Mapping of the \( supP \) (\( Su^6 \)) Amber Suppressor Gene in \( Escherichia coli \)

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The \( supP \) (\( Su^6 \)) amber suppressor gene has been mapped on the clockwise side of the \( valS \) locus near min 95 on the \( Escherichia coli \) chromosome.

The \( supP51 \) (formerly \( Su^6 \)) amber suppressor was originally described by Chan and Garen (3) and shown to cause the amber triplet UAG to code for leucine. Fractionation of leucyl tRNA from \( supP^* \) (wild type) and \( supP51 \) strains (5) revealed two fractions of UUG-binding leucyl-tRNA in the \( supP^* \) strain, whereas only one of these fractions was found in the \( supP51 \) strain. In the \( supP51 \) strain the other fraction had been replaced by UAG-binding leucyl tRNA (suppressor tRNA). Thus, the \( supP51 \) mutation appears to have transformed one of two UUG-binding leucyl tRNA fractions normally present in \( Escherichia coli \) into amber suppressor tRNA.

The map position of the \( supP \) gene has not been known, but it has been reported to be located in the region covered by the F' factor F14 (9). Recently an amber suppressor gene, \( supJ \), has been mapped in the corresponding region of the \( Salmonella typhimurium \) chromosome, closely linked to the \( cya \) locus (11).

Little is known about the distribution of leucyl-tRNA genes on the \( E. coli \) chromosome. However, approximate mapping of tRNA genes in \( E. coli \) (6) has indicated the presence of leucyl tRNA genes in 3 widely separated chromosomal regions: min 66–69, 82–85, and 90–93.

After having obtained mapping results which indicated that the \( supP \) gene is not located in the F14 region, we carried out conjugation experiments to determine its approximate map position. These experiments showed that \( supP \) is located in the clockwise neighborhood of the \( purA \) gene, which is near min 93.5 on the \( E. coli \) map (1). Further mapping was done by P1-mediated transduction involving the \( purA \), \( pyrB \), \( argI \), and \( valS \) genes. The last three genes are closely linked to each other near min 95 on the map (1). Their order has been determined in \( E. coli B \) and found to be as shown above (8). The recipients all carry the amber mutations \( his-85 \) and \( trpA9605 \). Selection and scoring for \( supP51 \) transductants was carried out on minimal media without histidine and tryptophan.

Our transduction mapping (Table 1 and Fig. 1).

Table 1. Mapping of \( supP51 \) (\( Su^6 \)) by transduction with \( P1^* \)

<table>
<thead>
<tr>
<th>Recipient strain</th>
<th>Selection</th>
<th>No. of transductants scored</th>
<th>Unselected marker</th>
<th>No. of cotransductants</th>
<th>% Cotransduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>GE554</td>
<td>( supP51 )</td>
<td>295</td>
<td>argI*</td>
<td>140</td>
<td>47.5</td>
</tr>
<tr>
<td></td>
<td>( purA^* )</td>
<td>300</td>
<td>argI*</td>
<td>1</td>
<td>0.34</td>
</tr>
<tr>
<td>GE556</td>
<td>( supP51 )</td>
<td>272</td>
<td>pyrB*</td>
<td>100</td>
<td>36.8</td>
</tr>
<tr>
<td></td>
<td>( purA^* )</td>
<td>282</td>
<td>pyrB*</td>
<td>1</td>
<td>0.37</td>
</tr>
<tr>
<td>GE557</td>
<td>( supP51 )</td>
<td>250</td>
<td>valS*</td>
<td>211</td>
<td>84.4</td>
</tr>
<tr>
<td></td>
<td>( valS^* )</td>
<td>233</td>
<td>purA*</td>
<td>169</td>
<td>72.5</td>
</tr>
</tbody>
</table>

*Strain CGSC4487 (strain S26, \( Su^6 \), of A. Garen; Hfr P02A phoA4 relA1 tonA22 \( supP51 = Su^6 \)) was used as donor in all experiments. All recipient strains are \( supP^* \) and carry the amber mutations \( his-85 \) and \( trpA9605 \). Other characteristics of the recipient strains: GE554 \( F^- purA54 argF58 arg161 thr-25 rpsL190 tpx-84 \); GE556 \( F^- purA54 pyrB9 argF58 rpsL190 tpx-84 \); GE557 \( F^- purA54 valS7 argF58 rpsL190 tpx-84 \). Selection and scoring for \( supP51 \) were carried out on minimal medium without tryptophan and histidine; \( valS^* \) transductants were selected on L-agar at 42°C. Selection for \( pyrB^* \) in strain GE556 resulted in preferential recovery of \( purA^+ \) \( supP^* \) cotransductants (data not shown). Transduction procedures were as described by Thorbjarnardóttir et al. (10).

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1) shows that the supP gene is cotransducible at high frequency with the pyrB, argI, and valS genes and at very low frequency (0.2 to 0.3%) with the purA gene. The order purA-(pyrB, argI, valS)-supP is established, placing the supP gene near 95 min on the map. This map position of the supP gene indicates that it may be very closely linked to the ts-210 mutation which has recently been mapped by Isono and Kitakawa (7). One tRNA locus, glyV, has previously been mapped within the region shown in Fig. 1 (1, 4, 6). However, its exact position has not been reported. According to the approximate mapping referred to above (6), a leucyl tRNA locus, leuV, lies in the counterclockwise neighborhood of purA. The leuV locus is known to specify a CUG-binding leucyl-tRNA, tRNA-Leu (2, 6).

It can be concluded that the supP locus is neither identical to the supF locus of S. typhimurium nor to any previously mapped leucyl tRNA locus in E. coli.

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**LITERATURE CITED**


