Ribitol Teichoic Acid Synthesis in Bacteriophage-resistant Mutants of *Staphylococcus aureus* H*

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**SUMMARY**

Teichoic acid synthesis was examined in two types of phage-resistant mutants of *Staphylococcus aureus* H that have altered cell wall composition. In type I mutants (52A2, 52A4, 52B2) which lack N-acetylglucosamine (GlcNAc) on the cell wall ribitol teichoic acid, the CDP-ribitol:acceptor phosphoribitoltransferase was normal but no UDP-GlcNAc:poly(ribitol phosphate) GlcNAc-transferase activity was detected. Revertants of type I mutants recovered the normal levels of GlcNAc in the teichoic acid as well as GlcNAc-transferase activity.

Type II mutants (52A5, 52A7, M3, M9) lacked ribitol teichoic acid in the cell wall and no ribitol phosphate polymer was detected in any other cell fraction or in the spent medium. Despite the lack of ribitol teichoic acid, normal activities in vitro of phosphoribitoltransferase and GlcNAc-transferase were found in these mutants. Compared to the parent strain, trichloracetic acid extracts of type II mutants contained high levels of CDP-ribitol. The lack of ribitol teichoic acid in the cell wall is caused presumably by some defect in the membrane or in some unknown factor required in the polymerization or attachment step of the teichoic acid to murein.

Analysis placed the mutants in two categories. Type I mutants lack GlcNAc residues on the teichoic acid but approximately half of the ribitol residues have a D-alanine substituent on the 2-hydroxyl (5). They are presumed to be the result of one step point mutations since phage-sensitive revertants, which contain normal levels of GlcNAc, can be obtained. Type II mutants lack ribitol teichoic acid and, although they exhibit many pleiotropic effects, they are also point mutants because revertants, both spontaneous and chemically induced, are wild type in all respects (6).

Baddiley et al. (7-9) showed that the teichoic acid of *S. aureus* H cell wall is a polymer of 4 O-acetylglucosaminyl-D-ribitol 5-phosphate residues, substituted also with ester D-alanine (the proportion depending on growth conditions). More than 95% of the GlcNAc residues have the β configuration (10) and an average chain length of 8 units was found for teichoic acid isolated by acid extraction of cell walls (11). The pathway of biosynthesis is analogous to that found by Burger and Glaser (12-14) for glycerol and ribitol teichoic acids in *B. subtilis* and *Lactobacillus arabinosus*. The enzyme activities are associated with particles that are presumed to be fragments of bacterial membrane. A phosphoribitoltransferase synthesizes poly-(ribitol phosphate) chains from CDP-ribitol and a GlcNAc-transferase catalyzes the addition of GlcNAc substituents (10, 15, 16).

$$
\text{Acceptor} + n\text{GDP-ribitol} \rightarrow \\
(\text{ribitol phosphate})_n\text{acceptor} + n\text{CMP} \quad (1)
$$

$$
(\text{Ribitol phosphate})_n\text{acceptor} \rightarrow \\
+ n\text{UDP-GlcNAc} \quad (2)
$$

The present paper describes the activities of teichoic acid-synthesizing enzymes in phage-resistant mutants and revertants.

**METHODS AND MATERIALS**

**Growth of Bacteria**—The isolation and maintenance of the streptomycin-resistant parent strain, *S. aureus* H(SmR), and of the two types of phage-resistant mutant strains, type I (strains 52A2, 52A4, 52B2) and type II (strains 52A5, 52A7, M3, and M9), as well as the isolation of revertants from type II mutants,

1 The abbreviation used is: GlcNAc, N-acetylglucosamine.
were described previously (4, 6). Conditions of growth were as follows. Strains were grown in 0.5% phytone (Baltimore Biological Laboratories), 0.5% yeast extract (Difco), 0.3% K$_2$HPO$_4$, and 0.2% glucose (pH 7.2) (PYK medium) in a rotary shaker at 37°. In all growth experiments 3 to 5% of a 13- to 15-hour culture was used as an inoculum. Growth was monitored by measuring the optical density of the culture at 585 nm.

**Isolation of Revertants**—Wild type phage-sensitive revertants from type I phage-resistant mutants were obtained as follows. Strain 52A4 and strain 52A2 exponential phase cells grown in PYK medium were harvested and washed in 0.06 m potassium phosphate buffer, pH 6.8. The cells were resuspended in 0.1 m potassium phosphate buffer, pH 5.8, at a concentration of 3 x 10$^7$ cells per ml, was added and the cells were incubated on a rotary shaker at 37° for 30 min, during which period 88% were killed. The cells were harvested and washed twice with 0.05 m phosphate buffer, pH 6.8, diluted with 0.85% NaCl, and about 200 viable cells plated together with about 10$^6$ 52A4 type phages on nutrient agar plates. Detection of phage-sensitive colonies was by the nilling technique described by Rovel (17). After incubation at 37° for 18 hours, 0.5% of the colonies was found to be nilled. Wild type cells were obtained from these colonies after streaking them on nutrient agar plates. In addition to phage sensitivity, the revertants were found to have the same streptomycin resistance as the parent strain (1 mg per ml) (4), were coagulase positive, and fermented mannitol.

**Isolation of Cell Walls**—Cell walls were prepared essentially by the method of Sharon and Janoloz (18). Cells grown in PYK medium to an optical density of 3.0, harvested, and washed with 0.1 m potassium phosphate buffer, pH 6.8. Cells were disrupted with glass beads in a Sorvall Omni-Mixer (30 min, -2°, top speed). After breakage deoxyribonuclease and ribonuclease were nibbled. Wild type cells were obtained from these colonies after streaking them on nutrient agar plates. In addition to phage sensitivity, the revertants were found to have the same streptomycin resistance as the parent strain (1 mg per ml) (4), were coagulase positive, and fermented mannitol.

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**Particulate Enzyme**—The particulate enzyme preparation employed was that fraction of mechanically disrupted cells that sedimented between 12,000 and 100,000 x g (19). The pellet was washed twice with 0.05 M Tris-HCl buffer, pH 7.2, and were then heated to 100° for 15 min to inactivate lytic enzymes. The boiled walls were suspended in 0.1 M Tris-HCl buffer, pH 8.0, and were digested with trypsin (Worthington) (100 µg per ml, 37°, 3 hours). The walls were washed three times with water, homogenized with chloroform-methanol (2:1) to extract any lipids, filtered on Whatman No. 1 paper, and washed on the filter with chloroform-methanol. Cell walls were finally suspended again in water, washed, and lyophilized.

**Incubation of Cells with [32P]Phosphate**—Cells grown in PYK medium to an optical density of 1.8, were harvested, and were transferred to a defined medium that contained the four cell wall amino acids and glucose (19) but with 0.02 M Tris-HCl, pH 7.2, replacing 0.05 M potassium phosphate. [32P]Orthophosphate (New England Nuclear) (30 µCi per µmole, 40 µmole per liter of medium) was added and the cells were incubated with shaking for 90 min at 37°. The pH of the medium was maintained at pH 7.2 by the dropwise addition of 1 n NaOH. The cells were harvested, disrupted, and fractionated as described above.

**Nucleotides—CDP-[3H]ribitol** was prepared essentially as described by Glaser in 1964. μ-Ribose 5-phosphate was reduced by [3H]-NaBH$_4$ (New England Nuclear) to give [3H]ribitol phosphate which was converted to CDP-[3H]ribitol by the action of the pyrophosphorylase in dialyzed crude extracts of S. aureus (20). CDP-[3H]ribitol was purified by column chromatography on Dowex 1-bicarbonate, with elution by a linear gradient of ammonium bicarbonate. The product was found to be chromatographically homogeneous in Solvents A and B. The specific activity was 20 cpm per pmole. Unlabeled CDP-ribitol was prepared in the same manner with the use of unlabeled NaBH$_4$.

UDP-[1-14C]Glucose was obtained from New England Nuclear and the specific activity was adjusted to 20 cpm per pmole.

UDP-N-acetyl-muramyl-(L-[14C]lysine) pentapeptide was prepared as described by Park and Chatterjee (19).

**Assay of CDP-Ribitol:Acceptor Phosphoribitoltransferase—** [3H]Poly(ribitol phosphate) formed by the action of particulate enzyme on CDP-[3H]ribitol was separated from residual substrate by a rapid chromatographic procedure.

The following reagents were added to glass tubes (6 mm x 50 mm): 5 µl of membrane preparation suspended in 50 mM Tris-HCl buffer, pH 8.0, containing 1 mm mercaptoethanol, 5 µl of 20 mM spermidine$^2$ hydrochloride adjusted to pH 8 with NaOH, and 10 µl of water. The mixture was placed in a water bath at 25° for 30 sec and then 5 µl of 20 mM CDP-[3H]ribitol, 20 cpm per pmole, were added to start the reaction. After incubation at 25° for 10 min, a 20 µl sample was streaked along the 4-cm wide starting line of a piece of Whatman No. 1 paper and was dried within 15 sec in a stream of warm air. Controls were performed with boiled enzyme. The paper was 10 cm long, a multiple of 4 cm wide, and the starting line was marked 1.5 cm from one end. Unreacted substrate was separated from polymeric product by ascending chromatography in Solvent C. The paper was placed between two sheets of glass (thin layer plates, 20 cm x 5 cm) which were clipped together and arranged in a Plexiglas trough so that one end of the paper dipped into solvent and the other end was exposed to air. The trough was constructed to take many plates simultaneously and an adjustable lid was placed to prevent undue evaporation of solvent. Cold air from a hair dryer helped dry the solvent as it emerged at the exposed upper end of the paper. Elution was continued for 1 hour or more than 0.1% of the unreacted radioactive substrate remained at the starting line. After this elution, the paper was dried and a rectangle (4 cm x 2 cm) was cut out to contain the starting line. Paper was placed in a scintillation vial. After 5 min at room temperature, 10 ml of ethoxyethanol scintillation fluid were added and the contents of the vial were subjected to gentle rotary agitation for 10 min. Tests showed that the counts were transferred completely from the paper to the solution.

3 A divalent cation is required for enzymic activity (see also Reference 16); spermidine was found to promote greater activity than other cations tested (e.g. spermine, putrescine, Mg$^{2+}$, Ca$^{2+}$, Mn$^{2+}$) (D. R. D. Shaw, unpublished data).
Under these conditions, and in contrast to the results of Ishimoto and Strominger (16), the progress of the reaction was linear until three quarters of the substrate had been consumed, and the reaction velocity was proportional to enzyme concentration.

**Assay of UDP-GlcNAc:Poly(Ribitol Phosphate) GlcNAc-transferase—**Poly(14C]GlcnAc:ribitol phosphate) formed by the action of particulate enzymes on CDP-ribitol and UDP-[14C]-GlcNAc was separated from residual substrate in the manner described above.

The following reagents were added to glass tubes (6 mm × 50 mm): 5 μl of membrane preparation suspended in 50 mM Tris-HCl buffer, pH 8.0, containing 1 mM mercaptoethanol, 5 μl of 20 mM spermidine2 hydrochloride adjusted to pH 8.0 with NaOH, 5 μl of water, and 5 μl of 1.5 mM unlabeled CDP-ribitol. The mixture was incubated at 25°C for 10 min and then 0.2 μl of 2% UDP-[14C]GlcNAc, 20 cpm per pmole, were added. Incubation was continued for an additional 10 min after which a 20-μl sample was streaked along the starting line of a chromatographic paper as described above. The 14C-labeled product remaining at the origin of the paper was counted directly on the paper immersed in scintillation fluid. A control was performed with water replacing CDP-ribitol and control counts were subtracted from counts obtained with the complete reaction mixture to obtain the CDP-ribitol-dependent counts. Control counts were between 5% and 10% of the counts obtained with the complete reaction mixture. The unreacted UDP-[14C]GlcNAc remaining at the origin in controls using boiled enzyme was not more than 0.01% of the counts added to the reaction mixture. The velocity of the reaction was proportional to enzyme concentration.

**Analytical Methods—**Radioactive counts were measured in a Nuclear Chicago Mark I scintillation counter using aqueous samples a scintillation fluid composed of 2 ethoxyethanol (1 liter), toluene (2 liters), 2,5-diphenyloxazole (12 g) (Nuclear Chicago), and p-bis[2-(5-phenyloxazolyl)]benzene (POPOP) (0.38 g) (Packard Instrument Company). The radioactive material on paper was counted with a scintillation instrument (Nuclear Chicago), and p-bis[2-(5-phenyloxazolyl)]benzene (POPOP) (0.3 g) (Pilot Chemicals, Watertown, Massachusetts). The radioactive material on paper was counted with a scintillation fluid composed of toluene (3.8 liters), 2,5-diphenyloxazole (15.2 g), and 1,4-bis[2-(methyl-5-phenyloxazolyl)]benzene (di-POPOP) (0.38 g) (Packard Instrument Company).

Descending paper chromatography was on Whatman No. 1 paper with the following solvents: A, isobutyric acid-1 mM NaOH (5:3, v/v); B, ethanol-0.5 M ammonium acetate, pH 3.8 (5:2, v/v). Ascending chromatography was with Solvent C, methanol-0.1 M formic acid (7:3, v/v). Quantitative analysis of cell wall components was done with a Technicon amino acid analyzer. Walls were hydrolyzed by 6 N HCl at 100°C for 15 hours and the acid was evaporated in a vacuum over KOH pellets. Results were computed from a standard containing the wall amino acids and amido sugars that had been heated with HCl under identical conditions.

For the determination of ribitol and glycerol, walls containing the teichoic acid were hydrolyzed with 2 N NaOH at 100°C for 2 hours in stoppered polypropylene tubes. The hydrolysates were deionized with Dowex 50-H+ and the insoluble material, as well as the resin, was removed by filtering. Ribitol from CDP-ribitol was estimated after hydrolysis of the nucleotide in 1 N HCl at 100°C for 10 min. The dephosphorylation of ribitol phosphates and glycerol phosphates obtained after acid or alkaline hydrolysis was accomplished by incubating a sample in 0.1 M sodium carbonate buffer (1 ml, pH 9.6) with *Escherichia coli* alkaline phosphatase (50 μg) (Sigma). Ribitol was determined enzymatically in a reaction mixture (0.3 ml) containing 0.01 μl NAD+, 0.33 M sodium carbonate buffer, pH 9.5, and 0.5 unit of *Aerobacter aerogenes* ribitol dehydrogenase.

Glycerol was determined enzymatically in a reaction mixture (0.3 ml) with glycerokinase (20 μl of 5 mg per ml) (Boehringer Mannheim) and glycerol 1-phosphate dehydrogenase (10 μl of 10 mg per ml) (Boehringer Mannheim) according to the conditions described by Wieland (21).

To check on recoveries, standard solutions of ribitol, ribitol 5-phosphate, and glycerol were treated under the same conditions of hydrolysis.

Protein was estimated by the method of Lowry et al. (22); serum albumin served as standard. Total and inorganic phosphate were estimated by the method of Chen, Toribara, and Warner (23).

**RESULTS**

Table I compares the cell wall composition of parent, mutant, and revertant strains. The proportion of glucosamine to muramic acid in type I mutants is half of that found in the parent strain H. This is caused by the absence of GlcNAc residues on the teichoic acid (see also Reference 4). The phage-sensitive revertants of type I mutants have normal levels of GlcNAc, indicating that the teichoic acid has recovered GlcNAc substituents. The disappearance of teichoic acid from walls of type II mutants is shown by the low level of phosphate and the decrease in glucosamine and alanine. Repeated analyses by enzymic assay with ribitol dehydrogenase and paper chromatography failed to detect any ribitol or anhydroribitol in cell wall hydrolysate, so it must be concluded that type II mutants are without any ribitol teichoic acid. The cell wall analysis of the revertant from type II mutants was very similar in all components to that of the parent strain. These revertants recovered their phage sensitivity and all the other characteristic properties of *S. aureus H* (6). Small amounts of glycerol (about 0.05 μmole per mg of cell wall) were found in all isolated cell walls analyzed (Table I). In strain 52A5, the amounts of phosphate and glycerol are similar and the significance of the compounds containing these components is under investigation.4

Table II gives the levels of phosphoribitoltransferase and GlcNAc-transferase activities in the various strains. It can be seen that GlcNAc-transferase activity is absent from type I mutants, which have no GlcNAc on the teichoic acid, but that phage-sensitive revertants were restored both in transferase and cell wall content of GlcNAc. Phosphoribitoltransferase activities were similar in all strains. Some variations in specific activities were experienced among different preparations of enzyme from the same strain and no particular significance is attached to the variations shown in Table II other than the lack of GlcNAc-transferase in type I mutants.

Because the lack of ribitol teichoic acid in type II mutants cannot be attributed to an absence of phosphoribitoltransferase, the availability of CDP-ribitol was investigated in strain 52A5. Previous observations (6) indicated that the level of nucleotide-bound precursors of murin in strain 52A5 than in the parent strain. Column chromatography of the nucleotide fraction from 52A5 revealed appreciable amounts of CDP-ribitol (Fig. 1). Analysis of the isolated nucleotide gave phosphate.

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1 D. R. D. Shaw and D. Mirelman, unpublished data.
2 D. Mirelman, unpublished data.
TABLE I

Cell wall composition

All values are corrected for losses during hydrolysis.

<table>
<thead>
<tr>
<th>Component</th>
<th>Strain H, wild type</th>
<th>Strain 52A4, type I</th>
<th>Strain 52A4, type II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Molar ratio (µmoles/mg wall)</td>
<td>Molar ratio (µmoles/mg wall)</td>
<td>Molar ratio (µmoles/mg wall)</td>
</tr>
<tr>
<td>Muramic acid</td>
<td>0.54</td>
<td>0.46</td>
<td>0.42</td>
</tr>
<tr>
<td>Glucosamine</td>
<td>1.16</td>
<td>0.55</td>
<td>0.54</td>
</tr>
<tr>
<td>Glutamic acid</td>
<td>0.50</td>
<td>0.59</td>
<td>0.51</td>
</tr>
<tr>
<td>Glycine</td>
<td>2.48</td>
<td>2.38</td>
<td>2.27</td>
</tr>
<tr>
<td>Alanine</td>
<td>1.23</td>
<td>1.26</td>
<td>1.05</td>
</tr>
<tr>
<td>Lysine</td>
<td>0.71</td>
<td>0.97</td>
<td>0.86</td>
</tr>
<tr>
<td>Phosphate</td>
<td>0.92</td>
<td>0.97</td>
<td>0.90</td>
</tr>
</tbody>
</table>

Fig. 1. Chromatography of nucleotides from S. aureus strain 52A5. Cells were grown in PYK medium to an optical density of 3.0, were harvested, and were washed with 0.1 M potassium phosphate buffer, pH 7.0. The cells were homogenized in 5% trichloroacetic acid and were kept in the cold with occasional mixing for 30 min. The cell suspension was centrifuged at 12,000 x g for 20 min, the supernatant was decanted, and the precipitate was washed twice with 5% trichloracetic acid. The combined supernatants were treated with Nuchar C charcoal (West Virginia Pulp and Paper Company), were washed with water, and the adsorbed nucleotides were eluted with ethanol-0.1 M NH₄OH (1:1, v/v). The ethanolic ammonia was evaporated under reduced pressure and the nucleotide mixture was chromatographed on a Dowex 1-formate column with a linear gradient of ammonium formate-formic acid as eluent at a rate of 10 ml per hour as described by Park (24). The buffer concentration was determined with conductivity meter (Radiometer, Copenhagen). Labeled CDP[3H] ribitol and UDP-A-acetylmuramic acid-(L-[14C] lysine) pentapeptide were used as internal markers and chromatographed together with the cell nucleotide mixture. Optical density was monitored at 280 nm (△—△) and 260 nm (○—○). The gradient is shown by △—△. MurNAc, N-acetylmuramic acid.
**Table II**

Specific activities of teichoic acid-synthesizing enzymes in particulate preparations from *S. aureus* strains

Specific activities are given in nanomoles per min per mg of protein. The GlcNAc-transferase values are calculated from the CDP-ribitol-dependent incorporation of [*4C*]GlcNAc.

<table>
<thead>
<tr>
<th>Strain</th>
<th>Phospho-ribitoltransferase</th>
<th>GlcNAc-transferase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parent strain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H</td>
<td>0.63</td>
<td>0.42</td>
</tr>
<tr>
<td>Type I mutants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>52A2</td>
<td>0.13</td>
<td>&lt;0.006</td>
</tr>
<tr>
<td>52A4</td>
<td>0.93</td>
<td>&lt;0.006</td>
</tr>
<tr>
<td>52B2</td>
<td>0.30</td>
<td>&lt;0.006</td>
</tr>
<tr>
<td>Revertants of type I mutants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>52A2-R1</td>
<td>0.23</td>
<td>0.49</td>
</tr>
<tr>
<td>52A2-R2</td>
<td>0.34</td>
<td>0.85</td>
</tr>
<tr>
<td>52A4-R1</td>
<td>0.56</td>
<td>0.57</td>
</tr>
<tr>
<td>52A4-R2</td>
<td>0.21</td>
<td>0.28</td>
</tr>
<tr>
<td>Type II mutants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>52A0</td>
<td>0.82</td>
<td>0.51</td>
</tr>
<tr>
<td>52A7</td>
<td>0.60</td>
<td>0.19</td>
</tr>
<tr>
<td>M3</td>
<td>0.54</td>
<td>0.83</td>
</tr>
<tr>
<td>M9</td>
<td>0.45</td>
<td>0.86</td>
</tr>
</tbody>
</table>

per mg of membrane protein). The small amount of ribitol found in hydrolysates of the 100,000 X g mutant fraction was similar to the amount of ribitol obtained by extraction of whole cells and identified previously as CDP-ribitol. There is no indication, therefore, that ribitol teichoic acid was excreted into the medium nor that any accumulated in a soluble or particulate fraction of strain 52A5.

**DISCUSSION**

A defect in any of the steps involved in the synthesis of teichoic acid results in the inability of certain phages to adsorb to the cell wall. The production of type I phage-resistant mutants of *S. aureus* H is the consequence of a defect in the UDP-GlcNAc:poly(ribitol phosphate)-GlcNAc-transferase. Phage-sensitive revertants containing normal levels of GlcNAc in the cell walls and GlcNAc-transferase activity (Tables I and II) have been obtained after chemical mutagenesis. Young (2) has observed a similar defect in a phage-resistant mutant of *B. subtilis* 168 that lacks glucose on the teichoic acid and has an inactive UDP-Glc:poly(glycerol phosphate) glucosyltransferase.

Type II phage-resistant mutants of *S. aureus* H have a cell wall that completely lacks ribitol teichoic acid. These mutants exhibit other phenotypic differences, some of which may be secondary effects caused by the absence of poly(ribitol phosphate) (6). The normal levels of phosphoribitoltransferase and GlcNAc-transferase found in enzyme preparations from type II mutants (Table II) exclude the possibility that these enzymes were themselves impaired. However, no synthesis of ribitol teichoic acid appears to take place in vivo, because no evidence was found for any excretion of free teichoic acid into the medium or any accumulation of teichoic acid within the cells.

**REFERENCES**


* D. R. D. Shaw, unpublished data.
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