Location of the *prlC* (*opdA*) Gene on the Physical Map of *Escherichia coli*

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Mutations at the *prlC* locus were first identified as extragenic suppressors of the protein localization defect caused by certain *lamB* signal sequence mutations (5). We have shown that *prlC* is the *Escherichia coli* homolog of the *Salmonella typhimurium* gene *opdA*, which encodes metalloproteinase oligopeptidase A (4). We have sequenced both of these genes and shown that their nucleotide sequences are 84% identical (4). Sequence comparisons with other proteases have shown that OpdA and PrlC are members of a class of metalloproteinases which includes enzymes from rats, pigs, *Schizophyllum commune*, and *Saccharomyces cerevisiae*. Another peptidase, dipeptidyl carboxypeptidase, the product of the *dcp* gene found in *E. coli* and *S. typhimurium*, is also a member of this group of enzymes (2, 3). Recent work in our laboratory has shown that *opdA* is a previously uncharacterized σ^{32} -dependent heat shock gene (3). Since the promoter region of *prlC* is identical to that of the corresponding region of *opdA*, we believe that *prlC* is also a heat shock gene.

Phage P22 cotransduction of genetic markers clearly placed *opdA* at 76 map units on the *Salmonella* chromosome, between *asd* (75 map units) and *xylB* (78 map units) (2). However, phage P1 cotransduction in the MC4100 strain background suggested that *prlC* was located at 71 min on the *E. coli* chromosome (11, 12). To localize *prlC* on the physical map of *E. coli*, we made a probe from a 708-bp internal fragment of *opdA* the sequence of which is 88% identical to *prlC*. This probe was hybridized to the Kohara miniset (7). After high-stringency washes ($1\times$ SSC [$1\times$ SSC is 0.15 M NaCl plus 0.015 M sodium citrate]–0.1% sodium dodecyl sulfate) (10), 65°C), a hybridization signal was obtained from only one phage clone, 12E4(608), and not from the neighboring clones 4D9(607) and 6A4(609) (Fig. 1). These results suggested that the probe sequence was entirely contained in the 1.4-kb gap between 4D9(607) and 6A4(609). To confirm this location, restriction enzyme sites in the *prlC* sequence were compared with those in the *E. coli* chromosome with the MapSearch program (8, 9). The sites matched perfectly the region from 3663.9 to 3666 kbp on the physical map of Rudd (8). (The published *prlC* sequence contains an error which predicts a *Bgl*I site not present on the physical map. Restriction digests do not show this site, however, and resequencing of this region resulted in correction of an error [CG at positions 461 and 462 should be GC] which removes the *Bgl*I site.) These results (Fig. 1) show that *prlC* is located at 78.4 map units; this location is between the positions on the *E. coli* chromosome of *asd* at 75.7 map units and *xylB* at 80.1 map units (1) and is thus analogous to the 76-map-unit location of *opdA* in *S. typhimurium*. The *prlC* gene is transcribed in the counterclockwise direction in both species. This position differs from the map position determined

by cotransduction of genetic markers (11, 12). We do not understand the basis for this difference although it may be worth noting that the strain used for the transductional mapping, MC4100, has been shown to differ from W3110 in this region (6).

FIG. 1. Restriction map of *E. coli* chromosome from 3663 to 3666 kbp showing the location and direction of transcription of the *prlC* gene, position of the probe used to hybridize to the Kohara miniset of phage clones, and regions contained on clones 4D9(607), 12E4(608), and 6A4(609).

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