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Genetic Evidence for Modulation of the Activator by Two Regulatory Proteins Involved in the Exogenous Induction of Phosphoglycerate Transport in *Salmonella typhimurium*

SHU-QIN JIANG, GUAN-QIAO YU,† ZHI-GANG LI, AND JEN-SHIANG HONG*

Department of Cell Physiology, Boston Biomedical Research Institute, 20 Staniford Street, Boston, Massachusetts 02114

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Previous work from this laboratory has identified in a fragment of DNA, cloned from *Salmonella typhimurium*, two genes involved in the exogenous induction of phosphoglycerate transport. These two genes, the transporter gene, *pgtP*, and the activator gene, *pgtA*, are closely linked physically; they are only 3.4 kilobases apart. In the accompanying paper, we describe the determination of the nucleotide sequence of this 3.4-kilobase DNA segment and show that this segment contains two genes, *pgtB* and *pgtC*, encoding two polypeptides of 593 and 397 amino acid residues, respectively. This paper presents an analysis of the effects of insertions and deletions in *pgtBC* on the expression of *pgtP* gene and on the expression of *lacZ* fused to the *pgtP* gene. The results indicate that both *pgtBC* genes are necessary for expression of the *pgtP* gene. Strikingly, deletion of both genes resulted in a constitutive phenotype, suggesting that PgtB and PgtC polypeptides modulate PgtA activity. The expression of the *pgtP* gene appears to be regulated by the *pgtA* gene product, which acts as an activator. A model of induction is proposed in which the central feature is the interaction of the three regulatory proteins in the membrane such that the activity of the activator (PgtA) is subject to modulation by the binding of an inducer.

We previously reported the cloning of a 14.4-kilobase (kb) DNA fragment from *Salmonella typhimurium* (4). This fragment, which carries the genetic information for the exogenously induced phosphoglycerate transport system and its regulatory components, allowed us to identify and sequence the transporter gene, *pgtP*, and a regulator gene, *pgtA* (3, 10). The nucleotide sequence of the 3.4-kb segment located between the *pgtP* and *pgtA* genes has been determined and has been shown to contain two genes, *pgtB* and *pgtC*, which encode two polypeptides of 593 and 397 amino acid residues, respectively (see accompanying paper [9]). This paper presents an insertion and deletion analysis of the *pgtBC* genes. It is concluded that, in addition to *pgtA*, these two regulatory genes are necessary for the inducibility of the *pgtP* gene expression. A model of induction by phosphoglycerates in which the *pgtA* gene product acts as a derepressor is proposed and discussed.

MATERIALS AND METHODS

General methods and materials used in this work were as described in the accompanying paper (9).

3-PG transport assays. Strain CSR603, harboring particular plasmids, was grown at 37°C in minimal medium (medium E) (7) containing 0.5% succinate as a carbon source and supplemented with thiamine, threonine, leucine, proline, arginine, and the appropriate antibiotics. When growth reached exponential phase, cells were collected by centrifugation, washed twice with medium E, and suspended in medium E to an optical density at 660 nm of 3.0. When induction of the *pgt* transport system was required, 0.2% 3-phosphoglycerate (3-PG) was added to exponentially growing cells, and the cells were harvested 2 h later.

3-PG transport was measured as follows; an aliquot (25 μl) of cell suspension prepared as described above was incubated at 37°C for 2 min, when 1 μl of 250 mM glucose was added. After 15 s, 1 μl of [^{14}C]3-PG (2.3 mM; specific activity, 55 mCi/mM) was added, and the incubation was continued for desired intervals. To terminate transport, the mixture was diluted with 2 ml of medium E. Cells were collected on cellulose acetate membranes (pore size, 0.45 μm; Schleicher & Schuell, Inc.) and washed once with 2 ml of the same medium. Membranes were dried and counted in toluene-based Omnifluor (Dupont, NEN Research Products) in a liquid scintillation counter.

RESULTS

Analysis of high-copy-number *lac* fusion plasmids. To determine whether the *pgtB* and *pgtC* genes were required for the expression of *pgtP*, *lac* fusion clones were constructed in vitro with the *lac*-bearing *Bam*HI sequence isolated from the plasmid pMC931 of Casadaban et al. (2). The fusion clones with fusion sites located outside of the *pgtP* and *pgtA* genes are shown in Fig. 1. The fusion clone pJH529 had the fusion site in the *pgtC* gene and was unable to confer 3-PG transport (Table 1), suggesting that the *pgtC* gene was required for expression of the *pgtP* gene. In contrast, the clones pJH525 to pJH527, pJH532, pJH536, and pJH537 with fusion sites in the *pgtB* gene conferred a constitutive 3-PG transport phenotype (data for only pJH525 and pJH526 are shown in Table 1). These results suggest that the *pgtB* gene was required for the inducibility of *pgtP* expression.

Analysis of high-copy-number deletion plasmids. Deletion analysis was also carried out to confirm the results from the insertion analysis described above. To this end, three plasmids with short deletions were derived from plasmid pJH6 (Fig. 2). In pSJ1 (*pgtC*) a *Nsi*I-*Bgl*II deletion removed 168 base pairs (bp) from the *pgtC* gene, presumably creating a truncated PgtC polypeptide; in pSJ2 (*pgtBC*) the *Bgl*II-*Sna*BI deletion removed the sequence of 323 bp that in-

* Corresponding author.
† Present address: Department of Molecular Genetics, Shanghai Institute of Plant Physiology, Academia Sinica, Shanghai, People's Republic of China.

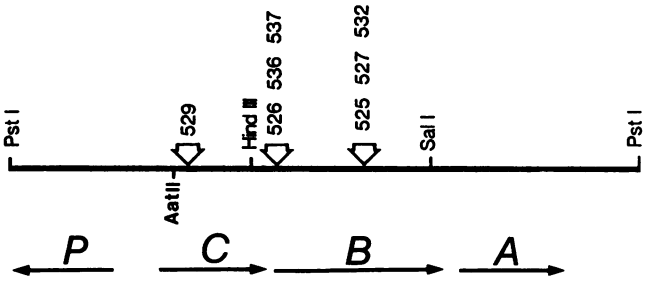

FIG. 1. Locations of *lac* fusions. *lac* fusion clones were centrifuged as follows. Plasmid pJH6 was partially cut with *Sau*3A1, and full-length linearized DNA was purified, dephosphorylated with calf intestinal phosphatase, and ligated to the *lac*-bearing *Bam*HI sequence isolated from plasmid pMC931 constructed by Casadaban et al. (2). Strain JSH1000 was transformed with the ligation mixture, and tetracycline-resistance colonies were selected and screened for those having the *lac* fragment in the 7.6-kb *Pst*I fragment. Fusion clones with fusion sites located in the 2.0-kb *Pst*I-*Aat*II fragment which contained the *pgtP* gene and those with sites in the 2.7-kb *Sal*I-*Pst*I which contained the *pgtA* gene were not further analyzed. The approximate locations of the fusion sites were established by restriction fragment analysis. The letters above the thin arrows specify the *pgt* genes.

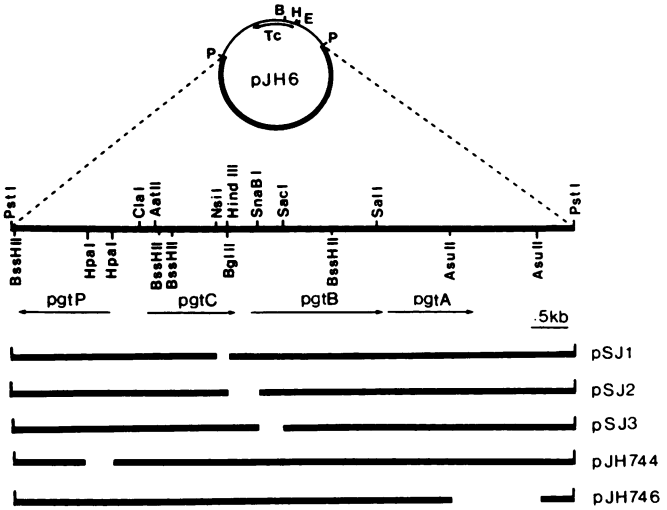

FIG. 2. Construction of short internal deletion plasmids. Plasmids pSJ1, pSJ2, and pSJ3 were constructed by removing the *Nsi*I-*Bgl*II, *Bgl*II-*Sna*BI, and *Sna*BI-*Sac*I fragments, respectively, and trimming the ends with nuclease S1, followed by ligation. Plasmids pJH744 and pJH746 were constructed by deleting the *Hpa*I and *Asu*I fragments, followed by ligation. Abbreviations: B, *Bam*HI; E, *Eco*RI; H, *Hin*dIII; P, *Pst*I; Tc, tetracycline resistance.

cluded 53 bp from a *pgtC* gene and 45 bp from the *pgtB* gene, presumably generating a truncated PgtC polypeptide and eliminating the *pgtB* gene completely; in pSJ3 (*pgtB*) a *Sna*BI-*Sac*I deletion removed 402 bp from the *pgtB* gene, thereby completely eliminating the *pgtB* gene. Deletion plasmids pJH744, in which a 316-bp *Hpa*I fragment was removed from *pgtP*, and pJH746, in which a 1.2-kb *Asu*II fragment was deleted, were also constructed from pJH6.

pSJ1 (*pgtC*) conferred a non-transport phenotype (Table 2), indicating that the *pgtC* gene was required for expression of the *pgtP* gene. In contrast, plasmids pSJ2 (*pgtC pgtB*) and pSJ3 (*pgtB*) conferred a constitutive transport phenotype, suggesting that the *pgtB* gene was dispensable for expression of the *pgtP* gene, but necessary for inducibility of the *pgtP* transport system. These deletion results are similar to those observed with the high-copy-number insertion plasmids described above. As expected, pJH744 (*pgtA*) and pJH746 (*pgtP*) conferred a non-transport phenotype (Table 1).

Analyses of low-copy-number insertion and deletion plasmids. In order to examine the genetic control of the *pgt* system in a genetic background more closely resembling its natural conditions, i.e., to avoid complications that might arise as a result of high gene dosage, the wild-type *pgt* sequence from the high-copy-number plasmid pJH6 and the fusion sequence from pJH526 (also a high-copy-number plasmid) were recloned into the low-copy-number mini-F vector pTN1105 (6), generating pJH567 and pJH577, respectively. Likewise, low-copy-number mini-F plasmids with inserts essentially identical to those in high-copy-number plasmids pJH6, pSJ1, pSJ2, pSJ3, and pJH746 were constructed as in Fig. 3 to generate their respective mini-F equivalents pJH66, pSJ11, pSJ12, pSJ13, and pSJ14.

TABLE 1. 3-PG transport conferred by high-copy-number *pgt-lac* fusion and deletion plasmids[a]

Plasmid	3-PG transport (nmol/mg of protein/30 s)	
	Uninduced	Induced
pJH6 (wild type)	0.5	21.5
pJH525 (*pgtB*)	9.2	5.6[b]
pJH526 (*pgtB*)	14.6	4.1[b]
pJH529 (*pgtC*)	0.21	0.20
pSJ1 (*pgtC*)	0.51	0.30
pSJ2 (*pgtBC*)	11.20	4.6[b]
pSJ3 (*pgtB*)	8.22	3.51[b]
pJH744 (*pgtP*)	0.34	0.24
pJH746 (*pgtA*)	0.62	0.61
pBR322	0.22	0.21

[a] Transport assays were performed as described in Materials and Methods.
[b] We have no explanation for why transport activity was higher in uninduced than induced cultures.

Low-copy-number plasmid pJH567 (wild type) conferred an inducible transport phenotype as expected (Table 2). However, in contrast to the high-copy-number fusion plasmid pJH526 (*pgtB*), the low-copy-number mini-F equivalent pJH577 was unable to confer 3-PG transport, suggesting that the *pgtB* gene was necessary for expression of the *pgtP* gene when these genes were present in low copy number, as in the chromosome. Like its high-copy-number equivalent pSJ1 (*pgtC*), mini-F deletion plasmid pSJ11 (*pgtC*) conferred a non-transport phenotype. Similarly, mini-F deletion plasmids pSJ12 (*pgtC pgtB*) and pSJ13 (*pgtB*) also conferred a non-transport phenotype, in marked contrast to the constitutive phenotype observed with the high-copy-number equivalents pSJ2 and pSJ3. Thus, both *pgtB* and *pgtC* genes appeared to be indispensable for the expression of the *pgtP* gene in these low-copy-number mini-F *pgt* plasmids. Plasmid pSJ14, in which part of the activator gene was deleted, conferred a non-transport phenotype, as expected from previous experiments (3).

Constitutive *pgt* mutant. When both the *pgtB* and *pgtC* genes were essentially completely deleted (Fig. 4), strikingly, the resulting plasmid, pSJ18, conferred a constitutive transport phenotype, rather than the non-transport phenotype observed with the *pgtBC* gene deletion plasmid, pSJ12 (Table 2). The deletion in plasmid pSJ18 was 2.4 kb long and extended from the *Aat*II site located at the beginning of *pgtC* gene to the *Bss*HII site located in the middle of *pgtB* gene. Plasmid pSJ18 thus differed from plasmid pSJ12, in that the

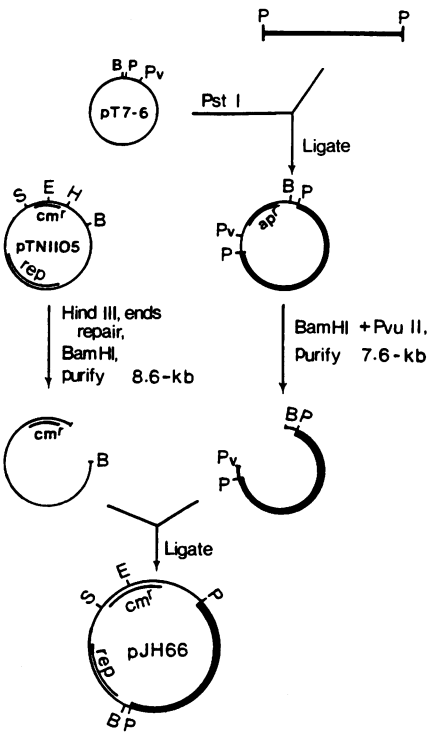

FIG. 3. Construction of low-copy-number mini-F deletion plasmids. The mini-F plasmids, pJH66, pSJ11, pSJ12, pSJ13, and pSJ14, were constructed as is illustrated for pJH66. The 7.6-kb *Pst*I fragment was isolated from high-copy-number pJH6 and ligated to pT7-6 at the *Pst*I site. From the resulting plasmid, the 7.6-kb *Bam*HI-*Pvu*II fragment was purified and ligated to the mini-F plasmid pTN1105 (6), which had been cut with *Hin*dIII, end-repaired, and further cut with *Bam*HI. Mini-F plasmids pJH66, pSJ11, pSJ12, pSJ13, and pSJ14 were equivalent to high-copy-number plasmids pJH6, pSJ1, pSJ2, pSJ13, and pJH746, respectively, in terms of insert DNA. Abbreviations of restriction sites used are as in Fig. 2. Pv, *Pvu*II; S, *Sal*I; cm^r, chloramphenicol resistance; ap^r, ampicillin resistance; rep, replication origin.

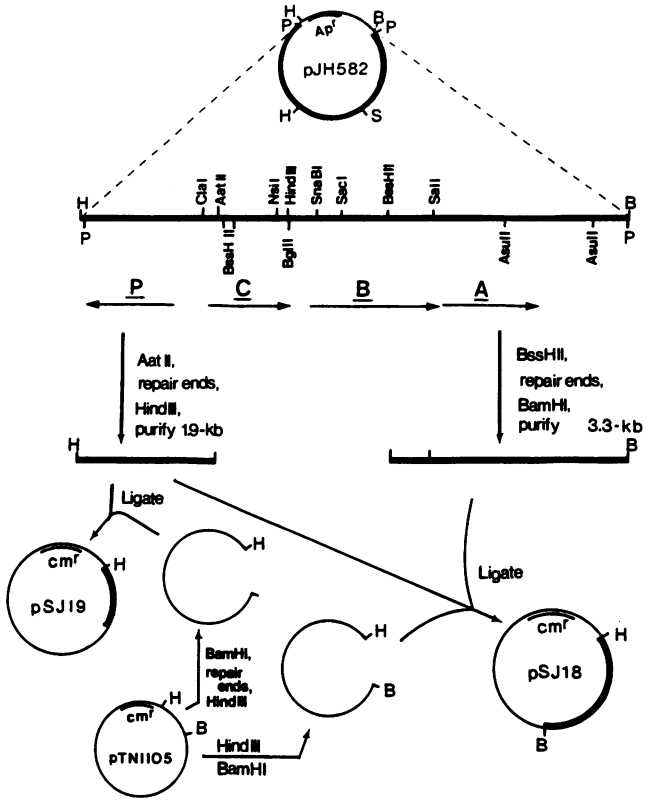

FIG. 4. Construction of mini-F plasmids pSJ18 and pSJ19. The 1.9-kb fragment carrying the *pgtP* gene with *Hin*dIII site on one end and a blunt end on the other was prepared from the *Hin*dIII-*Aat*II fragment of pJH582. The 3.3-kb fragment carrying the *pgtA* gene and part of the *pgtB* gene with *Bam*HI site on one end and a blunt end on the other was prepared from *Bss*HII-*Bam*HI fragment of pJH582. Ligation of the purified 1.9-kb fragment to mini-F plasmid pTN1105 which had been cut with *Bam*HI, end-repaired, and cut with *Hin*dIII yielded pSJ19. Ligation of both 1.9- and 3.3-kb fragments to pTN1105 which had been cut with both *Hin*dIII and *Bam*HI yielded pSJ18. Abbreviations of restriction enzymes used are as in the legends to Fig. 1 and 2.

latter still retained the bulk of *pgtC* gene, since the deletion began near the end of the *pgtC* gene and extended into the *pgtB* gene (Fig. 2). These results suggest that the activity of the *pgtA* gene product was affected not only by intact PgtB and PgtC proteins but also by the truncated PgtC protein of pSJ12.

Constitutive expression of *pgtP* (in pSJ18) required the *pgtA* gene product, even when the *pgtB* and *pgtC* gene products were absent. Thus, plasmid pSJ19 containing only the *pgtP* gene (in a 1.9-kb *Hin*dIII-*Aat*II fragment [Fig. 4]) conferred a non-transport phenotype, whereas pSJ18, which harbored both *pgtP* and *pgtA*, conferred a constitutive phenotype (Table 2).

TABLE 2. 3-PG transport conferred by low-copy-number mini-F *pgt* insertion and deletion plasmids[a]

Plasmid	3-PG transport (nmol/mg of protein/1 min)	
	Uninduced	Induced
pJH567 (wild type)	0.81	8.6
pJH577 (*pgtB*)	0.92	0.61
pJH66 (wild type)	1.45	20.9
pSJ11 (*pgtC*)	0.44	0.40
pSJ12 (*pgtBC*)	1.10	1.30
pSJ13 (*pgtB*)	1.10	0.91
pSJ14 (*pgtA*)	0.49	0.66
pSJ18 (Δ*pgtBC*)	13.8	13.4
pSJ19 (*pgtABC*)	0.37	0.43

[a] Transport assays were performed as described in Materials and Methods.

PgtABC polypeptides are not involved in posttranslational modulation of PgtP activity. While the results presented above strongly suggest that the three regulatory proteins, PgtABC, are most likely involved in the regulation of expression of the *pgtP* gene, alternatively, it is possible, although highly unlikely, that these polypeptides might be involved in posttranslational modulation of the PgtP activity, that is, PgtP might be always expressed but its activity would depend on PgtABC polypeptides. To test this possibility, a *lacZ-pgtP* fusion plasmid, pJHL30, was constructed as follows. A 760-bp *Bss*HII-*Nde*I fragment, harboring the 5′-flanking region of the *pgtP* gene plus the first 66 bp of the gene, was prepared from plasmid pJH585 (9), and its ends were blunted with S1 nuclease. This fragment was then ligated to *lacZY*-bearing plasmid pMC1403 (2) that had been cut with *Sma*I and dephosphorylated. By sequencing, the fusion plasmid pJHL30 (ampicillin resistant [Ap^r]) was found to have the 760-bp *Bss*HII-*Nde*I inserted in the desired

TABLE 3. Effects of low-copy-number deletion plasmids on the expression of *lacZ* in *lacZ-pgtP* fusion plasmid pJHL30

Plasmid	β-galactosidase activity[a]	
	Uninduced	Induced
pJHL30	370 (1.0)	278 (0.8)
pJHL30 + pJH66	255 (0.7)	6471 (17.5)
pJHL30 + pSJ11	344 (0.9)	289 (0.8)
pJHL30 + pSJ12	248 (0.6)	270 (0.7)
pJHL30 + pSJ13	229 (0.6)	274 (0.7)
pJHL30 + pSJ14	292 (0.8)	300 (0.8)
pJHL30 + pSJ18	2557 (6.9)	2338 (6.3)

[a] Exponentially growing cultures in minimal succinate medium were assayed for β-galactosidase activity as described previously (5), and each value, in units as defined by Miller (5), was an average from three determinations. Conditions for induction were described in Materials and Methods. Numbers in parentheses are those relative to the control (pJHL30) under uninduced conditions.

orientation with the *Nde*I end proximal to the *lacZ* coding region. It had 24 codons preceding codon 8 of the *lacZ*, 22 of which were the first 22 codons of *pgtP* and codons 23 and 24 being from the polylinker in pMC1403. Strain YMC9 with the *lac* operon deleted (1) was the recipient for transformation with plasmids. The expression of the fused *lacZ* of this plasmid was examined in cells lacking the *lac* operon and bearing one of the following low-copy-number mini-F deletion plasmids: pJH66, pSJ11, pSJ12, pSJ13, pSJ14, and pSJ18 (Table 3). Mini-F-derived plasmids and pJHL30 are compatible, thus allowing complementation to be performed. It is clear from Table 3 that the effects of the deletions on the expression of *lacZ* are essentially identical to those observed on the expression of *pgtP* measured by 3-PG transport (Table 2). With plasmid pJH66 carrying intact *pgtABC* genes, the expressed *lacZ* level was very low under uninduced conditions, was slightly lower than the control without pJH66, and was increased 17.5-fold under induced conditions. With deletion plasmids pSJ11, pSJ12, pSJ13, and pSJ14, very low expression of *lacZ* was observed under both uninduced and induced conditions. However, with plasmid pSJ18, the *lacZ* expression was constitutive. These results indicate that PgtABC are involved in the regulation of expression of *pgtP* gene, but not in modulation of the activity of PgtP polypeptide.

DISCUSSION

The results presented in this paper indicate that both *pgtB* and *pgtC* are regulatory genes necessary for the induction of the *pgtP* gene. These two genes are flanked by *pgtA* and *pgtP* genes. Thus, altogether the *pgt* system of *S. typhimurium* is organized in a cluster of four genes in the order *pgtP*, *pgtC*, *pgtB*, and *pgtA*. The *pgtP* gene, which encodes the transporter, is transcribed from right to left, and the regulatory genes, *pgtC*, *pgtB*, and *pgtA*, are transcribed in the opposite direction (3, 9, 10). The three regulatory genes do not appear to form an operon, since the expression of *pgtA* was unaffected by deletions or insertions in *pgtB* and deletion in *pgtBC* (pSJ2). However, it is possible that *pgtB* and *pgtC* constitute an operon.

A detailed understanding of the function of the three gene products, PgtA, PgtB, and PgtC, in relation to expression of *pgtP* will require further investigation. However, the results presented here enable certain conclusions to be made. It seems plausible that the role of PgtA is to positively regulate the expression of the *pgtP* gene, since in both high-copy-number plasmids and low-copy-number mini-F plasmids, deletion of *pgtA* always resulted in a non-transport phenotype. PgtA may be regarded therefore as an activator or derepressor.

PgtB and PgtC are also involved in induction of *pgtP*, since expression of the *pgtP* gene was (i) inducible only when all three regulatory genes were all intact, (ii) constitutive when both *pgtB* and *pgtC* genes were totally deleted, and (iii) not expressed when either the *pgtB* or *pgtC* gene (in the low-copy-number plasmids) were deleted. It is possible that the activity of PgtA protein in controlling *pgtP* expression is subject to modulation by PgtB and PgtC proteins. The mechanism for this modulation is unknown. The membrane location of these three proteins (9, 10) and the fact that induction of *pgtP* expression responds to exogenous inducer suggests that a mechanism involving protein-protein interactions between the PgtC, PgtB, and PgtA proteins occurs within the membrane.

The data obtained with *lacZ-pgtP* fusion plasmid pJHL30 (Table 3) supports the conclusion that PgtB and PgtC are involved in induction, and also renders highly improbable the possibility that PgtB and PgtC might be involved in modulation of PgtP activity, since no *lacZ* was expressed under uninduced conditions when the PgtABC were all functional and since PgtABC were all required for the expression of the *lacZ* gene under induced conditions.

A working model of induction that can form the basis for future experiments is as follows. Under noninducing conditions, the derepressor, PgtA, cannot assume the required conformation to promote DNA binding and therefore cannot facilitate *pgtP* gene transcription. Under inducing conditions, the derepressor can adopt an active form as a result of a conformational change that occurs when inducer binds to the receptor site on the periplasmic side of the membrane. PgtB and PgtC are presumably involved in transmission of a transmembrane signal to PgtA in response to inducer binding. PgtA can be freed from constraints imposed by PgtB and PgtC not only as a result of inducer binding but also when these proteins are removed by deletion as in plasmid pSJ18 (Δ*pgtBC*).

According to this model of induction, a defective receptor or defective signal transmission could explain the non-expression phenotype exhibited by plasmids pJH577, pSJ11, pSJ12, and pSJ13 that harbor insertions or deletions in either the *pgtB* or *pgtC* gene. Moreover, total deletion of both *pgtB* and *pgtC* genes could account for the constitutive phenotype conferred by pSJ18. The latter finding rules out the possibility that induction might involve chemical modification of the derepressor mediated by either PgtB or PgtC protein (or both), since such a mechanism would predict a non-expression phenotype, rather than the observed constitutive phenotype of pSJ18.

There exists a discrepancy between the constitutive transport phenotype conferred by the high-copy-number *pgt* deletion plasmids, pSJ2 and pSJ3, and insertion plasmid pJH526 on the one hand (Table 1) and the non-transport phenotype exhibited by their low-copy-number mini-F equivalents, pSJ12, pSJ13, and pJH577 on the other (Table 2). It may be, in the context of the proposed model, that when these regulatory proteins were expressed in the high-copy-number plasmids, some derepressor molecules were free and uncomplexed, and therefore were active, but when expressed in low-copy-number plasmids, all derepressor molecules were complexed and therefore inactive. Whatever the reason, it is clear from the contradicting observations made here that to avoid complications that may arise with

high-copy-number plasmids, it is important to conduct regulatory studies with low-copy-number plasmids, a situation which, in terms of gene number, closely resembles a chromosomal location. Similar cautions have been made by Weston and Kadner (8) and others.

Kadner and co-workers (7) have extensively analyzed the exogenously induced *uhp* system involved in hexose-6-phosphate transport in *Escherichia coli*. Like *pgtP* expression, the expression of *uhpT* encoding the transporter requires three regulatory genes. It is not unknown if similar regulatory mechanisms operate for these two systems.

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LITERATURE CITED

1. **Backman, K., Y. M. Chen, and B. Magasanik.** 1981. Physical and genetic characterization of the *glnA-glnG* region of the *Escherichia coli* chromosome. Proc. Natl. Acad. Sci. USA **78:** 3743–3747.
2. **Casadaban, M. J., J. Chou, and S. N. Cohen.** 1980. In vitro gene fusions that join an enzymatically active β-galactosidase segment to amino-terminal fragments of exogenous proteins: *Escherichia coli* plasmid vectors for the detection and cloning of translational initiation signals. J. Bacteriol. **143:**971–980.
3. **Goldrick, D., G.-Q. Yu, S.-Q. Jiang, and J.-S. Hong.** 1988. Nucleotide sequence and transcription start point of the phosphoglycerate transporter gene of *Salmonella typhimurium*. J. Bacteriol. **170:**3421–3426.
4. **Hugenholz, J., J.-S. Hong, and H. R. Kaback.** 1981. ATP-driven active transport in right-out membrane vesicles. Proc. Natl. Acad. Sci. USA **78:**3446–3449.
5. **Miller, J. H.** 1972. Experiments in Molecular Genetics, p. 162–172. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.
6. **Nohno, T., T. Saito, and J.-S. Hong.** 1986. Cloning and complete nucleotide sequence of the *Escherichia coli* glutamine permease operon (glnHPQ). Mol. Gen. Genet. **205:**260–269.
7. **Vogel, H. J., and D. M. Bonner.** 1965. Acetylornithinase of *Escherichia coli*: partial purification and some properties. J. Biol. Chem. **218:**97–102.
8. **Weston, L. A., and R. J. Kadner.** 1987. Identification of Uhp polypeptides and evidence for their role in exogenous induction of the sugar phosphate transport system of *Escherichia coli* K-12. J. Bacteriol. **169:**3546–3555.
9. **Yang, Y.-L., D. Goldrick, and J.-S. Hong.** 1988. Identification of the products and nucleotide sequences of two regulatory genes involved in the exogenous induction of phosphoglycerate transport in *Salmonella typhimurium*. J. Bacteriol. **170:**4299–4303.
10. **Yu, G.-Q., and J.-S. Hong.** 1986. Identification and nucleotide sequence of the activator gene of the externally induced phosphoglycerate transport system of *Salmonella typhimurium*. Gene **45:**51–57.