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Chromosomal Location of a Gene for Fructose 6-Phosphate Kinase in *Escherichia coli*

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Pfk lies between *rha* and *glpK*.

We have recently described the selection and characteristics of *Escherichia coli* mutants lacking fructose 6-phosphate kinase ("phosphofructokinase"); these mutants grow slowly or not at all on carbon sources whose major route of degradation is via fructose 6-phosphate (3). This note describes genetic mapping of the locus *pfk*.

The three mutants AM-1, 2, and 3, carrying the mutations *pfk-1*, *2*, and *3*, were selected from strain K-10 (Hfr Cavalli, str^s). In a conjugation of AM-1 with DF1933 (F^-, $arg(BCEH)^-$, $metA^-$, str^r) (*see* Fig. 1 for map positions of known markers), 70% of arg^+ str^r recombinants were pfk^-. In a P1 transduction between the same strains with selection for arg^+ (Table 1, experiment 1), *pfk* showed 5% cotransduction; with selection for met^+ (*data not shown*) there was no cotransduction of *pfk*. These results are best in accord with the order *pfk-arg-metA*. Similar results were obtained in transductions between this recipient and AM-2 and 3.

Transductions using as recipient a strain carrying $glpK^-$ and $metB^-$ (1) showed cotransduction of *pfk* with both of these markers (Table 1, experiments 2 and 3), and for each of the *pfk* alleles the distribution of unselected markers best fits the order *pfk-glpK-metB*.

The next known marker counterclockwise to *glpK* being *rha* (5), we then used (Table 1, experiments 4 and 5) as recipient a strain carrying rha^- and $metB^-$ (4). These data show the cotransduction of *pfk* with *rha*, and the pattern of inheritance of unselected markers in both experiments best fits the order *rha-pfk-metB*. The results thus establish the order *rha-pfk-glpK* as shown in Fig. 1 which also gives cotransduction frequencies from these experiments. It is not yet known whether the *pfk* locus is for a structural gene.

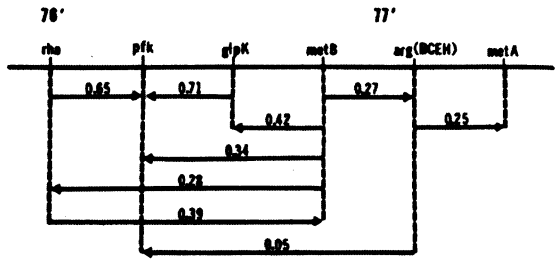

FIG. 1. *Genes at 76 to 77 min on the E. coli map (according to reference 5, except for pfk). Cotransduction frequencies from the experiments reported here are given as the number of recombinants with unselected marker inherited from donor/number of recombinants with selected marker inherited from donor, the selected marker being at the tail of the arrow and the unselected marker at the head.*

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TABLE 1. *Transduction of pfk*[a]

Expt	Donor	Recipient	Selected marker	No. scored	Unselected markers			
1	AM-1(*pfk-1*)	DF1933 [*arg*(*BCEH*)$^-$, *metA*$^-$]	*arg*$^+$	300	*metA*$^+$ *pfk*$^+$ 64	*metA*$^+$ *pfk*$^-$ 0	*metA*$^-$ *pfk*$^+$ 221	*metA*$^-$ *pfk*$^-$ 15
2	AM-1 (*pfk-1*)	161(*glpK*$^-$, *metB*$^-$)	*glpK*$^+$	287	*metB*$^+$ *pfk*$^+$ 37	*metB*$^+$ *pfk*$^-$ 101	*metB*$^-$ *pfk*$^+$ 47	*metB*$^-$ *pfk*$^-$ 102
3a	AM-1(*pfk-1*)	161(*glpK*$^-$, *metB*$^-$)	*metB*$^+$	266	*glpK*$^+$ *pfk*$^+$ 37	*glpK*$^+$ *pfk*$^-$ 75	*glpK*$^-$ *pfk*$^+$ 154	*glpK*$^-$ *pfk*$^-$ 0
3b	AM-2(*pfk-2*)	161(*glpK*$^-$, *metB*$^-$)	*metB*$^+$	300	48	89	163	0
3c	AM-3(*pfk-3*)	161(*glpK*$^-$, *metB*$^-$)	*metB*$^+$	300	46	89	161	4
4	AM-1(*pfk-1*)	*rhaA50*, *metB*$^-$	*metB*$^+$	300	*pfk*$^+$ *rha*$^+$ 27	*pfk*$^+$ *rha*$^-$ 156	*pfk*$^-$ *rha*$^+$ 51	*pfk*$^-$ *rha*$^-$ 66
5	AM-1(*pfk-1*)	*rhaA50*, *metB*$^-$	*rha*$^+$	270	*metB*$^+$ *pfk*$^+$ 7	*metB*$^+$ *pfk*$^-$ 98	*metB*$^-$ *pfk*$^+$ 88	*metB*$^-$ *pfk*$^-$ 77

[a] Media and transduction with phage P1 were as described earlier (2). Strain DF1933 is an *str*^{r} derivative of AB1932 from E. A. Adelberg's collection. Strain 161 was kindly supplied by E. C. C. Lin, and the *rhaA50*, *metB*$^-$ strain by E. Englesberg. Selection and scoring for nutritional markers were on the appropriately supplemented gluconate minimal plates, as *pfk* mutants grow normally on gluconate (3). *GlpK*$^+$ selection was on plates with glycerol as carbon source, and *rha*$^+$ selection used rhamnose as carbon source. Recombinants were purified by single-colony reisolation on the selective media, and unselected markers were scored by replica plating. *Pfk* was scored on mannitol minimal plates in experiments 4 and 5, and on glucose minimal plates in experiments 1–3. (Mannitol is the preferred medium for this as *pfk* mutants do not grow at all on mannitol; the recipients in experiments 1–3, however, carried mutations in the specific mannitol pathway.) Scoring of *pfk* was confirmed by enzyme assay of selected recombinants.