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Maltose Transport in *Escherichia coli* K-12: Involvement of the Bacteriophage Lambda Receptor

SEVEC SZMELCMAN* AND MAURICE HOFNUNG
Unité de Génétique Moléculaire, Institut Pasteur, 75015 Paris, France

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Mutants affected in *lamB*, the structural gene for phage λ receptor, are unable to utilize maltose when it is present at low concentrations (≤10 μM). During growth in a chemostat at limiting maltose concentrations, the *lamB* mutants tested were selected against in the presence of the wild-type strain. Transport studies demonstrate that most *lamB* mutants have deficient maltose transport capacities at low maltose concentrations. When antibodies against purified phage λ receptor are added to a wild-type strain, transport of maltose at low concentrations is significantly reduced. These results strongly suggest that the phage λ receptor molecule is involved in maltose transport.

The transport of maltose in *Escherichia coli* K-12 is ensured by a specific mechanism that is energy dependent (19) and allows accumulation of maltose against a concentration gradient.

It is known that the genes involved in maltose utilization are located in two regions, *malA* and *malB* (15). These two regions are formed by three operons positively controlled by the gene *malT* (Fig. 1). The products of *malF*, *malK*, and *malE*—the latter recently shown to be a "periplasmic" maltose-binding protein (10)—are necessary for maltose transport (7). The product of *lamB* was shown to be the phage λ receptor, a protein of the outer membrane (14).

The location of the *lamB* gene in a region otherwise devoted to the transport of maltose suggested that the *lamB* product could play a role, as yet undetected, in this process.

It was recently reported (4) that *lamB* mutants are affected in their taxis towards maltose. Here we present evidence that the *lamB* product is directly involved in the transport of maltose but is essential only at low external concentrations of this sugar.

In the accompanying paper (5) Hazelbauer reaches similar conclusions.

MATERIALS AND METHODS

Chemicals. [U-^{14}C]maltose with a specific activity of 7 mCi/mmol was purchased from the Radiochemical Centre (Amersham, England). [^{14}C]thiomethyl β-D-galactoside (0.25 mCi/mmol; New England Nuclear Corp.) was kindly supplied by A. Ullman.

Media and bacterial strains. The strains of *E. coli* K-12 used are listed in Table 1. Minimal medium M63 (13) was supplemented with 0.01% of the necessary L-amino acids and 0.4% of the carbohydrates used as energy sources. Strains unable to utilize maltose were grown on 0.4% glycerol with 50 mM maltose as inducer.

For measurement of thiomethyl β-D-galactoside transport, cells were induced with 10 mM isopropyl thiogalactoside.

Transport assays. Cells grown at 37 C were harvested in the exponential phase, washed with M63, and suspended in the same medium containing 40 μg of chloramphenicol per ml. After adjustment of the absorbance to 0.6 at 600 nm, the cells were incubated at 25 C for 15 min. At time zero, 3 ml of the bacterial cell suspension was withdrawn and quickly mixed with the radioactive substrate. Harvesting and subsequent steps were performed at 25 C. At different time intervals, 0.5-ml samples were taken and passed through membrane filters (Millipore Corp.; type HA; 0.45-μm pore size), washed with 5 ml of M63 at room temperature, dried, and counted in a Intertechnique liquid scintillation counter with toluene-based scintillation fluid. It must be pointed out that, in contrast to the old technique (19) in which maltose penetration was studied over 15 to 30 min, we used a fast kinetic method in which maltose transport was followed for 15 to 180 s. Under these conditions, the contribution of maltose metabolism is negligible (at 30 s all of the accumulated radioactivity could be displaced by unlabeled maltose).

Bacterial growth in a chemostat. The chemostat consisted of a growth chamber (New Brunswick Micro Ferm) kept in a constant-temperature water bath at 37 C. The flow rate (14 ml/h) of the culture medium was regulated with two well-controlled peristaltic pumps.

Bacteria were grown at limiting carbohydrate concentrations, in a constant volume (100 ml) of M63, with a mean generation time of usually 7 h. The culture was well aerated and continuously mixed with a magnetic stirrer. At regular time intervals, samples were withdrawn under sterile conditions; one fraction

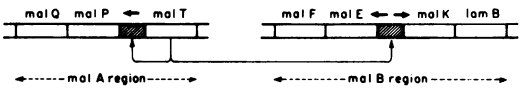

FIG. 1. *malA and malB regions of E. coli K-12. Drawn according to the recombination and complementation data of Hofnung et al. (6, 7). Genes malP and malQ are the respective structural genes for maltodextrin phosphorylase and amylomaltase. The region located between malE and malK comprises the promoters and initiators for the two divergent operons, malK-lamB and malE-malF; the region located between malT and malP comprises the promoter and initiator of the operon malP-malQ.*

TABLE 1. *Bacterial strains*[a]

Name	*lamB* mutations	Other relevant markers
pop 1048		*metA lacZ*^{am}
pop 1093	*lamB 13* missense	*metA lacZ*^{am}
pop 1067	*lamB5* missense	*metA lacZ*^{am}
pop 1071	*lamB9* nonsense	*metA lacZ*^{am}
pop 1050	*lamB200* nonsense	*metA lacZ*^{am} *galE galy*
pop 1078		*trp*^{am}*galE galy*
pop 1075	*lamB 204* nonsense	*trp*^{am}*galE galy*
pop 1076	*lamB5* missense	*trp*^{am}*galE galy*
pop 1077	*lamB9* nonsense	
pop 1021		*metA trp*^{am} *galE galy*
pop 1079	*lamB101* missense	*metA trp*^{am} *galE galy*
pop 1081	*lamB103* missense	*metA trp*^{am} *galE galy*
pop 1082	*lamB104* missense	*metA trp*^{am} *galE galy*
pop 1083	*lamB105* missense	*metA trp*^{am} *galE galy*
pop 1084	*lamB106* missense	*metA trp*^{am} *galE galy*
pop 1085	*lamB107* missense	*metA trp*^{am} *galE galy*
pop 1086	*lamB108* missense	*metA trp*^{am} *galE galy*
pop 1087	*lamB109* missense	*metA trp*^{am} *galE galy*
pop 1088	*lamB110* missense	*metA trp*^{am} *galE galy*
pop 1090	*lamB112* missense	*metA trp*^{am} *galE galy*
pop 1091	*lamB113* missense	*metA trp*^{am} *galE galy*
pop 1000		*his malQ7*
pop 1730	*malBΔ17* deletion	*his malBΔ17*

[a] Strains pop 1075 through pop 1078 were obtained from strain pop 1021 by P1 transduction, so that they are essentially isogenic. The characterization of the *lamB* mutations will be described elsewhere (M. Hofnung, A. Jezierska, and C. Braun, manuscript in preparation). *galy* is a mutation located in the *gal* region and confers galactose resistance to the *galE* mutant.

was spread on eosin methylene blue-maltose agar plates, and the rest was used for measuring the absorbance at 600 nm. The eosin methylene blue plates were then incubated at 37 C overnight, and 60 colonies from each plate were tested for their sensitivity to phage λv.

Treatment of bacterial cells with antisera. The antiserum made against phage λ receptor (generously provided by M. Schwartz) gives only one detectable immunoprecipitation band with a *lamB*$^+$ strain extract as detected on double-diffusion plates (H. Shuman, personal communication). The preparation and specificity of the antiserum to maltose-binding protein were described previously (10). Before incubation with antiserum, bacterial cells were washed thoroughly with a saline solution (0.9% NaCl in water) and then suspended in saline with chloramphenicol (40 μg/ml). The final dilution of the antiserum was 1:30.

Abbreviations. The genetic nomenclature follows that suggested by Taylor and Trotter (17): λ^s, λ sensitive; λ^r, λ resistant; Mal$^+$, able to grow on maltose at high maltose concentration (1 mM); Mal$^-$, unable to grow on maltose at high concentration (1 mM).

RESULTS

Growth at limiting maltose concentrations. Previously, mutations located inside *lamB* had not been found to affect maltose metabolism, and the resulting phenotype was accordingly noted as λ^r Mal$^+$. Indeed, *lamB* mutants are able to grow on maltose at the concentrations routinely used (1 mM), and no difference was detected either in the growth rate or yield between a λ^s Mal$^+$ strain and an isogenic λ^r Mal$^+$ derivative.

However, when two such strains are mixed and grown together in a chemostat under conditions of limiting carbon source, the following observations can be made. When maltose is the carbon source, the growth of the λ^r strain is dramatically affected. In the experiment illustrated in Fig. 2, the λ^r strain did not grow, whereas the λ^s strain grew and reached almost 100% of the population after four generations.

When the carbon source is lactose or glycerol, the growth behavior of both λ^s and λ^r strains is identical. In Fig. 3 the ratio of λ^s to λ^r strains is

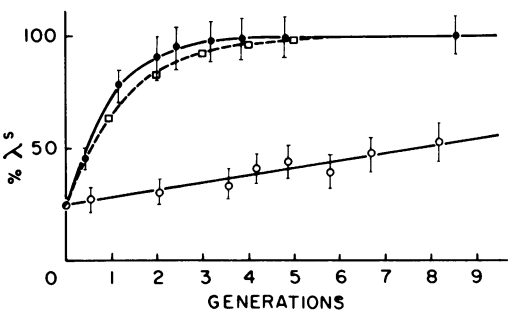

FIG. 2. *Growth in chemostat of λ^s and λ^r strains at limiting concentrations of maltose or glucose. The wild-type λ^s strain pop 1078 was mixed 1:3 with the isogenic strain pop 1075 carrying a lamB nonsense mutation and grown in a chemostat at 37 C. At regular intervals samples were withdrawn and plated on eosin methylene blue-maltose agar plates, and 60 colonies from each plate were scored for their sensitivity to phage λv. Symbols: ●, growth on maltose; ○, growth on glucose; □, theoretical curve obtained assuming that the λ^r strain is not growing when maltose is the limiting factor; I, statistical sampling error.*

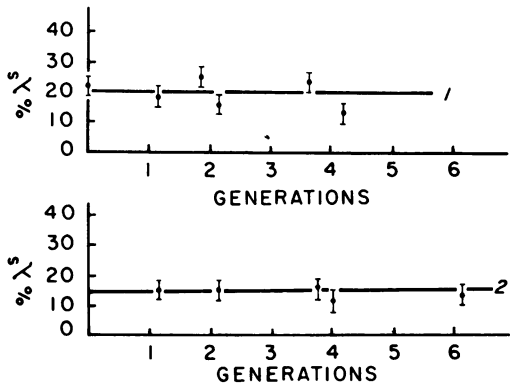

FIG. 3. *Growth in chemostat of λ^s and λ^r strains at limiting concentrations of lactose or glycerol. The wild-type λ^s strain pop 1078 was mixed 1:4 with the isogenic strain pop 1075 carrying a lamB nonsense mutation and grown in a chemostat at 37 C. At regular intervals, samples were withdrawn and plated on eosin methylene blue-maltose agar plates, and 60 colonies from each plate were scored for their sensitivity to phage λv. Curve 1, Growth on lactose; curve 2, growth on glycerol;* $\overline{\mathrm{I}}$ *, statistical sampling error.*

constant. When the carbon source is glucose (Fig. 2), a slow increase of this ratio is observed.

It can thus be concluded that λ^r Mal^+ mutants are impaired in their ability to grow at low maltose concentrations. This impairment does not extend to growth on low concentrations of lactose or glycerol. The possible reasons for the slight effect observed on glucose will be examined in the Discussion.

Transport capacities of lamB mutants at different maltose concentrations. The above observations could be accounted for if the *lamB* product, which is localized on the outer membrane of the bacterium (14), played a role in maltose entry. To test this assumption, transport studies at a maltose concentration of 3.5 μM were performed with *lamB* mutants and compared with those of the wild-type strain.

The initial rates for maltose transport measured in two strains (pop 1071 and pop 1050) harboring *lamB* nonsense mutations (Fig. 4) are less than 2% that of the wild type. They reach 30% of the normal level in the presence of a nonsense suppressor (Fig. 5). With all the other nonsense mutation tested, the rate was less than 5% that of the wild type. This would mean that the *lamB* nonsense mutations affect transport either through the inactivation of the *lamB* gene itself or through a polar effect exerted on a yet unknown distal gene.

The initial rates of entry in strains carrying missense mutations (pop 1063 and pop 1067, Fig. 4 and Table 2) range from 6 to 91% that of the induced wild type. Since no polar effect is expected for missense mutations, it is likely that *lamB* mutations affect transport by altering the *lamB* product.

The thiomethyl β-D-galactoside transport kinetics in strains carrying *lamB* nonsense mutations are identical to that of the wild type (Fig. 6). The same results were obtained with α-methylglucoside (data not shown). Thus, a pleiotropic effect of the *lamB* mutations on different transport activities can be excluded.

All the *lamB* mutants used in the present

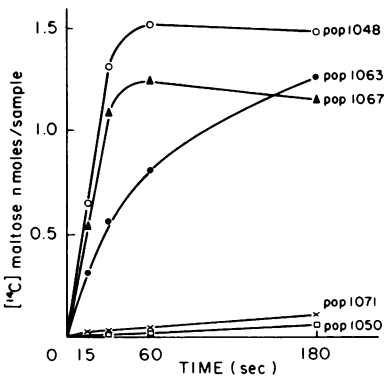

FIG. 4. *Uptake kinetics of maltose by lamB mutants. After adjustment of the absorbance to 0.6 at 600 nm in the presence of chloramphenicol (40 μg/ml), 3 ml of bacteria was rapidly mixed with [^{14}C]maltose (3.5 μM final concentration) and incubated at 25 C. At given time intervals, 0.5-ml samples were taken, quickly filtered, washed with 5 ml of M63, dried, and counted. Symbols:* ○, *wild-type pop 1048;* ×, *lamB nonsense pop 1071;* ●, *lamB missense pop 1063;* □, *lamB nonsense pop 1050;* ▲, *lamB missense pop 1067.*

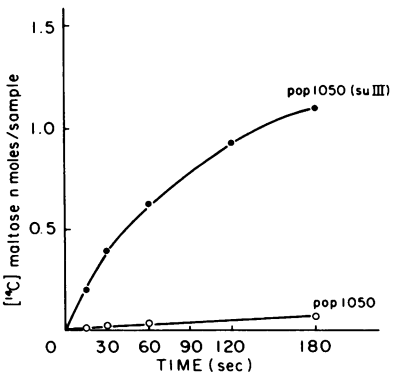

FIG. 5. *Uptake kinetics of maltose by a lamB strain carrying a nonsense mutation in the absence and in the presence of a nonsense supressor. For details see Fig. 4 and the text.*

TABLE 2. *Initial rates of maltose transport in lamB missense mutants*[a]

Strains	Initial rates[b] (nmol/min per sample)	% Wild type
Wild type		
pop 1048	2.2	
Mutants		
pop 1087	0.14	6.5
pop 1086	0.44	21
pop 1079	0.53	24
pop 1090	0.55	25
pop 1085	0.72	33
pop 1091	1.02	46.5
pop 1083	1.27	58
pop 1082	1.47	67
pop 1084	1.51	68.5
pop 1081	1.58	72
pop 1088	1.76	80
pop 1067	2.0	91

[a] The transport assays were made with 3.5 μM [^{14}C]maltose. At given intervals, 0.5-ml samples were withdrawn and filtered through membrane filters (Millipore Corp.), dried, and counted.

[b] The initial rate values were calculated from the 30-s points.

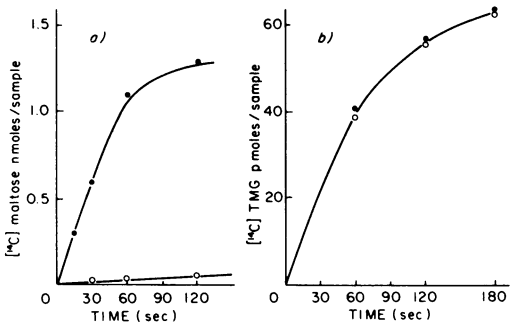

FIG. 6. *Maltose and thiomethyl β-D-galactoside (TMG) transport in a lamB strain carrying nonsense mutation pop 1077 and in the wild-type pop 1078. For details see the text and Fig. 4. For TMG uptake, the cells were induced with 10 mM isopropyl thiogalactoside. The final concentration of [^{14}C]TMG was 10 μM. (a) Maltose uptake; (b) TMG uptake. Symbols: ●, wild-type pop 1078; ○, lamB mutant pop 1077.*

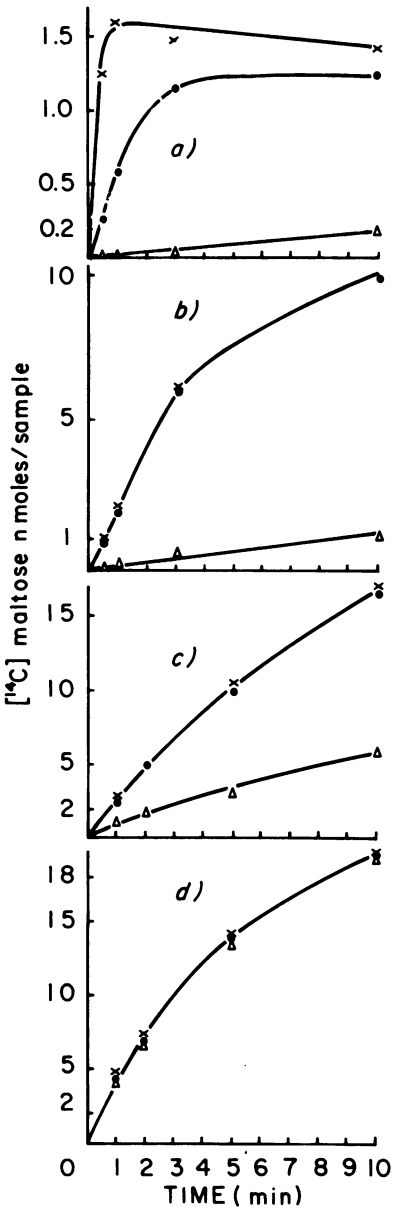

FIG. 7. *Transport kinetics of strains carrying lamB mutations at different maltose concentrations. The transport assays were performed at different external concentrations of maltose: (a) 3.5 μM, (b) 35 μM, (c) 0.1 mM, (d) 1 mM. Symbols: ×, wild-type pop 1048; ●, lamB missense pop 1067; Δ, lamB nonsense pop 1071.*

study were selected as having a Mal$^+$ phenotype, i.e., as growing well at 10 mM maltose on agar plates and in liquid medium, but showed defective maltose uptake at low concentrations (3.5 μm). Thus, it seemed interesting to follow the initial rates of transport of these strains at different external maltose concentrations. The results (Fig. 7) indicate that when the external concentration of maltose is increased from 3.5 μM to 1 mM the difference in the initial rates of the *lamB* mutants and the wild type decreases, the two rates reaching identical values at 1 mM maltose.

This change in the initial rates of transport in *lamB* mutants might reflect an increase in the apparent K_m for maltose transport in these strains without affecting the V_{max} for maltose uptake (see Discussion).

Direct involvement of the phage λ receptor in maltose transport. Further evidence for the direct involvement of the phage λ receptor in maltose uptake (rather than in the synthesis or assembly of transport components) was provided by the utilization of specific antibodies to purified phage λ receptor. Antiserum prepared against purified phage λ receptor was exhausted by incubation with whole bacterial cells bearing a deletion (*amlBΔ17*) extending from the *lamB* gene into part of the *malK* gene. This antiserum was added to a λ^s strain (pop 1000) and incubated for 1 h at 25 C. The bacteria were then washed, and maltose transport at an external concentration of 3.5 μM was tested.

The transport kinetics of strain pop 1000 after incubation with the exhausted antiserum show an initial rate of maltose transport reduced almost seven times as compared with the control (Fig. 8, curves 1 and 4). On the other hand, when the same antiserum was exhausted with a wild-type λ^s strain ($HfrG_6$), this antiserum had lost most of its capacity to inhibit maltose uptake (Fig. 8, curve 2).

The inactivation of the phage λ receptor did not affect thiomethyl β-D-galactoside transport (Fig. 9). An identical result was obtained with the transport of α-methylglucoside.

Thus, it can be concluded that the treatment of whole cells by antibodies against phage λ receptor mimics the effect of some *lamB* mutations and that the genetic inactivation of the phage λ receptor, as well as its inactivation by specific antibodies, acts only on the maltose transport mechanism, not affecting the other transport systems tested.

Addition of specific antiserum against the maltose-binding protein, an essential element of maltose transport (10), did not significantly change the initial rate of maltose transport (Fig. 8, curve 3). Since in vitro experiments had shown that the purified maltose-binding protein is inactivated by its specific antibodies, this result suggests that in whole cells the maltose-binding protein, in contrast to the phage λ receptor, is inaccessible to the specific antibodies probably because it is hidden by the outer membrane.

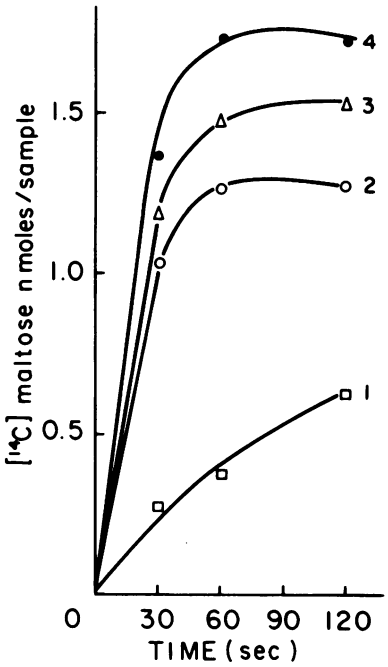

FIG. 8. *Inhibition of maltose transport by specific antibodies against phage λ receptor. λ^s strain pop 1000 was grown on glycerol with 50 mM maltose as inducer, harvested in exponential phase, and washed with saline. The cells were then suspended in saline with chloramphenicol (40 μg/ml), and the absorbance was adjusted to 0.66 at 600 nm. After 10 min of incubation at 25 C, 2.7 ml of the cell suspension was incubated with 0.3 ml of antiserum for 1 h at 25 C. The cells were centrifuged and then suspended in 3 ml of M63 in the presence of 10 mM glycerol and 40 μg of chloramphenicol per ml. At time zero, the cells were mixed with [^{14}C]maltose (3.5 μM final concentration), and maltose transport was measured as described in the text and in Fig. 4. Curve 1, In the presence of antiserum against phage λ receptor exhausted by the λ^r strain pop 1730 bearing the deletion malBΔ17 (see Results). Curve 2, In the presence of antiserum against phage λ receptor exhausted by the wild-type λ^s strain $HfrG_6$. Curve 3, In the presence of antiserum against the maltose-binding protein. Curve 4, Control in the presence of saline.*

DISCUSSION

The involvement of phage λ receptor in maltose utilization is demonstrated by the selective disadvantage displayed by *lamB* mutants under conditions of maltose limitation. This handicap is not apparent when these mutants are grown in 1 to 10 mM maltose, the concentrations normally used in culture media, so that *lamB* mutants are phenotypically Mal^+. No selective disadvantage was detected when glycerol or lactose was the limiting carbon source. The slight counterselection of *lamB* mutants under conditions of glucose limitation could be due to

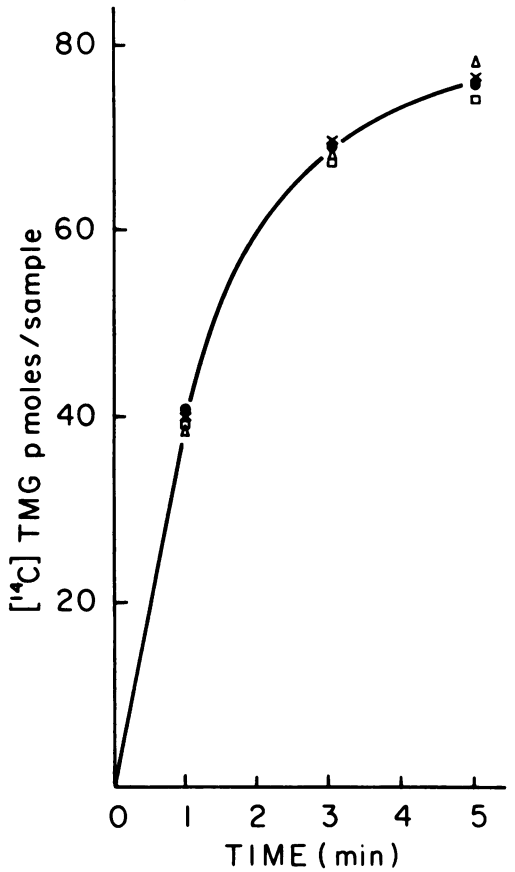

FIG. 9. *Action of specific antibodies against phage λ receptor on thiomethyl β-D-galactoside (TMG) transport. λ⁺ strain pop 1000 was grown on glycerol, with 10 mM isopropyl thiogalactoside as inducer, and submitted to the procedure described in the legend of Fig. 8, and thiomethyl β-D-galactoside (TMG) transport at 10 μM final concentration was tested. Symbols: □, In the presence of antiserum against phage λ receptor exhausted by the λʳ strain pop 1730 bearing the deletion malBΔ17; ×, in the presence of antiserum against phage λ receptor exhausted by the wild-type λ⁺ strain HfrG₆; Δ, in the presence of antiserum against the maltose-binding protein; ●, control in the presence of saline.*

a small contamination of glucose by maltose or to the inability of *lamB* mutants to recapture maltose or maltodextrins synthesized from glucose and released by the bacterial population. The possibility that phage λ receptor plays some role in glucose transport or catabolism seems unlikely for two reasons. (i) No involvement of the receptor in glucose transport could be demonstrated (unpublished data). (ii) The strains growing on lactose in the chemostat experiment can only utilize the glucose moiety of the lactose molecule because of two mutations in the *gal* operon (Table 1); however, no counterselection of *lamB* mutants is observed.

In all strains carrying *lamB* nonsense mutations, as well as in some of the strains carrying missense mutations, the initial rate of transport is significantly reduced with respect to the wild type at low maltose concentrations (below 0.1 mM), whereas it is essentially normal at high maltose concentration (above 1 mM). Thus, the selective disadvantage of *lamB* mutants at low maltose concentration can be accounted for by a defect in the maltose transport system.

The specific inhibition of maltose transport by anti-λ receptor antibodies provides further evidence for a role of the phage λ receptor in the permeation process. Furthermore, it suggests that the λ receptor molecules that take part in transport are located in the outer membrane—and are thus accessible to antibodies (16)—rather than deeper in the bacterial envelope. Indeed, proteins located below the outer membrane, such as the "periplasmic" maltose-binding protein, are not accessible to specific antibodies.

What could be the function of the phage λ receptor in maltose transport? The determination of kinetic parameters for maltose uptake in the wild type and in a *lamB* nonsense mutant give apparent K_m values of 1 μm and 1 mM, respectively, while the V_{max} is identical (S. Szmelcman, T. Silhavy and W. Boos, manuscript in preparation). These data suggest that the phage λ receptor bears specific binding sites for maltose. However, preliminary experiments fail to reveal any affinity of the receptor for this sugar. An alternative hypothesis would be that the phage λ receptor, rather than binding maltose, plays a role in positioning the maltose-binding protein so as to increase the accessibility of its binding site to external maltose. If such were the case, a specific interaction between phage λ receptor and the maltose-binding protein might be revealed in future studies.

Recently, Hazelbauer (4) has shown that the maltose-binding protein and the phage λ receptor are implicated in maltose taxis. These results provide further evidence of the close relationship between chemotaxis and transport, the "chemoreceptor" elements (the maltose-binding protein and phage λ receptor) being common to the two systems.

The bifunctional role played by the phage λ receptor in phage adsorption and in maltose

transport is not a unique case. Di Masi et al. (3) and Kadner and Liggins (9) reported that the common receptor for E colicins and bacteriophage BF23 is also involved in the transport of vitamin B_{12}. Wang and Newton presented evidence that in *tonB* mutants the apparent K_m for iron transport is increased 10-fold, while the V_{max} is unchanged. These authors suggested the existence of a common receptor on the outer membrane for phage T1 and for iron.

The role played by the outer membrane in the permeation process is not yet clear. Originally it was suggested by Cohen and Monod (1) that the cell wall, which comprises the outer membrane, "is not a significant element of the permeability barrier." Later, Mitchell proposed (11) that this outer membrane is a "molecular sieve," filtering molecules according to their size and charges. This proposition is reinforced by recent data on the chemical structure of the outer membrane (2, 8, 12). However, the inward penetration of extraneous molecules is, in some cases, mediated by elements of a specific transport system that are located on the outer membrane (transport of vitamin B_{12}, iron, and maltose). It is known, on the other hand, that the maltose transport machinery is also needed for the entry of maltodextrins. It appears possible, therefore, that the phage λ receptor might be more stringently needed for the entry of higher dextrins than for that of maltose. Indeed, strains carrying *lamB* nonsense mutations do not transport maltotriose even at an elevated concentration (10 mM) (Szmelcman et al., manuscript in preparation).

The selective advantage displayed by *lamB*$^+$ over *lamB* strains under maltose limitation is likely to play a role in the usual ecological niche of the bacteria. Thus, one might think that the favorable evolutionary process provided by the *lamB* product is not phage λ sensitivity but ability to concentrate maltose and maltodextrins. In agreement with this hypothesis is the fact that most Mal$^+$ strains of *E. coli* and *Shigella*, even though they do not support the growth of phage λ and often do not adsorb this phage, still have phage λ receptor protein which can be detected in cell extracts (16).

ACKNOWLEDGMENTS

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