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Morphology of Complexes Formed Between Bacteriophage Lambda and Structures Containing the Lambda Receptor

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Two types of complexes can be formed between bacteriophage lambda and structures bearing the lambda receptor, either liposomes or rod-shaped particles. Type 1 complexes involve binding between the tip of the lambda tail fiber and the receptor, so that the hollow tail is positioned an average of 17 nm from the surface of the receptor-bearing structures. In type 2 complexes, the hollow tail is in direct contact with the membrane of the liposome or surface of the rod-shaped particle. Type 1 complexes are the precursors for type 2 complexes whose formation is necessary for normal DNA ejection.

Relatively little is understood about the initial stages of infection by most types of bacteriophage, including lambda, especially with respect to the mechanism by which DNA crosses host membranes. For bacteriophage T4, morphological changes are readily observable in the electron microscope during infection, and so the mechanics of the adsorption process are clear. Bacteriophage T4 consists of a DNA-containing head and a tail composed of an outer contractile sheath and an inner hollow core, ending in a baseplate to which are attached six long, bent tail fibers. The tail fiber tips bind to diglucosyl groups of surface lipopolysaccharides so that the baseplate is initially positioned 70 to 90 nm from the surface. The baseplate then moves closer and becomes irreversibly attached to the surface of the cell. When the tail sheath contracts, the inner core is forced about 12 nm into and through the outer membrane. The T4 DNA then exits from the head via the core (for a recent review, see reference 6). How the injected T4 DNA crosses the bacterial inner membrane in addition to the outer membrane is not clear, although it has been suggested that the DNA is injected at sites of fusion between the inner and outer membrane (1).

Other bacteriophages such as lambda are unlikely to utilize a mechanism that is identical to that employed by T4 since they do not have contractile tails. Study of these bacteriophages may therefore reveal the nature of alternate methods employed for the entry of DNA into cells. The receptor for bacteriophage lambda is the *lamB* protein, which facilitates entry of maltose and maltodextrins into bacterial cells, presumably by providing appropriate aqueous channels (for a recent review, see reference 14). It is not known whether the entry of lambda DNA depends in any significant way on the transport properties of this protein. Bacteriophage lambda initially binds to the *lamB* protein, which is an integral membrane protein located in the outer membrane (11). Since neutralizing antibodies bind to the tail fiber (3, 4), a spike-shaped 20-nm projection from the lambda tail that is composed of several copies of protein J (M_r, ~130,000), and since host range mutations map in the part of the lambda gene, *J* (16), which codes for the carboxyl terminus of protein J, it is likely that an affinity exists between the tail fiber and the *lamB* protein.

Several experiments have shown that the isolated *lamB* protein can be incorporated into artificial membranes. Yamada et al. (17) have developed a reconstitution system utilizing lipopolysaccharide and have assembled the *lamB* protein into a hexagonal lattice structure. Boehler-Kohler et al. (2) found that the protein could form pores in black lipid membranes of a diameter of 1.6 nm as measured by conductivity. Nakae (10) showed that the *lamB* protein formed channels in vesicles which permitted the transmembrane diffusion of a variety of compounds. Luckey and Nikaido (8) also demonstrated this pore-forming activity, using liposomes.

The lambda receptor isolated from *Shigella* sp. differs from that isolated from most *Escherichia coli* strains. The free *Shigella* receptor is capable of directly causing DNA ejection from bacteriophage lambda (15), whereas the *E. coli* receptor causes DNA injection only if chloroform is added (11). Bacteriophage host range mutants called λ*h* exist which do eject their DNA in the absence of chloroform after binding to the *E. coli* receptor (11). The use of either λ*h* or the *Shigella* receptor is necessary to study DNA ejection across a membrane in in vitro

systems since addition of $CHCl_3$ would destroy the membrane.

Recently, we demonstrated that bacteriophage lambda binds to and injects its DNA into liposomes bearing the *lamB* protein from *Shigella* sp. in the membrane (12a). Here, we report on an electron microscopic comparison of the complexes observed between bacteriophage lambda and structures bearing either the *E. coli* K-12 or the *Shigella lamB* protein. Two types of complexes were found. In type 1 complexes, the bacteriophage was held to the receptor by the tip of the lambda tail fiber. In type 2 complexes, which probably form from type 1 complexes, the base of the bacteriophage tail was attached directly. DNA ejection appears to occur after formation of type 2 complexes.

MATERIALS AND METHODS

Bacteria and bacteriophage. *E. coli* K-12 substrain Y mel is *lamB*$^+$ (λ*h*$^{+s}$) *suIII*$^+$.

E. coli C600(λ *c*I857 *S*7) was grown to 5×10^8 per ml, and bacteriophage development was induced by heating at 43°C for 20 min, followed by vigorous shaking for 2 h at 37°C. The cells were centrifuged and concentrated 20-fold in 10 mM $MgSO_4$–10 mM Tris-hydrochloride, pH 7.5. The cells were lysed by freezing and thawing, and 10 μg of DNase I per ml was added to reduce the viscosity of the lysed cells. The lysate was layered onto a step gradient consisting of three layers (10% glycerol; CsCl, density 1.4; and CsCl, density 1.6) and centrifuged for 3 h at 25,000 rpm in an SW28.1 rotor. The band of bacteriophages located at the top of the density 1.6 CsCl layer was collected, further purified on a CsCl equilibrium gradient, and stored at 4°C in CsCl. In some experiments, bacteriophages were added directly from the storage medium and in others they were dialyzed before use.

Radioactive ^{3}H-labeled bacteriophages were prepared by inducing 10 ml of *E. coli* C600(λ *c*I857 *S*7) Thy$^-$ in M9 salts medium containing 1% glucose, 0.5% Casamino Acids, 3 μg of vitamin B_1 per ml, and 3 μg of thymine per ml. Shortly after induction, 500 μCi of [^{3}H]thymidine (60 Ci/mmol; Schwarz/Mann) was added.

λ*h* was obtained from Maxime Schwartz (Institut Pasteur), and a stock was prepared from plate lysates.

Receptor preparation. Receptors were extracted from substrain Y mel or an *E. coli* strain carrying the *Shigella* receptor (Pop 154 or Pop 157, which is Pop 154 Pel$^-$, obtained from Dorothea Scandella, Institut Pasteur) with EDTA and cholate as described previously (11). Two liters of cells in tryptone broth plus 0.4% maltose were grown to a density of 5×10^8 cells per ml and collected by centrifugation. The cells were suspended in 100 ml of 10 mM Tris-hydrochloride, pH 7.5–2.0 mM EDTA–1.0% cholate and incubated at 37°C in a shaking water bath for 30 min. The cells were centrifuged for 30 min at 10,000 × *g*, and the supernatant was dialyzed extensively against 10 mM Tris-hydrochloride, pH 7.5–2.0% cholate. The EDTA-cholate extract was further extracted with 11 volumes of butanol. The precipitate that formed was collected by centrifugation at 10,000 × *g* for 10 min, dried under a stream of nitrogen, and dissolved in 10 mM Tris-hydrochloride, pH 7.5–2% cholate.

Preparation of liposomes containing lambda receptor. Liposomes bearing the receptor for bacteriophage lambda were prepared as previously described (12a) by detergent dialysis. Briefly, a receptor-containing extract in 2% cholate was used to dissolve 10 mg of phosphatidylcholine (Avanti) that had been dried on the walls of a conical centrifuge tube. The receptor-lipid solution was dialyzed against two 1-liter changes of Hanks phosphate-buffered saline. The receptor-containing vesicles were then purified on a Sepharose 4B column.

Bacteriophage-receptor complex formation. Bacteriophages (10^{10} PFU in 10 μl) were mixed with enough receptor or liposomes containing receptor to completely inactivate the phage (usually 10 μl) in a total volume of 1.0 ml of 10 mM Tris-hydrochloride, pH 7.5, and CsCl, NaCl, and $MgSO_4$ as indicated. Incubation was for 30 min at 4 or 37°C.

Electron microscopy. The bacteriophage-receptor complexes were adjusted to 0.8 μg/ml of cytochrome *c*, and 10 μl was adsorbed to a carbon-coated grid for 5 min. The droplet was absorbed with filter paper, and 10 μl of 2% uranyl acetate (pH 4.5) was applied to the grid. After 30 s, this droplet was absorbed with filter paper. The grid was allowed to air dry and was examined in a Hitachi H-500 electron microscope. Length measurements were made with a Numonics graphics calculator from negatives projected onto frosted opal glass.

RESULTS

The morphological appearance of complexes between bacteriophage lambda and liposomes or rod-shaped particles bearing the lambda receptor was studied by electron microscopy. Artifical phospholipid vesicles (liposomes) served as models to assess the role of membranes in bacteriophage lambda adsorption. Rod-shaped particles bearing the lambda receptor have been observed previously (9, 12), and extraction of these with butanol provided the means to observe complexes formed with delipidated structures. Complexes formed readily with both liposomes and rod-shaped particles (Fig. 1 and 2), and for both, two types of complexes were observed.

Type 1 complexes. Type 1 complexes were characterized by a gap of 10 to 22 nm (average, 17 nm) between the base of the banded region of the bacteriophage tails and the rod-like particles (Fig. 1A) or liposomes (Fig. 1B and C). In some cases, a thin tail fiber can be seen traversing this gap (Fig. 1B and C). Measurements on free bacteriophages yielded a mean value and standard deviation of 150 ± 4 nm for the length of the banded region of the tail (Fig. 3A) and 171 ± 4 nm for the entire tail including the tail fibers (Fig. 3B). The tail fiber thus measures about 21 nm long, which is about equal to the widest gaps measured. The mean and standard deviation for

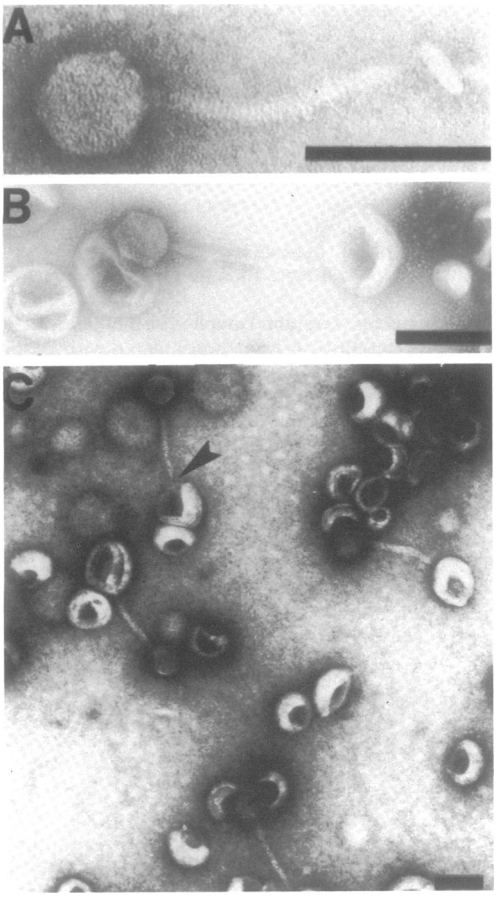

FIG. 1. Type 1 complexes. Electron micrographs of type 1 complexes between λh^+ and rod-shaped particles (A) or liposomes (B, C) bearing the *E. coli* receptor were prepared as described in the text. Note the presence of a gap at the banded region of the tail which at times can be seen to be traversed by the lambda tail fiber (arrow). Bar, 100 nm.

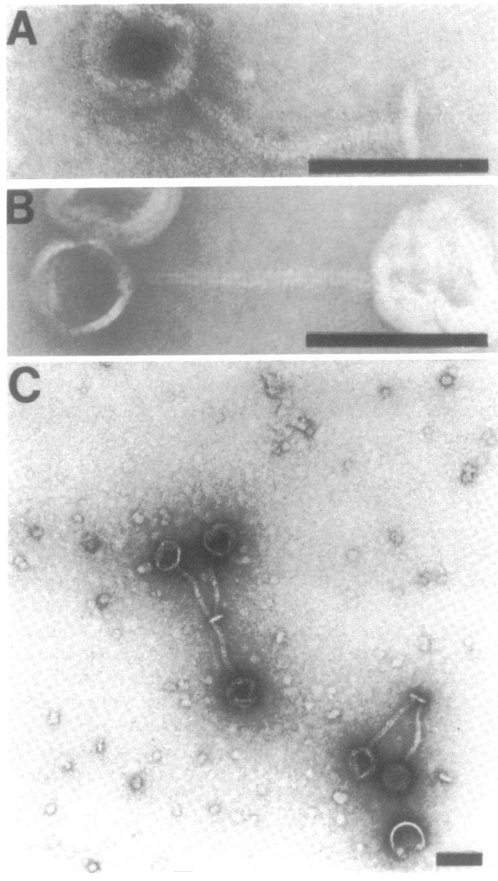

FIG. 2. Type 2 complexes. Electron micrographs of type 2 complexes between λh^+ and rod-shaped particles (A, C) or liposomes (B) bearing the *Shigella* receptor were prepared as described in the text. Bar, 100 nm.

the distance separating the heads of the bacteriophages from the particles or liposomes in type 1 complexes formed under a variety of conditions was 170 ± 6 nm (Fig. 4A through D). These measurements suggest that type 1 complexes are formed by an interaction between the distal tip of the tail fiber and the receptor.

A few of the complexes represented in Fig. 4 have short separation distances of less than 150 nm and do not have a gap between the end of the tail and the liposomal membrane. It is possible that these complexes are type 2 complexes (see below), but it is also possible that the insertion point of these tails may have been obscured by the overlying vesicle so that the distances measured are too short. We found an approximately equal number of bacteriophages which overlay

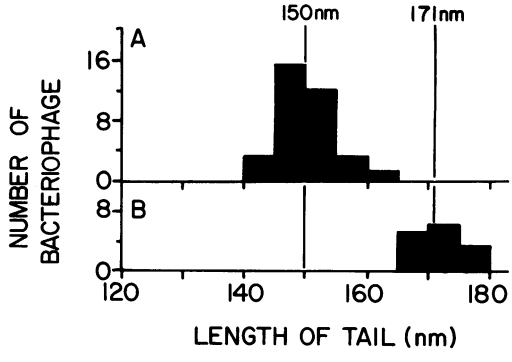

FIG. 3. Length of the lambda tail and tail fiber. The length of the banded region of lambda tails (A) and of the entire tails including the tail fibers (B) of free bacteriophages were determined from electron micrographs. Vertical lines indicate the mean values of 150 and 171 nm.

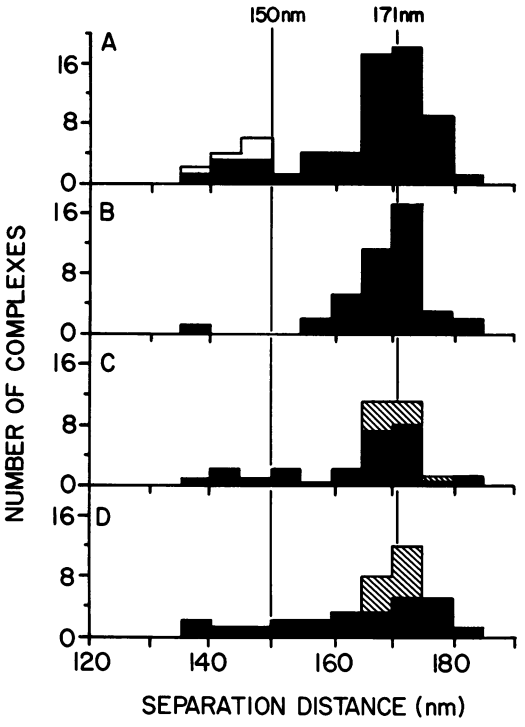

FIG. 4. Separation distances for type 1 complexes. Type 1 complexes were formed in several ways, and the distance separating the bacteriophage heads from the surface of the liposomes or rod-shaped particles was determined from electron micrographs. Filled and hatched squares represent bacteriophages whose heads contain DNA. Open squares represent bacteriophages which do not contain DNA. Hatched areas correspond to complexes in which the bacteriophage tail continued beyond the edge of the vesicle so that the tail overlay the vesicle. This is presumed to be a consequence of the geometry of the mode of attachment. The entire tail length was measured rather than the distance from the head to the rim of the liposome. Vertical lines indicate the length of the banded region of the lambda tail (150 nm) and the length of the entire tail including the tail fiber (171 nm). (A) Complexes of λh^+ and particles carrying the *E. coli* receptor formed at 37°C in 10 mM Mg^{2+}–40 mM CsCl. (B) Complexes of λh^+ and particles carrying the *Shigella* receptor formed at 4°C in 10 mM Mg^{2+}–40 mM CsCl–100 mM NaCl. (C) Complexes of λh^+ and liposomes bearing the *E. coli* receptor formed at 37°C in 2 mM Mg^{2+}–40 mM CsCl. (D) Complexes of λh^+ and liposomes bearing the *Shigella* receptor formed at 4°C in 10 mM Mg^{2+}–40 mM CsCl–100 mM NaCl.

the vesicle and whose tails continued past the edge of the vesicles. In these cases, the length of the entire visible tail and tail fiber has been measured. These bacteriophages are represented in Fig. 4 by hatched squares.

DNA in bacteriophage heads in type 1 complexes. Incubation of wild-type bacteriophages (λh^+) with preparations of free receptor protein from *E. coli* leads to reversible binding but not to inactivation of the bacteriophages (11, 13). Similar results were obtained when λh^+ was incubated with liposomes or rod-like particles bearing the *E. coli* receptor. Nearly 100% of the bacteriophages remained viable and were able to form a plaque when incubated with plating bacteria despite preincubation with the liposomes. Depending on the concentrations of divalent and monovalent ions, 33 to 81% of the λh^+ bacteriophages could be seen by electron microscopy to be bound to the liposomes (Table 1, lines 1 and 2). The higher frequency of complexes observed at 2 mM Mg^{2+} is in agreement with results reported by Schwartz (13), who found that the complex has a half-life of about 40 min in 2 mM Mg^{2+} but only 1 to 2 min in 10 mM Mg^{2+}. Table 1 (lines 1 and 2) and Fig. 1 show also that the heads of nearly all of the attached bacteriophages contained DNA, as would be anticipated since attachment is reversible and the bacteriophages remain viable. All of the complexes which had measured separation distances near 170 nm contained DNA (Fig. 4A and C).

When λh^+ is incubated with free receptor preparations from *Shigella* sp. at 37°C, the bacteriophage is inactivated, and the DNA is ejected (15). The bacteriophages were also inactivated when λh^+ was incubated with liposomes or rod-shaped particles bearing the *Shigella* receptor since by plaque assay more than 99% of the bacteriophages were inactivated. Inactivation and DNA ejection did not occur at 4°C. When bacteria to be plated were incubated for 30 min with the mixture of liposomes and bacteriophages at 4°C before plating at 37°C, nearly all of the bacteriophages formed plaques, indicating that the complexes formed at 4°C are reversible. Examination in the electron microscope of the complexes formed at 4°C with the *Shigella* receptor demonstrated that they were type 1 complexes and that all of the bacteriophage heads contained DNA (Fig. 4B and D).

Type 2 complexes. In type 2 complexes, the banded portion of the bacteriophage tail appeared to be directly attached to the surface of the rod-like particle (Fig. 2A and C) or to the liposomal membrane (Fig. 2B) with no visible gap. The mean and standard deviation for distance separating the bacteriophage heads from the surface of the particles or liposomes in type 2 complexes formed under a variety of conditions was 145 ± 6 nm (Fig. 5A through C).

DNA in bacteriophages in type 2 complexes. Complexes formed at 37°C between λh^+ and liposomes or particles bearing the *Shigella* receptor were observed in the electron microscope to be type 2 complexes in which the bacterio-

TABLE 1. Bacteriophage-liposome complexes[a]

Bacteriophage	Vesicles	Ions	Attached		Unattached	
			% Empty heads	% Full heads	% Empty heads	% Full heads
λh^+	*E. coli*	Standard[b]	<1	33	<2	65
λh^+	*E. coli*	2 mM Mg^{2+}	6	75	1	18
λh^+	*Shigella*[c]	Standard	61	19	17	3
λh	*E. coli*	Standard	32	22	34	12

[a] Complexes were formed and prepared for electron microscopy as described in the text. Incubation was for 30 min at 37°C.

[b] Standard conditions are 10 mM Tris-hydrochloride, pH 7.5, 10 mM $MgSO_4$, 100 mM NaCl, and 40 mM CsCl.

[c] Similar results were obtained with vesicles bearing the *Shigella* receptor isolated from Pel^- bacteria (Pop 157).

phage heads were largely empty of DNA (Fig. 5). Most (76%) of the attached bacteriophages had empty heads (Table 1, line 3). Only 3% of the bacteriophages were unattached and had full heads. About 17% of the bacteriophages were unattached but nevertheless had empty heads, although bacteriophages visualized without the addition of receptor or receptor-bearing liposomes contained less than 1% empty heads. The substantial increase in unattached, empty-headed bacteriophages may indicate that the liposome preparations contain some free receptor, perhaps derived from disintegrated liposomes, or that some of the bacteriophages were released from the liposomes after DNA ejection.

A host range mutant of lambda, λh, has the ability to form plaques on certain strains of *E. coli* to which λh^+ cannot adsorb. When λh is incubated with free receptor preparations from *E. coli*, the bacteriophages are inactivated, and the DNA is ejected (11). Similar results were obtained when λh was incubated with liposomes bearing the *E. coli* receptor. By plaque assay, 90 to 100% of the bacteriophages were inactivated. With the electron microscope, only 12% of the bacteriophages were found to be unattached with full heads (Table 1, line 4). An additional 22% were bound but had full heads (Table 1, line 4), which is about the same percentage observed with λh^+ and liposomes bearing the *Shigella* receptor (Table 1, line 3). These bacteriophages could be either reversibly or irreversibly adsorbed, although the high fraction of bacteriophages that were inactivated as measured by plaque assay suggests that the bacteriophages may be irreversibly adsorbed despite the fact that their DNA has not been ejected. About 66% of the bacteriophages have empty heads, and of these, about half are attached to liposomes and half are free. The high frequency of free bacteriophages with empty heads observed for λh is probably due to the increased frequency of spontaneous ejection for λh bacteriophages since about 20% of λh bacteriophages in preparations visualized without the addition of receptor or receptor-bearing liposomes have empty heads. Randall-Hazelbauer and Schwartz (11) have also commented upon the diminished stability of host range mutants of lambda.

Length measurements demonstrate that type 2 complexes form at 37°C either with λh and the *E.*

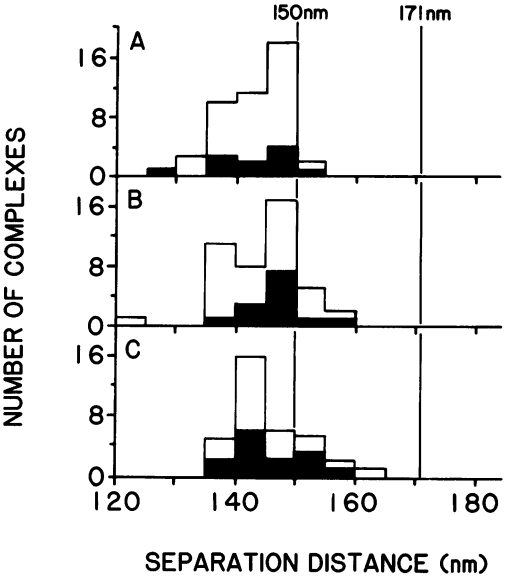

FIG. 5. Separation distances for type 2 complexes. Type 2 complexes were formed in several ways, and the distance separating the bacteriophage heads from the surface of the liposomes or particles was determined from electron micrographs. Closed and open squares represent bacteriophages whose heads do and do not contain DNA, respectively. (A) Complexes of λh^+ and particles carrying the *Shigella* receptor formed at 37°C in 10 mM Mg^{2+}–40 mM CsCl. (B) Complexes of λh and liposomes bearing the *E. coli* receptor formed at 37°C in 10 mM Mg^{2+}–40 mM CsCl–100 mM NaCl. (C) Complexes of λh^+ and liposomes bearing the *Shigella* receptor formed at 37°C in 10 mM Mg^{2+}–40 mM CsCl–100 mM NaCl.

coli receptor or λh^+ and the *Shigella* receptor (Fig. 5). Most of the bacteriophages represented in Fig. 5 have empty heads (indicated by empty squares), but 24% of those in Fig. 5A (λh^+, particles containing *Shigella* receptor), 29% of those in Fig. 5B (λh, liposomes containing *E. coli* receptor), and 40% of those in Fig. 5C (λh^+, liposomes containing *Shigella* receptor) have full heads. This observation suggests that the type 2 complex forms before DNA ejection or at least does not require DNA ejection for formation.

Conversion of type 1 complexes to type 2 complexes. Complexes formed by λh^+ and the *Shigella* receptor at 4°C in the presence of 2 mM Mg^{2+} appear to be irreversible since these incubation conditions lead to bacteriophage inactivation. Examination of these complexes revealed that the base of the bacteriophage tail was separated from the surface of the receptor-bearing particle by a gap similar to that observed in the reversible type 1 complexes formed in the presence of 10 mM Mg^{2+} and 100 mM NaCl and that the bacteriophage heads still contained DNA (Table 1, lines 1 and 2). Thus, the reversibility of type 1 complexes is dependent upon the ionic conditions used for their formation. The irreversible type 1 complexes formed in 2 mM Mg^{2+} have separation distances somewhat shorter than those in the reversible complexes formed at higher salt concentrations (compare Fig. 6A with Fig. 4A). This might indicate a structural change in the bacteriophage-receptor complex which causes bacteriophage inactivation without DNA ejection.

When the irreversible type 1 complexes were shifted to 37°C, they were almost quantitatively converted to type 2 complexes without a gap between the base of the bacteriophage tail and the surface of the receptor-bearing particle (Fig. 6A and B). Similar results were obtained in experiments with λh^+ and the rod-shaped form of the *E. coli* receptor (Fig. 6C and D). In this latter case, it was necessary to add chloroform to obtain irreversible type 1 complexes during the initial incubation at 4°C and for their subsequent conversion to type 2 complexes after the shift to 37°C. With either receptor, the majority of the bacteriophages had ejected their DNA during or after conversion of the type 1 to type 2 complexes (Fig. 6B and D).

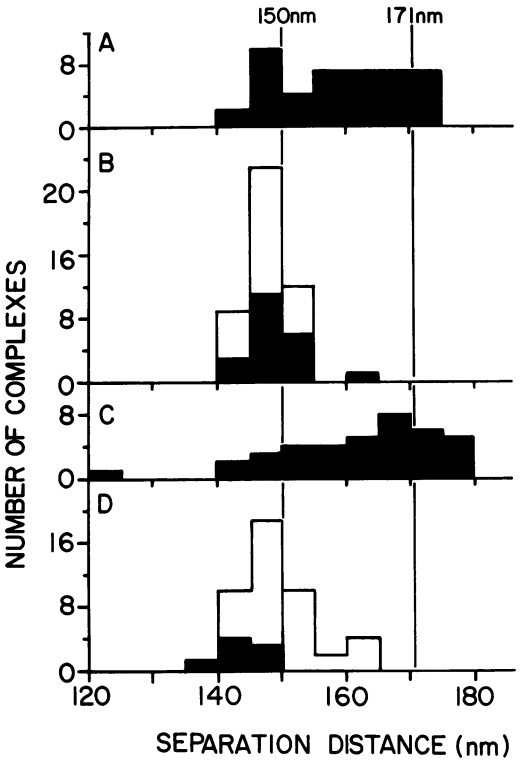

FIG. 6. Conversion of type 1 complexes to type 2 complexes. Histograms were constructed as in Fig. 4 and 5. Closed and open squares are as in Fig. 5. (A) Complexes of λh^+ and particles carrying the *Shigella* receptor formed at 4°C in 2 mM Mg^{2+}. (B) The above complexes from (A) then incubated at 37°C. (C) Complexes of λh^+ and particles carrying the *E. coli* receptor formed at 4°C in 2 mM Mg^{2+} and incubated with $CHCl_3$ at 4°C. (D) The above complexes from (C) subsequently incubated at 37°C.

DISCUSSION

Bacteriophage lambda was observed to form two types of complexes after binding to structures bearing the lambda receptor, either liposomes or rod-shaped particles found in crude cholate extracts of bacterial membranes. Both types of complexes can be observed with either *E. coli* or *Shigella* receptors. In type 1 complexes, the bacteriophages are bound to the receptor by the tip of the tail fiber, and the bacteriophage head is separated from the receptor-bearing structure by about 170 nm. In some type 1 complexes, the thin 21-nm-long lambda tail fiber can be seen extending between the distal end of the banded part of the tail and the liposomal membrane or the rod-shaped particle. Since the banded part of the bacteriophage tail is about 150 nm long, it appears that binding occurs near the end of the tail fiber. In most type 1 complexes, the tail fiber cannot be visualized, and so these complexes appear to have a gap whose measured width has a mean value of 17 nm. Since these complexes are observed under conditions in which binding is reversible and the bacteriophages are not inactivated, we believe that the type 1 complexes are the result of a simple and reversible binding between the receptor protein and the tip of the tail fiber, as studied kinetically by Schwartz (13).

In type 2 complexes, the distal end of the banded part of the bacteriophage tail is directly attached to the liposomal membrane or rod-shaped particle. Tail fibers cannot be visualized in type 2 complexes. The mean distance separating the bacteriophage heads from the liposomes or particles is 145 nm, which is shorter than that observed for type 1 complexes by a length approximately equal to the 21-nm length of the tail fiber. Type 2 complexes can be formed from type 1 complexes and seem to represent a subsequent and more direct interaction between the surface of the receptor-bearing particles and the hollow tubes of the lambda tails through which DNA exits from the head. It is likely that this direct association is a prerequisite for normal DNA injection across the membrane to occur. DNA ejection, however, cannot be a requirement for type 2 complex formation since the heads in 30 to 40% of the type 2 complexes still contain DNA.

There may exist intermediates between type 1 and type 2 complexes. Irreversible type 1 structures were observed which were found to have the heads and receptors separated by distances between 145 and 170 nm. Complexes of λh^+ formed at 4°C in 2 mM Mg^{2+} in the presence of chloroform with rod-like particles bearing the *E. coli* receptor or in the absence of chloroform with structures bearing the *Shigella* receptor do not dissociate to yield viable bacteriophages, although the bacteriophage heads still contain DNA. The absence of reversible adsorption does not simply reflect the higher affinity that exists in 2 mM Mg^{2+}, compared with affinity at higher ionic strengths (13), since some of these complexes have measured separation distances that are shorter than 170 nm. These structures may represent intermediates on the pathway to DNA ejection since they can be converted by a temperature shift to type 2 complexes which are mostly empty headed.

When type 1 complexes are converted to type 2 complexes, at least three fates for the tail fibers can be imagined. The tail fibers may extend through the membranes into the internal aqueous space, they may be flattened onto the inner or outer surface of the liposomal membranes or rod-shaped particles, or they may be released from the bacteriophage tails, as occurs when bacteriophage lambda is incubated in 5 M LiCl (7). A ratchet-like mechanism could draw the tail fiber in if the fiber had several high-affinity contact zones for receptor arranged linearly, as might occur by end-to-end association of J proteins. The tail fiber might be carried into or through the membrane, since receptor molecules capable of binding the tail fiber seem to be located on both the outer and the inner aspects of the *E. coli* outer membrane (5). The distal tip of the banded region of the tail could be drawn into or through the membrane so that the DNA could directly exit on the opposite side, as suggested by the fact that the banded region of the tail is 150 nm long and the measured separation distance in type 2 complexes is 145 nm. As an alternative, DNA entry could occur through the membrane pore formed by the lambda receptor to facilitate entry of maltodextrines or through a pore created by the lambda J protein alone or in conjunction with the lambda receptor.

LITERATURE CITED

1. **Bayer, M. E.** 1968. Adsorption of bacteriophages to adhesions between wall and membrane of *Escherichia coli*. J. Virol. **2**:346–356.
2. **Boehler-Kohler, B. A., W. Boos, R. Dieterle, and R. Benz.** 1979. Receptor for bacteriophage lambda of *Escherichia coli* forms larger pores in black lipid membranes than the matrix protein (porin). J. Bacteriol. **138**:33–39.
3. **Buchwald, M., and L. Siminovitch.** 1966. Production of serum-blocking material by mutants of the left arm of the λ chromosome. Virology **38**:1–7.
4. **Dove, W. F.** 1966. Action of the lambda chromosome. I. Control of functions late in bacteriophage development. J. Mol. Biol. **19**:187–201.
5. **Gabay, J., and K. Yasunaka.** 1980. Interaction of the *lamB* protein with the peptidoglycan layer in *Escherichia coli* K12. Eur. J. Biochem. **104**:13–18.
6. **Goldberg, E.** 1980. Bacteriophage nucleic acid penetration, p. 115–141. *In* L. L. Randall and L. Philipson (ed.), Virus receptors. Chapman and Hall, London.
7. **Konopa, G., and K. Taylor.** 1979. Coliphage λ ghosts obtained by osmotic shock or LiCl treatment are devoid of J- and H- gene products. J. Gen. Virol. **43**:729–733.
8. **Luckey, M., and H. Nikaido.** 1980. Specificity of diffusion channels produced by λ phage receptor protein of *Escherichia coli*. Proc. Natl. Acad. Sci. U.S.A. **77**:167–171.
9. **MacKay, D. J., and V. Bode.** 1976. Binding to isolated phage receptors and λ DNA release *in vitro*. Virology **72**:167–181.
10. **Nakae, T.** 1979. A porin activity of purified λ-receptor protein from *Escherichia coli* in reconstituted vesicle membranes. Biochem. Biophys. Res. Comm. **88**:774–781.
11. **Randall-Hazelbauer, L., and M. Schwartz.** 1973. Isolation of the bacteriophage lambda receptor from *Escherichia coli*. J. Bacteriol. **116**:1436–1446.
12. **Roa, M., and D. Scandella.** 1976. Multiple steps during the interaction between coliphage lambda and its receptor protein *in vitro*. Virology **72**:182–194.

12a. **Roessner, C. A., D. Struck, and G. Ihler.** 1983. Injection of DNA into liposomes by bacteriophage λ. J. Biol. Chem. **258**:643–648.

13. **Schwartz, M.** 1975. Reversible interaction between coliphage lambda and its receptor protein. J. Mol. Biol. **99**:185–201.
14. **Schwartz, M.** 1980. Interaction of phages with their receptor proteins, p. 59–94. *In* L. L. Randall and L. Philipson (ed.), Virus receptors. Chapman and Hall, London.
15. **Schwartz, M., and L. Le Minor.** 1975. Occurence of the bacteriophage lambda receptor in some Enterobacteriaceae. J. Virol. **15**:679–685.
16. **Shaw, J. E., H. Bingham, C. Fuerst, and M. Pearson.** 1977. The multisite character of host-range mutations in bacteriophage λ. Virology **83**:180–194.
17. **Yamada, H., T. Nogami, and S. Mizushima.** 1981. Arrangement of bacteriophage lambda receptor protein (LamB) in the cell surface of *Escherichia coli*: a reconstitution study. J. Bacteriol. **147**:660–669.