monspeliensis*. McKay, A. C., Ophel, K. M., Reardon, T. B., Gooden, J. M. (2001). *Plant Disease* 77:635-641.** Flood plain staggers, a recently discovered poisoning of livestock, has been linked to *Clavibacter toxicus* infection in the seedheads of blown grass, *Agrostis avenacea*, in northern New South Wales and annual beardgrass, *Polypogon monspeliensis*, in the southeast of South Australia (Australia). The same bacterium on annual ryegrass, *Lolium rigidum*, causes the poisoning of livestock known as annual ryegrass toxicity. Strains of *C. toxicus* from *A. avenacea* and *P. monspeliensis* were indistinguishable from strains from *L. rigidum* based on colony morphology, serological reactions, and bacteriophage specificity. Bacteriophages isolated from *C. toxicus* on the three hosts were indistinguishable from each other based on DNA restriction patterns. In allozyme studies, considerable variation was observed between the *C. toxicus* strains from the three hosts, but the variation was within the range exhibited by a single species. *C. toxicus* is carried into *L. rigidum* by a seed gall-forming nematode, *Anguina funesta*. Anguina nematodes are also associated with *C. toxicus* infection of *A. avenacea* and *P. monspeliensis*. Allozyme studies indicate that the same Anguina species probably infects both grasses, and that it is not *Anguina funesta*, *Anguina agrostis*, *Anguina tritici*, or the species found on velvetgrass (*Holcus lanatus*). This is the first recording of a nematode other than *Anguina funesta* as a vector for *C. toxicus*. The new vector broadens the range of grasses that the bacterium can infect.

33. **Characterization of two novel *Rhizobium leguminosarum* bacteriophages from a field release site of genetically-modified rhizobia. Mendum, T. A., Clark, I. M., Hirsch, P. R. (2001). *Antonie van Leeuwenhoek* 79:189-197.** Two *Rhizobium leguminosarum* biovar *viceae* bacteriophages with contrasting properties were isolated from a field site in which the survival of genetically modified *R. leguminosarum* inoculants had been monitored for several years. Inoculant strain RSM2004 was used as the indicator for phage isolation and propagation. One phage, RL1RES, was temperate and could not replicate in any of the 42 indigenous *R. leguminosarum* field isolates tested although nested PCR indicated that phage sequences were present in six of the isolates. The second phage, RL2RES, was virulent, capable of generalised transduction, contained DNA with modified cytosine residues, and was capable of infecting all field isolates tested although the GM inoculant strain CT0370 was resistant. Sequence with homology to RL2RES was detected by nested PCR in six of the 42 field-isolates. These were not

the same isolates that showed homology to RL1RES. The implication of these findings for the survival of rhizobial inoculants, and the ecology of phages and their host bacteria, are discussed

34. **Effect of phage on survival of *Salmonella enteritidis* during manufacture and storage of cheddar cheese made from raw and pasteurized milk. Modi, R., Hirvi, Y., Hill, A., Griffiths, M. W. (2001). *Journal of Food Protection* 64:927-933.** The ability of *Salmonella enteritidis* to survive in the presence of phage, SJ2, during manufacture, ripening, and storage of Cheddar cheese produced from raw and pasteurized milk was investigated. Raw milk and pasteurized milk were inoculated to contain 10^4 CFU/ml of a luminescent strain of *Salmonella enteritidis* (lux) and 10^8 PFU/ml SJ2 phage. The milks were processed into Cheddar cheese following standard procedures. Cheese samples were examined for *Salmonella enteritidis* (lux), lactic acid bacteria, molds and yeasts, coliforms, and total counts, while moisture, fat, salt, and pH values were also measured. *Salmonella enteritidis* (lux) was enumerated in duplicate samples by surface plating on MacConkey novobiocin agar. Bioluminescent colonies of *Salmonella enteritidis* were identified in the NightOwl molecular imager. Samples were taken over a period of 99 days. Counts of *Salmonella enteritidis* (lux) decreased by 1 to 2 log cycles in raw and pasteurized milk cheeses made from milk containing phage. In cheeses made from milks to which phage was not added, there was an increase in *Salmonella* counts of about 1 log cycle. Lower counts of *Salmonella enteritidis* (lux) were observed after 24 h in pasteurized milk cheese containing phage compared to *Salmonella* counts in raw milk cheese with phage. *Salmonella enteritidis* (lux) survived in raw milk and pasteurized milk cheese without phage, reaching a final concentration of 10^3 CFU/g after 99 days of storage at 8 degrees C. *Salmonella* did not survive in pasteurized milk cheese after 89 days in the presence of phage. However, *Salmonella* counts of approximately 50 CFU/g were observed in raw milk cheese containing phage even after 99 days of storage. In conclusion, this study demonstrates that the addition of phage may be a useful adjunct to reduce the ability of Salmonella to survive in Cheddar cheese made from both raw and pasteurized milk

35. **Phage conversion of Panton-Valentine leukocidin in *Staphylococcus aureus*: molecular analysis of a PVL-converting phage, phiSLT. Narita, S., Kaneko, J., Chiba, J., Piemont, Y., Jarraud, S., Etienne, J., Kamio, Y. (2001). *Gene* 268:195-206.** Staphylococcal Panton-Valentine leukocidin (PVL) is an important virulence factor, which causes leukocytolysis and tissue necrosis. Our previous report on the existence of the PVL genes (lukS-PV and lukF-PV) on the genome of prophage phiPVL in the *Staphylococcus aureus* strain V8 suggested the horizontal transmission of PVL genes by temperate bacteriophage among *S. aureus* (Kaneko, et al., 1998. Gene 215, 57-67). Here, we demonstrated the phage conversion of *S. aureus* leading to the production of PVL by discovery of a novel PVL-carrying phage, phiSLT (Staphylococcal Leukocytolytic Toxin) from a clinical isolate of *S. aureus*. phiSLT was able to lysogenize several clinical isolates of PVL-negative *S. aureus* strains as well as strain RN4220 at the conserved 29-bp sequence (attB) and all the lysogenized S. aureus strains had the ability to produce PVL. phiSLT had an elongated head of about 100x50 nm and a flexible tail of 400 nm long, that was quite different from phiPVL which had an isometric hexagonal head of about 60 nm diameter. The linear double-stranded phiSLT genome comprised 42,942 bp with 29-bp attachment core sequences and contained 62 open reading frames. Only 6.4 kbp region containing lysis cassette, PVL genes, attP, integrase, and orf204 of phiSLT was identical to that of phiPVL, while other regions were different from those of phiPVL. Thus, it can be concluded that PVL genes are carried by different temperate phages, which have the same attachment site

36. **Increased mutation rate of E. coli K12 lambda cultures maintained in continuous logarithmic growth. Northrop, J. H. (2001). *Journal of General Physiology* 50:369-377.** Continuous logarithmic growth of E. coli K12 lambda in an automatic culture cell resulted in marked increases in the proportion of several mutants. The P1 phage-resistant cells increased 10 to 3000 times, the T2 phage-resistant cells 1 to 1000 times, the neomycin-resistant cells 1 to 10 times, and the virus-producing cells 30 to 70 times. No change occurred in the penicillin-resistant cells. Calculation of the growth curves and direct determination of the mutation rates by the null fraction method showed that the increases in the proportion of mutants were due to increases in the mutation rates

37. **Pathogenicity and resistance islands of staphylococci. Novick, R. P., Schlievert, P., Ruzin, A. (2001). *Microbes Infect* 3:585-594.** Variable genetic elements including plasmids, transposons and prophages are involved in pathogenesis and antibiotic resistance, and are an important component of the staphylococcal genome. This review covers a set of newly described variable chromosomal elements, pathogenicity and resistance islands, carrying superantigen and resistance genes, especially toxic shock and methicillin resistance, respectively

38. **Diversification of *Escherichia coli* genomes: are bacteriophages the major contributors? Ohnishi, M., Kurokawa, K., Hayashi, T. (2001). *Trends in Microbiology* 9:481-485.** Determination of the genome sequence of enterohemorrhagic *Escherichia coli* O157 Sakai and genomic comparison with the laboratory strain K-12 has revealed that the two strains share a highly conserved 4.1-Mb sequence and that each also contains a larger amount of strain-specific sequence. The analysis also revealed the presence of a surprisingly larger number of prophages in O157, most of which are lambda-like phages that resemble each other. Based on these results, we discuss how bacteriophages contributed to this process. We also describe possible mechanisms by which O157 acquired many closely related phages, and raise the possibility that such bacteria might function as 'phage factories', releasing a variety of chemeric or mosaic prophages into the environment.

39. **Bacteriophage P4282, a parasite of *Ralstonia solanacearum*, encodes a bacteriolytic protein important for lytic infection of its host. Ozawa, H., TANAKA, H., Ichinose, Y., Shiraishi, T., Yamada, T. (2001). *MGG Molecular Genetics and Genomics* 265:95-101.** To enhance bacterial wilt resistance in tobacco expressing a foreign protein, we isolated the bacteriolytic gene from a bacteriophage that infects *Ralstonia solanacearum*. The bacteriolytic protein of phage P4282 isolated in Tochigi Prefecture was purified from a lysate of *R. solanacearum* M4S cells infected with the phage, and its bacteriolytic activity was assayed by following the decrease in the turbidity of suspensions of R. solanacearum M4S cells. The molecular weight of the bacteriolytic protein was approximately 71 kDa, and the sequence of the N-terminal 13 amino acids was determined. We used oligonucleotide probes based on this amino acid sequence to isolate the bacteriolytic gene from phage P4282 DNA. This gene of 2061 bp encodes a product of 687 amino acids, whose calaculated molecular weight was 70.12 kDa. The bacteriolytic gene was placed under the control of an inducible promoter, and the plasmid was transformed into *Escherichia coli* NM522. The soluble proteins extracted from *E. coli* NM522 cells harboring the plasmid with the bacteriolytic gene showed obvious bacteriolytic activities against several strains of R. solanacearum isolated in various districts in Japan. DNA fragments from five phages, isolated in Niigata, Aomori, Okinawa, Fukushima and Yamaguchi Prefectures, hybridized to the bacteriolytic gene of phage P4282. These observations indicate that the bacteriolytic protein shows nonspecific activity against *R. solanacearum* strains, and a sequence similar to that of the bacteriolytic gene is conserved in the DNA of other bacteriophages. These results indicate that the generation of transgenic (tobacco) plants expressing the bacteriolytic gene of phage P4282 might

result in enhanced resistance to bacterial wilt in tobacco

40. **Survival of bacteriophages of Lactococcus lactis in sodium hypochlorite and during storage. Parada, J. L., De Fabrizio, S. V. (2001). *Revista Argentina de Microbiologia* 33:89-95.** Survival of lytic bacteriophages active against Lactococcus lactis ssp. lactis and ssp. cremoris was determined after treatment with sodium hypochlorite and during at 4degreeC. Three phages were isolated from dairy plants in Argentina (ARG) and the other phages were isolated in the United States of America (US). All of them represent phages that infected cheese manufacture industries and belong to different morphological or serological groups. These phages showed higher survival in M17 broth, buffered with sodium glycerophosphate, than in trypteine soy broth (TSB). Phage populations did not decrease significantly during 14 weeks in M17 broth, whereas in TSB the titers of phage suspensions began to decline around 9 days. In addition, the effect of sodium hypochlorite was more marked in broth than in milk. A higher surviving fraction was obtained in milk, even when tenfold higher concentrations of chloride were used. The effect of hypochlorite on phages of the same serological group was quite similar and independent of phage morphology. However, phage 137-1, which belongs to other serological group, showed lower resistance to sodium hypochlorite. Comparing the hypochlorite inactivation for ARG and US phages, it was observed that they have their own inactivation values, independently of their origin and morphological group. Long periods of time and high concentrations of chlorine were necessary to reduce the surviving fraction in milk. This indicates that hypochlorite concentrations and times of contact can be critical for the efficiency of the operative sanitization processes

41. **Comparative study of nine *Lactobacillus fermentum* bacteriophages. Picozzi, C., Galli, A. (2001). *Journal of Applied Microbiology* 91:394-403.** Aims: To investigate the basic properties of six temperate and three virulent phages, active on *Lactobacillus fermentum*, on the basis of morphology, host ranges, protein composition and genome characterization. Methods and Results: All phages belonged to the Siphoviridae family; two of them showed prolate heads. The host ranges of seven phages contained a common group of strains. SDS-PAGE protein profiles, restriction analysis of DNA and Southern blot hybridization revealed a high degree of homology between four temperate phages; partial homologies were also detected among virulent and temperate phages. Clustering derived from host range analysis was not related to the results of the DNA hybridizations. Conclusions: The phages investigated have common characteristics with other known phages active on the genus Lactobacillus. Sensitivity to viral infection is apparently enhanced by the presence of a resident prophage. Significance and Impact of the Study: These relationships contribute to the explanation for the origin of phage infection in food processes where Lact. fermentum is involved, such as sourdough fermentation

42. **Evolutionary role of restriction/modification systems as revealed by comparative genome analysis. Rocha, E. P., Danchin, A., Viari, A. (2001). *Genome Research* 11:946-958.** Type II restriction modification systems (RMSs) have been regarded either as defense tools or as molecular parasites of bacteria. We extensively analyzed their evolutionary role from the study of their impact in the complete genomes of 26 bacteria and 35 phages in terms of palindrome avoidance. This analysis reveals that palindrome avoidance is not universally spread among bacterial species and that it does not correlate with taxonomic proximity. Palindrome avoidance is also not universal among bacteriophage, even when their hosts code for RMSs, and depends strongly on the genetic material of the phage. Interestingly, palindrome avoidance is intimately correlated with the infective behavior of the phage. We observe that the degree of palindrome and restriction site avoidance is significantly and consistently less important in phages than in their bacterial hosts. This result brings to the fore a larger selective load for palindrome and restriction site avoidance on the bacterial hosts than on their infecting phages. It is then consistent with a view where type II RMSs are considered as parasites possibly at the verge of mutualism. As a consequence, RMSs constitute a nontrivial third player in the host-parasite relationship between bacteria and phages

43. **Changes in bacterial community composition and dynamics and viral mortality rates associated with enhanced flagellate grazing in a mesoeutrophic reservoir. Simek, K., Weinbauer, M. G., Hornak, K., Dolan, J. R., Nedoma, J., Masin, M., Amann, R. (2001). *Applied and Environmental Microbiology* 67:2723-2733.** Bacterioplankton from a meso-eutrophic dam reservoir was size fractionated to reduce (<0.8-mum treatment) or enhance (<5-mum treatment) protistan grazing and then incubated in situ for 96 h in dialysis bags. Time course samples were taken from the bags and the reservoir to estimate bacterial abundance, mean cell volume, production, protistan grazing, viral abundance, and frequency of visibly infected cells. Shifts in bacterial community composition (BCC) were examined by denaturing gradient gel electrophoresis (DGGE), cloning and sequencing of 16S rDNA genes from the different treatments, and fluorescence in situ hybridization (PISH) with previously employed and newly designed oligonucleotide probes, Changes in bacterioplankton characteristics were clearly linked to changes in mortality rates. In the reservoir, where bacterial production about equaled protist grazing and viral mortality, community characteristics were nearly invariant, In the "grazer-free" (0.8-mum-filtered) treatment, subject only to a relatively low mortality rate (similar to 17% day(-1)) from viral lysis, bacteria increased markedly in concentration. While the mean bacterial cell volume was invariant, DGGE indicated a shift in BCC and FISH revealed an increase in the proportion of one lineage within the beta proteobacteria, In the grazing-enhanced treatment (5-mum filtrate), grazing mortality was similar to 200% and viral lysis resulted in mortality of 30% of daily production. Cell concentrations declined, and grazing-resistant flocs and filaments eventually dominated the biomass, together accounting for > 80% of the total bacteria by the end of the experiment. Once again, BCC changed strongly and a significant fraction of the large filaments was detected using a FISH probe targeted to members of the Flectobacillus lineage, Shifts of BCC were also reflected in DGGE patterns and in the increases in the relative importance of both beta proteobacteria and members of the Cytophaga-Flavobacterium cluster, which consistently formed different parts of the bacterial flocs. Viral concentrations and frequencies of infected cells were highly significantly correlated with grazing rates, suggesting that protistan grazing may stimulate viral activity.

44. **Quorum sensing is a global regulatory mechanism in enterohemorrhagic *Escherichia coli* O157:H7. Sperandio, V, Torres, A. G., Giron, J. A., Kaper, J. B. (2001). *Journal of Bacteriology* 183:5187-5197.** Enterohemorrhagic *Escherichia coli* (EHEC) O157:H7 is responsible for outbreaks of bloody diarrhea and hemolytic-uremic syndrome in many countries. EHEC virulence mechanisms include the production of Shiga toxins (Stx) and formation of attaching and effacing (AE) lesions on intestinal epithelial cells. We recently reported that genes involved in the formation of the AE lesion were regulated by quorum sensing through autoinducer-2, which is synthesized by the product of the luxS gene. In this study we hybridized an E. coli gene array with cDNA synthesized from RNA that was extracted from EHEC strain 86-24 and its isogenic luxS mutant. We observed that 404 genes were regulated by luxS at least fivefold, which comprises approximately 10% of the array genes; 235 of these genes were up-regulated and 169 were down-regulated in the wild-type strain compared to in the luxS mutant. Down-regulated genes included several involved in cell division, as well as ribosomal and tRNA genes. Consistent with this pattern of gene expression, the luxS mutant grows faster than the wild-type strain (generation times of 37.5 and 60 min, respectively, in Dulbecco modified Eagle medium). Up-regulated genes included several involved in the expression and assembly of flagella, motility, and chemotaxis. Using operon::lacZ fusions to class I, II, and III flagellar genes, we were able to confirm this transcriptional regulation. We also observed fewer flagella by Western blotting and electron microscopy and decreased motility halos in semisolid agar in

the luxS mutant. The average swimming speeds for the wild-type strain and the luxS mutant are 12.5 and 6.6 microm/s, respectively. We also observed an increase in the production of Stx due to quorum sensing. Genes encoding Stx, which are transcribed along with lambda-like phage genes, are induced by an SOS response, and genes involved in the SOS response were also regulated by quorum sensing. These results indicate that quorum sensing is a global regulatory mechanism for basic physiological functions of *E. coli* as well as for virulence factors

45. **Application of *Streptococcus thermophilus* DPC1842 as an adjunct to counteract bacteriophage disruption in a predominantly lactococcal Cheddar cheese starter: use in bulk starter culture systems. Stokes, D., Ross, R. P., Fitzgerald, G. F., Coffey, A. (2001). *Lait* 81:327-334.** A significant amount of Cheddar cheese manufactured world-wide relies on bulk starter cultures instead of direct vat set (DVS) cultures. While the inclusion of *S. thermophilus* is sometimes used to counteract failure due to lactococcal phage in the latter system, it is considered difficult to implement in bulk starter systems and is normally avoided. This stems from the problem in controlling the ratio of *S. thermophilus* to lactococci during the bulk starter preparation such that suitable acidification rates can be achieved. The current study demonstrates how *S. thermophilus* numbers can be controlled during growth in the bulk starter medium prior to inoculation of a culture, based on three lactococcal strains and *S. thermophilus* DPC1842, into the cheese vat. The concentration of inorganic phosphate necessary to inhibit the growth of strain DPC1842 in a whey-based bulk starter medium was found to be 0.18 mol.L^{-1}. Since higher levels of phosphate exist in different commercial bulk starter media used for mesophilic cultures, a number of these media can be used for propagation of this starter blend without domination of strain DPC1842 over its lactococcal counterparts. Strain DPC1842 is highly phage resistant and is particularly acid-fast in the cheese milk and reduces the pH efficiently at very low inocula. Cheddar cheese was manufactured in a commercial plant with this system and the resulting cheese had good flavour characteristics. This study demonstrates the effectiveness of the *S. thermophilus* DPC1842 component in rescuing the fermentation in the event of severe lactococcal phage attack.

46. **Therapy of infections in cancer patients with bacteriophages. Weber-Dabrowska, B., Mulczyk, M., Górski, A. (2001). *CLIN APPL IMMUNOL REV* 1:131-134.** Cancer patients are known to be immunocompromised and susceptible to infections. We have used bacteriophages matched for specific bacterial isolates to treat antibiotic-resistant infections in those patients. Cure of infection was achived in all cases indicating very highefficacy of BP therapy.

47. **Interaction of the PhiHSIC virus with its host: lysogeny or pseudolysogeny? Williamson, S. J., McLaughlin, M. R., Paul, J. H. (2001). *Applied and Environmental Microbiology* 67:1682-1688.** The marine phage PhiHSIC has been previously reported to enter into a lysogenic relationship with its host, HSIC, identified as *Listonella pelagia*. This phage produces a variety of plaques on its host, including turbid and haloed plaques, from which lysogens were previously isolated. These lysogens were unstable during long-term storage at -80° C and were lost. When HSIC was reinfected with phage PhiHSIC, pseudolysogen-like interactions between the phage and its host were observed. The cells (termed HSIC-2 or HSIC-2e) produced high viral titers (10^{11} ml^{-1}) in the absence of inoculating phage and yet reached culture densities of nearly 10^{9} ml^{-1}. Prophages were not induced by mitomycin C or the polyaromatic hydrocarbon naphthalene in cells harboring such infections. However, such cells were homoimmune to superinfection. Colonies hybridized strongly with a gene probe from a 100-bp fragment of the PhiHSIC genome, while the host did not. Analysis of chromosomal DNA preparations suggested the presence of a chromosomally integrated prophage. Phage adsorption experiments suggested that HSIC-2 was adsorption impaired. Because of the chromosomal prophage integration and homoimmunity, we interpret these results to indicate that PhiHSIC establishes a lysogenic relationship with its host that involves an extremely high level of spontaneous induction. This could be caused by a weak repressor of phage production. Additionally, poor phage adsorption of HSIC-2 compared to the wild type probably helped maintain this pseudolysogen-like relationship. In many ways, pseudolysogenic phage-host interactions may provide a paradigm for phage-host interactions in the marine environment

48. **Integrated management of bacterial leaf spot of mungbean with bacteriophages of Xav and chemicals. Borah, P. K., Jindal, J. K., Verma, J. P. (2000). *Journal of Mycology and Plant Pathology* 30:19-21.** The population of *Xanthomonas axonopodis* pv *vignaeradiatae* (Xav) was completely eliminated from mungbean seeds by lytic action of bacteriophage (XMP-1) and streptomycin when the seeds were treated with Xav, phages and streptomycin at a ratio/concentration of 1: 60 + 300 mug ml-1. These results confirmed the synergistic action between phage (XMP-1) and streptomycin, as their combination could eradicate Xav from mungbean seeds at a much lower concentration as compared to when used singly. Moreover, the seed treatment with phage lysate + streptomycin 300 mug ml-1 was also found most effective in checking seedling infection of mungbean by Xav. The seedling infection was 4 per cent as compared to 68 per cent in control. The percentage of seed germination was also increased to 86 per cent in comparison to 75 per cent in control

49. **Viruses of fungi and protozoans: Is everyone sick? Bruenn, J. A. (2000). pp. 297-317 in Hurst, C. J. (ed.) *Viral Ecology*. Academic Press, San Diego.** [no abstract?[

50. **Lateral gene transfer in prokaryotes. Campbell, A. M. (2000). *Theoretical Population Biology* 57:71-77.** Evolutionists have traditionally depicted organismal descent on a tree, where all lineages branch from a com- mon ancestor. Such a tree phylogeny implies that all genetic traits within a lineage are derived from its founder. Lateral (horizontal) gene transfer negates this exclusive relationship between ancestor and descendants by the introduction into a lineage of genes originating elsewhere.

51. **An introduction to the evolutionary ecology of viruses. DeFilippis, V. R., Villarreal, L. P. (2000). pp. 125-208 in Hurst, C. J. (ed.) *Viral Ecology*. Academic Press, San Diego.**

52. **Microvirus of *Chlamydia psittaci* strain Guinea pig inclusion conjunctivitis: Isolation and molecular characterization. Hsia, R. C., Ting, L. M., Bavoil, P. M. (2000). *Microbiology (Reading)* 146:1651-1660.** The authors report the isolation and molecular characterization of a bacteriophage, phiCPG1, which infects *Chlamydia psittaci* strain Guinea pig Inclusion Conjunctivitis. Purified virion preparations contained isometric particles of 25 nm diameter, superficially similar to spike-less members of the phiX174 family of bacteriophages. The single-stranded circular DNA genome of phiCPG1 included five large ORFs, which were similar to ORFs in the genome of a previously described *Chlamydia* bacteriophage (Chp1) that infects avian *C. psittaci*. Three of the ORFs encoded polypeptides that were similar to those in a phage infecting the mollicute *Spiroplasma melliferum*, a pathogen of honeybees. Lesser sequence similarities were seen between two ORF products and the major capsid protein of the phiX174 coliphage family and proteins mediating rolling circle replication initiation in phages, phagemids and plasmids. Phage phiCPG1 is the second member of the genus *Chlamydiamicrovirus*, the first to infect a member of a *Chlamydia* species infecting mammals. Similarity searches of the nucleotide sequence further revealed a highly conserved (75%

identity) 375 base sequence integrated into the genome of the human pathogen *Chlamydia pneumoniae*. This genomic segment encodes a truncated 113 residue polypeptide, the sequence of which is 72% identical to the amino-terminal end of the putative replication initiation protein of phiCPG1. This finding suggests that *C. pneumoniae* has been infected by a phage related to phiCPG1 and that infection resulted in integration of some of the phage genome into the *C. pneumoniae* genome

53. **Genomic sequences of bacteriophages HK97 and HK022: Pervasive genetic mosaicism in the lambdoid bacteriophages. Juhala, R. J., Ford, M. E., Duda, R. L., Youlton, A., Hatfull, G. F., Hendrix, R. W. (2000). *Journal of Molecular Biology* 299:27-51.** We report the complete genome DNA sequences of HK97 (39,732 bp) and HK022 (40,751 bp), double-stranded DNA bacteriophages of *Escherichia coli* and members of the lambdoid or lambda-like group of phages. We provide a comparative analysis of these sequences with each other and with two previously determined lambdoid family genome sequences, those of *E. coli* phage lambda and *Salmonella typhimurium* phage P22. The comparisons confirm that these phages are genetic mosaics, with mosaic segments separated by sharp transitions in the sequence. The mosaicism provides clear evidence that horizontal exchange of genetic material is a major component of evolution for these viruses. The data suggest a model for evolution in which diversity is generated by a combination of illegitimate and homologous recombination and mutational drift, and selection for function produces a population in which most of the surviving mosaic boundaries are located at gene boundaries or, in some cases, at protein domain boundaries within genes. Comparisons of these genomes highlight a number of differences that allow plausible inferences of specific evolutionary scenarios for some parts of the genome. The comparative analysis also allows some inferences about function of genes or other genetic elements. We give examples for the generalized recombination genes of HK97, HK022 and P22, and for a putative headtail adaptor protein of HK97 and HK022. We also use the comparative approach to identify a new class of genetic elements, the morons, which consist of a protein-coding region flanked by a putative sigma70 promoter and a putative factor-independent transcription terminator, all located between two genes that may be adjacent in a different phage. We argue that morons are autonomous genetic modules that are expressed from the repressed prophage. Sequence composition of the morons implies that they have entered the phages' genomes by horizontal transfer in relatively recent evolutionary time

54. **Ecology of bacteriophages in nature. Paul, J. H., Kellogg, C. A. (2000). pp. 211-246 in Hurst, C. J. (ed.) *Viral Ecology*. Academic Press, San Diego.**

55. **Genomic sequence and analysis of the atypical temperate bacteriophage N15. Ravin, V, Ravin, N., Casjens, S., Ford, M. E., Hatfull, G. F., Hendrix, R. W. (2000). *Journal of Molecular Biology* 299:53-73.** N15 is a temperate bacteriophage that forms stable lysogens in *Escherichia coli*. While its virion is morphologically very similar to phage lambda and its close relatives, it is unusual in that the prophage form replicates autonomously as a linear DNA molecule with closed hairpin telomeres. Here, we describe the genomic architecture of N15, and its global pattern of gene expression, which reveal that N15 contains several plasmid-derived genes that are expressed in N15 lysogens. The tel site, at which processing occurs to form the prophage ends is close to the center of the genome in a similar location to that occupied by the attachment site, attP, in lambda and its relatives and defines the boundary between the left and right arms. The left arm contains a long cluster of structural genes that are closely related to those of the lambda-like phages, but also includes homologs of umuD', which encodes a DNA polymerase accessory protein, and the plasmid partition genes, sopA and sopB. The right arm likewise contains a mixture of apparently phage- and plasmid- genes including genes encoding plasmid replication functions, a phage repressor, a transcription antitermination system, as well as phage host cell lysis genes and two putative DNA methylases. The unique structure of the N15 genome suggests that the large global population of bacteriophages may exhibit a much greater diversity of genomic architectures than was previously recognized

56. **Comparative analysis of Chlamydia bacteriophages reveals variation localized to a putative receptor binding domain. Read, T. D., Fraser, C. M., Hsia, R. C., Bavoil, P. M. (2000). *Microbial and Comparative Genomics* 5:223-231.** Three recently discovered ssDNA Chlamydia-infecting microviruses, phiCPG1, phiAR39, and Chp2, were compared with the previously characterized phage from avian *C. psittaci*, Chp1. Although the four bacteriophages share an identical arrangement of their five main genes, Chp1 has diverged significantly in its nucleotide and protein sequences from the other three, which form a closely related group. The VP1 major viral capsid proteins of phiCPG1 and phiAR39 (from guinea pig-infecting *C. psittaci* and *C. pneumoniae*, respectively) are almost identical. However, VP1 of ovine *C. psittaci* phage Chp2 shows a high rate of nucleotide sequence change localized to a region encoding the "IN5" loop of the protein, thought to be a potential receptor-binding site. Phylogenetic analysis suggests that the ORF4 replication initiation protein is evolving faster than the other phage proteins. phiCPG1, phiAR39, and Chp2 are closely related to an ORF4 homolog inserted in the C. pneumoniae chromosome. This sequence analysis opens the way toward understanding the host-range and evolutionary history of these phages

57. **The passage and propagation of fecal indicator phages in birds. Ricca, D. M., Cooney, J. J. (2000). *Journal of Industrial Microbiology & Biotechnology.* 24:127-131.** The presence of F-specific phages in the diet of birds influenced the presence of these fecal indicators in their feces. F-specific phage concentrations in the feces of Canada geese and pigeons, which are normally low, increased greatly the same day coliphage MS2 was added to their diets. F-specific phage concentrations decreased to the original low levels a week after the phage-spiked feed was removed. Geese kept in pens that were cleaned regularly to reduce fecal-oral contamination had significantly lower somatic coliphage concentrations in their feces than wild geese had in their feces. Somatic coliphage concentrations in feces of feral pigeons were typically low with an occasional fecal sample having high numbers of either one of the two types of phages seen in this population of birds. Sometimes many birds had high numbers of only one type of phage in their feces. This lasted only a day and was probably due to fecal contamination of the feeding pans by the pigeons. The degree to which birds are a source of phage indicators of fecal pollution can change in a short period of time. Thus the presence of contaminated feeding sites should be considered before ruling out animals as a possible source of fecal indicators. F-specific phages may be useful tracers for modeling viral transmission and tracking feeding habits in birds

58. **Screening environmental samples for source-specific bacteriophage hosts using a method for the simultaneous pouring of 12 petri plates. Ricca, D. M., Cooney, J. J. (2000). *Journal of Industrial Microbiology & Biotechnology.* 24:124-126.** A simple apparatus was developed to allow 12 petri plates to be poured simultaneously by hand. It was used when screening bacterial isolates from sewage and dog feces for their ability to detect phages from these sources. This was done to assess the ease with which source-specific phage hosts can be isolated from these sources of fecal pollution. Host bacteria that consistently detected phages from sewage were easily isolated from sewage. These bacterial isolates did not detect phages from dog feces. Host bacteria were not isolated from dog feces even after screening hundreds of colonies from fecal samples from six dogs

59. **The genome sequence of the plant pathogen Xylella fastidiosa. Simpson, A. J. G., Reinach, F. C., Arruda, P., Abreu, F. A., Acencio, M., Alvarenga, R., Alves, L. M. C., Araya, J. E., Baia, G. S., Baptista, C. S., Barros, M. H., Bonaccorsi, E. D., Bordin, S., Bove, J. M., Briones, M. R. S., Bueno, M. R. P., Camargo, A. A., Camargo, L. E. A., Carraro, D. M., Carrer, H., Colauto, N. B., Colombo, C., Costa, F. F., Costa, M. C. R., Costa-Neto, C. M., Coutinho, L. L., Cristofani, M., Dias-Neto, E., Docena, C., El-Dorry, H., Facincani, A. P., Ferreira, A. J. S., Ferreira, V. C. A., Ferro, J. A., Fraga, J. S., Franca, S. C., Franco, M. C., Frohme, M., Furlan, L. R., Garnier, M., Goldman, G. H., Goldman, M. H. S., Gomes, S. L., Gruber, A., Ho, P. L., Hoheisel, J. D., Junqueira, M. L., Kemper, E. L., Kitajima, J. P., Krieger, J. E., Kuramae, E. E., Laigret, F., Lambais, M. R., Leite, L. C. C., Lemos, E. G. M., Lemos, M. V. F., Lopes, S. A., Lopes, C. R., Machado, J. A., Machado, M. A., Madeira, A. M. B. N., Madeira, H. M. F., Marino, C. L., Marques, M. V., Martins, E. A. L., Martins, E. M. F., Matsukuma, A. Y., Menck, C. F. M., Miracca, E. C., Miyaki, C. Y., Monteiro-Vitorello, C. B., Moon, D. H., Nagai, M. A., Nascimento, A. L. T. O., Netto, L. E. S., Nhani, A., Jr., Nobrega, F. G., Nunes, L. R., Oliveira, M. A., de Oliveira, M. C., de Oliveira, R. C., Palmieri, D. A., Paris, A., Peixoto, B. R., Pereira, G. A. G., Pereira, H. A., Jr., Pesquero, J. B., Quaggio, R. B., Roberto, P. G., Rodrigues, V, de, M. R., de Rosa, V. E., Jr., de Sa, R. G., Santelli, R. V., Sawasaki, H. E., da Silva, A. C. R., da Silva, A. M., da Silva, F. R., Silva, W. A., Jr., da Silveira, J. F. (2000). *Nature (London)* 406:151-157.** *Xylella fastidiosa* is a fastidious, xylem-limited bacterium that causes a range of economically important plant diseases. Here we report the complete genome sequence of *X. fastidiosa* clone 9a5c, which causes citrus variegated chlorosis-a serious disease of orange trees. The genome comprises a 52.7% GC-rich 2,679,305-base-pair (bp) circular chromosome and two plasmids of 51,158 bp and 1,285 bp. We can assign putative functions to 47% of the 2,904 predicted coding regions. Efficient metabolic functions are predicted, with sugars as the principal energy and carbon source, supporting existence in the nutrient-poor xylem sap. The mechanisms associated with pathogenicity and virulence involve toxins, antibiotics and ion sequestration systems, as well as bacterium-bacterium and bacterium-host interactions mediated by a range of proteins. Orthologues of some of these proteins have only been identified in animal and human pathogens; their presence in *X. fastidiosa* indicates that the molecular basis for bacterial pathogenicity is both conserved and independent of host. At least 83 genes are bacteriophage-derived and include virulence-associated genes from other bacteria, providing direct evidence of phage-mediated horizontal gene transfer

60. **Prophage, phiPV83-pro, carrying panton-valentine leukocidin genes, on the *Staphylococcus aureus* P83 chromosome: comparative analysis of the genome structures of phiPV83-pro, phiPVL, phi11, and other phages. Zou, D., Kaneko, J., Narita, S., Kamio, Y. (2000). *Bioscience, Biotechnology, and Biochemistry* 64:2631-2643.** *Staphylococcus aureus* P83 has Panton-Valentine leukocidin (PVL)-like genes, lukM and lukF-PV. Here, lukM and lukF-PV genes were found on the genome of a prophage, which was designated as phiPV83-pro. The precise genome size was 45,636 bp with att core sequences of 10 base pairs. Sixty-four ORFs were identified on the phiPV83-pro genome, including two extra operons, lukM-lukF-PV and orfs63-64. The lukM-lukF-PV cluster was located 2.1 kb upstream of the attL site. The most striking feature of the phiPV83-pro genome was a constituent of at least 4 regions from phi11, phiPVL, and other phages, i.e., (i) att sites identical with those of phi11, (ii) a cos sequence and the genes encoding packaging and head proteins of phiPVL (occupied half region of phiPV83-pro), and (iii) the other two regions which showed no significant similarity with known phages (occupied about 40% of phiPV83-pro). Furthermore, two insertion sequences, ISSA1 and ISSA2 were integrated into attL site and orf44, respectively. PhiPV83-pro was not induced as phage particles from *S. aureus* P83 regardless of its treatment with mitomycin C. The insertion of ISSA1 into the attL site was one of the reasons of the failure of the induction of the phage particles by mitomycin C treatment of the strain P83

61. **Flow cytometric analyses of virus infection in two marine phytoplankton species, *Micromonas pusilla* (Prasinophyceae) and *Phaeocystis pouchetii* (Prymnesiophyceae). Brussaard, C. P. D., Thyrhaug, R., Marie, D., Bratbak, G. (1999). *Journal of Phycology* 35:941-948.** Cell characteristics of two axenic marine phytoplankton species, *Micromonas pusilla* (Butscher) Manton et Parke and *Phaeocystis pouchetii* (Hariot) Lagerheim, were followed during viral infection using flow cytometry, Distinct differences between noninfected and infected cultures were detected in the forward scatter intensities for both algal species. Changes in side scatter signals on viral infection were found only for *P, pouchetii*. Chlorophyll red fluorescence intensity per cell decreased gradually over time in the infected cultures, DNA analyses were performed using the nucleic acid-specific fluorescent dye SYBR Green I. Shortly after infection the fraction of algal cells with more than one genome equivalent increased for both species because of the replication of viral DNA in the infected cells. Over time, a population of algal cells with low red autofluorescence and low DNA fluorescence developed, likely representing algal cells just prior to viral lysis, The present study provides insight into basic virus-algal host cell interactions. It shows that flow cytometry can be a useful tool to discriminate between virus infected and noninfected phytoplankton cells.

62. **Biocontrol of *Erwinia amylovora* using bacteriophage. Gill, J. J., Svircev, A. M., Myers, A. L., Castle, A. J. (1999). *Phytopathology* 89:S27.**

63. **Cyanophages. Martin, E. L., Kokjohn, T. A. (1999). pp. 324-332 in Granoff, A., Webster, R. G. (eds.) *Encyclopedia of Virology second edition*. Academic Press, San Diego.**

64. **Bacteriophage therapy of *Clostridium difficile*-associated intestinal disease in a hamster model. Rdamesh, V., Fralick, J. A., Rolfe, R. D. (1999). *Miroecol. Anarobes[sic?]* 5:69-???**

65. **Dissolved esterase activity as a tracer of physoplankton lysis: Evidence of high phytoplankton lysis rates in the northwestern Mediteranean. Agustí, S., Satta, M. P., Mura, M. P., Benavent, E. (1998). *Limnology and Oceanography* 43:1836-1849.** Phytoplankton cell lysis is perceived to be an important loss process in the sea, although a quantification of this process has proved elusive. A recently developed method, based on the measurement of dissolved esterase activity (EA), was used to estimate the release of esterases following phytoplankton cell lysis in an effort to evaluate the importance of this process as a loss factor in the summer phytoplankton of the northwestern Mediterranean Sea. Implicit in this method was the assumption that only the lysis of phytoplankton cells caused these enzymes to be released to the medium. This assumption was tested by analyzing the presence and release of esterases by marine bacteria, heterotrophic flagellates, and heterotrophic ciliates, all isolated from the Blanes Bay (northwestern Mediterranean, Spain), and by phytoplankton grown in culture (*Synechococcus elongatus*, *Dunaliella* sp., *Chlorella* sp., *Phaeodactyllum tricornutum*, and *Chaetoceros decipiens*). The dissolved EA found during the growth, stationary, and decay phases of microheterotrophs (bacteria, flagellate, and ciliate) was negligible when compared to that found for phytoplanktonic cultures. Differences in cell volume explained the differences in cell EA among the organisms, but heterotrophs showed lower cell EA (10-50-fold) than phytoplanktonic cells of similar cell size. These results support the assumption that microheterotrophs do not contribute significant amounts of EA to the dissolved pear, allowing the use of the method to estimate phytoplankton lysis. Independent estimates of cell, loss in phytoplankton cultures, derived from cell cycle analysis, confirmed the estimates of cell lysis obtained from the measurement of dissolved EA.

During the study conducted in the Mediterranean Sea, the water column was strongly stratified, showing a deep (40-55 m) chlorophyll a (Chl a) maximum (DCM; 1.25 +/- 0.09 mu g liter^{-1}) and low surface Chl a concentrations (0.09 +/- 0.008 mu g liter(-1)). Phytoplankton lysis rates ranged between 0.026 d^{-1} and 1.9 d^{-1}, and they declined significantly with depth; the fastest rates were found in surface waters and the slowest ones at the DCM. Despite the fast gross growth rates of surface phytoplankton las calculated from phytoplankton biovolume and oxygen production), the calculated lysis rates represented a considerable proportion of gross phytoplankton growth rate (50%) at the surface, whereas they were comparatively less important at the DCM (7%). These results provide strong evidence that phytoplankton lysis can be an important loss factor in the surface waters of this stratified, oligotrophic sea. Phytoplankton lysis could provide the loss factor needed to explain the low phytoplankton biomass despite fast growth and low grazing rates in the northwestern Mediterranean surface waters. The high lysis rate of phytoplankton in surface waters represents an important path by which primary production may fuel the growth of microheterophic organisms, consistent with the high respiration rate of the surface community examined. The conclusion that phytoplankton lysis rates can occur at rates high enough to influence food web dynamics and biogeochemical cycles in the oligotrophic ocean should stimulate research on this largely neglected loss factor in phytoplankton ecology.

66. **Polyvirulent rhizobiophage from a soybean rhizosphere soil. Ali, F. S., Hammand, A. M. M., Loynachan, T. E. (1998). *Soil Biology and Biochemistry* 30:2171-2175.**

67. **Viral lysis of *Phaeocystis pouchetii* and bacterial secondary production. Bratbak, G., Jacobsen, A., Heldal, M. (1998). *Aquatic Microbial Ecology* 16:11-16.** In this experimental study we investigated the effect of viral infection on primary production and carbon now in a phytoplankton-DOC-bacteria food chain during viral lysis of the phytoplankton population. The phytoplankter host-virus system used was *Phaeocystis pouchetii* (Prymnesiophyceae) and the virus PpV01. Viral infection allowed primary production in the cells to continue throughout most of the lytic cycle. In non-infected algal cultures, net production of DOC and bacterial biomass was low and at the end of the experiment the DOC concentration was 10 to 20%, and the bacterial biomass 0.5 to 4 % of the algal carbon biomass. The amount of DOC released during viral lysis of the algal cells implies that the entire algal biomass was converted to DOG. Growth of bacteria succeeding cell lysis and release of DOC in Virus infected cultures demonstrated that the net effect of the virus infection was an efficient conversion of algal biomass into bacterial biomass.

68. **Virus production in *Phaeocystis pouchetii* and its relation to host cell growth and nutrition. Bratbak, G., Jacobsen, A., Heldal, M., Nagasaki, K., Thingstad, T. F. (1998). *Aquatic Microbial Ecology* 16:1-9.**

69. **Ultrastructural analysis of viral invection in the brown-tide alga, *Aureococcus anophagefferens* (Pelagophyceae). Gastrich, M. D., Anderson, O. R., Benmayor, S. S., Cosper, E. M. (1998). *Phycologia* 37:300-306.** The DNA-containing virus (BtV) is known to lyse laboratory cultures of *Aureococcus anophagefferens* Hargraves et Sieburth, an alga known to cause blooms devastating to shellfish and eelgrass beds. Ultrastructural study of the infection of *A. anophagefferens* by this virus shows a progressive degradation of host algal cells. Healthy uninfected algal cells (c. 2.0 mu m) exhibit organelles typical of the Pelagophyceae and are surrounded by a prominent fibrous glycocalyx. All laboratory cultures of *A. anophagefferens* inoculated with the BtV virus were lysed within 24-48 h, leaving no living cells. Infected brown-ride cells had an unusually electron-dense, crenated plasma membrane and lacked a glycocalyx. During early stages of infection, the vacuole disappeared, and the nucleus was disrupted by the formation of viroplasm. The organelles disappeared, with the chloroplast being the last to degrade. A few intracellular viral capsids (c. 140-160 nm) were observed during the degeneration of the organelles. In the final stages of infection, the entire host cell was filled with viroplasm and viral capsids, and no organelles remained.

70. **Biological control of bacterial blight of geranium with h-mutant bacteriophages. Harbaugh, B. K., Jones, J. B., Jackson, L. E., Somodi, G., Flaherty, J. E. (1998). *95th Annual International Conference of the American Society for Horticultural Science* 33:519.**

71. **Effect of temperature on the algicidal activity and the satability of HaV (*Heterosigma akashiwo* virus). Nagasaki, K., Yamaguchi, M. (1998). *Aquatic Microbial Ecology* 15:211-216.** The effect of temperature on the algicidal activity and stability of HaV (*Heterosigma akashiwo* virus), which infects the harmful bloom causing alga, *H. akashiwo* (Raphidophyceae), was determined by growing *H. akashiwo* culture inoculated with HaV under various conditions. Temperature and growth stage of the host culture are considered to be important factors determining the algicidal activity of HaV. The optimum temperature for the algicidal activity of HaV ranged from 20 to 25 degrees C. Comparing the viral susceptibility of *H. akashiwo* strains and the algicidal activity of the HaV clones at different temperatures, both were suggested to be phenotypically diverse. Effect of temperature on the stability of HaV was also evaluated. HaV showed a relatively rapid decrease in infectious titer even when preserved at 5 degrees C in the dark. The data is discussed in relation to the behavior of HaV in natural environments and the disintegration mechanism of *H. akashiwo* red tide.

72. **Lysogeny of *Oenococcus oeni* (syn. *Leuconostoc oenos*) and study of their induced bacteriophages. Poblet-Icart, M., Bordons, A., Lonvaud-Funel, A. (1998). *Current Microbiology* 36:365-369.** A large number of strains of *Oenococcus oeni* (formerly *Leuconostoc oenos*) that had been isolated from wines were checked for lysogeny with mitomycin C as inducer. As a result of this test, 45% of the strains proved to be lysogenic, suggesting that lysogeny is widespread among bacteria isolated from wines during malolactic fermentation. The sensitivity of bacteria to phages was very different, depending on the strain. All the lysogenic strains were resistant to infection by the temperate phage they released. Some phages infected none of the strains. Phages of Oenoc. oeni had a classical morphology, an isometric head, and a long striated tail. With the broadest host strain as an indicator, phages were detected in wines after malolactic fermentation

73. **Comparative analysis of the effect of energy process inhibitors on the efficacy of phage infection in staphylococci. Polishko, T. N. (1998). *Mikrobiolohichnyi Zhurnal* 60:36-42.** The study of the effect of KCN, DCCD and CCCP as inhibitors of the energy yielding processes showed that the efficacy of phage infection depended on respiration, protone ATPase, and protone electrochemical potential of hydrogen ions. There was a 49.5-68.0% decrease of the efficacy of phage infection after addition of the above mentioned inhibitors at the period of the contact of cells with bacteriophages at the stage of the phage nucleic acid transfer. The Ambden - Meirhoff - Pamas route inhibitors NaF and CH2ICOOH less affected the efficacy of phage infection. The same effect was observed during addition of Na3AsO4 as the ATP synthesis inhibitor. This efficacy decrease was probably due the inhibition of the processes of the substrate level phosphorylation and the deplete of the intracellular ATP content

74. **Seasonal abundance in Skagerrak-Kattegat coastal waters and host specificity of viruses infecting the marine photosynthetic flagellate *Micromonas pusilla*. Sahlsten, E. (1998). *Aquatic Microbial Ecology* 16:103-108.** Seawater sampled in the Skagerrak and Kattegat coastal waters during the period October 1995 to September 1996 were screened for the occurrence of viruses lytic to marine microalgae. Viruses lytic to the photosynthetic marine picoflagellate *Micromonas pusilla* (Butcher) Manton & Parke (Prasinophyceae) were detected in all seawater samples screened. Evidence for viral lysis of any other of the 11 algal species tested was not obtained. Several viruses infecting different strains of M. pusilla were isolated. Ten isolated viruses which were tested for host specificity were found to be species specific to *M. pusilla* and even strain specific to 1-3 of the 6 strains of *M pusilla* used in the experiment. In the Skagerrak and Kattegat the seasonal abundance of viruses infectious to a *M. pusilla* strain isolated from the Oslofjord, Norway, was at least 1 order of magnitude higher (average 2.5 x 10^5 l^{-1}) than viruses infecting 2 *M. pusilla* strains isolated from Gull of Maine, USA (average 2.2 x 10^4 and 4.6 x 10^3 l^{-1} respectively).

75. **Vertical distribution of virus-like particles (VLP) and viruses infecting *Micromonas pusilla* during late summer in the southeastern Skagerrak. Sahlsten, E., Karlson, B. (1998). *J. Plankton Res.* 20:2207-2212.** Vertical profiles were made at one offshore station and one coastal station, on 4-5 September 1996, in the south-eastern Skagerrak. The surface water of the two stations differed significantly with respect to both temperature and salinity, as the outer station (A) was situated in high-saline water originating from the North Sea, while the low-saline surface water at the inner station (B) was influenced by the Baltic current. Virus-like particle (VLP) abundance was 5 x 10^9-25 x 10^9 l(-1) in the 0-50 m water column. Maximal VLP values were found in the surface water, although a lower number was detected in the low-saline surface water (0 m depth) at station B. Viruses infective to *Micromonas pusilla* were estimated to similar to 0.01% of the VLP number. The ambient concentrations of dissolved inorganic nutrients were typical for a stratified summer situation, i.e. generally low in the surface waters, although a raised ammonium concentration was associated with the sharp halocline at 5 m depth at station B, and all nutrient levels were increasing below 30 m depth.

76. **Morphology and abundance of free and temperate viruses in Lake Superior. Tapper, M. A., Hicks, R. E. (1998). *Limnology and Oceanography* 43:95-103.** The morphology and abundance of free viruses were measured in spring, summer, and fall at one site in Lake Superior. Free viral head sizes ranged from 10 to 70 nn and tail length ranged from 10 to 110 nm. The vast majority (98%) of free viral head sizes were less than or equal to 60 nm, smaller than reported in most freshwater habitats. Most of these free viruses (70%) had polyhedral heads and tails, indicative of bacteriophage. Free viral abundance only ranged from 0.1 to 9 x 10(6) viruses ml(-1) in the surface microlayer (top 20 mu m) and subsurface water (20 m) in Lake Superior, but viruses were 2-15 times more abundant in the surface microlayer. This difference may be due to the enrichment of bacterial hosts, higher levels of UV light that induce temperate phage, or differences in viral burst sizes in the surface microlayer relative to subsurface water. Bacterioplankton were always more abundant than free viruses in both the surface microlayer and subsurface water, which resulted in some of the lowest virus-to-bacterium ratios reported for marine or freshwater environments. Temperate viruses from both habitats responded equally to mitomycin-C and UV light treatments used to induce prophage into lytic cycles. An estimated 0.1-7.4% of the bacterioplankton from this site in Lake Superior contained temperate prophage depending on viral burst sizes that were assumed. Three times more bacteria in the surface microlayer may contain temperate viruses compared to bacterioplankton in subsurface waters. In the western arm of Lake Superior, bacterioplankton infected by temperate phage may be more important for the survival of bacteriophage populations than as future carbon sources for new microbial production.

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Acknowledgements

May we all have a happy, productive, and peaceful 2002!

Contact Steve Abedon (microdude+@osu.edu) with suggestions, criticisms, comments, or anything else that might help make this a better site.