Biofilm 7 (2024) 100170

Contents lists available at ScienceDirect

Biofilm

journal homepage: www.sciencedirect.com/journal/biofilm

Controlling of foodborne pathogen biofilms on stainless steel by bacteriophages: A systematic review and meta-analysis

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ARTICLE INFO

Keywords:
Bacteriophage
Biofilm
Stainless steel
Foodborne pathogens
Biocontrol
Systematic review and meta-analysis

ABSTRACT

This study investigates the potential of using bacteriophages to control foodborne pathogen biofilms on stainless steel surfaces in the food industry. Biofilm-forming bacteria can attach to stainless steel surfaces, rendering them difficult to eradicate even after a thorough cleaning and sanitizing procedures. Bacteriophages have been proposed as a possible solution, as they can penetrate biofilms and destroy bacterial cells within, reducing the number of viable bacteria and preventing the growth and spread of biofilms. This systematic review and meta-analysis evaluates the potential of bacteriophages against different biofilm-forming foodborne bacteria, including *Cronobacter sakazakii*, *Escherichia coli*, *Staphylococcus aureus*, *Pseudomonas fluorescens*, *Pseudomonas aeruginosa* and *Listeria monocytogenes*. Bacteriophage treatment generally causes a significant average reduction of 38 % in biofilm formation of foodborne pathogens on stainless steel. Subgroup analyses revealed that phages are more efficient in long-duration treatment. Also, applying a cocktail of phages is 1.26-fold more effective than applying individual phages. Phages at concentrations exceeding 10^7 PFU/ml are significantly more efficacious in eradicating bacteria within a biofilm. The antibacterial phage activity decreases substantially by 3.54-fold when applied at 4 °C compared to temperatures above 25 °C. This analysis suggests that bacteriophages can be a promising solution for controlling biofilms in the food industry.

1. Introduction

Food safety is an increasingly critical and priority concern, particularly regarding foodborne bacteria and their impact on individuals and public health [1–5]. *Salmonella* spp., *Listeria* spp., *E. coli, Bacillus cereus,* and *Staphylococcus aureus* are major pathogens that can contaminate many food commodities. They are considered a great concern for individual health since they can cause outbreaks that lead to diseases and even death ([6–10]; H.-x. [11–15]). Besides the health consequences of foodborne pathogens, including vomiting, diarrhea, fever, and even death. Moreover, their economic losses are substantial [16–20]. Food-originated outbreaks can lead to costly product recalls, lower customer trust in the food products, and ultimately cause harm to the credibility of food industries as a whole ([21]; Z. [22]). In addition, healthcare expenses to treat foodborne diseases can be considerable, especially once the illness results in prolonged hospitalization or severe health complications [20,23–26]. Therefore, the safety of food products should be guaranteed not only for the sake of customers' safety but also for the stability of the food industry [27,28]. Therefore, it is necessary to investigate all options to prevent and inhibit the spreading of

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https://doi.org/10.1016/j.bioflm.2023.100170
Received 12 October 2023; Received in revised form 27 November 2023; Accepted 10 December 2023
Available online 17 December 2023
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food-originated diseases [2,29,30].

One of the most well-known materials used as a food-contact surface in the food industry is stainless steel, widely used in food preparation and processing areas, including kitchens and restaurants, due to its unique properties [31,32]. Cleaning and sanitation of stainless-steel equipment and surfaces are uncomplicated, reducing the risk of cross-contamination and spreading foodborne pathogens [33,34]. Additionally, stainless steel is corrosion-resistant, which prevents the formation of rust and other leachable contaminants in food, potentially posing health hazards [32,34,35]. The smooth, non-porous surface of stainless steel makes it less likely to harbor bacteria and other non-bacterial pathogens, reducing the risk of foodborne illness ([33,35]; M. [34]). However, it is worth mentioning that even though stainless steel is highly appropriate for food-contact applications, it cannot be entirely protected from the buildup of bacteria and other pathogens in the form of biofilm ([36]; X. [37]). Biofilm-forming bacteria can attach to stainless steel surfaces and form a protective layer resistant to cleaning and sanitizing agents, resulting in the survival of bacteria and other pathogens even after thorough cleaning procedures ([38,39]; X. [37]; R. [40]). Therefore, it is critical to implement efficient measures for preventing and controlling biofilm formation on stainless steel surfaces, such as regular cleaning and sanitizing, using effective anti-bacterial agents, and applying appropriate treatments for contact surfaces.

Biofilm is a complex dynamic matrix of microorganisms that can adhere to surfaces, forming a protective barrier and harboring pathogenic bacteria [41–43]. It can carry a persistent risk of food contamination by foodborne pathogens, sequentially leading to foodborne illness outbreaks, affecting the economy through losses in production and reputation and creating more need for regulatory scrutiny. Biofilms protect biofilm-forming bacteria from sanitizers by their matrices that make a physical barrier mainly formed from extracellular polymeric substances (EPSs), which makes it difficult to reach and remove [44,41,45]. In addition, biofilm can damage equipment and cause food safety and quality issues, including spoilage, off-flavors, and the growth of foodborne pathogens [46–49].

To maintain food safety, controlling the biofilm formation of foodborne pathogens on stainless steel surfaces in the food industry is crucial [50]. To achieve this, a multi-disciplinary approach has been employed, which includes integrating various physical, chemical, and biological methods [51–53]. Physical methods encompass abrasive materials, high-pressure water jets, irradiation, and ultrasonic cleaning to remove or disrupt the biofilm [54–56]. Even though physical methods are effective, their application in the food industry has drawbacks [57–59]. For example, using radiation for sterilization has received low customer acceptance, and ultrasound has a low potential for decontamination ([60,61,62]). Chemical methods, for instance, using sanitizers such as chlorine, can make the stainless steel less receptive to biofilm formation, but it can lead to chemical reactions and residue [63,64]. Nowadays, new methods of controlling biofilm on the surface of food industry facilities, such as applying bacteriophages to control biofilm formation, have attracted the attention of researchers [65,47,66].

Bacteriophages, phages, or viruses of microbes specifically infect bacteria [67]. Phages are the most abundant organisms in the biosphere, and they have been used in the health fields since the 1920s when they were first discovered by the French-Canadian microbiologist Félix d'Hérelle [68,69]. The application of bacteriophage has been considered a promising approach for controlling the biofilm formation of foodborne pathogens on the food industry surfaces [70,71]. Because phages can only infect bacteria, they are generally recognized as low toxic to human and eukaryote cells [72,73]. The Food and Drug Administration (FDA) approved the application of bacteriophage as an additive for poultry and beef in 2006 [74]. Bacteriophages can effectively penetrate and destroy bacterial cells within biofilms, reducing the number of viable bacteria and preventing the growth and spread of biofilm [75,76]. Some bacteriophages possess depolymerase polysaccharides that enable them to penetrate polysaccharides, which are the main protective layer of biofilm and facilitate reaching phage to bacteria in biofilm [77,78]. This approach is especially useful in the food industry, where conventional methods such as chemical sanitizers and physical cleaning may not effectively remove or kill the bacteria within the biofilm. Another noticeable advantage of phage application is their natural and environmentally friendly essence, which reduces the risk of chemical residue and environmental pollution compared to chemicals [79,80]. The efficacy of utilizing bacteriophages as a viable strategy to control foodborne pathogens was thoroughly examined across diverse food commodities, encompassing milk, poultry, vegetables, and eggs, and the effectiveness of this approach was confirmed [81–85]. The co-application of bacteriophages with other biofilm control strategies, including probiotics and surface modifications, has enhanced their efficacy in controlling biofilm formation [75,86,87].

The present systematic review and meta-analysis aimed to evaluate different bacteriophages and their potential ability to control foodborne pathogen biofilms on stainless steel in the food industry. This study will systematically assess the pooled data from the eligible research on the application of bacteriophage for controlling biofilm of foodborne pathogens on stainless steel and evaluate the different factors that may influence its efficacy.

2. Methods

2.1. Search strategy and study selection

A search strategy was designed to comprehensively identify all relevant original research focused on the effects of bacteriophage on the bacterial count in biofilm formed on stainless steel. To ensure the search's validity, the search strategy's reliability was first evaluated before being applied to five databases, including three general Web of Science, Science Direct, PubMed, Scopus, and two specific databases of Agricola and AGRIS. The search strategy utilized a consistent approach across all five databases. To avoid missing related studies, it included two sets of terms, including bacteriophage* OR phage* AND "stainless steel" OR "stainless steels" OR stainless OR steel OR steels. Additionally, non-specific terms were used to increase the search's comprehensiveness. The Agricola and AGRIS databases were screened only by checking the titles of studies for relevant terms, while both the titles and abstracts were screened in Web of Science, PubMed, and Scopus. The timeframe of this study was restricted to the period of 2000–2022 to reflect the most recent advancements in the field. The screening for study selection and data extraction were performed by three independent co-authors for more caution, and a referee co-author solved disagreements.

2.2. Inclusion and exclusion criteria and screening of relevant studies

Identifying relevant studies on bacteriophage efficacy against biofilm formation on stainless steel involved several well-defined steps. Firstly, a comprehensive search strategy was executed in five major databases, including Web of Science, PubMed, Scopus, AGRIS, and Agricola, incorporating terms such as bacteriophage, stainless steel, and related terms. Duplicate studies were removed as the same study may have been included in multiple databases. In the second step, meticulous scrutiny of the title and abstract of each article was performed to eliminate irrelevant studies that did not meet the predefined criteria. Based on these criteria, studies had to be conducted on phage application against biofilm on stainless steel and written in English. Review studies, including narrative or systematic ones, book chapters, or those that did not mention specific experiments, were also excluded. The third and final step involved a thorough full-text reading of the remaining studies to ensure relevance and compliance with the selection criteria. This procedure facilitated an enhanced and comprehensive analysis of the study design, results, and conclusions, ensuring the highest quality and accuracy in the selected studies.

2.3. Screening full texts

The selection process involved several key steps to ensure that only relevant and credible studies were included for further analysis. Firstly, all studies with unavailable full text were discarded, leaving only those with accessible full text to be considered. Explicit selection criteria were established and followed to ensure that the studies met certain standards of quality and relevance to the study to identify the most eligible studies for data extraction. Studies had to meet the following criteria in order to be considered for analysis: 1) original research studies, no reviews nor book chapters; 2) published between the years 2000 and 2022; 3) focused on the effects of bacteriophage on biofilm bacterial count on stainless steel, with neither planktonic nor live cells being considered; 4) reporting the bacterial count in the treatment and control groups in text, tables or graphs; 5) accurate analytical methods clarifying the number of replications and standard deviation or standard error reported for each examined group and 6) written in English. By adhering to these selection criteria, only the most relevant and reliable studies were identified and considered, enhancing the credibility of the analysis and the findings.

2.4. Data extraction

The procedure encompassed the screening of text, followed by the examination of tables, and in cases where data was represented through graphs, the Digitizer software (version 5.4.9) was utilized for extraction. To establish confidence in the extracted data, it was tested in triplicate. The extracted data included details such as the name of the first author, year of publication, bacterial species, phage name, method of the bacterial count, number of phages, method of biofilm treatment with phages, source of phage isolation, temperature of biofilm formation, the temperature of biofilm treated with phages, age of biofilm, duration of phage treatment on biofilm, the titer of initial phage, initial bacterial count, count of bacteria in the treated group, count of bacteria in the control group, standard deviation or standard error for the treatment group and control group, number of replicates for both treatment and control groups, using or not using food matrix and type of bacteria based on Gram-staining.

2.5. Statistical analysis

Within the framework of our study, we developed a meta-analysis approach to evaluate the ability of bacteriophages to control foodborne pathogen biofilm on stainless steel. To quantitatively measure the observed effects, a response ratio (R) was computed for both methodologies [88–90].

The estimation of R and its variance regarding the impact of the treatments was performed by utilizing the values of both the control and treatment groups. This was carried out through the application of the following equation (*Eq.* (1)):

$$R = \frac{X_{initial(treatment)}}{X_{initial(control)}} \quad (1)$$

Where $X_{initial\ (control)}$ refers to the initial values of the control groups, and $X_{initial\ (treatment)}$ signifies the initial values of the treatment groups. Next, the variable R was subjected to a natural logarithmic transformation (L) in order to attain a normalized distribution (*Eq.* (2)):

$$L = \ln(R) \quad (2)$$

The variance for L (V_{lnR}) and approximate standard error (SE_{lnR}) was calculated through the equations below (*Eqs.* 3-4):

$$V_{lnR} = \left(SD_{pooled}\right)^2 \times \left(\frac{1}{\left(control \times X^2_{initial(control)}\right)} + \frac{1}{\left(n_{treatment} \times X^2_{initial(treatment)}\right)}\right) \quad (3)$$

$$SE_{lnR} = \sqrt{V_{\ln R}} \quad (4)$$

The calculation of the overall L, which serves as a measure of effect size across the included studies, was executed by implementing the random-effects model. This statistical framework was deemed appropriate in light of the variability in the design of the constituent studies. Herein, the contribution of each study's L value was weighted by the individual experiment's variance and the random-effects model. Finally, the conversion of the weighted overall L (L*) to the weighted overall R (R*) was carried out using the following equation (*Eq.* (5)):

$$R^* = \exp(L^*) \quad (5)$$

The assessment of heterogeneity among the included studies involved the utilization of Cochran's Q test to examine the evidence of variation. Additionally, the extent of heterogeneity was quantified using the *I*-squared (I^2) index. A significance level of $P \leq 0.05$ for Cochran's Q test indicated heterogeneity, while an I^2 value exceeding 50 % reflected a notable degree of heterogeneity across the studies [91,92].

Within the ambit of the present study, subgroup analyses were meticulously carried out. The examination of potential publication bias entailed the implementation of two statistical tests: the Begg and Mazumdar adjusted rank correlation test as well as Egger's regression asymmetry test [93–95]. Stata software version 11.2 (Stata Corp., College Station, TX) was utilized for conducting the meta-analysis, with a significance threshold set at $P \leq 0.05$ to ascertain statistically significant findings.

3. Result and discussion

3.1. Literature search and study characteristics

A comprehensive literature search was conducted in this study to assess the potential of bacteriophages in controlling the formation of biofilm by foodborne pathogens on stainless steel. As shown in Fig. 1, 391 studies were identified, of which 207 were screened after removing

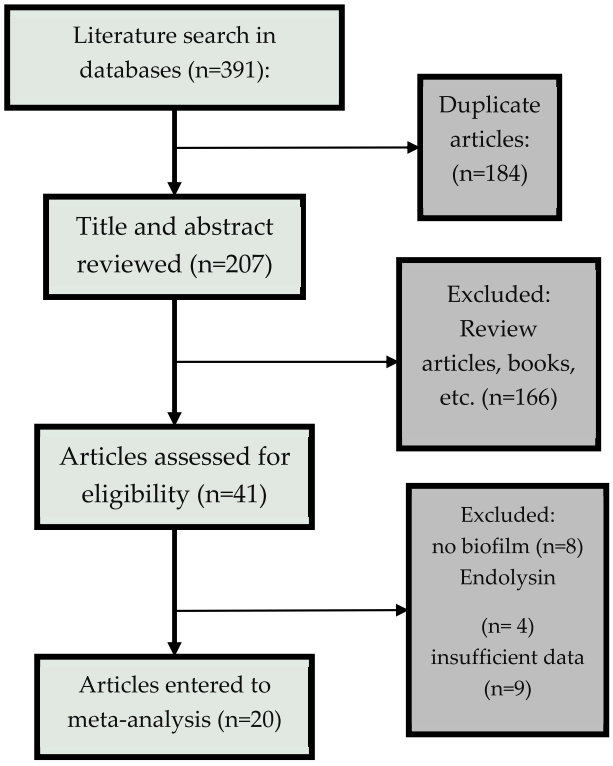

Fig. 1. Flow-diagram of screening stages.

duplicates. Following a comprehensive evaluation of the title and abstract and excluding review articles, books, and letters, 41 studies were deemed eligible for further scrutiny in the next step. Then, 21 studies were excluded, of which 8 studies investigated viable cells, not biofilms, 4 studies investigated the potential of endolysins, which are phage enzymes, instead of direct application of bacteriophages, and 9 studies did not report sufficient data that could be analyzed by meta-analysis.

Therefore, only 20 studies remained for systematic review and meta-analysis, with the collected data being presented in Table 1. The efficiency of bacteriophages against seven different biofilm-forming bacteria, including *E. coli*, *L. monocytogenes*, *Salmonella* spp., *C. sakazakii*, *Pseudomonas* spp., *S. aureus*, *Citrobacter freundii*, and *Hafnia alvei*, was evaluated in the included studies. Based on the studies, the biocontrol was carried out using one up to nine phages with treatment conditions, including temperatures varying between 4 and 40 °C, biofilm ages of 2–168 h, phage treatment durations ranging from 0.5 to 168 h, and initial phage titers of 10^5 to 10^12 PFU/ml. In addition, the presence or absence of a food matrix was checked to evaluate the effect of remaining food on the application of bacteriophage.

3.2. Phage application can reduce biofilm formation on stainless steel by 38 %

By obtaining an overall estimation in this meta-analysis, as the main finding, it showed that bacteriophage treatment caused a significant average reduction of 38 % (R* = 0.620) in bacterial count of foodborne pathogens within biofilm formed on stainless steel. It is crucial to consider the results of this meta-analysis in the context of other relevant studies, as the efficacy of phage biocontrol may vary based on the individual strains of the foodborne pathogen and the conditions in which it is present. The subgroup analysis of data conducted in this meta-analysis can help provide more insight into the specific factors influencing the efficacy of bacteriophage treatment (see Table 2). These factors involve phage and bacteria intrinsic features, environmental aspects, and treatment situations such as exposure duration. These factors are important to consider when evaluating the potential use of bacteriophage for controlling biofilm formation on stainless steel surfaces. Therefore, each part will be discussed in detail in the following sections.

3.3. Subgroup analysis

3.3.1. Bacteria

Biofilm formation represents a significant challenge within the food industry, as it substantially impedes hygiene protocols by reducing thermal conduction, obstructing conduits, clogging filtration systems, and inducing surface degradation [114–116]. In addition to technical issues, forming biofilms by foodborne pathogens engenders a multitude of diseases and associated health implications [117]. This meta-analysis has explored the potential of bacteriophages against different biofilm-forming foodborne bacteria. Among these pathogens, *C. sakazakii*, *E. coli*, S. *aureus*, *P. fluorescens*, *P. aeruginosa,* and *L. monocytogenes* contribute significantly to food contamination. These bacteria can form biofilms on food contact surfaces, making their eradication difficult and posing a health risk to consumers [65,97,107,112,118,66].

The findings of the efficacy of bacteriophages against formed *E. coli* and *Salmonella* biofilms on stainless steel revealed a reduction of 43.4 % (R* = 0.566) and 33 % (R* = 0.670) in the biofilm, respectively. The prevalence of *E. coli* contamination is frequently associated with the meat industry, primarily due to its ability to adhere to surfaces in slaughterhouse environments that come into direct contact with meat products [119,120]. Considering that stainless steel is the primary material used in slaughtering and meat manipulation surfaces, STEC can form biofilm on it, leading to cross-contamination. Shiga-toxigenic *Escherichia coli* (STEC) strains can cause serious complications such as hemolytic uremic syndrome, hemorrhagic colitis, and thrombotic thrombocytopenic purpura [121,122]. Due to poor hygiene practices and procedures in meat processing plants, *E. coli* contamination is prevalent in the food industry. Just like *E. coli*, *Salmonella* spp. are described as being environmentally persistent and able to form biofilms on the surfaces and equipment in the food industry, making this a hazardous situation for human health [123–125]. It has been proven that standard cleaning will not be effective against previously formed biofilms. Therefore, this can represent the importance of finding alternatives for combating biofilms [100,126–128].

Studies that applied bacteriophages for biofilm reduction of *L. monocytogenes* and *S. aureus* showed a reduction of 30.2 % (R* = 0.698) and 42.4 % (R* = 0.576) in biofilms formed on stainless steel, respectively. Listeriosis caused by *L. monocytogenes* is a significant foodborne illness affecting vulnerable groups, including pregnant women, neonates, immunocompromised individuals, and the elderly [129,130]. Its ability to cross various human body barriers, including blood, brain, intestine, and placenta, contributes to the severity of the disease with a mortality rate of 20–30 % [131,132]. *L. monocytogenes* can survive and form biofilms at refrigeration temperatures and may resist disinfectants, contributing to its persistence in food-associated environments and subsequent transmission to humans [133]. *S. aureus*, characterized as an opportunistic pathogen, can infiltrate the food chain to form biofilms on surfaces within food industry facilities and consequently infect humans and animals. This pathogenic species significantly contributes to numerous instances of food poisoning [134,135].

The subgroup analysis of bacterial type showed a significant reduction of 41.3 % (R* = 0.587) in the biofilm of *C. sakazakii* on stainless steel. Various food products, including cereals, flour, dairy, fermented and ready-to-eat products, may be contaminated with *C. sakazakii* [136,137]. If an infant consumes contaminated infant formula, they may become infected with *C. sakazakii*, resulting in enterocolitis, meningitis, or even septicemia, with mortality rates ranging from 50 to 80 %. The ability of *C. sakazakii* to form biofilms depends on its survival on food-contact surfaces [138,139]. Conventional treatments, such as thermal and chemical treatments, have some disadvantages. For instance, they may negatively impact the nutritional value of dairy products and cause environmental and human health problems [140–142]. In addition, using other controlling methods like radiation leaves adverse effects on the customer's health and affects their acceptance, or ultrasound has a low decontamination capacity [143,144,145].

The subgroup analysis for *Pseudomonas* spp. it has revealed a biofilm reduction of 38.2 % (R* = 0.618) after phage treatment. *P. aeruginosa*, as another opportunistic bacterium, can form biofilm on moist surfaces, which means it can be protected from most current antimicrobial agents [146,147]. *P. aeruginosa* can cause infections in immunocompromised individuals and is considered a problem in hospitals and thermal spas by colonization in vulnerable people's wounds, ears, eyes, lungs, and urinary tracts [148–151]. In addition, *P. aeruginosa* is a serious foodborne pathogen that can contaminate various foods and make resistant forms of biofilm in areas with a lower ability to clean during sanitization and lead to human infection [152]. *Pseudomonas fluorescens* is a spoilage-causing bacterium in various food-related environments [152,153]. It is one of the most commonly isolated psychrotrophic bacteria, particularly in the dairy industry. It can produce heat-stable extracellular lipases, proteases, and lecithinases, making it survive thermal processings resulting in spoilage [154,155]. *P. fluorescens* is recognized as a major bacteria associated with the spoilage of fresh poultry, and some of its strains can assist with the colonization of *L. monocytogenes* on inert surfaces and protect it from disinfectants [156–158]. Refrigerated meat products are particularly susceptible to spoilage by *P. fluorescens*, which results in changes in appearance and off-odor [159,160].

B biofilms formed by these kinds of foodborne pathogens burden food safety since they protect biofilm-forming bacteria from sanitizers and can damage equipment. As such, it is critical to find ways to eliminate them.

Table 1
Characteristics of included studies.

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
Bumunang, E. W [96].	*Escherichia coli* O154: H10	1	22	24	3	13	5.36	3.26	0.07	0.93	3	3	NO
	Escherichia coli O154: H10	1	22	48	3	13	6.54	5.4	0.17	0.27	3	3	NO
	Escherichia coli O154: H10	1	22	72	3	13	7.01	5.71	0.07	0.19	3	3	NO
	Escherichia coli O113: H21	1	22	24	3	13	6.24	3.7	0.33	0.1	3	3	NO
	Escherichia coli O113: H21	1	22	48	3	13	6	5.7	0.22	0.95	3	3	NO
	Escherichia coli O113: H21	1	22	72	3	13	5.4	4.42	0.15	0.18	3	3	NO
Islam, M. S [97].	*Salmonella* ATCC 14028	3	30	72	24	7	8.7	3.2	0.13	0	5	5	NO
	Salmonella ATCC 14028	3	30	72	24	8	8.7	2.28	0.13	0.4	5	5	NO
	S. Typhimurium ATCC 14028 and *S.* Enteritidis ATCC 13076	3	30	72	24	7	8.91	3.68	0.05	0.74	5	5	NO
	S. Typhimurium ATCC 14028 and *S.* Enteritidis ATCC 13076	3	30	72	24	8	8.91	2.91	0.05	0.73	5	5	NO
Kim, H. S [98].	*Cronobacter sakazakii*	1	20	24	2	–	6.12	5.34	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	20	24	4	–	6.12	3.31	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	20	24	6	–	6.12	2.81	0.15	0.23	3	3	infant formula milk
	Cronobacter sakazakii	1	30	24	2	–	6.12	5.53	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	30	24	4	–	6.12	4.68	0.15	0.1	3	3	infant formula milk
	Cronobacter sakazakii	1	30	24	6	–	6.12	3.1	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	40	24	2	–	6.12	5.5	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	40	24	4	–	6.12	4.7	0.15	0.1	3	3	infant formula milk
	Cronobacter sakazakii	1	40	24	6	–	6.12	3.23	0.15	0.1	3	3	infant formula milk

(*continued on next page*)

Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Cronobacter sakazakii	1	20	24	2	–	6.1	5.05	0.15	0.14	3	3	infant formula milk
	Cronobacter sakazakii	1	20	24	4	–	6.1	3.72	0.15	0.2	3	3	infant formula milk
	Cronobacter sakazakii	1	20	24	6	–	6.1	2.33	0.15	0.33	3	3	infant formula milk
	Cronobacter sakazakii	1	30	24	2	–	6.1	5.02	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	30	24	4	–	6.1	4.38	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	30	24	6	–	6.1	3.09	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	40	24	2	–	6.1	5.51	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	40	24	4	–	6.1	4.38	0.15	0	3	3	infant formula milk
	Cronobacter sakazakii	1	40	24	6	–	6.1	3.69	0.15	0.2	3	3	infant formula milk
Sadekuzzaman, M [99].	*S.* Enteritidis	2	30	72	2	8	5.28	1.92	0.2	0.1	3	3	NO
	S. Enteritidis	2	10	144	2	8	3.26	0	0.23	0	3	3	NO
	S. Typhimurium	2	30	72	2	8	5.1	2.1	0.21	0.12	3	3	NO
	S. Typhimurium	2	10	144	2	8	3.22	0	0.25	0	3	3	NO
Gong, C [100].	10 *Salmonella* strain	6	23	6	6	9	7.11	6	0.38	0.33	2	2	NO
	10 *Salmonella* strain	6	23	6	48	9	7.65	5.13	0.35	0.26	2	2	NO
	10 *Salmonella* strain	6	23	6	96	9	6.97	4.23	0.31	0.24	2	2	NO
	10 *Salmonella* strain	6	23	6	168	9	4.93	2	0.26	0.13	2	2	NO
	S. Typhimurium strain 8243	6	23	6	6	9	6.97	5.46	0.34	0.25	2	2	NO
	S. Typhimurium strain 8243	6	23	6	48	9	7.06	4.49	0.34	0.25	2	2	NO
	S. Typhimurium strain 8243	6	23	6	96	9	5.97	3.24	0.3	0.15	2	2	NO
	S. Typhimurium strain 8243	6	23	6	168	9	3.78	0.77	0.16	0.05	2	2	NO
Montanez-Izquierdo, V. Y [101].	*Listeria monocytogenes*	1	23	72	2	5	5.32	5.27	0.19	0	6	6	NO
	Listeria monocytogenes	1	23	72	8	5	5.13	5.25	0.22	0.16	6	6	NO
	Listeria monocytogenes	1	23	72	24	5	5.08	5.4	0.26	0	6	6	NO
	Listeria monocytogenes	1	23	72	48	5	4.85	4.89	0.33	0.19	6	6	NO
	Listeria monocytogenes	1	23	72	2	6	5.32	5.22	0.19	0	6	6	NO
	Listeria monocytogenes	1	23	72	8	6	5.13	5.01	0.22	0.42	6	6	NO
	Listeria monocytogenes	1	23	72	24	6	5.08	0.07	0.26	0	6	6	NO
	Listeria monocytogenes	1	23	72	48	6	4.85	0.19	0.33	0.23	6	6	NO
	Listeria monocytogenes	1	23	72	2	7	5.32	5.22	0.19	0	6	6	NO

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Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Listeria monocytogenes	1	23	72	8	7	5.13	1.81	0.22	0.3	6	6	NO
	Listeria monocytogenes	1	23	72	24	7	5.08	0	0.26	0	6	6	NO
	Listeria monocytogenes	1	23	72	48	7	4.85	0	0.33	0	6	6	NO
	Listeria monocytogenes	1	23	72	2	8	5.32	5.04	0.19	0	6	6	NO
	Listeria monocytogenes	1	23	72	8	8	5.13	2.02	0.22	0.37	6	6	NO
	Listeria monocytogenes	1	23	72	24	8	5.08	0.16	0.26	0	6	6	NO
	Listeria monocytogenes	1	23	72	48	8	4.85	0	0.33	0	6	6	NO
	Listeria monocytogenes	1	23	72	2	5	5.22	5.22	0.14	0.1	6	6	NO
	Listeria monocytogenes	1	23	72	8	5	5.61	5.58	0.25	0	6	6	NO
	Listeria monocytogenes	1	23	72	24	5	5.28	5.47	0.16	0.17	6	6	NO
	Listeria monocytogenes	1	23	72	48	5	5.52	5.3	0.3	0.19	6	6	NO
	Listeria monocytogenes	1	23	72	2	6	5.22	5.47	0.14	0	6	6	NO
	Listeria monocytogenes	1	23	72	8	6	5.61	5.35	0.25	0.34	6	6	NO
	Listeria monocytogenes	1	23	72	24	6	5.28	3.99	0.16	0.3	6	6	NO
	Listeria monocytogenes	1	23	72	48	6	5.52	3.72	0.3	0.24	6	6	NO
	Listeria monocytogenes	1	23	72	2	7	5.22	5.14	0.14	0	6	6	NO
	Listeria monocytogenes	1	23	72	8	7	5.61	4.53	0.25	0.27	6	6	NO
	Listeria monocytogenes	1	23	72	24	7	5.28	3.59	0.16	0.23	6	6	NO
	Listeria monocytogenes	1	23	72	48	7	5.52	3.85	0.3	0.28	6	6	NO
	Listeria monocytogenes	1	23	72	2	8	5.22	4.96	0.14	0.13	6	6	NO
	Listeria monocytogenes	1	23	72	8	8	5.61	4.16	0.25	0.24	6	6	NO
	Listeria monocytogenes	1	23	72	24	8	5.28	4.11	0.16	0.19	6	6	NO
	Listeria monocytogenes	1	23	72	48	8	5.52	4.2	0.3	0.16	6	6	NO
Soni, K. A [102].	*Listeria monocytogenes*	1	22	48	24	9	6.92	1.52	0.1	1.51	3	3	NO
	Listeria monocytogenes	1	22	168	24	9	6.53	2.94	0.16	1.42	3	3	NO
Sadekuzzaman, M [103].	*Escherichia coli* O157: H7 ATCC43889	1	30	72	2	8	5.16	1.7	0.18	0.35	3	3	NO
	Escherichia coli O157: H7 NCCP 11090	1	30	72	2	8	6.18	2.37	0.24	0.34	3	3	NO
	Escherichia coli O157: H7 NCCP 14541	1	30	72	2	8	5.39	1.82	0.2	0.3	3	3	NO
	Escherichia coli O157: H7 ATCC43889	1	10	144	2	8	4.41	1.41	0.22	0.28	3	3	NO
	Escherichia coli O157: H7 NCCP 11090	1	10	144	2	8	5.51	1.91	0.3	0.24	3	3	NO
	Escherichia coli O157: H7 NCCP 14541	1	10	144	2	8	4.81	1.62	0.14	0.15	3	3	NO
Gong, C [104].	*Citrobacter freundii* and *Hafnia alvei*	9	30	48	2	8	6.48	6.11	0.33	0.31	2	2	NO
	Citrobacter freundii and *Hafnia alvei*	9	30	48	4	8	6.65	5.67	0.34	0.29	2	2	NO
	Citrobacter freundii and *Hafnia alvei*	9	30	48	6	8	6.66	4.65	0.34	0.24	2	2	NO
Arachchi, G. J. G [105].	*Listeria monocytogenes*	3	15	168	1	9	3.9	2.8	66	0.05	4	4	NO
	Listeria monocytogenes	3	15	168	2	9	2.63	2.38	0.2	0.19	4	4	NO
	Listeria monocytogenes	3	15	168	3	9	2.33	1.43	0.17	0.12	4	4	NO
	Listeria monocytogenes	1	15	168	1	9	4.21	3.7	0.2	0.14	4	4	NO
	Listeria monocytogenes	1	15	168	2	9	3.2	2.5	0.07	0.05	4	4	NO
	Listeria monocytogenes	1	15	168	3	9	2.5	2.2	0.08	0.31	4	4	NO
	Listeria monocytogenes	1	15	168	1	9	4.27	2.47	0.06	0.11	4	4	NO
	Listeria monocytogenes	1	15	168	2	9	2.78	2.5	0.06	0.09	4	4	NO
	Listeria monocytogenes	1	15	168	3	9	2.08	1.07	0.32	0.21	4	4	NO

(*continued on next page*)

Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
Duc, H. M [106].	*Staphylococcus aureus*	1	37	24	2	10	6.52	3.93	0.29	0.4	3	3	NO
	Staphylococcus aureus	1	37	24	4	10	6.61	3.5	0.31	0.48	3	3	NO
	Staphylococcus aureus	1	37	24	6	10	6.56	2.96	0.24	0.33	3	3	NO
	Staphylococcus aureus	1	24	24	2	10	6.47	4.1	0.4	0.15	3	3	NO
	Staphylococcus aureus	1	24	24	4	10	6.57	4.2	0.35	0.33	3	3	NO
	Staphylococcus aureus	1	24	24	6	10	6.68	4.08	0.15	0.33	3	3	NO
Wang, L [66].	*Cronobacter sakazakii*	1			1	9	4.51	3.57	0.12	0.23	3	3	NO
	Cronobacter sakazakii	1			2	9	4.36	1.62	0.07	0.14	3	3	NO
	Cronobacter sakazakii	1			3	9	4.64	1.56	0.15	0.1	3	3	NO
	Cronobacter sakazakii	1			4	9	4.45	1.37	0.15	0.36	3	3	NO
	Cronobacter sakazakii	1			5	9	4.51	1.53	0.16	0.13	3	3	NO
	Cronobacter sakazakii	1			6	9	4.36	1.58	0.23	0.17	3	3	NO
Magin, V [107].	*Pseudomonas aeruginosa* PAO1	1	30	24	14	10	7.16	5.69	0.46	0.39	3	3	NO
	Pseudomonas aeruginosa PAO1	1	30	24	14	10	6.15	5.11	0.6	0.55	3	3	NO
	Pseudomonas aeruginosa PAO1	1	30	24	14	10	7.12	6.81	68	0.27	3	3	NO
	Pseudomonas aeruginosa PAO1	1	30	24	14	10	7.13	6.6	0.41	0.22	3	3	NO
	Pseudomonas aeruginosa D1	1	30	24	14	10	6.9	5.95	0.25	0.37	3	3	NO
	Pseudomonas aeruginosa D1	1	30	24	14	10	6.68	5.3	0.24	0.84	3	3	NO
	Pseudomonas aeruginosa D1	1	30	24	14	10	8.1	6.5	0.55	0.47	3	3	NO
	Pseudomonas aeruginosa D1	1	30	24	14	10	7.6	6.2	0.21	0.51	3	3	NO
Sadekuzzaman, M [108].	*L. monocytogenes* ATCC 19113		30	72	2	8	6.85	4.9	0.15	0.19	3	3	NO
	L. monocytogenes ATCC 19115		30	72	2	8	7.19	5.13	0.28	0.35	3	3	NO
	L. monocytogenes ATCC 13932		30	72	2	8	6.77	4.82	0.15	0.13	3	3	NO
	L. monocytogenes ATCC 19113		10	144	2	8	5.75	3.28	0.19	0.09	3	3	NO
	L. monocytogenes ATCC 19115		10	144	2	8	5.87	3.58	0.13	0.14	3	3	NO
	L. monocytogenes ATCC 13932		10	144	2	8	5.5	3.09	0.16	0.17	3	3	NO
Gonzalez-Gomez, J. P [65].	*Escherichia coli* MGA-EC-21	1	22	2	1	9	4.98	1.79	0.12	0.31	3	3	NO
	Escherichia coli MGA-EC-21	1	22	2	1	9	4.98	1.49	0.12	0.24	3	3	NO
	Escherichia coli MGA-EC-21	1	22	2	1	9	4.98	2.16	0.12	0.54	3	3	NO
	Escherichia coli MGA-EC-21	3	22	2	1	9	4.98	1.08	0.12	0.32	3	3	NO
	Escherichia coli MGA-EC-21	1	22	24	1	9	7.8	3.78	0.33	0.38	3	3	NO
	Escherichia coli MGA-EC-21	1	22	24	1	9	7.8	4.21	0.33	0.57	3	3	NO

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Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Escherichia coli MGA-EC-21	1	22	24	1	9	7.8	2.58	0.33	0.42	3	3	NO
	Escherichia coli MGA-EC-21	3	22	24	1	9	7.8	3.38	0.33	0.53	3	3	NO
	Escherichia coli MGA-EC-21	1	22	48	1	9	7.91	2.81		0.44	3	3	NO
	Escherichia coli MGA-EC-21	1	22	48	1	9	7.91	3.41		0.42	3	3	NO
	Escherichia coli MGA-EC-21	1	22	48	1	9	7.91	3.37		0.45	3	3	NO
	Escherichia coli MGA-EC-21	3	22	48	1	9	7.91	3.56		0.31	3	3	NO
	Escherichia coli MGA-EC-25	1	22	2	1	9	6	3.4	0	0.49	3	3	NO
	Escherichia coli MGA-EC-25	1	22	2	1	9	6	2.93	0	0.64	3	3	NO
	Escherichia coli MGA-EC-25	1	22	2	1	9	6	3.61	0	0.18	3	3	NO
	Escherichia coli MGA-EC-25	3	22	2	1	9	6	3.13	0	0.56	3	3	NO
	Escherichia coli MGA-EC-25	1	22	24	1	9	8.5	2.04	0.29	0.41	3	3	NO
	Escherichia coli MGA-EC-25	1	22	24	1	9	8.5	2.45	0.29	0.49	3	3	NO
	Escherichia coli MGA-EC-25	1	22	24	1	9	8.5	1.76	0.29	0.53	3	3	NO
	Escherichia coli MGA-EC-25	3	22	24	1	9	8.5	1.92	0.29	0.15	3	3	NO
	Escherichia coli MGA-EC-25	1	22	48	1	9	8.01	2.71	0.52	0.48	3	3	NO
	Escherichia coli MGA-EC-25	1	22	48	1	9	8.01	2.36	0.52	0.59	3	3	NO
	Escherichia coli MGA-EC-25	1	22	48	1	9	8.01	2.57	0.52	0.19	3	3	NO
	Escherichia coli MGA-EC-25	3	22	48	1	9	8.01	2.48	0.52	0.6	3	3	NO
	Escherichia coli MGA-EC-27	1	22	2	1	9	5.49	2.16	0.18	0.28	3	3	NO
	Escherichia coli MGA-EC-27	1	22	2	1	9	5.49	2.06	0.18	0.31	3	3	NO
	Escherichia coli MGA-EC-27	1	22	2	1	9	5.49	1.69	0.18	0.31	3	3	NO
	Escherichia coli MGA-EC-27	3	22	2	1	9	5.49	1.95	0.18	0.47	3	3	NO
	Escherichia coli MGA-EC-27	1	22	24	1	9	8	2.01	0.44	0.4	3	3	NO
	Escherichia coli MGA-EC-27	1	22	24	1	9	8	2.02	0.44	0.29	3	3	NO
	Escherichia coli MGA-EC-27	1	22	24	1	9	8	2.31	0.44	0.44	3	3	NO
	Escherichia coli MGA-EC-27	3	22	24	1	9	8	2.89	0.44	0.53	3	3	NO

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Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Escherichia coli MGA-EC-27	1	22	48	1	9	7.91	2.31		0.3	3	3	NO
	Escherichia coli MGA-EC-27	1	22	48	1	9	7.91	3.38		0.34	3	3	NO
	Escherichia coli MGA-EC-27	1	22	48	1	9	7.91	2.46		0.32	3	3	NO
	Escherichia coli MGA-EC-27	3	22	48	1	9	7.91	4.66		0.23	3	3	NO
	Escherichia coli MGA-EC-21	1	22	2	1	8	4.98	4	0.12	0.29	3	3	NO
	Escherichia coli MGA-EC-21	1	22	2	1	8	4.98	4.29	0.12	0.45	3	3	NO
	Escherichia coli MGA-EC-21	1	22	2	1	8	4.98	3.63	0.12	0.39	3	3	NO
	Escherichia coli MGA-EC-21	3	22	2	1	8	4.98	4.03	0.12	0.27	3	3	NO
	Escherichia coli MGA-EC-21	1	22	24	1	8	7.8	5.77	0.33	0.43	3	3	NO
	Escherichia coli MGA-EC-21	1	22	24	1	8	7.8	5.1	0.33	0.45	3	3	NO
	Escherichia coli MGA-EC-21	1	22	24	1	8	7.8	5.51	0.33	0.72	3	3	NO
	Escherichia coli MGA-EC-21	3	22	24	1	8	7.8	5.9	0.33	0.53	3	3	NO
	Escherichia coli MGA-EC-21	1	22	48	1	8	7.91	5.61		0.24	3	3	NO
	Escherichia coli MGA-EC-21	1	22	48	1	8	7.91	5.78		0.29	3	3	NO
	Escherichia coli MGA-EC-21	1	22	48	1	8	7.91	5.45		0.33	3	3	NO
	Escherichia coli MGA-EC-21	3	22	48	1	8	7.91	5.83		0.31	3	3	NO
	Escherichia coli MGA-EC-25	1	22	2	1	8	6	4.08	0.12	0.48	3	3	NO
	Escherichia coli MGA-EC-25	1	22	2	1	8	6	4.75	0.12	0.7	3	3	NO
	Escherichia coli MGA-EC-25	1	22	2	1	8	6	4.5	0.12	0.68	3	3	NO
	Escherichia coli MGA-EC-25	3	22	2	1	8	6	3.92	0.12	0.83	3	3	NO
	Escherichia coli MGA-EC-25	1	22	24	1	8	8.5	6.1	0.29	0.34	3	3	NO
	Escherichia coli MGA-EC-25	1	22	24	1	8	8.5	6.37	0.29	0.44	3	3	NO
	Escherichia coli MGA-EC-25	1	22	24	1	8	8.5	5.98	0.29	0.57	3	3	NO
	Escherichia coli MGA-EC-25	3	22	24	1	8	8.5	6.03	0.29	0.69	3	3	NO
	Escherichia coli MGA-EC-25	1	22	48	1	8	8.01	6.19	0.52	0.69	3	3	NO
	Escherichia coli MGA-EC-25	1	22	48	1	8	8.01	6.44	0.52	0.39	3	3	NO

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Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Escherichia coli MGA-EC-25	1	22	48	1	8	8.01	5.56	0.52	0.47	3	3	NO
	Escherichia coli MGA-EC-25	3	22	48	1	8	8.01	6.57	0.52	0.43	3	3	NO
	Escherichia coli MGA-EC-27	1	22	2	1	8	5.49	3.92	0.18	0.42	3	3	NO
	Escherichia coli MGA-EC-27	1	22	2	1	8	5.49	3.15	0.18	0.34	3	3	NO
	Escherichia coli MGA-EC-27	1	22	2	1	8	5.49	2.63	0.18	0.61	3	3	NO
	Escherichia coli MGA-EC-27	3	22	2	1	8	5.49	3.09	0.18	0.25	3	3	NO
	Escherichia coli MGA-EC-27	1	22	24	1	8	8	5.6	0.44	0.61	3	3	NO
	Escherichia coli MGA-EC-27	1	22	24	1	8	8	5.72	0.44	0.49	3	3	NO
	Escherichia coli MGA-EC-27	1	22	24	1	8	8	5.59	0.44	0.52	3	3	NO
	Escherichia coli MGA-EC-27	3	22	24	1	8	8	5.7	0.44	0.53	3	3	NO
	Escherichia coli MGA-EC-27	1	22	48	1	8	7.91	5.71		0.18	3	3	NO
	Escherichia coli MGA-EC-27	1	22	48	1	8	7.91	5.78		0.64	3	3	NO
	Escherichia coli MGA-EC-27	1	22	48	1	8	7.91	5.94		0.23	3	3	NO
	Escherichia coli MGA-EC-27	3	22	48	1	8	7.91	5.74		0.32	3	3	NO
Gutierrez, D [109].	*Listeria monocytogenes* Lm1		12	72	4	7	6.1	5.32	0.39	0.03	3	3	NO
	Listeria monocytogenes Lm2		12	72	4	7	6	5.63	0.52	0.26	3	3	NO
	Listeria monocytogenes Lm3		12	72	4	7	6.21	5.06	0.06	0.08	3	3	NO
	Listeria monocytogenes Lm22		12	72	4	7	6.2	5.88	0.22	0.15	3	3	NO
	Listeria monocytogenes Lm37		12	72	4	7	5.45	4.7	0.13	0.19	3	3	NO
	Listeria monocytogenes Lm41		12	72	4	7	5.47	5.11	0.1	0.14	3	3	NO
	Listeria monocytogenes S2		12	72	4	7	6.2	5.81	0.05	0.12	3	3	NO
	Listeria monocytogenes S12-1		12	72	4	7	6.8	5.25	0.13	0.14	3	3	NO
	Listeria monocytogenes S4-2		12	72	4	7	6.15	5.35	0.03	0.28	3	3	NO
	Listeria monocytogenes S7-2		12	72	4	7	5.54	5.16	0.06	0.05	3	3	NO
	Listeria monocytogenes INIA 2530		12	72	4	7	6.29	5.35	0.23	0.33	3	3	NO
	Listeria monocytogenes Lm22	1	12	72	4	9	6.2	2.52	0.22	0.5	3	3	NO

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Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Listeria monocytogenes Lm37	1	12	72	4	9	5.45	2.74	0.13	0.6	3	3	NO
	Listeria monocytogenes S2	1	12	72	4	9	6.2	1.25	0.05	0.35	3	3	NO
	Listeria monocytogenes S12-1	1	12	72	4	9	6.8	3.12	0.13	0.45	3	3	NO
	Listeria monocytogenes S4-2	1	12	72	4	9	6.15	1.78	0.03	0.39	3	3	NO
	Listeria monocytogenes S7-2	1	12	72	4	9	5.54	2.47	0.06	0.32	3	3	NO
	Listeria monocytogenes INIA 2530	1	12	72	4	9	6.29	0.1	0.23	0	3	3	NO
Wang, C [110].	*Escherichia coli* (STEC) O145	1	24	24	3	10.30	4.7	1.8	0.2	1.4	3	3	NO
	Escherichia coli (STEC) O145	1	24	48	3	10.30	5.4	3.5	0.2	0.5	3	3	NO
	Escherichia coli (STEC) O145	1	24	72	3	10.30	5.8	3.9	0.1	0.7	3	3	NO
Chaitiemwong, N [111].	*Listeria monocytogenes*	1	15	168	0.5	9	5.51	0	0.27	0	2	2	milk
	Listeria monocytogenes	1	15	168	0.5	9	5	0	0	0	2	2	ham
	Listeria monocytogenes	1	15	168	0.5	9	5.77	0	0.17	0	2	2	fish
	Listeria monocytogenes	1	15	168	0.5	9	4.96	0	0.12	0	2	2	vegetable
	Listeria monocytogenes	1	15	168	0.5	9	7.61	6.73	0.07	0.04	2	2	milk
	Listeria monocytogenes	1	15	168	0.5	9	6.81	4.86	0.37	1.35	2	2	ham
	Listeria monocytogenes	1	15	168	0.5	9	7.41	5.69	0.1	0.38	2	2	fish
	Listeria monocytogenes	1	15	168	0.5	9	6.6	5.46	0.03	0.41	2	2	vegetable
Sillankorva, S [112].	*Pseudomonas fluorescens*	1	30	24	4	7	6.19	3.52	0.19	0.23	6	6	NO
	Pseudomonas fluorescens	1	30	72	4	7	7.04	3.08	0.26	0.24	6	6	NO
	Pseudomonas fluorescens	1	30	120	4	7	7.5	3.72	0.12	0.12	6	6	NO
	Pseudomonas fluorescens	1	30	168	4	7	8.49	3.17	0.11	0.2	6	6	NO
	Pseudomonas fluorescens	1	30	24	4	7	5	2.7	0.27	0.29	6	6	NO
	Pseudomonas fluorescens	1	30	72	4	7	7.26	3.1	0.19	0.21	6	6	NO
	Pseudomonas fluorescens	1	30	120	4	7	7.92	4.53	0.05	0.17	6	6	NO
	Pseudomonas fluorescens	1	30	168	4	7	7.99	2.5	0.19	0.11	6	6	NO
	Pseudomonas fluorescens	1	30	24	4	7	4.1	2.98	0	0.1	6	6	NO
	Pseudomonas fluorescens	1	30	72	4	7	6.1	3.25	0.05	0.11	6	6	NO
Sharma, M [113].	*Escherichia coli* O157: H7 ATCC 43985	1	4	24	24	9	4.56	3.02	0.17	0.17	8	8	NO
	Escherichia coli O157: H7 ATCC 43985	1	4	24	48	9	4.44	3.12	0.11	0.16	8	8	NO
	Escherichia coli O157: H7 ATCC 43985	1	4	24	72	9	3.92	3.19	0.14	0.17	8	8	NO

(*continued on next page*)

Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Escherichia coli O157: H7 ATCC 43985	1	4	24	96	9	3.77	3.36	0.13	0.16	8	8	NO
	Escherichia coli O157: H7 ATCC 43985	1	4	24	24	9	4.56	2.82	0.17	0.26	8	8	NO
	Escherichia coli O157: H7 ATCC 43985	1	4	24	48	9	4.44	2.73	0.11	0.26	8	8	NO
	Escherichia coli O157: H7 ATCC 43985	1	4	24	72	9	3.92	3.8	0.14	0.25	8	8	NO
	Escherichia coli O157: H7 ATCC 43985	1	4	24	96	9	3.77	3.65	0.13	0.25	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	24	9	3.63	3.95	0.15	0.16	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	48	9	4.12	3.7	0.14	0.18	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	72	9	3.96	3.87	0.14	0.18	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	96	9	3.93	0.45	0.12	0.18	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	24	9	3.63	4.34	0.15	0.15	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	48	9	4.12	3.98	0.14	0.14	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	72	9	3.96	3.94	0.14	0.13	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	96	9	3.93	3.54	0.12	0.15	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	24	8.47	2.92	2.07	0.19	0.11	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	48	8.47	3.11	2.26	0.18	0.15	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	72	8.47	2.17	2.45	0.13	0.14	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	96	8.47	2.18	1.93	0.19	0.14	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	24	8.47	2.92	2.78	0.19	0.14	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	48	8.47	3.11	2.55	0.18	0.14	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	72	8.47	2.17	1.97	0.13	0.15	8	8	NO
	Escherichia coli O157: H7 FRIK ATCC 43895	1	4	24	96	8.47	2.18	2.59	0.19	0.14	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	24	8.47	2.95	2.32	0.2	0.1	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	48	8.47	2.83	2.56	0.2	0.1	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	72	8.47	2.63	2.52	0.11	0.1	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	96	8.47	1.86	2.3	0.2	0.11	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	24	8.47	2.95	2.58	0.2	0.1	8	8	NO

(*continued on next page*)

Table 1 (*continued*)

Study	Type of bacteria	Number of phages	Temperature of treatment with phage	Biofilm age (h)	Duration of phage treatment(h)	Initial phage titer (PFU/ml)	Bacterial count in control group (log)	Bacterial count in phage treatment group (log)	SD/SE control group (log)	SD/SE treatment group (log)	N control group	N treatment group	Food matrix
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	48	8.47	2.83	2.28	0.2	0.1	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	72	8.47	2.63	2.83	0.11	0.1	8	8	NO
	Escherichia coli O157: H7 FRIK 816-3	1	4	24	96	8.47	1.86	2.55	0.2	0.1	8	8	NO

Table 2
Overall and subgroup analysis.

Subgroup	No. of trials	R* (95 % CI)	*P* value	I^2 (%)	Q-statistic (*P*)
Biofilm old					
≤6 h	32	0.475 (0.442–0.511)	<0.001	86.9	<0.001
>6 h	227	0.654 (0.631–0.678)	<0.001	98.3	<0.001
Duration of phage treatment					
<12 h	215	0.599 (0.578–0.635)	<0.001	98.4	<0.001
≥12 h	50	0.668 (0.592–0.730)	<0.001	98.2	<0.001
Initial phage titer					
≤ 10^7 (PFU/ml)	77	0.760 (0.710–0.813)	<0.001	99.2	<0.001
> 10^7 (PFU/ml)	170	0.559 (0.535–0.584)	<0.001	99.2	<0.001
Phage treatment temperature					
≤4 °C	18	0.862 (0.829–0.897)	<0.001	58.4	0.001
>4 to ≤ 25 °C	193	0.628 (0.607–0.649)	<0.001	97.2	<0.001
>25 °C	48	0.589 (0.540–0.643)	<0.001	98.9	<0.001
Number of phage type					
One phage	207	0.626 (0.602–0.651)	<0.001	98.0	<0.001
More than one phage	40	0.528 (0.479–0.582)	<0.001	96.8	<0.001
Type of bacteria					
Escherichia coli	87	0.566 (0.548–0.585)	<0.001	82.5	<0.001
Salmonella spp.	52	0.670 (0.644–0.752)	<0.001	98.4	<0.001
Cronobacter sakazakii	24	0.587 (0.520–0.664)	<0.001	98.3	<0.001
Listeria monocytogenes	75	0.698 (0.666–0.731)	<0.001	96.6	<0.001
Citrobacter freundii and *Hafnia alvei*	3	0.826 (0.697–0.980)	<0.001	75.9	0.016
Staphylococcus aureus	6	0.576 (0.520–0.638)	<0.001	80.6	<0.001
Pseudomonas spp.	18	0.618 (0.548–0.698)	<0.001	99.1	<0.001
Food matrix or not					
In vitro	239	0.628 (0.605–0.652)	<0.001	98.3	<0.001
Food matrix added	26	0.559 (0.506–0.618)	<0.001	98.2	<0.001
Overall estimate	265	0.620 (0.599–0.643)	<0.001	98.3	<0.001

Abbreviations: CI, confidence interval; R*, weighted overall response ratio.

3.3.2. Biofilm age

An analysis based on the subgroup of biofilm age revealed that the reduction of foodborne pathogens after applying bacteriophages on biofilms at the age of >6 h and ≤6 h was calculated to be 52.5 % (R* = 0.475) and 34.5 % (R* = 0.654), respectively. Obviously, in the case of older biofilms, the antibacterial effect of phages would be significantly lower by 1.52 folds compared to less mature biofilms. Once a biofilm is formed, it can protect bacterial cells from environmental, antibacterial, and abiotic factors and the immune system. This is achieved by the formation of a mixture of polymeric compounds including polysaccharides, proteins, nucleic acids, and lipids, with dense microcolonies being separated by channels that distribute water, nutrients, oxygen, enzymes, and cellular debris [43,52,115,161–163]. As biofilms age, they can become increasingly resistant to antibacterial agents [164,165]. This can be due to the biofilm matrix, made up of extracellular polymeric substances (EPS), becoming denser and more complex over time, making it harder for antibacterial agents, including bacteriophages, to penetrate and reach the bacteria within the biofilm.

Accordingly, bacterial cells in an old biofilm may be less susceptible to bacteriophage infection. For example, the bacteria within the biofilm can develop mutations or transfer the resistance mechanisms from other bacteria in a mixed biofilm that make them resist phage infection, such as producing bacteriophage-degrading enzymes or blocking the entry of the phage to the cell [166,167].

On the other hand, younger or less mature biofilms may be more susceptible to treatment with bacteriophages since younger biofilms are less complex and may contain lower levels of EPSs, facilitating the penetration and infection of targeted bacteria by phages [168,169]. Therefore, the maturity of the biofilm can substantially weaken the bacteriophage's antibacterial activity, as exponentially growing cells are more vulnerable to environmental stress than those in the stationary phase. Thus, applying bacteriophage at the early stages of biofilm formation can increase its efficiency in controlling the growth and spread of biofilm. Despite all this, foodborne pathogens become more resistant by forming a biofilm on surfaces. Phages still effectively reduce the bacterial counts of biofilms on stainless steel at the age of seven days [111, 105]. However, treatment of old biofilms may require longer exposure times, higher doses of bacteriophages, and/or a combination of different bacteriophages or other treatments to overcome the resistance of the biofilm.

3.3.3. Application of phages individually or in a cocktail

The subgroup analysis based on the number of phage types revealed significant reductions of 37.4 % (R* = 0.626) and 47.2 % (R* = 0.528) in biofilm formation of foodborne pathogens on stainless steel after treatment with one and more than one bacteriophage type, respectively. As a key finding, treating more than one bacteriophage type was more effective (1.26 folds higher) than one bacteriophage type. A considerable body of literature supports the privileges of applying bacteriophage in cocktails against pathogens. Using a phage cocktail, differences in the host range of bacteriophages can provide a broader control of bacterial strains in complicated infections [170]. Therefore, based on the conducted meta-analysis, using a cocktail of bacteriophages for biocontrol of biofilm of foodborne pathogens is more likely to bring a higher bacterial count reduction than individual phage. However, some studies suggest that the phage cocktail has no noticeable effect on reducing the bacterial count of biofilm compared with individual phages [65]. Also, some reports suggest that evaluating each phage before selecting a phage cocktail is a decisive factor for biocontrol and therapy purposes. In addition, applying a phage cocktail implies another positive effect in the biocontrol of pathogens as it suppresses the resistance of bacteria against phage [171,172]. In other words, evaluating each phage before creating a phage cocktail is important for its effectiveness in biocontrol and therapy of specific bacteria, and using a cocktail can also help prevent the development of bacterial resistance to phages. However, different bacterial species may need different numbers of phages to cover most of their trains. A recent study showed the possibility of impairment of *Pseudomonas aeruginosa* inhibition as a host in the case of overuse of various phages in a cocktail preparation [173]. They found that utilizing a mixture of four phages in a cocktail proved more effective than individual phages or a three-phage cocktail. However, the addition of another phage to create a five-phage cocktail resulted in a decrease in the cocktail's efficacy. In this case, receptor binding proteins (RBPs) may contribute by blocking the receptors by more than one phage of a cocktail, reducing the cocktail's antibacterial effect by making receptors unavailable for other phages [173,174]. Therefore, it is crucial to consider different aspects of phage dynamics in a cocktail, such as antagonistic and synergistic interactions, to obtain a more efficient application of bacteriophage as a cocktail of several phages for biocontrol and therapy.

3.3.4. The concentration of phage inoculation

As reported by most of the included studies, we preferred to employ the term phage concentration instead of calculating MOI (multiplicity of infection) since MOI could not be determined based on the available data. The result of the subgrouping meta-analysis based on initial phage titers showed a substantial reduction of 24 % (R* = 0.760) and 44.1 % (R* = 0.559) in biofilm formation of foodborne pathogens on stainless steel after treatment with $\leq 10^7$ PFU/ml and $> 10^7$ PFU/ml, respectively. Following our expectation, phage titers above 10^7 proved more effective (1.83 folds higher) in reducing pathogen count than those of less than $\leq 10^7$ PFU/ml. Findings in this section suggest that using a phage concentration above 7 logs PFU/ml can result in a greater antibacterial effect on biofilms formed on stainless steel. Additionally, there are two concepts, "lysis from within" and "lysis from without," with the first occurring by the injection of the phage genome into the bacterial cell, resulting in more phage production, and the latter by the adsorption of more than 100 phages around the bacterial cell, resulting in the death of the bacteria by abortive infection of phage. Therefore, applying a lower phage concentration is more suitable for applications with enough time since the reduction of host cells depends on the lytic cycle (producing new phage particles), which is time-consuming. Conversely, in the cases with less time, respecting the concept of lysis from without, the phage can kill bacteria quickly and decisively, as adsorption of phage mostly happens in the first few moments of interaction with the bacterial cell [175,176]. Several factors need to be taken into account to determine the concentration of bacteriophages for application, such as bacterial strain susceptibility to phage, contamination level with foodborne bacteria, food matrix (the physical and chemical characteristics of the food product, and presence of antimicrobial compounds, such as preservatives), the environmental conditions (temperature, pH and water activity (aw) and phage stability [177–180]. Also, applying high-titer phage on surfaces increases the probability of phage-bacteria interaction, even at a very low number of bacteria cells, leading to higher bacteria contamination to phage [181]. Based on the present analysis of the pooled data, applying 7 log PFU/ml or higher concentration of phage seems beneficial for controlling biofilm formation of foodborne pathogens on stainless steel. However, its possible disadvantages should be investigated accurately and taken into consideration.

3.3.5. Temperature of treatment

Another subgroup was considered to assess the potential effects of three temperature ranges, including 4 °C and lower, 4 < to ≤25 °C, and more than 25 °C. The results showed a significant reduction of 13.8 % (R^* = 0.862), 37.2 % (R^* = 0.628), and 48.9 % (R^* = 0.589) in the biofilm bacterial counts on stainless steel for the mentioned ranges, respectively. As observed, by increasing temperature from 4 °C to 37 °C, the antibacterial effect of bacteriophage increased significantly. The anti-biofilm effect of phage in temperatures over 25 °C was 3.54 and 1.31 folds higher than temperatures ≤4 °C and 4 < to ≤25 °C, respectively. Temperature is a key factor in biofilm formation in food processing environments [50,182]. Literature indicates that higher temperatures can promote the growth and formation of biofilms, as bacteria can reproduce more quickly [183]. This can be particularly problematic in cooling systems, where warm water creates an ideal biofilm formation environment. On the other hand, [184,184]) showed that most tested *Salmonella* strains formed biofilms at refrigerator temperatures. A protective layer by bacteria at low temperatures confers an increased resilience against cleaning and disinfection measures. Maintaining a proper temperature is an important control measure to prevent biofilm formation in food processing environments [50]. In parallel with regular cleaning and disinfection to remove existing biofilms, this may involve refrigeration or cooling systems to keep temperatures low. As supporting evidence, studies have also shown that bacteriophage biocontrol is more effective at higher temperatures, making the conditions favorable so that bacteriophage can replicate more quickly and spread to other bacterial cells [185,186]. Therefore, it can be claimed that bacteriophages are highly active as long as bacterial cells are active, so phages can replicate and kill them within the biofilm matrix. Although the cold temperature has a positive effect on the inhibition of

biofilm formation, some bacteria can adapt to cold environments and can continue to grow and form biofilms at low temperatures [133,187,188]. Additionally, biofilms that have already formed may remain stable at cold temperatures, which makes them difficult to remove [133,187,188]. Therefore, even though cold temperatures can prevent biofilm formation, they should not be relied upon as the sole control measure. The literature demonstrates that the efficiency of phage is associated with the maturity of biofilm since the exponentially growing bacteria are more susceptible to environmental stress than stationary bacteria because of their high energy demand and intense regulation of growth components [189], and this explains why bacteriophage is less effective in the temperatures lower than 5 °C [190]. Lower temperatures may reduce the bacteriophage infection and replication rate but may not eliminate their activity.

3.3.6. Duration of phage treatment

The result of subgroup analysis based on different treatment duration demonstrated a substantial reduction of 33.2 % (R* = 0.668) and 40.1 % (R* = 0.599) in the bacterial counts of biofilms formed by foodborne pathogens on stainless steel after <12 h and ≥12 h treatment with phage, respectively. The biofilm reduction after application of bacteriophage for 12 h or more was 1.2 folds higher than those treated for less than 12 h. A prolonged treatment time with phage can lead to a higher bacterial reduction within the biofilm. The optimization of phage application against foodborne bacteria that form biofilms necessitates meticulous consideration of treatment durations, as it significantly influences the efficacy of phage-based antibacterial interventions [175,191]. In general, longer treatment times may be required to treat biofilms with bacteriophages effectively [192]. For example, in a study by Ref. [100], a cocktail of six phages reduced the biofilm cell count of *Salmonella* after 6 h with approximately one log, while after 7 days, the amount of reduction was nearly 3 logs [100]. This is why the longer the treatment time, the more time the bacteriophage has to replicate and spread to other bacterial cells, which increases its efficiency but at the same time increases the chance of developing resistance bacteria to phage [175,193].

Bacteria in biofilm can apply various approaches, like increased mutability compared to free cells with high levels of gene exchange between bacterial communities that lead to increasing growth and rapid changes to environmental stress, such as antibiotics and bacteriophage, which make them more resilient. As a result, it would be a challenge to apply phage against such hosts, but it should be noted that phages and bacteria have been in a race with each other for millennia. Using a cocktail of bacteriophages would be beneficial in reducing the risk of bacterial resistance to phage. Moreover, bacteriophages are ubiquitous, so searching for novel phages can be an option when new resistant colonies are identified.

Although bacteriophages were more effective in prolonged treatment, this meta-analysis reaffirms the antibacterial ability of phage in short-term treatment. Also, phages can show a great antibacterial impact even a few moments after their application, as they can kill the host by their adsorption, which happens in the early moment [175,176]. However, optimizing the duration of treatment can be investigated by applying phage in combination with conventional or novel antibacterial methods to gain the optimum results based on the required time in the industry.

3.3.7. Food matrix

This subgroup was also allocated to evaluate the overall effect of any food matrix, which can be found in food processing facilities even after sanitization, on the antibacterial activity of phage along the treatment. The subgroup analysis showed a significant reduction of 37.2 % (R* = 0.628) and 44.1 % (R* = 0.559) in the presence and absence of food matrix, respectively. The efficiency of bacteriophage when applied in the absence of a food matrix was 1.18 folds higher. The presence of food matrices on food-contacting surfaces can be a considerable concern for the spreading of foodborne pathogens since it is a suitable matrix for bacteria to attach, accumulate, and propagate on surfaces, and finally be a source of cross-contamination of food products [194,195]. The study by Chaitiemwong et al. (1996) investigated the effect of food matrix and grooves on the surface of stainless steel on the efficiency of applying bacteriophage to biofilm-forming pathogens. Their study revealed that bacteriophage can reduce *L. monocytogenes* below the detection limit even in the presence of ham, fish, milk, and vegetables as food matrix. However, in this study, the efficiency of using phage dramatically decreased when the depth of groves on stainless steel increased from 0.2 to 0.5 mm. Therefore, the food matrix on the surface of stainless steel can lower the efficiency of bacteriophage and detergents, especially once the surface is not smooth. Also, biofilm can be a physical barrier for penetrating antibacterial agents like bacteriophages to reach pathogens [196]. Therefore, in general, pre-cleaning of the surfaces and removal of the food matrixes by washing, scrubbing, or other means, especially in vulnerable places with the risk of the remaining food matrix, is crucial firstly as the main goal of food safety and then for providing direct contact of phage-bacteria for a more efficient antibacterial effect of phage. Otherwise, bacteriophages may be less effective or require longer exposures to penetrate and eliminate the bacteria in the biofilm.

3.4. Publication bias

Fig. 2 represents the results of publication bias for the anti-biofilm effect of bacteriophage on stainless steel. Based on the Begg and Mazumdar adjusted rank correlation test (P = 0.091) and Egger's regression asymmetry test (P = 0.068), no sign of publication bias was found for the response ratio.

4. Conclusion

In conclusion, the use of bacteriophages shows great potential for controlling biofilms formed by foodborne pathogens on stainless steel surfaces in the food industry. The findings of this systematic review and meta-analysis highlight the effectiveness of bacteriophages in killing different types of bacteria within the biofilm matrix. Phage cocktails can increase antibacterial efficiency, but an accurate evaluation is required to ensure the stability of their efficiency. High phage concentrations can also increase lytic activity; however, the age of the biofilm should be considered, as older biofilms are less susceptible to phages. Giving more time to phage can substantially increase its lytic function as it has more time to replicate and kill bacteria. However, the food matrix can adversely affect phage lytic activity, which needs to be considered. Further research in this area is needed to fully understand the potential of bacteriophages for controlling biofilms in the food industry.

Funding

None to declare.

Institutional review board statement

Not applicable.

Informed consent statement

Not applicable.

CRediT authorship contribution statement

Rahim Azari: Conceptualization, Methodology, Conducting, Resources, Writing – original draft, Writing – review & editing. **Mohammad Hashem Yousefi:** Conceptualization, Investigation, Conducting, Resources, Writing – original draft, Writing – review & editing, Supervision, Project administration. **Aziz A. Fallah:** Methodology, Data

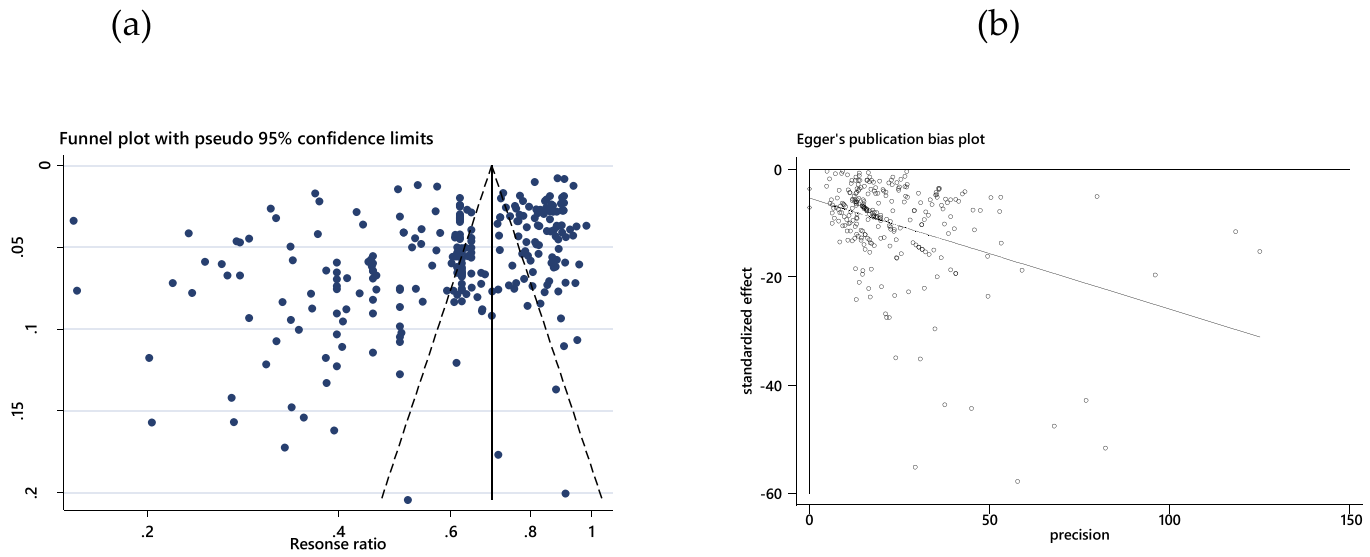

Fig. 2. Begg's funnel plot (a) and Egger's plot (b) for response ratio of the effect of bacteriophage on biofilms formed by foodborne pathogens.

curation, Formal analysis. **Arezo Ali Mohammadi:** Methodology, Conducting. **Nastaran Nikjo:** Methodology, Conducting. **Jeroen Wagemans:** Writing – review & editing. **Enayat Berizi:** Writing – review & editing. **Saeid Hosseinzadeh:** Writing – review & editing. **Mohammad Ghasemi:** Data curation. **Amin Mousavi Khaneghah:** Conceptualization, Methodology, Investigation, Conducting, Resources, Writing – original draft, Writing – review & editing, Supervision, Project administration, Funding acquisition. **Amin Mousavi Khaneghah:** Conceptualization, Data curation, Formal analysis, Investigation, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

Acknowledgments

The authors thank Prof. Rob Lavigne (KU Leuven) for his critical appraisal of this work.

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