Morphological features and lipopolysaccharide attachment of coliphages specific to *Escherichia coli* O157:H7 and to a broad range of *E. coli* hosts

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Abstract The objective of the present study was to analyze host-phage adsorption of bacteriophages infecting *Escherichia coli* O157:H7 and the other *E. coli* strains. Out of 55 coliphage strains, we selected seven coliphages infectious only to 23 *E. coli* O157 and seven other coliphages of broad specificity to *E. coli* O157:H7 and other 61 *E. coli*. *Escherichia coli* O157-specific phages and the broadly specific phages all belonged to the Siphoviridae and Myoviridae family, respectively. *Escherichia coli* O157-specific phages infected *E. coli* O157:H7, but not *E. coli* O157:H7ΔrfαL, deletion mutant of O-antigen ligase gene for lipopolysaccharide. Five coliphages among the broadly specific phages infected *E. coli* O103, but not *E. coli* O103ΔrfαG, deletion mutant of the glycosyltransferase gene. *E. coli* O157:H7-specific phages among Siphoviridae recognized O-antigen of *E. coli* O157, but the broadly specific coliphages of Myoviridae may recognize O-antigen and/or a part of the lipopolysaccharide core as an adsorption site in various *E. coli*. The receptor of the two coliphage groups interacts with some part of lipopolysaccharide, and the tail morphology of the coliphages may be related to their adsorption to and recognition of a different part of lipopolysaccharide. In particular, specificity of *E. coli* O157:H7-specific phages carrying the long tail of Siphoviridae for O-antigen as a receptor seems to be high.

Keywords Bacteriophage · *E. coli* O157:H7 · Tail · Receptor · Lipopolysaccharide

Introduction

Bacteriophages may exist in all habitats on earth with the estimated population of 10³¹, and tailed phages have been classified into three families. Myoviridae possessing a complex contractile tail account for 25 % of tailed phages such as T4 and Mu, whereas Siphoviridae (characterized by a long noncontractile and flexible tail) account for 61 % of tailed phages like λ and SPP1 (Bebeacua et al. 2013). Podoviridae bearing a short noncontractile tail account for 15 % of tailed phages, and T7 phage is a well-known member of this family (Ackermann 2009).

The viral infection process can consist of several sequential stages, and the first stage is phage adsorption to a host cell’s surface. Adsorption is known as a key stage of a virus’s recognition of a sensitive host cell, and specificity of phage infection is determined at this moment by the specific interaction between the phage and a special receptor on the host cell surface (Traurig and Misra 1999; Mudhakir and Harashima 2009; Rakhube et al. 2010). Furthermore, the host range of a bacteriophage, which depends on its ability to recognize different receptors, is one of many biological characteristics of a phage (Morita et al. 2002). Coliphages are a group of phages that infect *Escherichia coli* and are categorized into somatic and male-specific coliphages. Somatic coliphages infect through the receptors on the surface of *E. coli*, whereas male-specific coliphages (which are known as RNA
viruses) infect *E. coli* via F-pili of male cells. These two types of coliphages show not only different infection types but also different morphological characteristics and nucleic acids (DNA and RNA) (Lee et al. 2009, 2013).

Localization and density of the attachment receptor on the host cell’s surface also plays an important role in phage adsorption. Various components of the bacterial outermost cell layer have been reported to serve as bacteriophage receptors, and two flagellotrophic phages and one pilus-specific phage have been characterized in detail (Pate et al. 1979; Rakhuba et al. 2010). Proteins localized in the cell membrane and various parts of lipopolysaccharide (LPS) also serve as bacteriophage receptors in the cell wall of gram-negative bacteria (Rakhuba et al. 2010). Park et al. (2012) reported that O-antigen of LPS is the attachment receptor for SFP10 phage. The roles of OmpC and OmpF, which are major outer-membrane proteins of *E. coli* cells, in the adsorption of bacteriophages T4 and T2, respectively, have been studied by some research groups (Yu and Mizushima 1982; Riede et al. 1985). There are studies on the adsorption mechanism or the phage–receptor interaction of bacteriophage λ (Randall-Hazelbauer and Schwartz 1973; Friedman and Court 2001; Chatterjee and Rothenberg 2012).

Kutter et al. (2011) recently stated that there are few well-characterized phages infecting *E. coli* serotype O157:H7 although numerous species of phages targeting *E. coli* have been isolated and studied. Furthermore, there have been few studies on the receptor of bacteriophages specific for *E. coli* O157:H7, and characterization of such phages and of the host recognition mechanism of bacteriophages with a broad host range among *E. coli* strains (hereafter: broadly *E. coli*-specific phages) has not yet been conducted. Moreover, there are no comparative studies of *E. coli* O157:H7-specific bacteriophages and the broadly *E. coli*-specific phages.

Therefore, the objective of the present study was to characterize and compare morphological and adsorption receptor of both bacteriophages specific for *E. coli* O157:H7 and the broadly *E. coli*.

### Materials and methods

**Selection of *E. coli* bacteriophages and analysis of the host spectrum**

Seven *E. coli* O157:H7-specific phages and five broadly *E. coli*-specific phages were selected from the phage collection of 55 isolates (Kim 2015) on the basis of the host spectrum characteristics. Host spectrum analysis was performed by a spot assay involving 24 strains of *E. coli* O157:H7 from American Type Culture Collection (ATCC) and National Collection of Type Cultures (NCTC), and ten strains of *E. coli* serotypes O55, O91, O103, O104, O111, and O117 from the National Culture Collection for Pathogens (NCCP), Korea. In addition, 51 strains of non-pathogenic *E. coli* were used as a host for infection. One-hundred microliters of each test bacterial culture was added to 5 mL of LBC soft agar (LBC broth with 0.8 % agar) and overlaid on LBC agar. 10 ml of each phage lysate (107–109 plaque-forming units [PFU]/mL) was spotted on the overlaid plates, and the plates were incubated at 37 °C for 24 h. After the incubation, sensitivity of the tested bacteria to a phage was confirmed if we saw formation of a clear plaque on the plates (Kutter 2009). This test was performed in triplicate.

**Morphological analysis**

Phage particles at approximately 109–1010 PFU/mL that were concentrated with 20 % polyethylene glycol (PEG) 8000 (Sigma-Aldrich, St Louis, MO, USA) and 2 M NaCl were negatively stained with 2 % (w/v) uranyl acetate (Ackermann 2009). Morphological analysis of the bacteriophages was conducted by transmission electron microscopy (JEOL JEM-100S, Japan Electronics and Optics Laboratory; Tokyo, Japan), and morphological features of each bacteriophage were identified and used for classifying the phages, according to Ackermann (2009).

**Construction of an *E. coli* mutant**

The *E. coli* O103 NCCP 13937 mutant Δrfag was newly constructed by transposon mutagenesis as follows: Competent cells for construction of phage-resistant mutants were prepared by a modified method of Wu et al. (2010). 1 ml of overnight culture (8–9 log10 colony-forming units [CFU]/mL) was inoculated into 100 mL of Luria–Bertani (LB) broth and incubated at 37 °C for 2 h, with shaking at 300 rpm. After optical density at wavelength 600 nm (OD600) reached 0.5–0.7, the culture was chilled on ice for 20 min. To harvest the cells, we transferred the culture to a cold centrifuge bottle and centrifuged the culture at 4000 × g for 15 min at 4 °C. After the supernatant was discarded, the cell pellet was gently resuspended in 100 mL of ice-cold 10 % glycerol and centrifuged under the same conditions. The pellet was washed three times with 50, 10, and 1 mL of ice-cold 10 % glycerol using the method described above. The final cell pellet was stored at −70 °C. Random gene mutagenesis was performed using the EZ-Tn5 <R6 Kory/KAN-2> Tn5 transposome kit (Epicentre, Madison, WI, USA). For electroporation, 1 μL of Tn5 transposon (33 ng/μL) was added to 100 μL of competent cells, with brief mixing. The mixture was chilled on ice for 1 min and transferred into an ice-cold 2 mm
Electroporation was conducted at 2.5 kV on a MicroPulser system (Bio-Rad, Hercules, CA, USA). After the electroporation, 1 mL of the SOC medium (Super Optimal broth with catabolite repression; Invitrogen, Carlsbad, CA, USA) was added immediately, and the culture was incubated, at 37 °C for 1 h, with shaking at 225 rpm. A total of 300 mutants were selected on LB agar containing 50 µg/mL kanamycin sulfate (Sigma-Aldrich, St. Louis, MO, USA). The randomly selected mutants were stored in 10 % glycerol at −70 °C. Selection of phage-resistant mutants (by means of phage NOECP10) was conducted using a modified method of Park et al. (2012). Each selected mutant was inoculated into two 96-well plates containing 200 µL of LB broth with 50 µg/mL kanamycin sulfate, and the plates were incubated at 37 °C for 1 h, with shaking at 120 rpm. After that, one of the plates was inoculated with 10 µL of a phage lysate (at multiplicity of infection [MOI] of 1.0), and the other plate was not. Both plates were then incubated at 37 °C for 3 h, with shaking at 120 rpm. OD620 of the two plates was measured and compared to select the phage-resistant mutants. Plaque and spot assays were also conducted to confirm the phage-resistant mutants.

Analysis of Tn5 insertion sites in the mutants

Rescue cloning of the transposed genomic DNA of E. coli O103 NCCP 13937 ΔrfaG was performed by means of the EZ-Tn5 <R6 Kyori/KAN-2> Tnp transposome kit. 1 mg of genomic DNA from the mutant was digested with EcoRV (Takara, Otsu, Japan), and self-ligation of the fragmented genomic DNA was performed by means of T4 DNA ligase (Takara, Otsu, Japan) at room temperature for 1 h. The reaction was terminated by heating it at 70 °C for 10 min. After self-ligation, electroporation of E. coli EC100D pir+ competent cells was performed as described above. A KanR rescued clone was selected on LB agar containing 50 µg/mL kanamycin sulfate and stored in 10 % glycerol at −70 °C. To confirm the transposon insertion sites of the phage-resistant mutant, partial sequencing of plasmid DNA (200 ng/µL) from the rescued clone was conducted with transposon-specific primers supplied with the EZ-Tn5 <R6 Kyori/KAN-2> Tnp transposome kit. Homology analysis was conducted with the sequencing data by means of a National Center for Biotechnology Information (NCBI) nucleotide blast search; thus, the insertion site was identified.

Screening for an adsorption receptor on LPS mutants

To analyze the host-phage recognition, two mutants of E. coli O157:H7 ATCC43890 ΔrfaL and E. coli O103 NCCP 13937ΔrfaG were infected with the bacteriophages. E. coli O157:H7 ΔrfaL was obtained from Park et al. (2012) and infected with E. coli O157:H7-specific phages. E. coli O103 ΔrfaG was infected with the broadly E. coli-specific phages, and these infection samples were analyzed for plaque-forming activity in a spot assay.

Results and discussion

Selection of bacteriophages for various types of infection of E. coli

A total of 55 coliphages from our phage collection, 27 phages from swine feces, 16 from sewage, six from bovine feces, and six from soil, were used to select E. coli O157:H7-specific phages and broadly E. coli-specific phages. According to the host spectrum analysis, we selected seven phages that specifically infect E. coli O157:H7- and seven broadly E. coli-specific bacteriophages. E. coli O157:H7-specific phages (the narrow-specificity group) infected 23 of 24 E. coli O157:H7 strains at the 95 % infection rate and infected none of the 10 E. coli strains of serotypes NCPP O55, O91, O103, O111, O117, and O179, and none of the 51 nonpathogenic E. coli strains (Table 1). In line with our results, Park et al. (2012) reported similar results, namely that Salmonella and E. coli O157:H7 does not, or very poorly, infect other serotypes of E. coli such as O1, O126, and O112. In contrast, the seven phages of the broad-specificity group showed widely varying infection rates (25–95 %) in relation to the 24 E. coli O157:H7 strains and a >50 % infection rate toward 10 strains of other serotypes. As for the nonpathogenic E. coli strains, the infection rates there varied (4–40 %). Phage ECP19 showed a 96 % infection rate toward the 24 E. coli O157:H7 strains and infected the other serotypes at the 50 % rate. BECP4 also showed a broad host range, infecting E. coli O157:H7 and other E. coli serotypes at the 42 and 90 % rate, respectively. Raya et al. (2006) reported similar results, namely that CEV1 infects not only 17 strains of E. coli O157:H7 but also other serotypes including O43 and O126. Nevertheless, the E. coli O157:H7-specific phages here infected only this serotype and did not show effective infection of other E. coli strains.

Morphological characteristics

Transmission electron microscopy of seven E. coli O157:H7-specific phages showed flexible and noncontractile tails, suggesting that these phages all belonged to the Siphoviridae family (Fig. 1). The diameter of the head and tail length were within the ranges 50–90 and
140–260 nm, respectively. Li et al. (2010) reported a similar result, i.e., that coliphage EEP (which belongs to the Siphoviridae family) has a 62 ± 5 nm head and a 138 ± 16 nm tail. In contrast, the broadly E. coli-specific group had inflexible and contractile tails, suggesting that these phages all belonged to the Myoviridae family (Fig. 2). The diameter of the head and uncontracted-tail length were within the ranges 65–78 and 100–120 nm, respectively. Park et al. (2012) also reported that SFP10, which belongs to Myoviridae, has a 69 nm head and a

| Bacteriophage | Isolation source | No. of plaque-forming strains/total no. of E. coli strains (hosts) | E. coli O157:H7 | Other serotypes* | Nonpathogenic E. coli |
|---------------|------------------|---------------------------------------------------------------|----------------|-----------------|---------------------|
| ECP1          | Bovine feces     | 23/24                                                         | 0/10           | 0/51            |
| ECP3          | Bovine feces     | 23/24                                                         | 0/10           | 0/51            |
| ECP4          | Bovine feces     | 23/24                                                         | 0/10           | 0/51            |
| ECP6          | Soil             | 23/24                                                         | 0/10           | 0/51            |
| ECP7          | Soil             | 23/24                                                         | 0/10           | 0/51            |
| ECP9          | Soil             | 23/24                                                         | 0/10           | 0/51            |
| ECP13         | Sewage           | 23/24                                                         | 0/10           | 0/51            |
| ECP15         | Sewage           | 20/24                                                         | 8/10           | 4/51            |
| ECP19         | Sewage           | 23/24                                                         | 5/10           | 11/51           |
| ECP20         | Sewage           | 10/24                                                         | 5/10           | 3/51            |
| BECP3         | Sewage           | 3/24                                                          | 6/10           | 3/51            |
| BECP4         | Sewage           | 10/24                                                         | 9/10           | 2/51            |
| NOECP10       | Swine feces      | 9/24                                                          | 9/10           | 21/51           |
| NOECP16       | Swine feces      | 6/24                                                          | 7/10           | 13/51           |

* E. coli NCCP O55, O91, O103, O111, O117, and O179

Fig. 1 Morphological characteristics of E. coli O157:H7-specific phages according to transmission electron microscopy. (A) phage ECP1, (B) ECP3, (C) ECP4, (D) ECP6, (C) ECP7, (F) ECP9, (G) ECP13
131 nm uncontracted tail. These results revealed that morphological characteristics were different between \(E.\ coli\) O157:H7-specific phages and the broadly \(E.\ coli\)-specific phages.

**Construction of \(E.\ coli\) O103/\(rfaG\)**

We constructed this phage-resistant mutant for screening for the adsorption receptor in the broadly \(E.\ coli\)-specific phages. \(E.\ coli\) O103 NCCP 13937, which is not a resistant to kanamycin sulfate at 50 \(\mu\)g/mL, was chosen for this experiment. On the basis of OD\(_{620}\), six phage-resistant mutants of \(E.\ coli\) O103 were selected from the random library of mutants, which was composed of 300 independent mutants. After the plaque and spot assays were conducted, one mutant resistant to phage NOECP10 was identified.

Some studies have shown selection of a phage-resistant mutant by means of random transposon mutagenesis. Reyes-Cortés et al. (2012) reported that nine coliphage (mEp213)-resistant \(E.\ coli\) K-12 mutants were successfully selected by this method. Shin et al. (2012) reported that a library of Tn5 random mutants (≈1000 independent mutants) was prepared, and receptor screening was performed among 25 \(Salmonella\) phages. Choi et al. (2013) reported that six \(Salmonella\) phage (iEPS5)-resistant \(S.\ typhimurium\) mutants were successfully selected by the Tn5 transposon mutagenesis.

To confirm the insertion site of Tn5 in our mutant, we conducted DNA homology analysis. Sequencing with primers Kan-2 FP-1 forward and R6KAN-2 RP-1 reverse showed 99 and 100 % nucleotide homology in the genome locations 4,674,059–4,674,384 and 4,673,467–4,674,067, respectively, with the corresponding region in the complete genome of \(E.\ coli\) O103:H2 strain 12009 (sequence ID: dbj|AP010958.1). Data from the NCBI database also showed that the \(rfaQ\) and \(rfaG\) genes in the complete genome of \(E.\ coli\) O103:H2 strain 12009 are located in the regions 4,672,649–4,673,671 and 4,673,668–4,674,792, respectively. The sequences of the forward and reverse primers were consistent with the \(rfaG\) gene in the regions 392–717 and 1–400, respectively, suggesting that the overlapping nine bases between gene positions 392 and 400 could form complementary base pairing.

For composite transposons such as Tn5, Tn9, and Tn10, there are thousands of possible integration sites in the \(E.\ coli\) chromosome, but some preferable sites (hot spots) have been reported (Lodge et al. 1988; Goryshin et al. 1998). One of the characteristics frequently reported about Tn5 transposition is that 9-bp direct sequence repeats are
generated as a result of target sequence duplication. Another characteristic is that the preferred 9-bp direct sequence repeat contains G or C bases at each end (Lodge et al. 1988; Goryshin et al. 1998; Green et al. 2012). Accordingly, we chose nine bases (5′-CCTTCGAAC-3′) in the region 392–400 of the rfaG gene as a target sequence for Tn5 transposon and sequenced 9-bp direct repeats located at both ends of the inverted repeat by means of the above-mentioned transposon-specific primers.

Thus, the *E. coli* O103 NCCP13937 mutant resistant to phage NOECP10 was constructed and named *E. coli* O103ΔrfaG. Chang et al. (2010) reported that the *rfa* operon in the *E. coli* chromosome encodes all of the glycosyltransferases necessary for core elongation of LPS, and each gene product of the operon affects LPS structure. Many studies have shown that the glycosyltransferase encoded by *rfaG* is involved in the transfer of the first D-glucose to the heptose II group in the inner core of LPS (Austin et al. 1990; Parker et al. 1992; Genevaux et al. 1999; Yethon et al. 2000; Chang et al. 2010). Yethon et al. (2000) also demonstrated that disruption of the *rfaG* gene in *E. coli* F470 interrupts core elongation after the heptose residues and reduces the length of the LPS core. Therefore, the mutant *E. coli* O103ΔrfaG was expected to have a shortened core of LPS, where the O-antigen region was also deleted.

### Screening for a phage receptor

Adsorption receptor screening among *E. coli* O157:H7-specific phages and the broadly *E. coli*-specific phages was conducted by means of *E. coli* O157:H7ΔrfaL and *E. coli* O103ΔrfaG. Park et al. (2012) reported that the *rfaL* gene encoding O-antigen ligase in *E. coli* O157:H7 ATCC 43890 was successfully deleted by the TargeTron Intron System (Sigma-Aldrich). As shown in Table 2, the seven phages infecting *E. coli* O157:H7 ATCC43890 could not infect the *E. coli* O157:H7ΔrfaL. These results proved that the seven phages that infect *E. coli* O157:H7 may recognize O-antigen as the adsorption receptor when they adsorb to an *E. coli* O157:H7 cell. The receptor screening among the broadly *E. coli*-specific phages was conducted on *E. coli* O103ΔrfaG, which is the mutant with a shortened core of LPS, as shown in Table 3. The phages ECP19, ECP20, BECP4, NOECP10, and NOECP16 infected *E. coli* O103 NCCP 13937 but could not infect its mutant. Therefore, most of the broadly *E. coli*-specific phages, including ECP19, ECP20, BECP4, NOECP10, and NOECP16, may recognize the deleted part of the LPS core or O-antigen as an attachment site when they infect *E. coli* O103 NCCP 13937.

Although bacteriophages have several options for adsorption to their hosts, at least one structural phase protein (known as receptor-binding protein; RBP) must be in contact with one bacterial surface receptor (Vinga et al. 2006, 2012). RBPs are typical components of long and short fibers attached to the contractile tail of myoviruses and of base plates, fibers, spikes, or single straight fibers attached to the long noncontractile tail of siphoviruses (Vinga et al. 2012). The specific interaction between tail RBPs and receptors on the host cell’s surface determines importantly the host range of the bacteriophage (Vinga et al. 2012).

The key events initiating viral infection of the host cell are related to adsorption of a phage to its host. In our case, the bacteriophage tail may be the molecular machinery that recognizes the host; nevertheless, the three families—Siphoviridae, Myoviridae, and

### Table 2 Infection of *Escherichia coli* O157:H7 and its mutant by *E. coli* O157:H7-specific coliphages by assessment of plaque formation

| Bacteriophage | *E. coli* O157:H7 ATCC43890 | *E. coli* O157:H7 ATCC43890ΔrfaLa |
|---------------|--------------------------------|----------------------------------|
| ECP1          | +                              | –                                |
| ECP3          | +                              | –                                |
| ECP4          | +                              | –                                |
| ECP6          | +                              | –                                |
| ECP7          | +                              | –                                |
| ECP9          | +                              | –                                |
| ECP13         | +                              | –                                |

a infected by a phage; † not infected by a phage

### Table 3 Infection of *Escherichia coli* O103 and its mutant by broadly *E. coli*-specific coliphages by assessment of plaque formation

| Bacteriophage | *E. coli* O103 NCCP13937 | *E. coli* O103 NCCP13937ΔrfaGa |
|---------------|--------------------------|--------------------------------|
| Broadly *E. coli*-specific coliphages | | |
| ECP15         | –                        | –                              |
| ECP19         | +                        | –                              |
| ECP20         | +                        | –                              |
| BECP3         | –                        | –                              |
| BECP4         | +                        | –                              |
| NOECP10       | +                        | –                              |
| NOECP16       | +                        | –                              |

a infected by a phage; † not infected by a phage

a *E. coli* O103 NCCP13937ΔrfaG = mutant with a deletion of the glycosyltransferase gene.
Podoviridae—may have a different component that recognizes the host cell (Veesler and Cambillau 2011). LPS of the outer membrane in gram-negative bacteria is known as a receptor for bacteriophage adsorption. One of the two LPS types is the smooth type (S-type), which has a typical LPS structure, and the other type is the rough type (R-type) LPS, which contains lipid A and the core but lacks O-antigen (Rakhuba et al. 2010). According to Rakhuba et al. (2010), bacteriophages specific to S-type LPS show an extremely narrow host range, as a result of large variability of O-antigen’s structure in bacteria of different taxonomic groups. A bacteriophage that has a narrow host spectrum has been found (Sf6), and analysis showed that its adsorption to S. flexneri is associated with the O-chain of LPS (Lindberg et al. 1978). Our results are in agreement with these studies: the narrow-specificity coliphage group is specific only to E. coli O157:H7, and adsorption of these phages to E. coli O157:H7 may involve O-antigen of LPS.

In contrast, bacteriophages recognizing R-type LPS have a broad host range because the structure of the LPS core has been conserved in various species and genera of gram-negative bacteria (Rakhuba et al. 2010). Bacteriophage PVP-SE1, which has a broad host range, was reported to use the LPS inner core region as the attachment site (Santos et al. 2011). Our results are consistent with these reports: most of the broadly E. coli-specific phages infected various E. coli strains, and adsorption of these phages to E. coli O103 NCCP 13937 may involve O-antigen or the LPS core region that was deleted in our E. coli mutant.

Therefore, LPS may play an important role (in phage-host recognition) that is similar for our two coliphage groups, but the specific part of LPS that participates in the infection process may be different between our two coliphage groups. The morphological features of the tail of the selected phages belonging to the Siphoviridae or Myoviridae family may be involved in the adsorption of the phage to LPS on the surface of an E. coli cell; in the present work, these two families of phages showed a different host spectrum. The E. coli O157:H7-specific phages and the broadly E. coli-specific phages may recognize O-antigen or the deleted part of the core with O-antigen of LPS as the adsorption receptor, respectively. Specificity of the E. coli O157:H7-specific phages (carrying the long tail of Siphoviridae) to the O-antigen receptor seems to be high.

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