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LETTER

Large, double-stranded DNA viruses tend to suppress phytoplankton populations more effectively than small viruses of diverse genome type

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Scientific Significance Statement

Phytoplankton can suffer significant mortality from viral infection, with implications for community structure, trait evolution, primary productivity, and the carbon cycle. Phytoplankton viruses exhibit a wide range of sizes, but it is unclear whether the size of a virus determines how strongly it alters its host population. In this study, we compared diverse viruses using laboratory experiments, and we found that the strongest suppression of host populations was caused by larger viruses.

Abstract

Viruses infecting aquatic microbes vary immensely in size, but the ecological consequences of virus size are poorly understood. Here we used a unique suite of diverse phytoplankton strains and their viruses, all isolated from waters around Hawai'i, to assess whether virus size affects the suppression of host populations. We found that small viruses of diverse genome type (3–24 kb genome size, 23–70 nm capsid diameter) have very similar effects on host populations, suppressing hosts less strongly and for a shorter period of time compared to large double-stranded DNA viruses (214–1380 kb, 112–386 nm). Suppressive effects of larger viruses were more heterogeneous, but most isolates reduced host populations by many orders of magnitude, without recovery over the ~ 25-d experiments. Our results suggest that disparate lineages of viruses may have ecological consequences that are predictable in part based on size, and that ecosystem impacts of viral infection may vary with the size structure of the viral community.

Viruses are ubiquitous infectious agents that influence organismal health, the ecology and evolution of populations, and biogeochemical dynamics across domains of life and ecosystem types (Suttle 2007; Lefeuvre et al. 2019; Rowan-Nash et al. 2019). A primary axis of diversity among viruses is size: virus capsids vary ~ 100-fold in length, from 17 nm to ~ 1.5 μm, while virus genomes vary ~ 1,000-fold in size, from ~ 1 kb to 2.5 Mb (Campillo-Balderas et al. 2015). Genome size is strongly correlated with virion volume (Cui et al. 2014), and genome type is also correlated with both of these traits, with RNA and single-stranded DNA (ssDNA) viruses restricted to smaller sizes (< 50 kb, and generally much smaller than that), while double-stranded DNA (dsDNA) viruses occur across the whole viral size spectrum (Cui et al. 2014;

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Campillo-Balderas et al. 2015). Virus discovery, accelerated by metagenomics, has documented a vast diversity of viruses across sizes, such as smaller RNA viruses (Steward et al. 2013; Greninger 2018; Dominguez-Huerta et al. 2022) and ssDNA viruses (Labonté and Suttle 2013; Malathi and Renuka Devi 2019) as well as "giant" dsDNA viruses (Schulz et al. 2020; Gaïa et al. 2023). The size of a virus may have a large effect on traits affecting viral fitness (Edwards et al. 2021; Hinson et al. 2023), and although virion size and genome size are strongly correlated, they have overlapping yet partially distinct functional implications. For example, the rate at which hosts are encountered via molecular diffusion should decline steeply with increasing virion size (Murray and Jackson 1992), regardless of genome size. In contrast, the advantages of larger size may derive primarily from additional genes that can facilitate attachment/entry into host cells, replication, countering host defenses, and virion survival. Comparative analysis shows that larger phytoplankton viruses tend to have smaller burst sizes, an expected cost of both larger virion size and larger genome size, but this penalty is mitigated by more effective production of virion mass during infection, which is likely driven by a greater number of genes for manipulating cellular processes (Edwards et al. 2021). There is also evidence that larger bacterioviruses tend to have broader host ranges, another fitness benefit that likely derives from more viral genes (Edwards et al. 2021). Although comparative analyses have started to uncover how virus size affects important virus traits, we do not know whether viruses of different sizes tend to have different effects on their hosts. For example, the tradeoffs associated with size could cause larger and smaller viruses to differ on average in lethality for the host organism, suppression of host population density, or success in antagonistic coevolution.

One ecosystem where diverse viruses exert a persistent, widespread, and consequential influence on population dynamics is the pelagic ocean (Suttle 2007), where unicellular phytoplankton perform nearly half of global primary production (Huang et al. 2021), and phytoplankton and other microbes drive biogeochemical cycling (Hutchins and Fu 2017). Viruses can be a major source of mortality for unicellular marine plankton (Proctor and Fuhrman 1990; Mojica et al. 2016), and selection to resist infection can be a source of rapid (co)evolution (Marston et al. 2012), as can gene transfer mediated by viral infection (Touchon et al. 2017). Both eukaryotic and prokaryotic microbes can be infected by viruses differing greatly in size. For example, viruses infecting the bacterium *Cellulophaga baltica* include ssDNA viruses with 6 kb genomes and dsDNA viruses ranging from 29 to > 242 kb in genome size (Šulčius and Holmfeldt 2016). Viruses infecting the microalgal genus *Tetraselmis* include a 9-kb single-stranded RNA(+) (ssRNA(+)) virus (Schvarcz 2018), a 31-kb dsDNA virus (Pagarete et al. 2015), and a 668-kb dsDNA virus (Schvarcz and Steward 2018). Viruses of disparate size infecting the same host taxon raise the question of whether these viruses coexist by exploiting distinct niches, and whether they play distinct roles in eco-evolutionary processes.

In this study, we use diverse virus isolates from one ocean region—waters around Hawai'i—to test experimentally whether virus size is correlated with effects on host populations. The hosts are 12 strains of flagellated phytoplankton, ~ 3–10 μm in length, from a variety of taxa (Cryptophyceae, Chlorophyta, Haptophyta, Dictyochophyceae, and Chlorarachnea). This relatively high phylogenetic diversity is typical of small eukaryotic phytoplankton that play a large role in most photic zone environments (Pierella Karlusich et al. 2020; Rii et al. 2022). The viruses in this study include 11 isolates encompassing much of the known range of viral size, with genome sizes 2.6–1380 kb and capsid diameters 23–386 nm. These viruses also represent considerable taxonomic diversity, including dsDNA, double-stranded RNA (dsRNA), ssDNA, and ssRNA(+) genome types. By utilizing this unique suite of viruses and hosts, which includes multiple instances of smaller and larger viruses infecting the same host taxon, we ask whether virus size affects how viruses suppress their host populations over 3–4 weeks, corresponding to ~ 25–50 host generations.

Materials and methods

Experimental virus–host combinations

This study utilizes 11 virus strains (Table 1) and 12 phytoplankton host strains (Table 2). Methods for isolation and characterization of the phytoplankton and virus strains were described previously (Schvarcz 2018: Chapter 2, Appendices A and B), and are summarized in Supporting Information Methods. For brevity and comparative purposes we will refer to six virus strains as "large" and five as "small." The large viruses all have dsDNA genomes, including five representatives from four giant virus families (Schizomimiviridae, Allomimiviridae, Mesomimiviridae, and Mimiviridae) in the Imitervirales order, ranging 488–1380 kb in genome size and 193–386 nm in capsid diameter. The 6th large virus (CryptoV) is the smallest in the group (214 kb genome size and 112 nm capsid diameter), with no clear sequence homology to known viruses. In contrast to the large viruses, the small viruses possess a variety of genome types (ssRNA(+), dsRNA, ssDNA, and dsDNA), and have genome sizes 2.6–24 kb and capsid diameters 23–70 nm. Taxonomically they include a dicistrovirus, a birnavirus, a virus distantly related to geminiviruses, one virus without clear sequence homology to known viruses, and one virus whose genome is not yet sequenced.

We used the large and small viruses to ask whether there are average differences in how large and small viruses affect host populations. In some cases, our culture collection included 2–3 host strains that could be infected by the same virus, and we used multiple hosts in the experiment to assess whether virus effects were consistent across hosts. These

Table 1. Virus strains used in the host suppression experiments. Strain name abbreviation is listed in parentheses after the full strain name. Genome size is in kb and capsid diameter in nm. "Undescribed" under virus family or host genus indicates that the strain is from a new family or genus that has not yet been taxonomically described. Virus family, genome type, and genome size are "unknown" for TetCR05V because that strain was only characterized via TEM. Genome size for CBV is listed as 3 + 3 because the genome is bisegmented.

Virus strain name	Virus family	Host genus	Host class	Genome type	Genome size (kb)	Capsid diam. (nm)
Florenciella sp. virus SA1 (FloV-SA1)	Schizomimiviridae	*Florenciella*	Dictyochophyceae	dsDNA	1380	386
Tetraselmis virus 1 (TetV-1)	Allomimiviridae	*Tetraselmis*	Chlorodendrophyceae	dsDNA	668	385
Chlorarachniophyte sp. virus SA1 (ChlorV-SA1)	Mimiviridae	Undescribed	Chlorarachniophyceae	dsDNA	515	273
Rhizochromulina sp. virus SA1 (RhiV-SA1)	Mesomimiviridae	*Rhizochromulina*	Dictyochophyceae	dsDNA	512	193
Florenciella sp. virus SA2 (FloV-SA2)	Mesomimiviridae	*Florenciella*	Dictyochophyceae	dsDNA	488	217
Cryptovirus (CryptoV)	Undescribed	*Proteomonas*	Cryptophyceae	dsDNA	214	112
Chlorarachniophyte clade Y virus (ChlorYV)	Undescribed	Undescribed	Chlorarachniophyceae	dsDNA	24	54
Tetraselmis sp. RNA virus 01 (TetRNAV01)	Dicistroviridae	*Tetraselmis*	Chlorodendrophyceae	ssRNA(+)	9	36
Cryptophyte birnavirus (CBV)	Birnaviridae	*Proteomonas*	Cryptophyceae	dsRNA	3 + 3	70
Pavlova sp. gemini-like virus (PsGLV)	Gemini like	*Pavlova*	Pavlophyceae	ssDNA	2.6	23
Tetraselmis sp. KB-CR05 virus (TetCR05V)	Unknown	*Tetraselmis*	Chlorodendrophyceae	Unknown	Unknown	58

Table 2. Host–virus combinations used in the host suppression experiments. Each row lists one host–virus strain combination, and hosts combined with multiple viruses occupy multiple adjacent rows. Genera "chlorarachniophyte X" and "chlorarachniophyte Y" indicate that these strains fall into genera not yet taxonomically described. Genome size for CBV is listed as 3 + 3 because the genome is bisegmented.

Host genus	Host strain	Virus strain	Virus family	Genome type	Genome size (kb)
Florenciella	UHM3020	FloV-SA1	Schizomimiviridae	dsDNA	1380
		FloV-SA2	Mesomimiviridae	dsDNA	488
	UHM3011	FloV-SA1	Schizomimiviridae	dsDNA	1380
		FloV-SA2	Mesomimiviridae	dsDNA	488
Tetraselmis	UHM1315	TetV-1	Allomimiviridae	dsDNA	668
		TetRNAV01	Dicistroviridae	ssRNA(+)	9
	UHM1310	TetV-1	Allomimiviridae	dsDNA	668
		TetCR05V	Unknown	Unknown	Unknown
Proteomonas	UHM4300	CryptoV	Undescribed	dsDNA	214
		CBV	Birnaviridae	dsRNA	3 + 3
Chlorarachniophyte clade X	UHM2000	ChlorV-SA1	Mimiviridae	dsDNA	515
Bigelowiella	UHM2020	ChlorV-SA1	Mimiviridae	dsDNA	515
Chlorarachniophyte clade Y	UHM2010	ChlorYV	Undescribed	dsDNA	24
	UHM2011	ChlorYV	Undescribed	dsDNA	24
Rhizochromulina	UHM3074	RhiV-SA1	Mesomimiviridae	dsDNA	512
Pavlova	UHM4200	PsGLV	Gemini-like	ssDNA	2.6

included two *Florenciella* strains infected by both FloV-SA1 and FloV-SA2 (UHM3011 and UHM3020, with 96% nucleotide identity over near-complete *18S rRNA* gene sequences); two *Tetraselmis* strains infected by TetV-1 (UHM1310 and UHM1315, 99.4% near-complete *18S rRNA* sequence identity); two chlorarachniophyte strains infected by ChlorV-SA1 (UHM2000 and UHM2020, 90% near-complete *18S rRNA* sequence identity); and two chlorarachniophyte strains infected by ChlorYV (UHM2010 and UHM2011, 98.9% near-complete *18S rRNA* sequence identity; Table 2). Furthermore, in some cases one host strain could be infected by multiple virus strains, allowing us to compare effects of different viruses on that host: viruses FloV-SA1 and FloV-SA2 both infect *Florenciella* strains UHM3020 and UHM3011; viruses TetV-1 and TetRNAV01 both infect *Tetraselmis* strain UHM1315; viruses TetV-1 and TetCR05V both infect *Tetraselmis* strain UHM1310; and viruses CryptoV and CBV both infect *Proteomonas* strain UHM4300 (Table 2).

Culturing methods

Phytoplankton stock cultures were passaged monthly in K or f/2 media (depending on the strain), at 25°C and 100 μmol photons m^{-2} s^{-1} irradiance (12 h : 12 h light : dark cycle). Virus stocks were maintained by infecting host cultures in exponential phase monthly and storing lysates at 4°C. Host concentrations in source cultures were counted with flow cytometry after incubating with glutaraldehyde for 15 min (final concentration 0.25%), flash freezing in liquid nitrogen, and storage at −80°C. Infectious virus concentrations in source stocks were estimated using most-probable-number assays, with host cultures in 96-well plates. Virus stocks were diluted with a series of tenfold dilutions (up to 10^9-fold total), with six replicate wells per dilution. In each well, 10 μL of (diluted) virus stock was combined with 5 μL host culture and 195 μL fresh medium and monitored for lysis/growth via in vivo chlorophyll *a* (Chl *a*) fluorescence (Tecan Spark microplate reader). Infectious particle concentration was estimated using the R package MPN (Ferguson and Ihrie 2019).

To compare host responses to viral infection, we inoculated duplicate cultures of all host–virus combinations (Table 2) at $\sim 10^4$ cells mL^{-1} and added infectious virus particles at $\sim 10^2$ virions mL^{-1}. The total culture volume was 100 mL, in 250-mL Erlenmeyer flasks, yielding a total host population of $\sim 10^6$ and a total infectious virion population of $\sim 10^4$. Duplicate host cultures without added virus were inoculated at the same time. To monitor host populations, samples for flow cytometry and bulk in vivo Chl *a* fluorescence were taken every 2–3 d. Populations were monitored for 16–28 d, being terminated either when the host populations without added virus started to decline from their stationary density or when the host populations with added virus recovered to a density similar to the density of the no-virus controls.

Resistance assays

Some host populations exhibited a pronounced recovery after the initial virus-driven decline, and we tested whether these populations had acquired resistance to further lysis by the corresponding virus. A new 20-mL stock culture was inoculated with 200 μL of recovered host population ("recovered population"). After the stock culture had grown up (2 weeks) it was used to inoculate cultures in 24-well plates to test for susceptibility to lysis and was compared to the original host culture that was never exposed to viral infection ("original population"). We created six replicates of each of the four experimental combinations: (1) original population, no virus added, (2) original population, virus added, (3) recovered population, no virus added, (4) recovered population, virus added. All host populations were inoculated with 100 μL into 1.9 mL of growth medium, and virus additions included 1 μL of virus stock lysate. Cultures were monitored for lysis by sampling in vivo Chl *a* fluorescence every 2–3 d for 9–12 d.

Inducible resistance assays

We used a subset of host–virus combinations to ask whether resistance to viral infection could be induced by a low-molecular-weight chemical signal present in lysed culture. An exponentially growing host culture (2 mL in 24-well plates) was infected with 1 μL of virus stock lysate and monitored for lysis via Chl *a* fluorescence. Immediately after lysis, the lysate was filtered through a GF/F filter (syringe filtration) then a 30-kDa Amicon Ultra centrifugal filter (5000 × *g* for 10 min) to create virus-free lysate (VFL). Host cultures were inoculated in 24-well plates (100 μL into 1.9 mL medium), with four replicates each of four treatments: (1) incubation with VFL, followed by addition of virus lysate; (2) incubation with VFL, no lysate added; (3) no incubation with VFL, lysate added; (4) no incubation with VFL, no lysate added. For the VFL incubations, 100 μL of VFL was added to host cultures for 24 h before the addition of virus lysate (1 μL). The well plates were monitored every 2–3 d for population growth/lysis using in vivo Chl *a* fluorescence.

Statistical methods

To test for differences in host population suppression between small and large viruses, we averaged host concentrations over replicates and timesteps and fit a mixed model with syntax $\log_{10}$(cell concentration) ~ treatment + (1|host) + (1|virus), where "treatment" is control/large virus/small virus, (1|host) is a random effect capturing differences in mean concentration across host strains, and (1|virus) is a random effect capturing differences in mean concentration across virus strains (some viruses were used with multiple host strains). The magnitude of residual variation was allowed to vary between treatments and host strains. We also fit this model to four subsets of data: samples from week 1, week 2, week 3, and the final sample in each time series. Models were fit in R Version 4.3.2 with package glmmTMB (Brooks et al. 2017), and contrasts between treatments were

estimated with package emmeans (Lenth 2023). Data and metadata are deposited on figshare (Edwards et al. 2025).

Results and discussion

Host population decline and recovery was remarkably similar across the small virus treatments—an initial decline over 5–14 d to a density of $\sim 10^3$ cells mL^{-1}, followed by a relatively quick recovery, reaching a concentration comparable to the host population with no added virus (Fig. 1). In contrast, most (4/6) of the large viruses caused a greater decline of the host population, reaching lower concentrations of only 1–100 cells mL^{-1}, and either no recovery or slow recovery over ~ 4 weeks. On average the large viruses suppressed host populations to a significantly lower level when averaging over the whole experiment ($p = 0.0008$), but the difference between these treatments increased over time (Fig. 2), with no difference within the 1st week ($p = 0.51$), a tenfold difference in the 2nd week ($p = 0.034$), a 100-fold difference in the 3rd week ($p = 0.0012$), and a 500-fold difference at the termination of the experiment ($p = 0.0001$).

In cases where the host populations recovered to near-control abundances, we found that the recovered host populations were resistant to further lysis by the virus (Supporting Information Fig. S1). It is important to note that infectious virions and susceptible cells may have still been present in the recovered populations, but the population did not appreciably decline after inoculation with viral lysate,

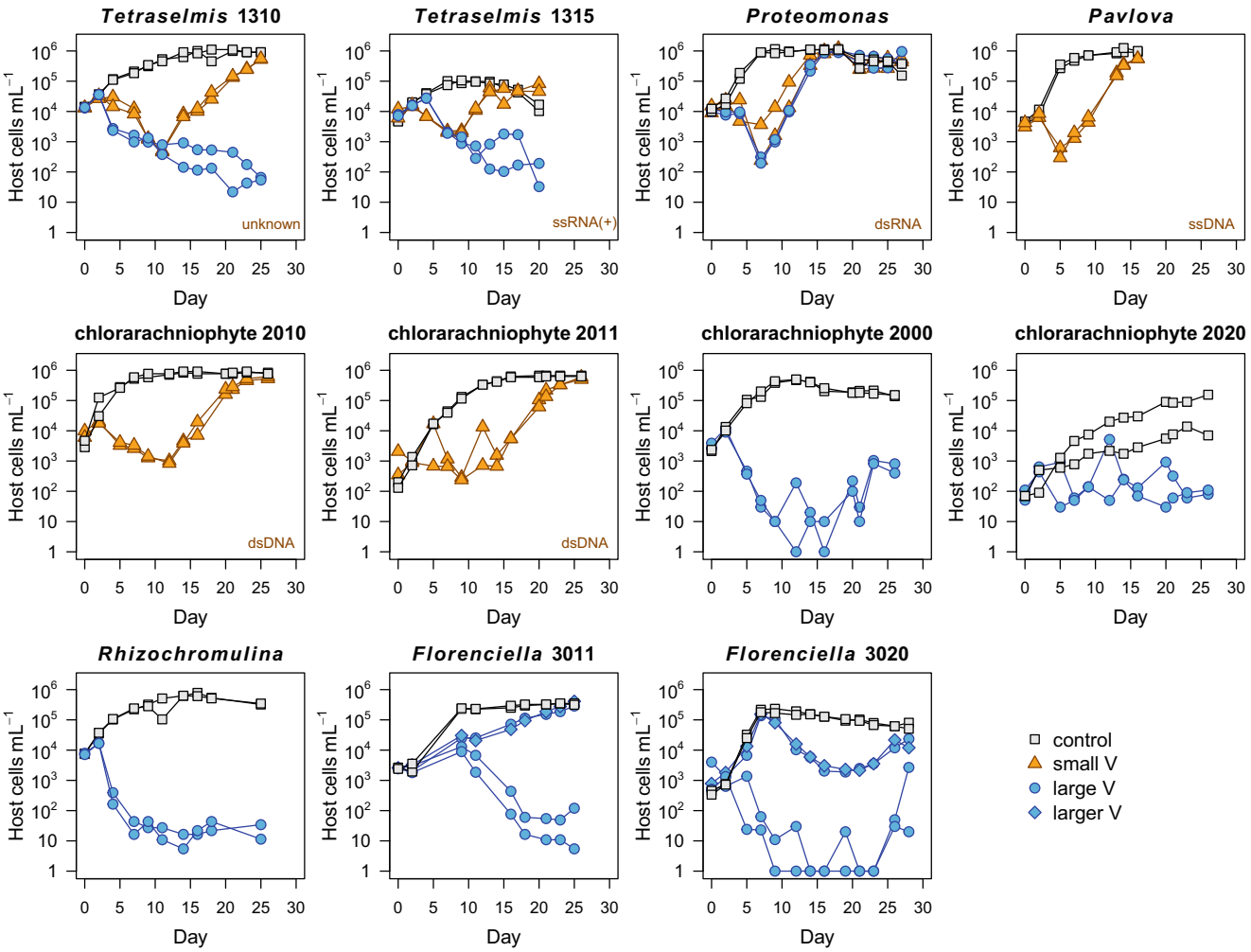

Fig. 1. Population trajectories of hosts infected with a small virus, a large virus, or no virus (control). Each panel displays one host strain. Host strains from genera with multiple representatives include the UHM strain number in the panel title. Some hosts were infected with both a large and a small virus, in separate treatments (the 1st three panels). Some viruses were combined with more than one host strain (large virus TetV-1 infecting *Tetraselmis* 1310 and *Tetraselmis* 1315; small virus ChlorYV infecting chlorarachniophyte 2010 and chlorarachniophyte 2011; large virus ChlorV-SA1 infecting chlorarachniophyte 2000 and chlorarachniophyte 2020; the two large viruses FloV-SA1 and FloV-SA2 infecting both *Florenciella* 3011 and *Florenciella* 3020). For host strains infected with a small virus, the text in the lower right indicates the genome type of the virus; all large viruses have dsDNA genomes. The two *Florenciella* panels each display two distinct large viruses, identified by circles vs. diamond symbols. All treatments were replicated twice.

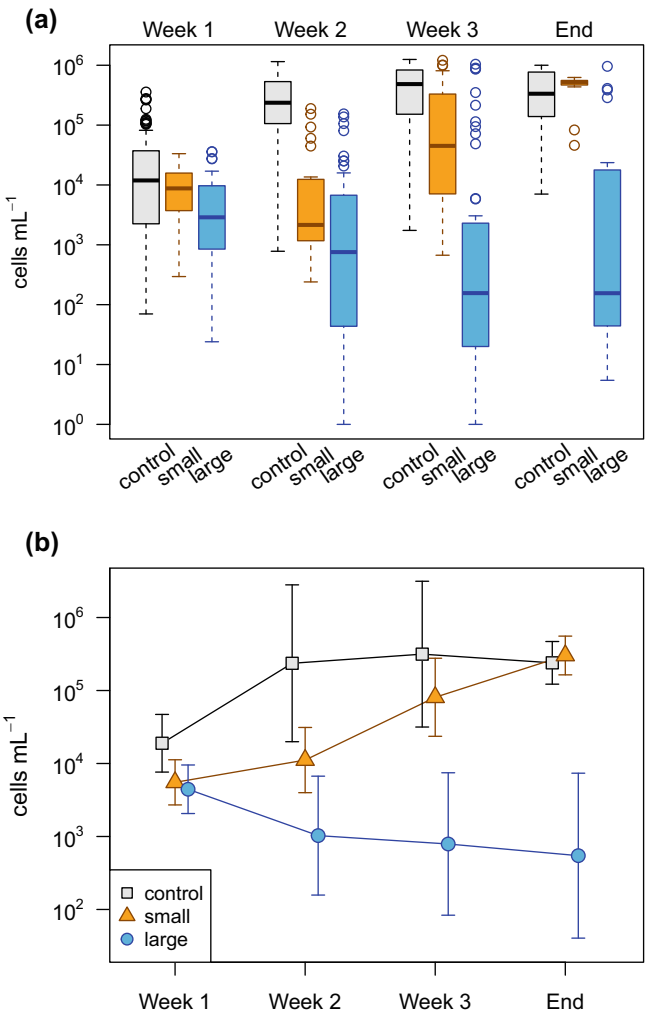

Fig. 2. (**a**) Boxplots of host concentrations in control, small virus, and large virus treaments over time. (**b**) Model-fitted host concentrations in control, small virus, and large virus treatments over time. Points are treatment means and errors bars are 95% CI from mixed models fit to subsets of the data—week 1, week 2, week 3, and the final sample in each trajectory ("End").

indicating the vast majority of cells could not be lysed. Because the suppressive effects of many viruses disappeared within 2–3 weeks, we selected three viruses (CryptoV and CBV, both infecting *Proteomonas* sp., and TetRNAV01, infecting *Tetraselmis* sp.) to assess whether resistance to infection could be induced by substance(s) released during infection and lysis. For all three cases, incubation of the host population with VFL for 24 h before addition of viral inoculum did not prevent or mitigate subsequent population decline (Supporting Information Fig. S2).

The hosts and viruses used in this experiment are diverse, including distant clades of the tree of life and a wide range of virus taxa and genome types. The consistent effects on host populations exhibited by small viruses suggest that small size leads to similar host–virus eco-evolutionary dynamics for eukaryotic phytoplankton, even though genome types and replication mechanisms of the viruses are disparate. Likewise, generally stronger suppression by large dsDNA viruses suggests that this functional group may, on average, have larger effects on host populations, even though this group also encompasses vast diversity (Schulz et al. 2020; Gaïa et al. 2023). In general, the largest viruses infecting either eukaryotes or prokaryotes are dsDNA viruses, while smaller viruses exhibit diverse nucleic acid types and replication mechanisms (Campillo-Balderas et al. 2015). This means the correlation between size and nucleic acid type in the viruses studied here is reflective of larger scale patterns of viral diversity, but it also means that effects of size per se are difficult to disentangle from effects of nucleic acid type and replication mechanism. It is interesting to note that the four viruses with the strongest suppressive effects in this experiment have a fairly narrow range of genome sizes (448–668 kb), and capsid diameters from 217 to 385 nm (Fig. 3). The largest virus, FloV-SA1, has a 1380-kb genome and 386-nm diameter capsid, but did not strongly suppress the two *Florenciella* strains used in the experiment (Fig. 3). It is possible that a very large genome size leads to significant costs during replication, while the persistence of the largest viruses may be supported by advantages in other traits, such as broader host range. However, this virus may also be unusual, and we cannot generalize about tradeoffs from experiments with only one isolate of this size. Although the viruses in this study are diverse in size and taxonomy, the level of replication is modest (11 viruses total), and future work should aim to test the robustness of the trends we have observed. Future work should also aim to disentangle the functional roles of virion size and genome size—although these traits are correlated, there is nonetheless some variation in capsid size among viruses of the same genome size, and vice versa (Cui et al. 2014; Edwards et al. 2021), implying there is some room for independent selection on these traits.

The population dynamics caused by small vs. large viruses suggest that host resistance to infection could be the most ecologically consequential difference between viruses of different sizes. The speed at which host populations recovered from initial decline caused by small viruses suggests that host populations have effective defense mechanisms that can be induced by infection, or that the populations maintain resistant genotypes at high enough frequencies to take over the population within 1–2 weeks. If defense is induced, it may be induced solely by the virus, or by other signals > 30 kDa in size, or it may require a high signal concentration, because we were unable to induce population-level resistance with < 30-kDa VFL added at a ratio of 1 : 20 for the three viruses tested. Evaluating mechanisms underlying small virus–host eco-evolutionary dynamics is an important future direction.

Most large virus treatments caused suppression of the host population by many orders of magnitude, with no or slow recovery over 3–4 weeks, corresponding to ~25–50 host

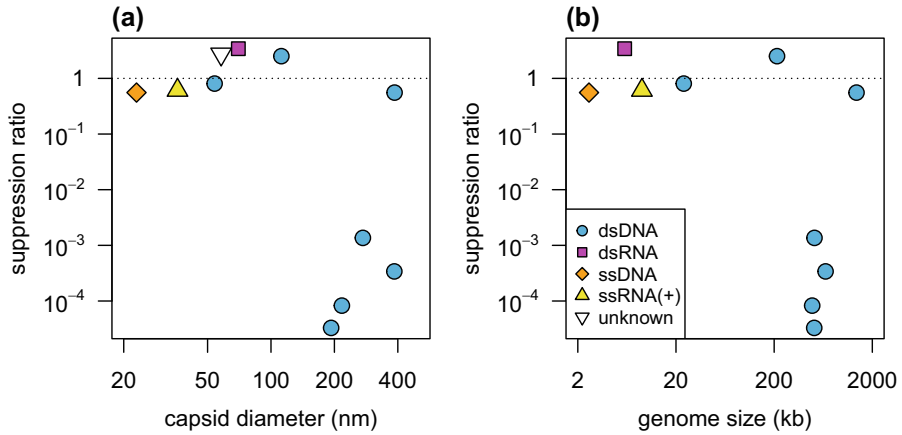

Fig. 3. Comparison of virus size and suppression of host populations. In both panels, the *y*-axis is the suppression ratio, defined as (host population density in the presence of virus)/(host population density in the absence of virus), measured at the end of the experiment. For viruses that were paired with multiple host strains, the geometric mean of the suppression ratios is shown. (**a**) Suppression vs. virion capsid diameter, (**b**) suppression vs. virus genome size. Panel (**b**) contains one fewer point because the genome size of TetCR05V (capsid diameter 58 nm) is unknown.

generations. These dynamics are consistent with longer-term studies investigating phytoplankton–virus coevolution. Marston et al. (2012) infected the cyanobacterium *Synechococcus* with a relatively large myovirus (RIM8, 173 kb genome), which suppressed the host by ~ 100-fold for 30–60 d, before resistant genotypes were able to recover to a density similar to no-virus controls. Frickel et al. (2016) infected the green alga *Chlorella* with a relatively large chlorovirus (PBCV-1, 331 kb), and observed cycles of 1000-fold decline over 30–40 d, before resistant genotypes recovered to densities that were 5–10 times lower than virus-free controls. Thyrhaug et al. (2003) also observed coexistence of the haptophyte *Phaeocystis* and a large phycodnavirus (PpV-01, 485 kb) for 1 year in batch culture, with long-term suppression of the host population by 10- to 100-fold, compared to stationary density of the host without virus. These outcomes provide further evidence for strong impacts of large viruses on host populations. They also suggest that the hosts in our study may eventually evolve resistance and increase in density, although growth rate and long-term density could be substantially reduced compared to the virus-free population. Quantifying in situ effects of specific viral populations is more challenging, but viruses implicated in large phytoplankton mortality events are generally large dsDNA viruses (e.g., Tarutani et al. 2000; Schroeder et al. 2003; Gastrich et al. 2004), although smaller RNA viruses infecting diatoms may also be significant sources of mortality (Kranzler et al. 2019). Testing the generality of our results will also benefit from future comparative experiments utilizing additional phytoplankton taxa. We have investigated viral effects on diverse flagellates, but phytoplankton communities are often dominated by non-motile diatoms (in very productive environments) or non-motile cyanobacteria and chlorophytes (in oligotrophic environments). The aforementioned coevolution studies with *Synechococcus* and *Chlorella* suggest that strong effects of large viruses are also important for these taxa, but the relative effects of smaller viruses are unknown. It is noteworthy that all known diatom viruses are ssRNA or ssDNA viruses < 10 kb in genome size (Arsenieff et al. 2022). If the patterns observed here translate to diatom viruses it could mean that diatoms are less strongly regulated by viruses compared to other phytoplankton, perhaps contributing to their dominance under productive conditions when higher host density and growth rates should otherwise enhance viral success. In general, a better understanding of virus size and natural phytoplankton mortality may benefit from an increased focus on impacts of RNA viruses, which are diverse (Dominguez-Huerta et al. 2022), can be abundant (Steward et al. 2013), and may often be cryptically present in algal cultures (Charona et al. 2022), but which have received relatively less attention in marine systems (Sadeghi et al. 2021).

Author Contributions

Kyle F. Edwards, Grieg F. Steward, and Christopher R. Schvarcz conceived the study. Christopher R. Schvarcz provided isolates and Kelsey A. McBeain performed the experiments. Kyle F. Edwards performed data analysis. All authors contributed to writing the manuscript.

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Supporting Information

Additional Supporting Information may be found in the online version of this article.

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