Systematic Review and Meta-Analysis of Decay Rates of Waterborne Mammalian Viruses and Coliphages in Surface Waters

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A manuscript for

Water Research

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Highlights

- Systematic review of virus decay rates (*k*) in surface waters identified 562 *k*
- Meta-analysis revealed *k* is different among viruses and coliphages
- *k* depended on temperature, light condition and enumeration method
- Limited data available for norovirus, hepatitis A and E, and astrovirus
- Common and novel indicators overpredict *k* of more persistent mammalian viruses

Abstract

Surface waters are essential natural resources. They are also receiving waters for a variety of anthropogenic waste streams that carry a myriad of pollutants including pathogens. Watershed and fate and transport models can help inform the spatial and temporal extent of microbial pollution from point and non-point sources and thus provide useful information for managing surface waters. Viruses are particularly important water-related pathogens because they often have a low infectious dose, which means that ingestion of even a small volume of water containing a low concentration of virions has the potential to cause disease. We conducted a systematic review of the literature, following best practices, to gather decay rate constants (k) of mammalian waterborne viruses (enteroviruses, adenoviruses, noroviruses, astroviruses, rotaviruses, and hepatitis A viruses) and coliphages in raw surface waters to aid in the parameterization of virus fate and transport models. We identified 562 k values from the literature, with the largest number identified for enteroviruses and coliphages and the smallest for astrovirus, hepatitis A virus, and norovirus. Average k values for each virus varied from 0.07 to 0.9 per day, in order from smallest to largest: *Norwalk virus* (i.e., noroviruses) < *Human astrovirus* < *Mastadenovirus* (i.e., adenoviruses) < *Hepatovirus A* (i.e., hepatitis A viruses) < *Rotavirus A* < coliphages < *Enterovirus*. A meta-analysis investigated how k varied among viruses for experiments conducted with different virus serotypes or species at different temperatures, salinities, and sunlight exposures, and for experiments that enumerated viruses using different methodologies. Virus species or serotype did not affect k among decay experiments. k values were generally larger for experiments conducted at higher temperatures, in sunlight, and in estuarine waters, and enumerated using culture methods. k values were statistically different between virus types with *Norwalk virus*, *Hepatovirus A*, and

Mastadenovirus having smaller *k* values than other viruses, controlling for experimental condition and enumeration method. While F+ coliphage *k* values were similar to those of *Enterovirus*, *Human astrovirus*, and *Rotavirus A*, they were different from those of the other mammalian viruses. This compilation of coliphage and mammalian virus *k* values provides essential information for researchers and risk assessors who model virus fate and transport in surface waters and identifies avenues for future research to fill knowledge gaps.

Keywords: viruses, coliphage, surface water, inactivation, rate constant, modeling

1. Introduction

Surface waters are essential drinking water sources, recreation sites, and animal and plant habitat. They are also receiving waters for a variety of anthropogenic waste streams that carry a myriad of pollutants including pathogens. In the USA, 67% of the public water supply and 58% of irrigation water comes from surface waters (USGS, 2005). Globally, 159 million people are dependent solely on surface water for their drinking water source (WHO, 2017). As of 2019, between 54% and 98% of assessed surface water bodies in the United States (depending on water body type and how the type was assessed - e.g. by area or length of coastline) were listed on the Clean Water Act 303(d) list due to pathogen pollution (USEPA, 2019). Swimming in pathogen contaminated surface waters is estimated to cause 90 million illnesses per year in the US with associated costs of $2.2 to $3.7 billion per year (DeFlorio-Barker et al., 2018).

Pathogens enter surface waters via raw and treated sewage inputs, open defecation, land-based runoff, and bather shedding. Once they enter surface waters, they are advected and dispersed by ambient currents, and subject to non-conservative processes including settling, predation, and inactivation (Hipsey et al., 2008; Nevers and Boehm, 2010; Thomann and Mueller, 1987). Inactivation is generally modeled as first-order decay with respect to pathogen concentration, although biphasic or delayed decay profiles have been observed under certain conditions (Boehm et al., 2018, 2012; Brooks and Field, 2016; Murphy, 2017).

Modeling pathogen fate and transport in surface waters can inform pathogen remediation efforts by aiding in the identification of contaminant sources (Dorner et al., 2006), providing an early warning of health-relevant pathogen concentrations (Liu et al., 2006), estimating risk at points of

contact based on the pathogen load measured elsewhere (Boehm et al., 2018; Derx et al., 2016), and allowing examination of the effects of hypothetical contamination events on pathogen concentrations in the environment (Mohammed et al., 2019). First-order decay rate constants are essential inputs to pathogen fate and transport models. Although there are numerous studies of pathogen inactivation in surface waters, there is a need to synthesize the results of these studies and identify data gaps. Boehm et al. (2018) recently conducted a systematic review to compile first-order decay rate constants (k) in surface waters of reference bacterial, protozoan, and viral pathogens commonly used in quantitative microbial risk assessment models. That study compiled k values for *Salmonella spp.*, *Campylobacter spp.*, *Escherichia coli* O157:H7, *Cryptosporidium spp., Giardia spp.*, and *Caliciviridae* from experiments conducted in raw surface waters. The goal of the present study was to specifically focus on viruses, and compile k values from the literature for water-associated mammalian viruses and coliphages that are often used as virus surrogates. This work is essential because viruses are typically more infectious than other waterborne pathogens (Haas et al., 1995) and are important causes of waterborne gastrointestinal illness (Kotloff et al., 2013; Scallan et al., 2011; Sinclair et al., 2009). The compiled values represent a resource for those modeling pathogen fate and transport in surface waters. Further, we conducted a meta-analysis to explore how experimental conditions and viral enumeration methods affect k values, and whether k values are distinct among viral genera and species. The systematic review and meta-analysis provide insight into data gaps in the study of viral inactivation in the environment, and best practices for conducting such experiments and associated meta-data reporting.

2. Materials and Methods.

106 **2.1 Systematic review**

107 The systematic review and meta-analysis followed PRISMA guidelines (Moher et al., 2009). The
108 goal of the review was to compile from the peer-reviewed literature quantitative information on
109 the decay of waterborne human viruses, their commonly used viral surrogates and coliphages in
110 surface waters under environmentally-relevant conditions. Pathogens included in the review
111 were human noroviruses (including their surrogate murine norovirus), adenoviruses, rotaviruses,
112 enteroviruses (including polioviruses, coxsackieviruses, and echoviruses), astroviruses, hepatitis
113 A viruses, and hepatitis E virus.

114

115 Web of Science core collection (search field = topic), Scopus (search field = article title, abstract,
116 keyword), and PubMed (search field = all fields) were searched in September 2018 (Table 1).
117 The search terms were "(X) AND (water OR seawater OR stormwater) AND (die-off OR
118 persistence OR survival OR inactivat* OR decay)" where X is the target-specific text (Table 1).
119 Identified articles were assembled and duplicates were removed. Details of the review process,
120 which involved two independent full-text reviews of papers, are provided in Boehm et al. (2018).
121 The inclusion criteria were that the paper: (1) contained quantitative data on the decay of the
122 target of interest in raw (unaltered) surface water, (2) was in English, (3) was not a review paper,
123 presented primary data, and was peer-reviewed, (4) did not contain data solely on disinfection
124 treatments such as addition of oxidants or SODIS, (5) included data from decay experiments
125 where the temperature was greater than or equal to 4°C and less than 30°C, and (6) described
126 methods to enumerate the target that are logical and justifiable.

127

Decay rate constants were extracted from papers by a single reviewer. First-order decay rate constants (k), in units per day (d^{-1}), calculated from natural log (ln)-transformed concentration data as used in Chick's law (Metcalf et al., 2003), were sought. If a study presented k values, then they were extracted from the paper along with any reported errors and model fit values (R^2 and/or root mean square error (RMSE)), and unit conversions were applied where appropriate. If a study reported decay parameters from a model that was not first-order (for example a shoulder log-linear model, or biphasic model), then we extracted those reported model parameters and any associated errors and model fit values. If a study only reported t_{90} or t_{99}, (i.e., time to 90% or 99% reduction in concentration, respectively), or other times for a specific amount of inactivation, then those times were converted to first-order decay rate constants assuming Chick's law applied. If no first-order decay rate constant was reported by the study authors, but data were available in graphs, then Plot Digitizer (http://plotdigitizer.sourceforge.net) was used to digitize the concentration time series appearing in graphs within the publication. To be clear, this included data from studies that only reported decay model parameters from other types of decay models (i.e., not first-order log-linear decay). k was then calculated as the regression slope of $\ln(C/C_o)$ versus time (in days) using linear least-squares regression in R. In this formulation C is the concentration at time t, and C_o is the concentration at the start of the experiment at t=0. k and its associated error, as well as model fit parameters, were recorded. In carrying out the linear regression, values reported at or below the detection limit were included if and only if they were not preceded by other consecutive values at or below the detection limit; the value directly reported by the author was used in these cases.

Once all data were compiled, datasets and model parameters were examined to assess whether a non-linear model was needed to describe decay. The goodness of the log-linear model fit to the data (R_2 and RMSE), and the number of data points that appeared to "deviate" from the log-linear model were considered. In general, if R_2 values were greater than 0.7 and RMSE was relatively small (~1 ln unit), only one data point visually deviated from a straight line fit between time and $\ln(C/C_o)$, or the non-log-linear model fit was no better than the log-linear fit, then a log-linear curve fit was deemed acceptable.

In addition to extracting information on the decay of the viral target, a record was kept as to whether the experiment was conducted in (1) freshwater, estuarine water, or seawater and (2) direct sunlight or the dark. If an experiment was reportedly carried out in sunlight, but at a depth in the water column greater than ~25 cm or in a container that was opaque to UVA and UVB light, then the experiment was categorized as carried out in the dark given the importance of these wavelengths for sunlight-mediated decay of viruses (Nelson et al., 2018). The temperature at which the experiment was conducted was also recorded. If a range of temperatures was provided, the mean of the reported range was used. Finally, the method used for virus enumeration was noted [i.e., culture, immunofluorescent methods, quantitative PCR (QPCR) or reverse-transcription QPCR (RT-QPCR), or ethidium monoazide (RT-)QPCR (EM-(RT)-QPCR)].

Fifteen percent of the papers from which data were extracted by a single reviewer were randomly chosen for a second round of data extraction by a different reviewer. Data extracted by the two

reviewers were compared to ensure consistency. A single reviewer conducted detailed review of all datasets to identify missing data, data outliers, and data entry mistakes.

2.2 Meta-analysis

Statistical distributions were fit to virus-specific *k* values. Goodness of fit was assessed by visual inspection of residual and Q-Q plots. This yielded satisfactory log-normal fits for all viruses with the number of *k* values n≥12. For congruity, log-normal distributions were also used when n<12 because there were too few values to justify a different distribution.

Linear models (equation 1) were used to model $\log_{10}k$ as a function of virus-species or -type (categorical; reference varies as described in results), water temperature (continuous; defined as reported temperature (T) minus 15°C), water matrix – fresh, estuarine, or marine (categorical with fresh being reference condition), sunlight (binary; dark is reference condition), and method used to enumerate the virus (categorical as culture, QPCR or RT-QPCR, EM-QPCR or EM-RT-QPCR, and immunological methods including ELISA or immunofluorescence microscopy, with culture being the reference condition):

$$log_{10}k = \beta_0 + \sum_{i=1}^{n} \beta_i x_i + \epsilon \quad (1)$$

where β_0 is the intercept and represents the model estimate for $\log_{10}k$ under reference conditions, β_i represents the coefficient for each of the model variables x_i (i.e., virus-species or -type dummy variables, T-15°C (where T = temperature), estuarine water matrix dummy, marine water matrix dummy, sunlight dummy, QPCR/RT-QPCR dummy, EM-QPCR or EM-RT-QPCR dummy, immunological method dummy), and ϵ is the error. For each of the dummy variables, x_i= 0 or 1. The temperature variable is the only continuous variable. Interaction terms (not

shown in Equation 1) were included in some instances, as described in the results. Post hoc Tukey contrasts, which adjust for multiple comparisons, were used to assess whether $\log_{10}k$ differed among viruses. Models for individual viruses as well as well as global model which combined data from all viruses were used, as described in more detail in the results section. Results with $p<0.05$ were considered statistically significant. Results where $0.05<p<0.1$ are also noted. All analyses were conducted in R using the 'lm' function.

3. Results

3.1 Systematic review

The study identified a total of 562 experiments describing decay of the target viruses in surface waters from a total of 73 unique papers (Table 2). Here, "experiment" is defined as an experiment-target combination. Therefore, if researchers carried out one experiment and enumerated two different targets relevant to our review, this counted as two experiments. The papers from which the 562 *k* values were extracted are provided in Tables 3 and 4.

Only 14 of 562 (2%) decay profiles were initially fit using a shoulder or biphasic decay model. However, the R_2 and RMSE values obtained from fitting extracted decay profiles with log-linear models indicated good fits. Therefore, first-order decay kinetics were assumed to apply to all experiments. As described in the methods, first-order decay constants (*k*) were calculated for experiments if they were not provided by the authors. The exceptions are the experiments from Wu et al. (2016) who did not report raw data for their F+ coliphage decay experiments and reported *k* values from their one-day delayed log-linear models.

Five of 562 *k* values (1%) were reported or calculated to be 0 (two adenovirus and three coliphages). Those values were replaced with 0.0008 d^{-1}, the lowest non-zero *k* value in the compiled data series, so that the data series of *k* values could be $\log_{10}$-transformed.

Half of the decay experiments were conducted in freshwater (281 of 562, 50%) with the remaining conducted in seawater (201 of 566, 36%) or estuarine water (80 of 566, 14%). Ninety-four (17%) experiments were carried out under the influence of sunlight; the rest (472, 83%) were carried out in the "dark" defined as experiments conducted in the dark or under conditions where UVA and UVB were likely not able to penetrate. Experiments were carried out at the full range of temperatures accepted for this review between 4°C and 29°C. Across all 562 experiments, *k* varied from 0 d^{-1} to 283 d^{-1} with a geometric mean of 0.63 d^{-1}.

Among the mammalian viruses, there were the greatest number of experiments for the enteroviruses: poliovirus, echovirus, and coxsackievirus (n=170, 38, and 50, respectively for a total of n=258 for enteroviruses). Of the poliovirus experiments, 114, 3, and 43 of the 170 experiments were conducted, respectively, with poliovirus 1, poliovirus 2, and poliovirus 3, and 10 experiments were conducted with unspecified poliovirus. Echovirus experiments were conducted using echovirus 1 (n=3), echovirus 6 (n=9), echovirus 7 (n=12), echovirus 12 (n=7), echovirus 32 (n=1), and unspecified echovirus (n=6). Coxsackievirus experiments were conducted using B1 (n=3), B2 (n=6), B3 (n=23), B5 (n=6), A9 (n=4), A13 (n=2), and unspecified coxsackievirus (n=6). The International Committee on Virus Taxonomy assigns all these viruses to the *Enterovirus* genus. All polioviruses and coxsackievirus A13 are assigned to the species *Enterovirus C*, and all echoviruses and other coxsackieviruses to *Enterovirus B*.

Nearly all enterovirus experiments (97%) reported enterovirus concentrations measured using cell culture. The remaining enterovirus experiments measured viruses using RT-QPCR (n=6) and ethidium monoazide (EM-)RT-QPCR (n=2).

There were 37 adenovirus decay experiments carried out using adenovirus 2 (n=11), adenovirus 5 (n=1), a recombinant adenovirus 5 (n=2), adenovirus 7 (n=1), adenovirus 40 (n=9), adenovirus 41 (n=7), and mixed assemblages of adenovirus obtained from wastewater (n=6). All the numbered adenoviruses are in the *Mastadenovirus* genus with adenovirus 2 and 5 classified to the *Human adenovirus C* species, adenovirus 7 to *Human adenovirus B,* and adenovirus 40 and 41 to *Human adenovirus F*. Most of the experiments quantified adenovirus using cell culture (n=23; 62%), while 32% used QPCR (n=12) and 5% used EM-QPCR (n=2).

There were 33 rotavirus decay experiments that were conducted with simian rotavirus SA11 (n=21), human rotavirus Wa (n=6), and bovine rotaviruses RF (n=1) and C486 (n=2), rhesus rotavirus RRV (n=2), and rotavirus-like particles (VLPs, n=1). All the rotaviruses belong to the same species *Rotavirus A,* aside for VLPs. Most experiments were conducted using cell culture (n=28; 85%). One experiment (3%) was conducted using RT-QPCR, and four (12%) with ELISA or immunofluorescent microscopy.

There were 12 hepatitis A virus experiments. Five were conducted using strain HM175, two with HM174, two with GBM, and three with an unspecified strain. All human hepatitis A viruses belong to the *Hepatovirus A* species. All experiments were completed using cell culture. We did not identify any hepatitis E virus experiments.

There were four astrovirus experiments. Two were carried out with astrovirus serotype 4, and two with serotype 8. Both these serotypes are in the *Mamastrovirus* genus and are the same species: *Human astrovirus*. Two experiments were carried out using an integrated cell-culture RT-QPCR assay, one using cell culture, and one using RT-QPCR.

There were 12 norovirus experiments. There were four *k* values for human norovirus, two of which were for norovirus GI and two for norovirus GII. All human norovirus experiments documented the decline in the number of copies of the gene located at the ORF1/ORF2 junction using RT-QPCR. There were eight *k* values available for murine norovirus, a culturable surrogate of human norovirus. Of these, five were measured using plaque assays (others used RT-QPCR). Human norovirus and murine norovirus are both in the *Norovirus* genus and are genotypes of the *Norwalk virus* species according to the International Committee on Viral Taxonomy (2018).

There were 206 coliphage experiments conducted using 28 coliphage species from seven different families (Table 5), as well as uncharacterized coliphage mixtures or isolates. These experiments were conducted using icosahedral (n=63), tailed (n=33), filamentous (n=3), and structurally uncharacterized (n=107) coliphages. Out of the total number of experiments, 104, 45, 15, and 42 were conducted using ssRNA, dsDNA, ssDNA, and genomically uncharacterized coliphages, respectively. 130 and 67 experiments were carried out using F+ and somatic coliphages, respectively, with the remaining 9 experiments insufficiently described to be placed in either of these categories. The majority of coliphage experiments (97%) were carried out using

288 either single or double agar layer methods with a bacterial host, with the remaining six (3%)
289 experiments conducted using RT-QPCR or QPCR.
290
291 Figure 1 shows a box and whisker plot illustrating the distribution of *k* values for all viral types.
292 It is important to note that these are empirical distributions of *k* values determined in experiments
293 carried out under diverse experimental conditions (including various temperatures, water
294 matrices, and sunlight irradiances) and potentially using diverse enumeration methods.
295
296 **3.2 Meta-analysis**
297 **3.2.1 Mammalian Virus-Specific Models.** We modeled $\log_{10}k$ values of each viral group as a
298 function of the following independent variables using multiple linear regression (Equation 1):
299 water temperature, water matrix, sunlight, method of viral enumeration, and virus species or
300 virus type.
301
302 Enterovirus species (*Enterovirus B* and *Enterovirus C*) was not significant in the enterovirus
303 model ($p>0.1$). Virus type was not significant in this model when the virus species variable was
304 replaced with a variable indicating whether the experiment was conducted using poliovirus,
305 coxsackievirus or echovirus ($p>0.1$). Adenovirus species was not significant in the adenovirus
306 model ($p>0.1$). All astrovirus, hepatitis A virus, norovirus, and rotavirus experiments were
307 carried out with viruses from the same species (*Human astrovirus*, *Hepatovirus A*, *Norwalk*
308 *virus*, and *Rotavirus A*, respectively) so the species variable was not relevant for those viruses.
309 For norovirus, we used a variable to indicate whether the experiment was conducted using
310 human or murine norovirus and that variable was not found to be significant in the $\log_{10}k$ model

311 ($p>0.1$). Given the lack of evidence that virus species or other relevant biological classifications
312 were associated with the decay rate constant values for mammalian viruses, we did not further
313 include those biological classification factors in the mammalian virus-specific models. This
314 allowed for inclusion of more experiments in the individual virus models as some experiments
315 were conducted using un-speciated viruses or mixtures of viral species. Hereafter, the
316 mammalian virus groups are referred to by the finest taxonomical classification of the viruses
317 they include: *Enterovirus*, *Mastadenovirus, Human astrovirus*, *Hepatovirus A*, *Norwalk virus*,
318 and *Rotavirus A.*
319
320 Model coefficients from the *Enterovirus*, *Mastadenovirus, Hepatovirus A*, *Norwalk virus*, and
321 *Rotavirus A* regression models are provided in Table 6. Given the very low number of *k* values
322 available for *Human astrovirus* (n=4), we did not create a model for it. The virus-specific models
323 included variables representing experimental condition and enumeration method. The reference
324 experimental condition is freshwater at 15°C in the dark with the virus enumerated using culture-
325 based methods. Therefore, the intercept (β_0) can be directly interpreted as $\log_{10}k$ under those
326 reference conditions. Under reference conditions, the model estimate for *k* in order from smallest
327 to largest is *Mastadenovirus<Norwalk virus <Hepatovirus A<Rotavirus A< Enterovirus.* Note
328 that this does not imply that *k* values are significantly different among each of these viruses, but
329 simply represents the ranking of the model intercepts. Note that this ranking differs from the
330 ranking of geometric mean *k* values of all those collected in the systematic review, which is
331 provided in the abstract because this ranking controls for the diverse conditions under which the
332 experiments were conducted.
333

The water temperature coefficient for each of the mammalian viruses was positive ($0.03>\beta_{temp}>0.08$; Table 6) indicating that $\log_{10}k$ increases by this amount for each one-degree increase in temperature, or that k increases by a factor of $10^{\beta_{temp}*(T-15)}$. The positive association between $\log_{10}k$ and temperature is visualized in Figure 2.

The three coefficients for the method dummy variables indicating whether the experiment enumerated viruses using methods other than culture methods were significant in some of the virus models. The model coefficient for the dummy variable indicating whether the virus was enumerated using QPCR or RT-QPCR (β_{meth1}) was statistically significant and negative for two viruses. This result indicates smaller k when these molecular methods were used compared to when culture methods were used. k was smaller by a factor of 6 or 9 ($10^{\beta_{meth1}}$) for *Enterovirus* and *Rotavirus A* when RT-QPCR was used to enumerate viruses. The model coefficient for the method dummy variable indicating that the experiment enumerated viruses by EM-RT-QPCR (β_{meth2}) was significant and negative in the *Enterovirus* model; its magnitude was similar to the value for β_{meth1}. These results are consistent with a visual examination of the $\log_{10}k$ values enumerated using the different methods (Figure 3).

A water matrix dummy variable coefficient was statistically significant and positive in the *Rotavirus A* and *Mastadenovirus* models, suggesting k in estuarine water was larger than k measured in freshwater by a factor of approximately 5 ($10^{\beta_{mat1}}$) for these two targets. However, neither water matrix dummy variable coefficients (β_{mat1} or β_{mat2}) were significant in the other virus models. These results are consistent with a visual inspection of the data distributions of $\log_{10}k$ measured in different water matrices (Figure 3).

The sunlight dummy variable coefficient was statistically significant in the *Enterovirus* and *Mastadenovirus* models. It was not significant in the *Rotavirus A* or *Norwalk virus* models, but the number of sunlight experiments conducted for these two viruses was small (n=2 and 1, respectively). Sunlight was not included as a factor in the other virus models as no experiments for those viruses were conducted under sunlit conditions. For both *Enterovirus* and *Mastadenovirus,* the model coefficient was positive indicating that k is larger by a factor of approximately 10 when sunlight was a parameter in the experiment. This is largely consistent with a visual inspection of the distribution of $\log_{10}k$ values observed under the different experimental conditions (Figure 3). Recall that experiments were classified as sunlit only if they were conducted under light exposure, and UVA and UVB were expected to penetrate into the experimental waters, given the importance of these sunlight regions for virus inactivation (Nelson et al., 2018).

3.2.2. Coliphages. Coliphage $\log_{10}k$ were modeled using the same technique as for the mammalian viruses, however, instead of coliphage species, we used a factor that indicated whether the coliphages were somatic or F+ coliphages. This was motivated by the fact that most applied research in surface waters differentiates between coliphage types using this classification. Coliphage characterization (F+ or somatic) was available for 197 of the 206 experiments; study authors did not provide enough information about their experiments to discern whether coliphages were F+ or somatic in the remaining 9 experiments. F+ or somatic factor was significant ($p<0.05$) in the regression model. We therefore separated the F+ and

somatic coliphage *k* values and created separate models for each to explore the importance of experimental conditions and method on *k* (Table 6).

The coliphage model results can be interpreted in much the same way as the mammalian virus models where the reference conditions for coliphages are the same as described above. Under the reference experimental conditions (freshwater at 15°C in the dark, measured by culture), F+ coliphage $\log_{10}k$ was larger than somatic $\log_{10}k$ as inferred from the model intercepts.

Model coefficients for temperature were positive and significant indicating larger $\log_{10}k$ at higher temperatures (Figure 2). Coefficient values were within the same range observed for the mammalian viruses (Table 6).

The model coefficient for the method dummy variable indicating that QPCR or RT-QPCR was used to enumerate coliphage was statistically significant in the F+ coliphage model with a negative coefficient indicating smaller *k* when F+ coliphages were enumerated using those molecular methods than culture methods by a factor of 5. This appears to be consistent with a visual examination of the data (Figure 3). No somatic coliphage experiments reported molecular-measured concentrations therefore the influence of this factor could not be discerned.

The model coefficients for the water matrix dummy variables were statistically significant and positive in both coliphage models (Table 6). This indicates that for both coliphage types, $\log_{10}k$ values measured in estuarine and marine waters were larger than the reference freshwater

401 condition. Both coefficients suggest that coliphage *k* in estuarine and marine water tend to be
402 about a factor of 2 to 6 larger than *k* in freshwater.
403 The sunlight factor was significant for both types of coliphage. The coefficient was positive,
404 consistent with sunlight promoting inactivation of both coliphages. *k* from experiments
405 conducted in sunlight were 3 and 14 times greater than *k* measured in the dark reference
406 condition for F+ and somatic coliphage, respectively
407
408 We re-ran the coliphage model using an indicator variable describing the genomic composition
409 of the coliphage (i.e., dsDNA, ssDNA, ssRNA) in lieu of the variable indicating whether the
410 coliphage was somatic or F+. This level of characterization was available for 164 of the 206 *k*
411 values, so a subset of the data was used for this model. Coliphage nucleic acid composition was a
412 statistically significant factor in the model. Post hoc Tukey comparisons that control for effects
413 of experimental conditions and methods indicate that *k* for ssRNA coliphages was larger than *k*
414 for dsDNA coliphages ($p<0.05$) and *k* for ssDNA coliphages ($p=0.1$), with no difference between
415 *k* for dsDNA and ssDNA coliphages ($p=0.8$).
416
417 We conducted an additional analysis where we replaced the coliphage nucleic acid composition
418 variable with a coliphage morphology variable (i.e., icosahedral versus filamentous versus
419 tailed); morphology was only available for 102 of 206 experiments. The morphology dummy
420 variable coefficients were statistically significant in the model. Post hoc Tukey comparison
421 indicated that *k* of tailed coliphages were smaller than *k* of icosahedral coliphages ($p<0.05$),
422 whereas other pairwise comparisons were not different.
423

3.2.3 Global model. $\log_{10}k$ values of the viruses, including *Human astrovirus* and coliphages were aggregated along with the associated variables describing experimental conditions and methods of enumeration. This approach allowed the decay rate constants of the viruses to be compared while controlling for the effects of the various independent variables describing experimental conditions that potentially affect *k*. In the global model, an indicator variable was used to specify the biological group (i.e., *Mastadenovirus, Norwalk virus, Human astrovirus, Hepatovirus A*, *Rotavirus A,* F+ coliphage, somatic coliphage). The model was used to test the following null hypotheses (1) *k* is the same among viral groups controlling for experimental conditions and enumeration methods, and (2) the model water temperature coefficient is the same among viral groups. We therefore included interaction terms in the model between viral group and temperature to test the second hypothesis. The reference experiment for the global model is enterovirus in freshwater at 15°C in the dark, enumerated using culture methods.

Viral group was a significant factor in the model; viral group dummy variables were significant ($p<0.05$ for all except for *Human astrovirus* ($p=0.07$) and F+ coliphage ($p=0.13$)). A post hoc Tukey test suggested two groupings of viruses that have similar $\log_{10}k$ values: (1) F+ and somatic coliphage, and (2) *Enterovirus, Human astrovirus*, *Rotavirus* A, and F+ coliphage. *Hepatovirus A, Norwalk virus,* and *Mastadenovirus* $\log_{10}k$ were lower and generally statistically different than $\log_{10}k$ of viruses in the two groupings, as suggested by Figure 1.

When we included an interaction term between temperature and viral group in the model, the interaction term was statistically significant for *Mastadenovirus*, F+ coliphage and somatic coliphage with positive coefficients. This suggests that the $\log_{10}k$ of these three viruses

447 (*Mastadenovirus* and the two coliphages) are more sensitive to temperature than the other viruses
448 considered in this review, although the effect size is small (factor of 1.1).
449
450 **4. Discussion**
451 The decay of mammalian viruses and coliphages in surface waters followed first-order decay
452 with decay rate constants, on average, between 0.07 to 0.9 d^{-1}. We identified 562 surface water
453 decay rate constants for the viruses, but the distribution of rate constants among viruses was
454 uneven. Most experiments were conducted with coliphages and enteroviruses. Far fewer
455 experiments have been conducted using the other mammalian viruses. *Norwalk virus* and
456 *Mastadenovirus* were the slowest decaying viruses in surface waters. Given the particularly low
457 number of experiments completed with *Norwalk virus*, more research is needed to better
458 understand its decay in surface waters. On the other hand, the large number of experiments with
459 *Enterovirus* suggests efforts to measure decay rate constants of mammalian viruses in surface
460 waters should focus on non-*Enterovirus* genera.
461
462 Given the results of the global model, under similar environmental conditions (e.g., temperature,
463 sunlight, water matrix) and enumeration methods, the decay of F+ coliphage was similar to that
464 of *Enterovirus, Rotavirus A*, and *Human astrovirus*, and also similar to decay of somatic
465 coliphage. However, F+ coliphages decayed faster than *Norwalk virus, Hepatovirus A,* and
466 *Mastadenovirus*. *Mastadenovirus* and F+ coliphage can differ in genomic structure
467 (*Mastadenovirus* have dsDNA while many F+ coliphage contain ssRNA) and capsid structure, so
468 differences in decay rate constants are not surprising. While F+ coliphages, *Norwalk virus,* and
469 *Hepatovirus A* have similar shapes (icosahedral), this does not necessarily mean that they will

exhibit similar decay characteristics, given differences in genome length and amino acid composition of the protein capsid (Meister et al., 2018; Sigstam et al., 2013; Silverman et al., 2013). Additional work to better characterize the decay of *Norwalk virus* and *Hepatovirus A* is warranted to better understand whether F+ coliphages are appropriate surrogates for estimating their decay rates or if there is something unique about the mammalian viruses that reduces their decay rate relative to the coliphages.

All viruses had decay rate constants that scaled with water temperature. The temperature reliance of organismal decay in water has been previously modeled using a temperature correction factor (Liu et al., 2006) such that $k=k'\theta_{T\text{-}20}$ where $\theta=1.07$, k' is the decay rate constant at 20°C, and T is the temperature in °C. The regression model we used to model k suggests that $k=k^{*}10^{\beta_{temp}(T\text{-}15)}$ where k^{*} is the decay rate constant at 15°C and β_{temp} is the regression coefficient. Our expression for k can be cast into a similar form as the equation involving θ. Doing so indicates that given our empirically derived β_{temp}, θ is between 1.07 and 1.17, consistent with the values previously reported in the literature (Hipsey et al., 2008). F+ and somatic coliphages and *Mastadenovirus* were slightly more sensitive to increases in temperature than the other viruses, based on the importance of the temperature - virus type interaction terms in the global model. The reason for the increased sensitivity of these viruses to temperature is uncertain, but could potentially be due to differences in capsid composition or morphology.

We did not find evidence that decay rate constants in marine waters were distinct from those in freshwater for the mammalian viruses. However, the limited experiments with mammalian viruses conducted in estuarine waters suggest higher decay rate constants in estuarine compared

493 to freshwater. Interestingly, there was no evidence of a water matrix effect on *Enterovirus* decay
494 rate constants despite the large number of *Enterovirus* experiments conducted in different water
495 matrices relative to other viruses. A previous meta-analysis of decay rate constants of pathogenic
496 waterborne bacteria, protozoa, and *Caliciviridae* found no clear effect of water matrix on decay
497 rate constants for these targets (Boehm et al., 2018).
498
499 Model results for coliphages, however, suggested larger decay rate constants in marine and
500 estuarine water relative to freshwater. Most authors unfortunately did not report the salinity of
501 their water matrix making it impossible to explore whether there is a predictable relationship
502 between *k* and salinity using a salinity correction factor, similar in form to the temperature
503 correction factor. Hipsey et al. (2008) parameterized *k* for coliform and enterococci bacteria to
504 account for a salinity effect using literature data, but concluded that there was lack of evidence of
505 a clear salinity effect on coliphages.
506
507 Sunlight, particularly light with wavelengths in the UVB region (i.e., 280-320 nm), has been
508 previously found to be an important environmental stressor causing enhanced inactivation of
509 viruses in surface waters (Nelson et al., 2018), including fresh (Elmahdy et al., 2018; Noble et
510 al., 2004; Sinton et al., 2002), estuarine (Burkhardt III et al., 2000; Johnson et al., 1997;
511 Silverman et al., 2013; Sinton et al., 2002), and marine (Fujioka and Yoneyama, 2002; Johnson
512 et al., 1997; Love et al., 2010; Noble et al., 2004; Sinton et al., 2002, 1999) water. This was
513 observed in the meta-analysis, which found that exposure to sunlight irradiance led to
514 significantly greater decay rates for *Enterovirus, Mastadenovirus,* and F+ and somatic coliphages
515 relative to exposure to dark conditions. While compiled *k* values for *Norwalk virus* and *Rotavirus*

516 *A* were suggestive of larger decay rate constants under sunlight exposure compared to the dark,
517 the difference in decay rates between the two conditions was not found to be statistically
518 significant, likely due to the small number of experiments conducted with sunlight exposure (n=1
519 and 2 for *Norwalk virus* and *Rotavirus A*, respectively). No sunlight experiments were were
520 identified for with *Hepatovirus A* and *Human astrovirus*; we suspect that sunlight exposure
521 would increase decay rates of these viruses as well, as compared to decay in the dark, but further
522 research is needed to determine this.
523
524 A number of factors modulate the effect of sunlight on decay rate constants of a particular virus
525 type (Nelson et al., 2018). These factors include the sunlight intensity and distribution of
526 wavelengths (Fisher et al., 2011; Silverman et al., 2015; Sinton et al., 1999), the quantum yield
527 of formation of photochemically-produced reactive intermediates and association between
528 photosensitizers and viruses (which jointly influence exogenous photoinactivation rates)
529 (Davies-Colley et al., 1999; Kohn et al., 2007; Romero-Maraccini et al., 2013; Silverman et al.,
530 2013), and the absorbance spectrum of the water and the depth and mixing of the water column
531 (Kohn and Nelson, 2007; Nguyen et al., 2014; Silverman et al., 2015), which influence the
532 amount of light that reaches the virus. The virus decay experiments identified in the systematic
533 review that included sunlight exposure were conducted under a range of irradiance and water
534 quality conditions. However, many of the included studies did not report information necessary
535 to directly compare sunlight inactivation rates, such as the irradiance and water absorbance
536 spectra that are required to normalize rate constants across different light exposure conditions
537 (Nelson et al., 2018). It was therefore not possible to directly compare *k* from experiments that
538 included sunlight exposure, and care should be taken in interpreting the sunlight data in Figure

539 3b as one virus having faster sunlight-exposed *k* than the others. A future systematic review and
540 meta-analysis could be conducted with a specific focus on sunlight inactivation. Such a review
541 would need to normalize rate constants based on exposure to UVA and UVB light, and also
542 control for the potential contribution of exogenous photoinactivation to *k*. There were a number
543 of experiments evaluating sunlight inactivation of viruses that were not included in the present
544 study because they did not meet inclusion criteria of being conducted in raw, natural surface
545 water; a systematic review comparing sunlight inactivation rates among viruses could include
546 additional experiments conducted in alternative water matrices (i.e., laboratory buffers,
547 wastewater effluent, filtered or autoclaved surface waters, solutions containing model natural
548 organic matter).
549
550 Decay rate constants of six viruses were measured using (RT-)QPCR, in addition to culture-
551 based methods. For three of those viruses (*Enterovirus*, *Rotavirus A*, and F+ coliphages),
552 modeling suggests that decay rate constants measured using RT-QPCR were significantly
553 smaller than those measured using culture methods. The median *Norwalk virus* decay rate
554 measured by RT-QPCR was lower than that measured by culture-based assay (0.04 versus 0.2 d^{-1},
555 respectively), although differences were not statistically significant. Previous work has found
556 significantly slower decay of DNA (Ho et al., 2016; Leifels et al., 2015) and RNA (Duizer et al.,
557 2004; Leifels et al., 2015; Pecson et al., 2009) viruses with exposure to disinfectants (e.g., heat,
558 chlorine, UV_{254}, sunlight) when measured with (RT-)QPCR methods instead of culture methods.
559 (RT-)QPCR quantification requires a nucleic acid target that is much smaller than the length of
560 the complete virus genome. As a result, the short RNA and DNA targets typically used for virus
561 quantification by (RT-)QPCR are not able to measure damage that occurred on another segment

562 of the genome or inactivation resulting from damage to the viral capsid, leading to the relatively
563 slow decay rates calculated using (RT-)QPCR-derived data.
564
565 *Mastadenovirus* was an exception to the trend of slower decay rates being reported for molecular
566 versus culture methods. *Mastadenovirus* decay measured by culture-based methods and QPCR
567 were similar. In fact, the median *k* obtained using QPCR was larger than the median *k* obtained
568 using cell-culture, although we did not find evidence that *k* values measured using the two
569 methods were significantly different. For inactivation mechanisms involving damage to nucleic
570 acids, slow decay kinetics have been previously reported for *Mastadenovirus* using culture
571 assays, which has been attributed to the ability of *Mastadenovirus* to repair its dsDNA genome
572 using host cell machinery while in cell culture (Eischeid et al., 2009; Guo et al., 2010).
573
574 Overall, there were a limited number of decay rate constants measured using (RT-)QPCR, and
575 there were no *k* values measured using (RT-)QPCR data for *Hepatovirus A* or somatic
576 coliphages. Due to their ease of use, versatility and reduced technical requirements compared to
577 infectivity assays, the application of molecular amplification methods for monitoring of
578 mammalian virus concentrations in water will continue to grow. Therefore, collection of
579 additional data on virus decay as measured by molecular methods may aid in interpreting these
580 measurements. Continuing efforts to develop techniques for inferring infectivity from molecular
581 measurements by targeting larger lengths of viral genomes (Pecson et al., 2011) will be useful
582 particularly for viruses that are very difficult to culture, like human *Norwalk virus*.
583

584 HF183 is a human-associated DNA marker of fecal pollution located in the genome of
585 *Bacteroidales* bacteria. A previous systematic review compiled data on its decay rate constants in
586 surface waters (Boehm et al., 2018) and found that the geometric mean *k* across all experiments
587 was 1.2 d^{-1} (range 0.12 to 5.6 d^{-1}). Those data were obtained and added to the data compilation
588 used in the global model. The global model was then re-run in the same manner as described
589 previously to investigate whether HF183 *k* values are different from the virus *k* values, while
590 controlling for variation in experimental conditions. A post hoc Tukey test indicated that HF183
591 *k* values were not distinct from *Enterovirus, Human astrovirus*, *Rotavirus A*, and F+ coliphage *k*
592 values, and were higher than *k* values of *Hepatovirus A, Norwalk virus, Mastadenovirus*, and
593 somatic coliphage, while controlling for the effects of experiment condition (temperature, water
594 matrix, and sunlight) and enumeration method.
595
596 Brooks and Field (2016) compiled data on the decay of sewage-sourced *Escherichia coli* and
597 enterococci in natural waters, and reported overall mean *k* of 0.74 d^{-1} and 0.84 d^{-1}, respectively
598 (or -0.13, and -0.08 if $\log_{10}$ transformed). CrAssphage is a nucleic-acid marker of human fecal
599 pollution located in a bacteriophage genome (Stachler et al., 2017). Currently, there is only one
600 study of crAssphage marker decay in surface waters where it is reported to decay with a first
601 order rate constant of 0.69 d^{-1} in freshwater and between 0.76 d^{-1} and 0.87 d^{-1} in marine water
602 (note: the authors did not report decay rate constants, so these values were converted from their
603 reported slopes of $\log_{10} C/C_o$ versus time) (Ahmed et al., 2019). Figure 4 compares modeled *k*
604 values of the mammalian viruses and coliphages under reference conditions (freshwater, 15°C
605 temperature, dark, and enumerated using culture methods) with *k* values of HF183, crAssphage,
606 *E. coli* and enterococci under similar reference conditions when possible. HF183, crAssphage, *E.*

607 *coli* and enterococci decay rate constants appear to be greater than those of the viruses described
608 in this review and thus may have limited utility in predicting the persistence of viruses in surface
609 waters.
610
611 Future surface water quality standards may include numerical limits for coliphages (United
612 States Environmental Protection Agency, 2015), therefore the decay of coliphages in surface
613 waters may be of increasing interest to the water quality engineering community. This study
614 suggests that F+ and somatic coliphage persistence in surface waters is similar, although distinct
615 from some mammalian viruses. Different genomic composition of coliphages was associated
616 with diverse decay rate constants. For example, on average, coliphages with DNA genomes were
617 found to decay more slowly than those with RNA genomes. Differences in decay of coliphages
618 with distinct genome composition in surface waters has been reported in individual studies
619 (Sinton et al., 2002, 1999) and specifically attributed to the potential for DNA genome
620 coliphages to take advantage of their hosts cellular machinery to repair damage to nucleic acids
621 (Rodriguez et al., 2014). Differences in coliphage morphology were also associated with
622 different decay rate constants with icosahedral coliphages having larger rate constants than tailed
623 coliphages, although this may be tied to predominance of icosahedral coliphage having ssRNA
624 genomes and the majority of tailed coliphage containing dsDNA.
625
626 There are limitations of this analysis that were not previously mentioned. First, we restricted our
627 review to raw surface waters in order to gain insight into decay rate constants expected in situ.
628 Many of the experiments were completed in the laboratory with the surface waters placed in
629 flasks; some were carried out by placing raw water in dialysis bags and placing them in situ. In

either case, the water, or some components of the water in the case of dialysis bags, was separated from the environment. The separation undoubtedly results in changes to the chemical and biological composition of the surface water during the experiment, which may subsequently alter the decay characteristics of the viruses. However, it is our opinion that studies using raw surface waters provide the best possible estimates of expected decay in the environment.

Second, the clustering of *k* values by study was not considered in the meta-analysis. While some studies presented one *k* value for a single virus under one set of conditions, others presented multiple *k* values for different viruses or different experimental conditions. Due to the inconsistency in the number of *k* values reported across studies, controlling for clustering among studies was not feasible.

Third, *k* values are likely affected by experimental factors other than those considered herein (i.e., water temperature, salinity, sunlight, enumeration method), including the biological composition of the water, which might contribute to biologically-mediated removal processes like predation or enzymatic degradation. We encourage authors to include information on the biological characteristics of raw surface waters in future studies including indicators like turbidity, total bacteria, or chlorophyll a concentrations – these data were not available consistently across the studies included in this review – so that the importance of biological composition can be considered in future reviews.

Fourth, there were limited data available for some of the viruses and certain experimental conditions, as described above, which may lead to simple regression models, as we used, being

underpowered. As more data become available on virus decay in surface waters, they may reveal that factors not identified as "significant" herein are actually important in controlling viral decay. As an example, we found that *k* values were not significantly different among viral species or genotypes within a specific virus group. In contrast, recent research on virus disinfection shows even small differences in virus genotypes can affect their persistence (Meister et al. 2018).

The data compiled in this review are available as supplementary material. The multiple regression model provided in this paper can be used to generate estimates for *k* values for specific viruses under specific environmental conditions (i.e, temperatures, water matrix, and enumeration method), where *k* is represented as a log-normal distribution with a mean and variance. This can be achieved using the "predict.lm" function in R which uses the model fit parameters generated by "lm" in R (as reported in Table 6), as well as the conditions for which one desires the predictions, as inputs.

5. Conclusions

- Decay rate constants of viruses are positively associated with temperature, and *Mastadenovirus* and the coliphage *k* values showed increased sensitivity to water temperature than the other mammalian viruses.
- Experiments conducted in sunlight yielded significantly larger *k* than those conducted in the dark. However, researchers rarely provided enough detail to account for light intensity and light screening which limits our ability to compare sunlight *k* values among studies.

- Enumeration methods can impact measured decay rate constants. In most cases, culture-based quantification methods provided larger rate constants than molecular methods. The exceptions to this were *Norwalk virus* and *Mastadenovirus* for which no significant difference between *k* measured by culture versus molecular methods was observed.
- Rate constants for coliphage, historically important indicators of viral fate and transport, were smaller in fresh versus estuarine and marine waters. However, this pattern was not observed for mammalian viruses, most of which had insufficient data to make a comparison.
- Information gaps revealed by the meta-analysis suggest future research is needed in the following areas: the decay of hepatitis A and E viruses and *Human astrovirus* in surface waters (including sunlight decay rates); the effect of salinity on coliphage *k* values (including the development of a salinity correction factor); the decay of *Norwalk virus* in estuarine waters and *Hepatovirus A* in freshwaters; and the measurement of human norovirus decay using novel cell culture methods.
- F+ and somatic coliphages have *k* values that were similar to some mammalian viruses, although may over predict decay rates of *Mastadenovirus, Hepatovirus A, Norwalk virus*, *Human astrovirus*.
- *k* of common and novel fecal indicators – including enterococci, *Escherichia coli*, HF183, and crAssphage – were generally larger than the mammalian viruses under reference conditions (temperature of 15°C, freshwater, dark, enumerated using culture methods).

Acknowledgements. AB was partially supported by the National Science Foundation (CBET-1804169). This publication was also prepared by authors under partial support from NOAA Grant #NA14OAR4170075, California Sea Grant College Program Project #R/RCC-06, through NOAA's National Sea Grant College Program, U.S. Dept. of Commerce. The statements, findings, conclusions and recommendations are those of the author(s) and do not necessarily reflect the views of California Sea Grant, NOAA or the U.S. Dept. of Commerce. The authors acknowledge W. Jennings and E. Andruszkiewicz for their input on the work.

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Search Date	Organism	Search terms	N identified in searches	Number unique papers identified through databases	Number identified from references of review or other papers	Number subject to full text review	Number papers included
9/27/18	norovirus	(norovir* OR norwalk vir* OR calicivir*)	WOS=481, Sc= 273, PM=250	579	0	26	4
9/28/18	adenovirus	(adenovir*)	WOS=315, Sc= 278, PM=215	448	0	32	10
9/28/18	rotavirus	(rotavir*)	WOS=177, Sc= 176, PM=154	333	3	22	10
9/28/18	enterovirus	(enterovir*)	WOS=265, Sc= 322, PM=221	541	15	65	NA
9/28/18	poliovirus	(poliovir*)	WOS=290, Sc= 399, PM=246	561	13	61	37
9/28/18	coxsackievirus	(coxsackievir*)	WOS=47, Sc= 84, PM=60	108	14	26	15
9/28/18	echovirus	(echovir*)	WOS=31, Sc= 67, PM=43	86	10	20	10
9/28/18	hepatitis E virus	(“hepatitis E”)	WOS=29, Sc= 29, PM=70	103	0	5	0
9/28/18	hepatitis A virus	(“hepatitis A”)	WOS=349, Sc= 203, PM=142	484	7	33	6
9/28/18	astrovirus	(astrovir*)	WOS=18, Sc= 25, PM=11	36	0	7	2
9/28/18	reovirus	(reovir*)	WOS=40, Sc= 68, PM=57	100	0	6	0
10/30/18	coliphage	(coliphage* OR bacteriophage*)	WOS=1156, Sc= 1151, PM=720	1702	6	95	32

Table 1. Search terms and statistics for the systematic literature review. NA in the most-right column for enterovirus indicates that full-text review papers that pass inclusion criteria were passed to coxsackievirus, poliovirus, and/or echovirus, as appropriate. WOS = Web of Science core collection, Sc = Scopus, and PM = PubMed.

	N	log_{10}-mean k	stdev log10k	fresh	estuarine	marine	dark	sunlit	T_{min}	T_{max}
Enterovirus	258	-0.07	0.57	102	39	117	240	18	4	29
Hepatitis A	12	-0.66	0.40	0	2	10	12	0	5	25
Astrovirus	4	-0.89	0.28	2	0	2	4	0	4	20
Norovirus	12	-1.15	0.59	12	0	0	11	1	4	25
Rotavirus	33	-0.34	0.55	20	5	8	31	2	4	29
Adenovirus	37	-0.69	1.01	17	10	10	27	10	4	26
Coliphage	206	-0.18	0.96	128	24	54	143	63	4	25
Total	562	-0.20	0.84	281	80	201	468	94	4	29

Table 2. The total number of experiments or *k* values collected (N) and their log_{10}-mean and standard deviation of the log_{10}-transformed *k* values. The number of experiments or k values collected under various conditions (fresh water, estuarine water, marine water, under dark conditions, under sunlit conditions), and the minimum (T_{min}) and maximum (T_{max}) (°C) under which experiments were conducted.

Table 3. Sources for mammalian virus decay rate constants in surface waters obtained from the systematic review.

Enterovirus	Hepatitis A	Astrovirus	Adenovirus	Rotavirus	Norovirus
(Akin et al., 1971) (Akin et al., 1976) (Bae and Schwab, 2008) (Blawat et al., 1976) (Bosch, 1995) (Callahan et al., 1995) (Chung and Sobsey, 1993) (Enriquez et al., 1995) (Fujioka et al., 1980) (Fujioka and Yoneyama, 2002) (Girones et al., 1989) (Herrmann et al., 1974) (Hurst et al., 1989) (Hurst and Gerba, 1980) (Johnson et al., 1997) (Jofre et al., 1986) (Joyce and Weiser, 1967) (LaBelle and Gerba, 1982) (Lycke et al., 1965) (Magnusson et al., 1966) (Magnusson et al., 1967) (Matossian and Garabedian, 1967) (McLean and Brown, 1968) (Metcalf and Stiles, 1967) (Nasser et al., 2003) (O'Brien and Newman, 1977) (Pancorbo et al., 1987) (Patti et al., 1987) (Patti et al., 1996) (Prevost et al., 2016) (Prier and Riley, 1967) (Shuval, 1970) (Silverman et al., 2013) (Smith et al., 1978) (Sobsey et al., 1987) (Toranzo and Metricic, 1982) (Vaughn and Metcalf, 1975) (Wait and Sobsey, 2001) (Walters et al., 2009) (Ward et al., 1986)	(Bosch, 1995) (Callahan et al., 1995) (Chung and Sobsey, 1993) (Patti et al., 1987) (Patti et al., 1996) (Sobsey et al., 1987)	(Bosch et al., 1997) (Espinosa et al., 2008)	(Ahmed et al., 2014) (Blawat et al., 1976) (Elmahdy et al., 2018) (Enriquez et al., 1995) (Eregno et al., 2018) (Liang et al., 2017) (Magnusson et al., 1966) (Moresco et al., 2016) (Prevost et al., 2016) (Silverman et al., 2013)	(Chung and Sobsey, 1993) (Espinosa et al., 2008) (Girones et al., 1989) (Hurst and Gerba, 1980) (Jofre et al., 1986) (Loisy et al., 2004) (Pancorbo et al., 1987) (Raphael et al., 1985) (Sattar et al., 1985) (Ward et al., 1986)	(Bae and Schwab, 2008) (Elmahdy et al., 2018) (Ngazoa et al., 2008) (Moresco et al., 2016)

Table 4. Sources for coliphage decay rate constants in surface waters obtained from the systematic review.

Coliphage	(Babich and Stotzky, 1980) (Bae and Schwab, 2008) (Berry and Noton, 1976) (Boehm et al., 2009) (Borrego and Romero, 1985) (Brion et al., 2002) (Burkhardt III et al., 2000) (Callahan et al., 1995) (Chung and Sobsey, 1993) (Craig et al., 2002) (Durán et al., 2002) (Eregno et al., 2018) (Gerba and Schaiberger, 1975) (Girones et al., 1989) (Jofre et al., 1986) (Lee and Sobsey, 2011) (Long and Sobsey, 2004) (Love et al., 2010) (Magnusson et al., 1966) (Mitchell and Jannasch, 1969) (Niemi, 1976) (Noble et al., 2004) (Ravva and Sarreal, 2016) (Schaper et al., 2002) (Silverman et al., 2013) (Sinton et al., 2002) (Vaughn and Metcalf, 1975) (Wu et al., 2016) (Yang and Griffiths, 2013) (Zaiss, 1981)

Family	**F+/ Somatic**	**Nucleic Acid Type**	**Morphology**	**Species**					
Inoviridae	F+	ssDNA	Filamentous	F1	fd	M13	OW	SD	ZJ/2
Leviviridae	F+	ssRNA	Icosahedral	F2	Dm	GA	Go1	MS2	Qβ
				SG1	SG4	SG42	SP		
Microviridae	Somatic	ssDNA	Icosahedral	PhiX174					
Myoviridae	Somatic	dsDNA	Tailed	MY2	P1	T2	T4		
Podoviridae	Somatic	dsDNA	Tailed	T7					
Siphoviridae	Somatic	dsDNA	Tailed	λ	SC12	SR51	SS13	T1	
Tectiviridae	Somatic	dsDNA	Icosahedral	PRD1					

Table 5. A list of families and species used in the coliphage experiments.

Virus	Intercept (β_0)	T-15 (β_{temp})	Method: QPCR/RT-QPCR (β_{meth1})	Method: EM-QPCR/EM-RT-QPCR (β_{meth2})	Method: Immuno-logical (β_{meth3})	Water: Estuarine (β_{mat1})	Water: Marine (β_{mat2})	Sunlight (β_{sun})	RSE	dof	R_2
Enterovirus	**-0.25**	**0.03**	**-0.77**	**-0.62**	na	0.12	0.00	**1.07**	0.43	242	0.46
Hepatovirus A	**-0.75**	*0.03*	na	na	na	*	-0.05	na	0.37	9	0.32
Norwalk virus	**-1.08**	**0.04**	-0.30	na	na	na	na	0.43	0.44	8	0.61
Rotavirus A	**-0.60**	**0.04**	**-0.96**	na	0.34	**0.67**	-0.05	0.51	0.38	26	0.61
Mastadenovirus	**-1.20**	**0.07**	-0.10	0.32	na	**0.80**	0.13	0.13	0.60	30	0.71
somatic coliphage	**-1.09**	**0.07**	na	na	na	**0.72**	**0.78**	**1.08**	0.62	62	0.63
F+ coliphage	**-0.47**	**0.06**	**-0.74**	na	na	**0.55**	*0.26*	**0.47**	0.56	124	0.50

Table 6. Model coefficients for virus-specific regression models. Variable name is provided on the top of each column along with the coefficient name. All coefficients are for dummy variables except for β_{temp} which is the coefficient for a variable that is calculated as temperature (in °C) minus 15°C. RSE is the model's residual standard error, "dof" is the degree of freedom of the model, and R_2 is the multiple R_2 value of the model. Coefficients are **bold** if they are statistically significant ($p<0.05$) and are *italicized* if $0.05<p<0.1$. na indicates that the variable was not used in the model because no experiments were conducted under the indicated conditions. * indicates that this served as the reference condition because no experiments were conducted in freshwater for this virus. A grey horizontal bar separates the mammalian viruses from the coliphage. The standard error for each coefficient can be found in the supplementary material

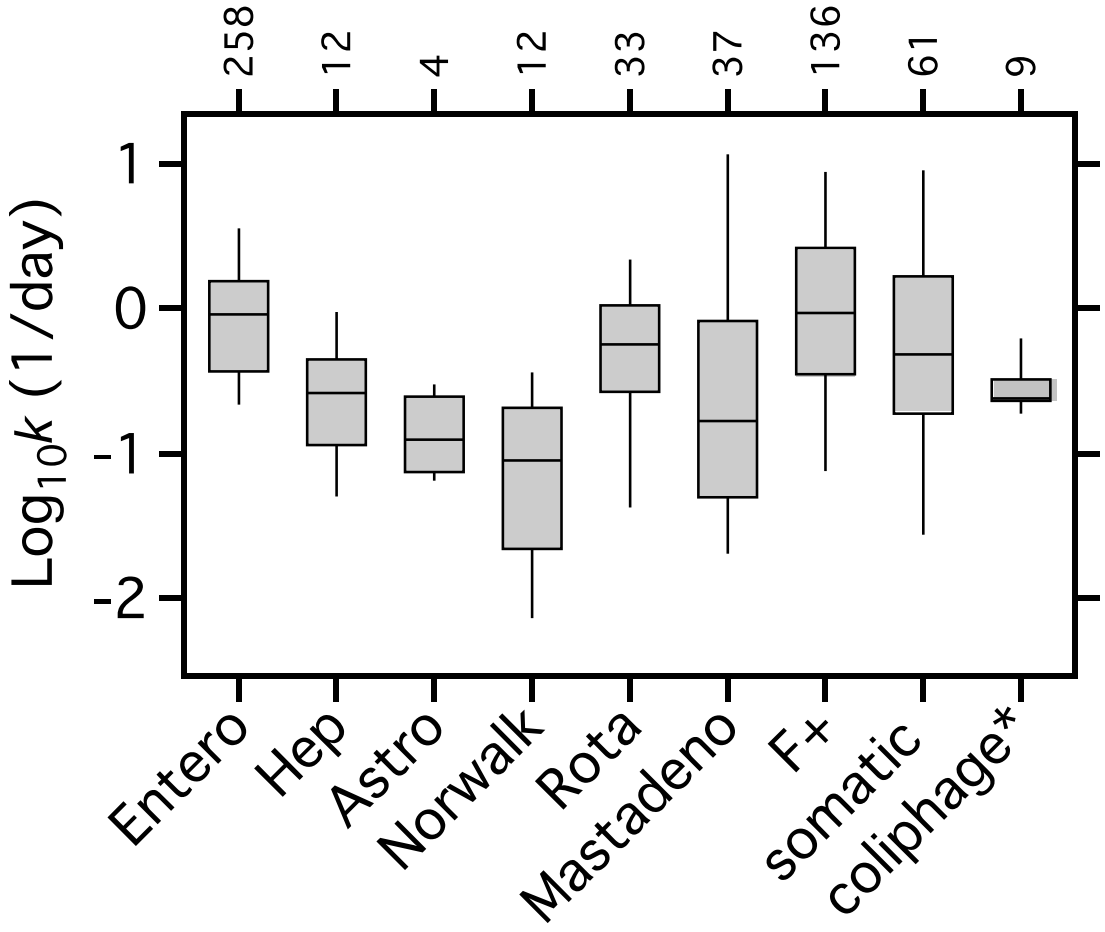

Figure 1. Box and whisker plot of $\log_{10}k$ values obtained in the systematic review. The horizontal line represents the median, and top and bottom of the box represent the 75th and 25th percentiles, respectively, and the top and bottom of the whisker represent the 10th and 90th percentile respectively. Entero is *Enterovirus*, Hep is *Hepatovirus A,* Astro is *Human astrovirus,* Norowalk is *Norwalk virus*, Rota is *Rotavirus A*, Mastadeno is *Mastadenovirus*, F+ is F+ coliphage, somatic is somatic coliphage, and coliphage* is coliphages that could not be characterized as F+ or somatic based on the information provided by the authors. Numbers on the top axis describe the number of *k* values used to create each box and whisker unit.

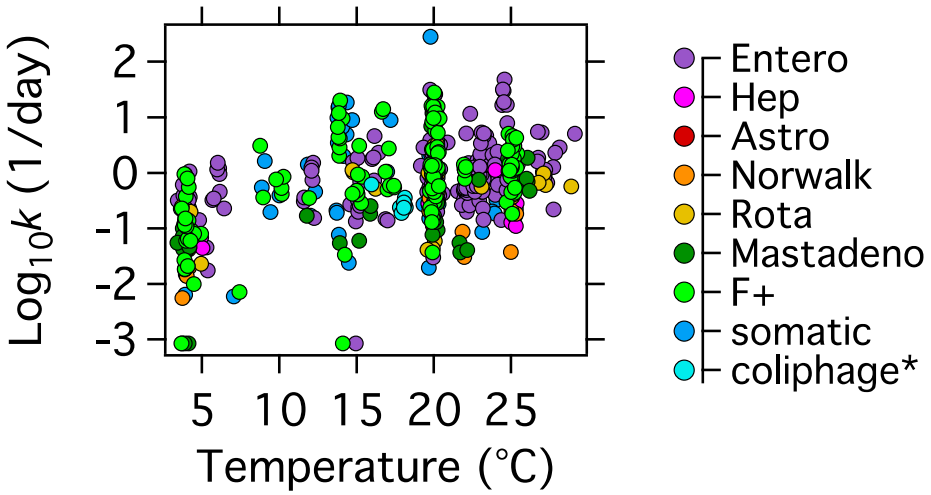

Figure 2. $Log_{10}k$ as a function of temperature, color coded by virus type. The five k values reported by authors as 0, which were replaced with 0.0008 per day, can be seen sitting just above the x-axis. See the caption of Figure 1 for a definition of the virus type shorthand provided in the legend. A small amount of jitter (Gaussian noise) was added to the temperature value for each experiment so that $log_{10}k$ values collected at common temperatures (4°C, 20°C, for example), were not directly on top of each other.

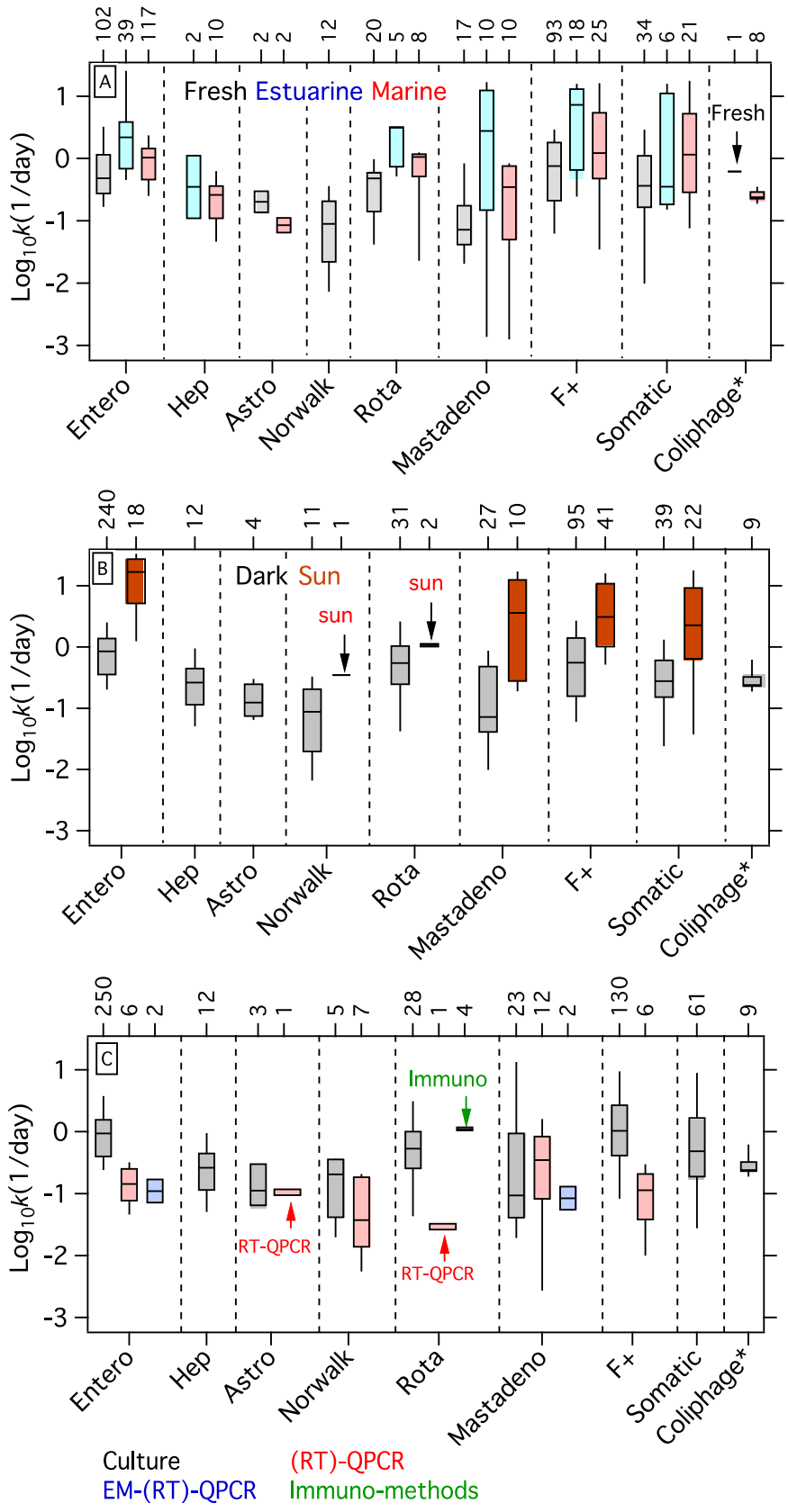
A
Fresh Estuarine Marine
Fresh
B
Dark Sun
sun
sun
C
Immuno
RT-QPCR
RT-QPCR
Log10k(1/day)
Entero
Hep
Astro
Norwalk
Rota
Mastadeno
F+
Somatic
Coliphage*
Culture
(RT)-QPCR
EM-(RT)-QPCR
Immuno-methods

Figure 3. Box and whisker plots comparing distribution of $\log_{10}k$ values for each virus group separated by (A) different water matrices – fresh, estuarine, and marine, (B) dark and sunlit waters, and (C) different measurement methods. Recall that experiments were classified as sunlit only if it they were conducted under light exposure and UVA and UVB were expected to penetrate into the experimental waters. The number of $\log_{10}k$ values used to create the box and whisker plot is shown on the top axis above each box. The midline of the box is the median, the top and bottom of the box are, respectively, the 75th and 25th percentiles, and the top and bottom of the whiskers show the 10th and 90th percentiles. No box and whisker is shown for a virus for a specific condition if there were no experiments conducted under that condition (i.e., there were no experiments that measured *k* for *Norwalk virus* in marine waters). Small boxes where the color cannot be seen are labeled with the condition. See the caption of Figure 1 for a definition of the virus type shorthand provided on the x-axis of each panel.

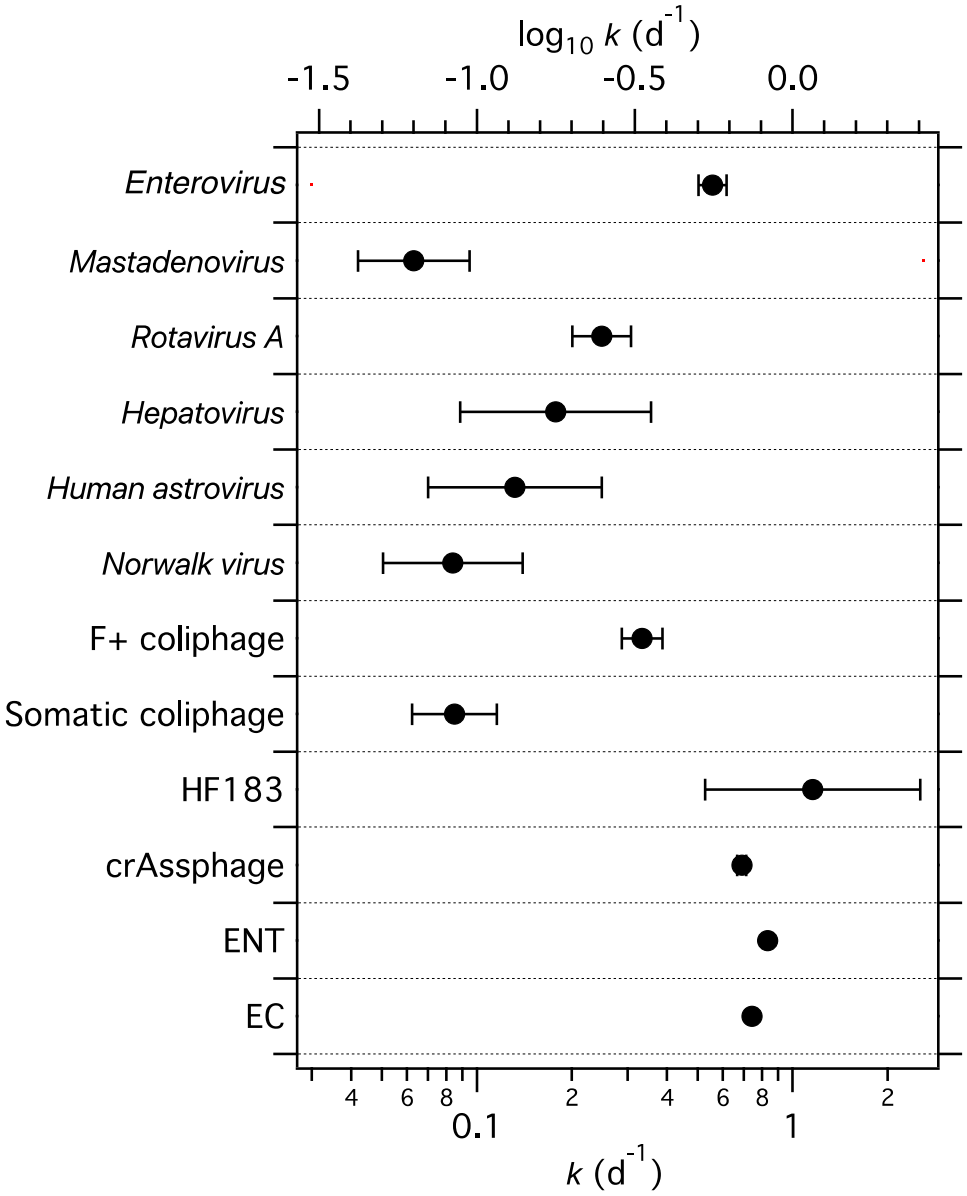

Figure 4. Estimates for *k* of mammalian viruses and common indicators. The values shown for the viruses included in this systematic review (except for *Human astrovirus*) are estimates for *k* and its standard error (SE) for the reference model condition (temperature of 15°C, freshwater, in the dark, enumerated using culture methods for all viruses except for *Hepatovirus* for which estuarine water serves as the reference condition in place of freshwater). For *Human astrovirus*, the geometric mean and its standard deviation for all 4 *k* values are shown. Values shown for HF183 represent the geometric mean and its standard deviation across 52 HF183 *k* values obtained in a systematic review by Boehm et al. (2018). The crAssphage *k* value and its SE were measured in a freshwater microcosm by Ahmed et al. (2019). The enterococci (ENT), and *E. coli* (EC) values are mean *k* values determined in a systematic review by Brooks and Field (2016). No error bars are shown on the ENT and EC values. The error bars on the crAssphage value are difficult to see because they are smaller than the symbol.