Regulation of Membrane Glycosyltransferases by the sfrB and rfaH Genes of Escherichia coli and Salmonella typhimurium

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The role of sfrB and rfaH genes in the regulation of expression of membrane glycosyltransferases was studied in Escherichia coli and Salmonella typhimurium. The transferase enzymes form part of a multienzyme system involved in biosynthesis of the polysaccharide core of Gram-negative bacterial lipopolysaccharides. Several sfrB mutants of E. coli showed reductions of 90–98% in the activities of two of the glycosyltransferases (UDP-galactose:(glucosyl)lipopolysaccharide 1,6-galactosyltransferase and UDP-glucose:(glucosyl)lipopolysaccharide 1,3-galactosyltransferase). Introduction of a recombinant ColE1 plasmid restored the transferase levels to normal and characterized other regions of the E. coli chromosome which were ineffective. An amber mutation of the sfrH gene (thought to be the homologue of the E. coli rfaH gene) resulted in 97% loss of activity of the Salmonella UDP-galactose:(glucosyl)lipopolysaccharide galactosyltransferase. Antibody precipitation studies showed that the loss of enzyme activity in the amber mutant was associated with a corresponding decrease in amount, but not in size, of the transferase protein, indicating that the gene is not the structural gene for the S. typhimurium galactosyltransferase. Taken together, these results indicate that the sfrB and rfaH gene products act as a positive regulatory element in expression of multiple glycosyltransferases in E. coli and S. typhimurium.

Mutations of the sfrB gene of Escherichia coli are associated with abnormalities in several F-factor functions. These include surface exclusion properties, formation of F-pili, and ability to act as donors in conjugal genetic crosses (1). Beutin et al. (2) have shown that the SfrB- phenotype is associated with premature termination of transcription of the traY → Z operon of the F-factor, suggesting that the sfrB gene product may regulate expression of this operon by acting as an anti-terminator of transcription. Consistent with this view is the observation that at least one gene product of the traY → Z operon, the TraT protein of the outer membrane, is diminished in amount in sfrB cells (1).

In addition to these changes in F-factor-mediated properties, sfrB mutants also show other alterations in cell envelope structure. These include changes in bacteriophage sensitivity patterns, alterations in electrophoretic mobility of lipopolysaccharide, and defects in flagellar function. These observations led Beutin and Achtman (1) to suggest that the sfrB gene product may act to regulate the expression of a number of operons responsible for synthesis of cell envelope components.

In Salmonella typhimurium, mutations of the rfaH gene result in defects in F-factor function that are similar to those of sfrB mutants of E. coli (3). It is well established that rfaH mutants are also defective in lipopolysaccharide biosynthesis (4, 5). Their similarities in phenotype and map position suggest that sfrB and rfaH represent homologous genes in the two organisms, as suggested by Sanderson and Stocker (3).

In this paper we present evidence that: (i) sfrB mutations result in loss of activity of two E. coli membrane proteins, UDP-galactose:(glucosyl)lipopolysaccharide 1,6-galactosyltransferase and UDP-glucose:(glucosyl)lipopolysaccharide 1,3-galactosyltransferase,1 enzymes required for biosynthesis of the complete core region of the E. coli lipopolysaccharide; (ii) both transferase activities are restored by introduction of a recombinant plasmid that also corrects the conjugational abnormalities of the sfrB cells; and (iii) an amber mutation of the rfaH gene of Salmonella typhimurium, the homologue of the sfrB gene of E. coli, is associated with a decrease in amount, but not in apparent size, of a Salmonella membrane protein, UDP-galactose:(glucosyl)lipopolysaccharide galactosyltransferase. The results implicate the genes responsible for synthesis of the membrane glycosyltransferases as likely additional targets for regulation by the sfrB gene product.

EXPERIMENTAL PROCEDURES

Materials—Radioactive nucleotide sugars and 35S-labeled H2SO4 were purchased from New England Nuclear. Unlabeled nucleotide sugars were obtained from Calbiochem. Nucleotide sugars were checked for purity as previously described (6). IgGorb was purchased from the Enzyme Center, Inc., and NCS tissue solubilizer from Amersham Corp.

Crude colicin E1 was prepared from the supernatant of a culture of E. coli W3110 (7). This crude extract, sterilized by filtration, was mixed with the growth medium and was applied to selective plates in appropriate dilutions as previously described (8). Lipopolysaccharides were purified as previously described (9).

Analytical Techniques—Ascending chromatography of sugars and nucleotide sugars was performed in 95% ethanol, 1 M ammonium acetate (7.3). Descending chromatography of sugars was performed in butanol/pyridine/water (6:4:3). SDS-gel electrophoresis was carried out in 11% gels using the system of Lugaenberg et al. (10). Colicin E1 sensitivity and bacteriophage sensitivities were determined by spot tests as described previously (8).

Organisms—E. coli and S. typhimurium strains are listed in Table I. The galE strains M1174T1, M1162T2, and M1170T3 were constructed by P1-mediated co-transduction of galE and radA from M1174 and M1162 to M1170, respectively.

TABLE I

| Strain          | Description                        |
|-----------------|------------------------------------|
| M1174T1         | Parental strain                     |
| M1162T2         | Parental strain                     |
| M1170T3         | Parental strain                     |
| M1174T1         | Parental strain                     |
| M1162T2         | Parental strain                     |
| M1170T3         | Parental strain                     |

1 For terminology of glycosyltransferases, see Ref. 8.
2 The abbreviation used is: SDS, sodium dodecyl sulfate.
**TABLE 1**

**Bacterial strains and plasmids**

| Strain/Plasmid | Relevant genotype | Ref. |
|---------------|------------------|------|
| **E. coli K12 strains** | | |
| M1174 | sfrB<sup>+</sup> trp str | (1) |
| M1162 | sfrB3 trp | (1) |
| M1170 | sfrB11 trp str | (1) |
| **M1174T1** | sfrB<sup>+</sup> trp galE nad::Tn10 | This paper |
| **M1162T2** | sfrB3 trp galE nad::Tn10 | This paper |
| **M1170T3** | sfrB11 trp galE nad::Tn10 | This paper |
| JA200 | F<sup>+</sup> trpE recA | (12) |
| KL181 | proA trp his | |
| **S. typhimurium strains** | | |
| G30 | galE | (9) |
| SL1060 | rfaH pmu met trp | (21) |
| TT2070 | zebo16::Tn10 supD35 | |
| SL3657 | his(Am) rfaH(Am) | |
| **SL3657-598** | his(Am) rfaH(Am) zebo16::Tn10 supD35 | This paper |
| **SL3657-599** | his(Am) rfaH(Am) zebo16::Tn10 supD351 | This paper |
| **Plasmids** | | |
| KLF23<sup>+</sup> | F123<sup>+</sup>-rac-trp<sup>+</sup> galU<sup>+</sup> | -<sup>*</sup> |
| pLC14-28<sup>+</sup> | ColE<sup>+</sup>-rfaH<sup>+</sup> | (13) |
| pLC14-27<sup>+</sup> | ColE<sup>+</sup>-rfaG<sup>+</sup> rm<sup>+</sup> | (8) |
| pLC14-27<sup>+</sup> | ColE<sup>+</sup>-rfaO<sup>+</sup> | (8) |

<sup>*</sup> Kindly provided by S. Normark, University of Umea.
<sup>+</sup> Kindly provided by B. A. D. Stocker, Stanford University.
<sup>?</sup> Kindly provided by J. Roth, University of Utah.
<sup>4</sup> To construct strains containing KLF23, E. coli KL181/KLF23 was used as donor in conjugational crossovers, using appropriate selective plates to select His<sup>+</sup> recombinants.
<sup>5</sup> For construction of strains containing hybrid plasmids, JA200/pLC strains were used as donors in conjugational crossovers as previously described (8). Streptomycin was used to select against the donor and colicin E1 to select against recipient cells which had not received a plasmid.

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**S. typhimurium** (14) and no attempt was made to distinguish between the two activities in the present study. One unit of enzyme activity is defined as the incorporation of 1 nmol of [3H]galactose into lipopolysaccharide from S. typhimurium 40 min at 37 °C. E. coli Glucosyltransferase II Assay—Glucosyltransferase II activity of E. coli extracts was determined by measuring the transfer of [14C]glucose from UDP-[14C]glucose into acceptor lipopolysaccharide. Acceptor lipopolysaccharide was preincubated with galactose-deficient lipopolysaccharide acceptor from S. typhimurium G30 in the standard galactosyltransferase reaction (see above) using nonradioactive UDP-galactose and wild type E. coli enzyme from strain M1174T1. After 60 min at 37 °C to permit addition of the branch 1,6-galactosyl residue (see Fig. 1), the wild type enzyme was inactivated by heating the reaction mixture at 100 °C for 2 min. Glucosyltransferase II activity was then determined by adding UDP-[14C]glucose (0.12 mM, 7,300 dpm/nmol) and the enzyme preparation to be assayed (5–60 μg of protein). Incorporation of [14C]glucose into acid-precipitable material was determined after incubation for 10 min at 37 °C.

E. coli Galactosyltransferase Assay—Galactosyltransferase activity of E. coli extracts was determined by measuring transfer of [3H]galactose from UDP-[3H]galactose into acceptor lipopolysaccharide from S. typhimurium G30 as described above for the S. typhimurium galactosyltransferase; nonradioactive UDP-galactose was also included in the reaction mixture because of evidence that this resulted in a slight increase in the rate of incorporation of [3H]galactose. Experiments were also performed in the absence of nonradioactive UDP-galactose; these gave similar results to those shown in Table I. Analysis of Reaction Products—Hydrolysis and chromatography of the acid-precipitated reaction products (5) confirmed that >95% of the incorporated radioactivity in the glucosyltransferase and galactosyltransferase assays co-chromatographed with authentic glucose and galactose, respectively.

**Measurement of Efficiency of Conjugal Genetic Transfer**—The donor strain E. coli KL181/KLF23 containing the F<sup>+</sup> trp<sup>+</sup> plasmid KLF23 was mixed with the recipient strain E. coli D1 at a ratio of 1:10 (donor:recipient). After 90 min at 37 °C, serial dilutions of the mating mixture were plated on appropriate selective media to determine the number of viable donor cells and the number of D1/KLF23 transconjugants. The number of transconjugants was expressed per 100 donor cells and was normalized to the value obtained for the sfrB<sup>+</sup> control donor strain M1174/KLF23.

**Immunoprecipitation**—Antiserum to purified S. typhimurium galactosyltransferase was obtained by immunization of female New Zealand white rabbits with purified UDP-galactose:[14C]lipopolysaccharide galactosyltransferase. The transferase was purified by a modification of the method of Romeo et al. (15). When chromatographed on Sephadex G-50, the purified enzyme showed a single band of activity that eluted in a position corresponding to a molecular weight of 40,000 and a minor peak, accounting for <5% of the total activity, whose elution position indicated an apparent molecular weight of 80,000. SDS-polyacrylamide gel analysis of the purified protein is described under “Results.” The galactosyldeoxy sugar linkage in the product of the reaction catalyzed by the purified transferase (originally thought to be the α1,3-galactosylglycol of the lipopolysaccharide core) has not been definitely established. Recent studies suggest that the product may instead be the branch α1,6-galactosylglycol (Fig. 1).

Extracts to be assayed for immunoprecipitable material were prepared from exponentially growing cells exposed to [14C]SO<sub>4</sub> for several generations. Cultures of 20 ml containing 1.6 mCi of [14C]SO<sub>4</sub> were grown in parallel with 120 ml of nonradioactive cultures. The two cultures were mixed before harvesting. The resulting cell pellet contained approximately 5 × 10<sup>8</sup> cpm/g of cells (wet weight). To obtain the supernatant and cell envelope fractions, the [14C]-labeled cells were resuspended in 0.2 M Tris·Cl, pH 8.0, 5 mM 2-mercaptoethanol (about 6.6 g of cells (wet weight)/ml) and treated with EDTA, sucrose, and lysozyme as described by Wittholt et al. (16). After a 2-fold dilution with water, the suspension was incubated at 37 °C for 30 min. Spheroplast suspension was washed and the supernatant treated with the purified transferase (originally thought to be the α1,3-galactosylglycol of the lipopolysaccharide core) was then collected on Filters 6a and sonicated for five 10-s bursts. Unbroken spheroplasts (<5% of total cells) were removed after pelleting by centrifugation at 500 × g for 2 min. The cell lysate was spun at 35,000 × g for 12 h at 4 °C and the supernatant fraction (solute)
fraction) was reserved for immunoprecipitation. The membrane pellet was extracted twice by resuspending in 10 mM Tris-Cl, pH 7.5, 2% Triton X-100, 10 mM EDTA, incubating at room temperature for 30 min, and spinning at 130,000 x g for 1 h at 4 °C. The combined supernatants of the two extractions were used as the cell envelope fraction for immunoprecipitation.

For 35S-labeled whole cell lysates, cells were harvested, frozen, and sonicated as previously described (8). The whole cell lysate was extracted twice with 2% Triton X-100, 10 mM EDTA as described above. The combined supernatants were used as total cell extract for immunoprecipitation.

Immunoprecipitation was carried out by the following modification of the method of Shuman et al. (17). To 0.4-ml aliquots of the 35S-labeled extracts (about 106 cpm) was added 0.20 ml of buffer (150 mM Tris-Cl, pH 7.5, 3 mM NaCl, 0.1% aprotinin, 3% Triton X-100, 5 mM EDTA). After addition of 0.03 ml of a 1:50 dilution of normal rabbit serum, samples were vortexed and incubated at 37 °C for 30 min. Samples were chilled, 0.03 ml of IgGsorb was added, and, after incubation at 0 °C for 20 min, the suspensions were spun for 3 min in a Microfuge (Beckman) to remove proteins that bind nonspecifically to rabbit serum or IgGsorb; the pellet was discarded. Following addition of either 0.06 ml of a 1:50 dilution of anti-galactosyltransferase antibody or 0.06 ml of a 1:50 dilution of normal rabbit serum, samples were vortexed and incubated at 0 °C overnight. Preliminary experiments established that this amount of antibody gave maximum precipitation of labeled antigen. Immunoprecipitates were collected by adding 0.3 ml of IgGsorb, vortexing, incubating at 0 °C for 20 min, and spinning in the Microfuge for 3 min. The resulting immunoprecipitates were washed twice in 50 mM Tris-Cl, pH 7.5, 1 M NaCl, 1% Triton X-100, 5 mM EDTA; twice in 50 mM Tris-Cl, pH 7.5, 1 M NaCl, 1% Triton X-100, 5 mM EDTA; twice in 50 mM Tris-Cl, pH 7.5, 0.5% SDS, 0.5 LiCl, 5 mM EDTA; twice in 50 mM Tris-Cl, pH 7.5, 2 M urea; and twice in 50 mM Tris-Cl, pH 7.5. The final pellets were resuspended in 0.075 ml of gel sample solubilizing buffer (0.0625 M Tris-Cl, pH 6.8, 2% SDS, 5% 2-mercaptoethanol, 10% glycerol), incubated at 100 °C for 5 min, and spun for 5 min in a Microfuge. Supernatant solutions were analyzed by SDS-gel electrophoresis. The location of the 35S-labeled peptides was determined by autoradiography of dried gels using Kodak (XAR-5) film.

For quantitation, appropriate strips were cut from the dried gel. The strips were solubilized by incubation at 48 °C for 60 min in 0.1 ml of water and 0.7 ml of NCS tissue solubilizer and were counted in a toluene-based scintillation counting mixture. Counts/min in the strip from the control immunoprecipitate (normal rabbit serum) were subtracted from the counts/min in the corresponding strip from the anti-galactosyltransferase immunoprecipitate. The results were expressed as a fraction of the counts/min in the original 35S-labeled cell extract.

RESULTS

F-factor Defects in sfrB Cells—As previously reported by Beutin and Achtman (1), the sfrB/F' strains were defective in their ability to act as donors in conjugal genetic transfers and were resistant to bacteriophages which utilize the F-plasmid for adsorption to the cell surface (Table IV, lines 1–3).

E. coli Glycosyltransferase Activity—Evidence that sfrB strains are defective in activity of UDP-galactose:glucosyl(1,6)-lipopolysaccharide α1,3-galactosyltransferase (E. coli galactosyltransferase) and of UDP-glucose:glucosyl(1,6)-lipopolysaccharide α1,3-galactosyltransferase (E. coli glucosyltransferase II) was obtained by assay of the transferase activities in cell extracts.

The E. coli galactosyltransferase catalyzes addition of the branch 1,6-galactosyl residue to the glucose I residue of the lipopolysaccharide of E. coli K12 (Fig. 1). The enzyme was assayed by measuring the activity of cell-free extracts to catalyze the transfer of galactose from UDP-galactose to an incomplete lipopolysaccharide acceptor prepared from a galE strain of S. typhimurium (strain G30). This galactose-deficient lipopolysaccharide provides a suitable acceptor for assay of the E. coli α1,6-galactosyltransferase because of the similarity of the inner core of the lipopolysaccharides of E. coli and S. typhimurium (Fig. 1).

The E. coli glucosyltransferase II catalyzes addition of the second glucosyl residue of the E. coli lipopolysaccharide core (GlcII in Fig. 1). Glucosyltransferase II activity was assayed by measuring the transfer of glucose from UDP-glucose into lipopolysaccharide acceptor prepared from S. typhimurium G30 which terminates with the glucose I residue of the core and therefore provides a suitable acceptor for assay of the E. coli glucosyltransferase II (Fig. 1). The activity of both enzymes was markedly reduced in the two sfrB strains tested (M1162T2 and M1170T3) as compared with the sfrB+ parental strain (M1174T1) (Table I, lines 4–6). The defect in activity of the two glucosyltransferases was accompanied by the previously reported (1) alteration in bacteriophage sensitivity patterns (Table III, lines 1–3).

Correction of Glycosyltransferase and F-factor Defects by Recombinant Plasmid pLC14-28—Simultaneous correction of the glycosyltransferase and F-factor defects was achieved by introduction of a recombinant ColE1 plasmid (pLC14-28) containing a cloned fragment of the E. coli chromosome that had previously been shown to correct the abnormal phenotype of rafaH mutants (13).

Introduction of pLC14-28 into E. coli sfrB strains M1162T2 and M1170T3 resulted in restoration of normal galactosyltransferase and glucosyltransferase II enzyme levels (Table II, lines 4–6). The abnormal patterns of sensitivity to lipopolysaccharide-specific bacteriophages U3 and C21 were also cor-
Proteins. The studies described above are consistent with this hypothesis but do not exclude the possibility that the sfrB gene may code for a common structural component of the affected cell envelope proteins.

If the model of Beutin et al. is correct, sfrB (rfaH) amber mutations should result in a decrease in amount but not in size of the affected cell envelope proteins. The availability of an rfaH(Am) mutant provided an opportunity to test these predictions for the S. typhimurium galactosyltransferase. An-
of lipopolysaccharide biosynthesis are present in both the soluble and cell envelope fractions obtained after mechanical disruption of cells (18). When immunoprecipitable galactosyltransferase was determined separately on the soluble and cell envelope fractions of rfaH(Am) and rfaH* cells (Fig. 4), the results were similar to those described above for the total cell extracts. Introduction of the suppressor gene resulted in a 6-fold increase in immunoprecipitable galactosyltransferase in both supernatant and cell envelope fractions (data not shown).

**DISCUSSION**

**Effects of sfrB and rfaH on Glycosyltransferase Activities**—The studies of Beutin et al. (2) indicate that the sfrB gene product acts as a positive regulatory element in transcription of the traY → Z operon. The present study indicates that the sfrB (rfaH) gene product also plays a role in expression of at least three cell envelope glycosyltransferases that are involved in lipopolysaccharide biosynthesis. This explains the observation that sfrB mutants show abnormal patterns of sensitivity to bacteriophages that use lipopolysaccharide as their adsorption sites (1) and indicates that the altered bacteriophage sensitivity patterns are due to changes in the levels of lipopolysaccharide biosynthetic enzymes rather than reflecting the secondary effects of other cell envelope alterations.

In the case of the S. typhimurium galactosyltransferase, an absolute decrease in cellular content of the protein in RfaH- cells was demonstrated by specific immunoprecipitation. The failure to see an increased cytoplasmic pool of the protein (Fig. 4) argues against the possibility that the rfaH (sfrB) defect affects cell envelope proteins by interfering with their entry into the membrane. The fact that there was no detectable change in size of the immunoreactive transferase protein of an rfaH(Am) mutant supports the conclusion that rfaH (and sfrB) are regulatory rather than structural genes for components of the affected glycosyltransferases. It should be noted, however, that a small change in size due to an amber mutation close to the 3′ end of a structural gene would not have been detected by this analysis.

All of the results are consistent with the suggestion of Beutin and Achtman (1) that the sfrB gene of E. coli (and presumably also the rfaH gene of S. typhimurium) acts to regulate expression of several operons involved in synthesis of cell envelope proteins. It is not known whether the structural genes for the glycosyltransferases are part of a single operon that is positively regulated by the sfrB (rfaH) gene product or whether expression of each of the structural genes is individually regulated. The present studies also do not indicate whether the regulation occurs at a transcriptional or post-transcriptional level although the studies of Beutin et al. (2) suggest that an effect on transcription is most likely.

**Effects of pLC10-7 on Glycosyltransferase Activities**—It has been shown previously that pLC10-7 contains genes required
for expression of several glycosyltransferase activities (8). Thus, pLC10-7 was capable of correcting the glucosyltransferase I defect of \( rfaG \) mutants of \( S. typhimurium \) and also induced the appearance of \( E. coli \) glucosyltransferase II (RfaM) activity when the plasmid was introduced into \( S. typhimurium \) host cells (8). In addition, pLC10-7 corrected the abnormal bacteriophage sensitivity patterns of \( rfaJ \) and \( rfaI \) mutants of \( S. typhimurium \) and therefore is likely to contain genetic information for expression of UDP-glucose:(galactosyl)lipopolysaccharide glucosyltransferase and UDP-galactose:(glucosyl)lipopolysaccharide \(-1,3\)-galactosyltransferase, enzymes that catalyze addition of the GlcII and GalI residues of the \( S. typhimurium \) lipopolysaccharide (Fig. 1).

An incidental finding in the present study was the observation that pLC10-7 induced a moderate increase in galactosyltransferase in both \( sfrB^+ \) and \( sfrB \) cells (Table II). This is consistent with the idea that pLC10-7 may also encode the \( rfaB \) gene, responsible for synthesis of UDP-galactose:(galactosyl)lipopolysaccharide \(-1,3\)-galactosyltransferase (14), the enzyme responsible for addition of the GalI residue of the polysaccharide core (Fig. 1).

Taken together, these data indicate that pLC10-7 includes genetic information for expression of at least five enzymes of lipopolysaccharide biosynthesis.

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* For nomenclature of \( rfa \) mutants, see Refs. 8 and 19.
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