The 6-Phosphogluconate Dehydrogenase Reaction in Escherichia coli*

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This study is an attempt to relate the in vivo use of the 6-phosphogluconate dehydrogenase reaction in Escherichia coli with the characteristics of the enzyme determined in vitro. 1) The enzyme was obtained pure by affinity chromatography and kinetically characterized; as already known, ATP and fructose-1,6-P₂ were inhibitors. 2) A series of isogenic strains were made in which in vivo use of the reaction might differ, e.g. a wild type strain versus a mutant lacking 6-phosphogluconate dehydrase, as grown on glucose; a phosphogluco-isomerase mutant grown on glucose or glycerol. 3) The in vivo rate of use of the 6-phosphogluconate dehydrogenase reaction was determined from measurements of growth rate and yield and from the specific activity of alanine after growth in ¹³C-labeled substrates. 4) The intracellular concentrations of 6-phosphogluconate, NADP⁺, fructose-1,6-P₂, and ATP were measured for the strains in growth on several carbon sources. 5) The metabolite concentrations were used for assay of the enzyme in vitro. The results allow one to calculate how fast the reaction would function in vivo if ATP and fructose-1,6-P₂ were its important effectors and if the in vitro assay conditions apply in vivo. The predicted in vivo rates ranged down to as low as one-tenth of the actual rates, and, accordingly, one cannot yet draw firm conclusions about how the reaction is actually controlled in vivo.

Neither the functions nor the control of the hexose monophosphate shunt are completely understood. It can be thought of as a biosynthetic pathway leading to ribose-5-P, erythrose-4-P, and sedoheptulose-7-P, as a source of reduced NADP⁺ for biosynthesis or as a cyclic pathway of carbohydrate metabolism forming glyceraldehyde-3-P. The problems of estimating rates and directions of metabolism in the shunt have been discussed extensively (1-3).

In Escherichia coli, there are several relevant experiments (4). First, the two dehydrogenases of the shunt, for glucose 6 P and 6-phosphogluconate, are found in all growth conditions. Second, mutants lacking phosphogluco-isomerase grow slower on glucose, and mutants lacking 6-phosphogluconate dehydrase grow slower on gluconate. In neither case are the dehydrogenase levels increased above wild type levels. Third, experiments with labeled glucose show that the oxidative branch of the shunt is used much less, if at all, anaerobically than aerobically. Fourth, a mutant lacking transketolase does not accumulate pentose-P.

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monitored with a Gilford model 300-N spectrophotometer.

Cell yields (Table II) were obtained from a series of growth experiments using a carbon source in the range of 0 to 10 mM and a plot of final ArXK versus initial carbon source concentration.

Enzyme Assays—Cells were harvested, washed with 0.9% NaCl, resuspended in 3 mM Tris-HCl, 0.03 mM dithiothreitol, pH 7.5, and disrupted by 1 to 2 min of treatment with a Biosonic III sonicator (Bronwill Scientific Co.). The 6-phosphogluconate dehydrogenase standard assay (Tris buffer assay) contained 50 mM Tris-HCl (pH 7.5), 10 mM MgCl₂, 1 mM 6-phosphogluconate, and 0.2 mM NADP⁺. The (phosphate buffer assay contained 0.1 mM potassium phosphate (pH 7.5) instead of Tris.) NADP⁺ reduction was monitored with a Gilford model 300-N spectrophotometer at 23°C unless otherwise specified. Glucose-6-P dehydrogenase was assayed like 6-phosphogluconate dehydrogenase (Tris buffer assay) but with 1 mM glucose-6-P as substrate. Phosphoglucose isomerase was assayed as described (13), and 6-phosphogluconate dehydrase was assayed by the procedure of Eisenberg and Dobrogosz (14). Protein was measured according to Lowry et al. (15) with bovine serum albumin as standard. Enzyme activities are usually given as nanomoles/min/mg of protein.

Purification of 6-Phosphogluconate Dehydrogenase—We used the strain with high level of 6-phosphogluconate dehydrogenase (RW226/pLC33-5) and affinity chromatography on Cibacron blue-Sepharose, following the report of Thompson et al. (16) that the yeast enzyme binds to such columns and could be eluted by glucose-6-P. The enzyme was partly purified by the method of Lowry et al. (19), slightly modified. Fifty-milliliter culture samples (approximately 0.5) were filtered (Millipore, 47 mm diameter, 0.45-μ pore size) and the filters with cells were frozen that for NADP⁺ (25).

Enzyme Kinetics—6-Phosphoglucuronate dehydrogenase was obtained pure by a new procedure using a strain with 5-fold normal amount of enzyme ("Experimental Procedures"). No activity was observed (1% would have been detected) with 1 mM glucose, glucose-6-P, fructose-6-P substituting for 6-phosphogluconate or with 4 mM NAD⁺ substituting for NADP⁺. In the latter characteristic 6-phosphoglucuronate dehydrogenase differs from glucose-6-P dehydrogenase of the same organism, which can use NAD⁺ with a Km 100 fold greater than that for NADP⁺ (25).

Table I shows the effects of some metabolites on the reaction. In agreement with the results of others (6, 7) ATP and fructose-1,6-P₂ were inhibitory; ribose-5-P and ADP also caused some inhibition.

Fig. 1 shows that the ATP inhibition curve was sigmoidal with half-maximal inhibition at 3 mM. The fructose-1,6-P₂ inhibition curve was hyperbolic with half-maximal inhibition at 0.025 mM. A similar fructose-1,6-P₂ inhibition curve was

![Graph](http://www.jbc.org/)
also obtained with toluenized cells (Fig. 1), which suggests that the partial inhibition is not an artifact of enzyme dissociation in dilute solution. Partial inhibition by fructose-1,6-P₂ is also a characteristic of the enzyme from Streptococcus faecalis (6).

The purpose of the present work was not to obtain a complete kinetic characterization of the enzyme. However, it was necessary to determine whether the rates depended greatly on the physical conditions of assay since we planned to later use the known in vivo metabolite concentrations on the enzyme in vitro. Since the cells were grown in a medium containing 0.1 M phosphate buffer, and there was a report of internal and external concentrations of phosphate then being similar (27), we chose as a second assay system one containing 0.1 M potassium phosphate. Vₘₐₓ values were similar (see Table IV) in the two buffer systems (Tris and phosphate).

Substrate affinities were similar for NADP⁺ (half-maximal rates at 1.7 and 2.8 X 10⁻⁵ M in Tris and phosphate, respectively), but differed considerably for 6-phosphogluconate (values of 1 X 10⁻⁵ and 1 X 10⁻⁴ M in Tris and phosphate, respectively). Inhibitions were less in the phosphate buffer, where 5 mM ATP inhibited only 20% (versus 100% in Tris), and concentration of fructose-1,6-P₂ about 10-fold higher was needed to give the same inhibition as in Tris. The pH versus activity curves differed somewhat for the two buffer systems (not shown), but a pH of 7.5 was used in accord with recent determinations of intracellular pH (28, 29).

These limited data show that E. coli 6-phosphogluconate dehydrogenase shows fairly complex kinetics and that fructose-1,6-P₂ and ATP are potential negative effectors in vivo. The differences in rate of the enzyme reaction seen in the two assay systems presented a serious potential difficulty for experiments where in vitro and in vivo rates were to be correlated, since the true in vivo enzyme environment is unknown, and they were done using both assay systems.

**Strains and Growth Characteristics**—Fig. 2 shows the relevant pathways. A series of isogenic strains were prepared blocked at steps other than 6-phosphogluconate dehydrogenase (Table II). The growth of these strains in minimal medium on several carbon sources (glucose, glucose-6-P, gluconate, and glycerol) is also indicated. The key data are as follows. Strains containing both 6-phosphogluconate dehydrogenase (edd⁺), the first enzyme of the Entner-Doudoroff pathway, and 6-phosphogluconate dehydrogenase (grd⁺) grow about twice as fast on gluconate as strains containing only the latter enzyme. Likewise, growth on glucose-6-P or glucose is about twice as fast in a strain having phosphoglucose isomerase (pgi⁺) as in a strain having only glucose-6-P dehydrogenase (zwf⁻). Strains lacking both phosphoglucose isomerase and either glucose-6-P dehydrogenase (DF1671DZI) or 6-phosphogluconate dehydrogenase (DF563) are completely unable to grow on glucose or glucose-6-P. A strain lacking only glucose-6-P dehydrogenase (DF565) grows well on glucose. All the strains grow on glycerol. These growth characteristics are in accord with previous results (4) and with the scheme of Fig. 2, but the comparison has not previously been made in a single series of isogenic strains. Table II also shows, for a few cases, the cell yields (cells formed (A₅₇₀)/ml/mmol of substrate used). Yields were similar for the several situations, e.g. growth on gluconate of an edd⁻ strain occurs with the same yield as the faster growth of an edd⁺ strain. This would be the result expected by guest on March 24, 2020http://www.jbc.org/Downloaded from
expected if the slower growth is caused by the inability of the cell to produce metabolic intermediates at normal rate.

Levels of Metabolites during Growth—The next step was to determine levels of pertinent metabolic intermediates in the various strains in growth on several carbon sources (Table III). We will not discuss each item, but make the following comments.

(i) There are several cases where certain metabolites were not found: for example, a pgi- mutant growing on glycerol contained no glucose-6-P or 6-phosphogluconate. Such expected results confirm the assignment of genotypes and metabolic pathways.

(ii) In certain cases metabolic levels were quite high. An example is strain DF564, which lacks both phosphoglucose isomerase and 6-phosphogluconate dehydrase and where, in growth on glucose, the concentration of glucose-6-P was 18.5 mM (versus 1.25 mM in the wild type) and the 6-phosphogluconate concentration was 6.6 mM (versus 0.1 mM in the wild type). Such results were also expected and apparently reflect accumulation of a metabolite at a point where a major pathway is blocked but a second pathway remains open. They also show the enzyme to function at a rate at least 50% of the Vmax.

(iii) The concentration of NADP+ did not vary much among strains and growth conditions. Its concentration probably would always be saturating for 6-phosphogluconate dehydrogenase.

(iv) 6-Phosphogluconate, the main substrate of interest in this work, would probably always (except for the special case of zero concentration) saturate 6-phosphogluconate dehydrogenase, if one uses the enzyme characteristics as determined in Tris buffer. However, as assayed in phosphate buffer, this substrate would be below the Km in growth on glucose or glucose-6-P.

(v) The concentrations of fructose-1,6-P2 are in a range which would be expected to appreciably inhibit the enzyme, even as assayed in phosphate.

The ATP concentrations are relatively constant (approximately 4 mM for the few cases measured). This might be expected to give 50 to 80% inhibition if the enzyme in vivo were in an environment best matched by the Tris assay system, but if the phosphate assay system is a truer reflection of the cell, then ATP inhibition would be less.

The Enzyme Activity Determined with in Vivo Substrate and Inhibitor Concentrations—Table IV gives the predicted rate (as % Vmax) of 6-phosphogluconate dehydrogenase using the in vivo substrate and inhibitor concentrations from Table III. For these experiments, the enzyme was a crude extract of the strain with a high level of 6-phosphogluconate dehydrogenase since the pure enzyme lost ATP sensitivity on storage. The experiments were done at the growth temperature (37°C) in both the Tris and phosphate assay systems. The results show the enzyme to function at a rate at least 50% of the Vmax.

| Strain         | Genotype | Medium   | Metabolite concentration |
|---------------|----------|----------|--------------------------|
|               |          |          | NADP+ | 6-P-gluconate | Glucose-6-P | Fructose-1,6-P2 | ATP | ADP |
| DF562         | pgi-     | Glucose  | 0.9  | 0.8           | 11.0        | 0.6            | N.D.* | N.D. |
| DF562         | pgi-     | Gluconate| 0.15 | 1.2           | 0.0         | 2.0            | 4.0  | 0.9  |
| DF562         | pgi-     | Gluconate (N2) | 0.1  | 7.4           | 0.0         | 2.2            | N.D. | N.D. |
| DF562         | pgi-     | Glycerol | 0.2  | 0.0           | 0.0         | 1.1            | N.D. | N.D. |
| DF563         | pgi-     | Gluconate | 0.2  | 0.6           | 0.0         | 0.75           | N.D. | N.D. |
| DF563         | pgi-     | Glycerol | 0.3  | 0.0           | 0.0         | 1.7            | N.D. | N.D. |
| DF564         | pgi-     | Glucose  | 0.1  | 6.1           | 18.5        | 1.2            | N.D. | N.D. |
| DF564         | pgi-     | Glucose-6-P | 0.06 | 10.5         | 35.4        | 0.9            | 3.2  | 0.9  |
| DF564         | pgi-     | Gluconate | 0.1  | 11.6          | 0.0         | 0.9            | 2.9  | 0.8  |
| DF564         | pgi-     | Glycerol | 0.3  | 0.0           | 0.0         | 2.7            | N.D. | N.D. |
| DF567         | zwf-     | Glucose  | 0.35 | 0.0           | 1.5         | 4.8            | N.D. | N.D. |
| DF565         | zwf-     | Gluconate | 0.1  | 15.9          | 0.5         | 1.9            | N.D. | N.D. |
| DF565         | zwf-     | Glycerol | 0.4  | 0.0           | 0.5         | 2.4            | N.D. | N.D. |
| DF566         | zwf-     | Glucose  | 0.1  | 0.1           | 0.8         | 4.9            | N.D. | N.D. |
| DF566         | zwf-     | Gluconate | 0.1  | 15.5          | 0.3         | 1.2            | N.D. | N.D. |
| DF566         | zwf-     | Glycerol | 0.2  | 0.08          | 0.07        | 1.3            | N.D. | N.D. |
| DF567         | (wild type) | Glucose | 0.4  | 1.1           | 1.2         | 6.2            | 4.2  | 1.0  |
| DF567         | (wild type) | Glucose-6-P | 0.15 | 0.1           | 4.5         | 2.5            | N.D. | N.D. |
| DF567         | (wild type) | Glucose-6-P | 0.10 | 0.2           | 6.0         | 5.8            | 3.4  | 1.3  |
| DF567         | (wild type) | Glycerol  | 0.25 | 0.85         | 0.6         | 1.7            | N.D. | N.D. |
| DF1671DZ1     | pgi-     | Glucose  | 0.25 | 0.08          | 0.4         | 3.2            | N.D. | N.D. |
| DF1671DZ1     | pgi-     | Glycerol | 0.35 | 0.0           | 0.0         | 1.2            | N.D. | N.D. |

* N.D., not determined.

(vi) The ATP concentrations are relatively constant (approximately 4 mM for the few cases measured). This might be expected to give 50 to 80% inhibition if the enzyme in vivo were in an environment best matched by the Tris assay system, but if the phosphate assay system is a truer reflection of the cell, then ATP inhibition would be less.
in the two conditions (lines 4 and 5) of metabolism restricted to the shunt and more slowly in the other cases.

To convert the % $V_{\text{max}}$ values into predicted in vivo rates, one must first know the amounts of enzyme in the several situations. Table IV also gives the enzyme levels ($V_{\text{max}}$) as assayed in the two buffer systems. The phosphate buffer gave slightly higher values than the Tris buffer, and the enzyme levels were similar in the several strains, as expected for a "constitutive" enzyme. The predicted in vivo rates would be the product of percent saturation and enzyme amount (see Table VI).

**In Vivo Use of 6-Phosphogluconate Dehydrogenase—** When all substrate utilization is via the 6-phosphogluconate dehydrogenase reaction (e.g. growth on gluconate or glucose or glucose-6-P of a mutant lacking both 6-phosphogluconate dehydrase and phosphoglucose isomerase (edd- $pgi$-)), the in vivo use of the reaction may be determined as growth rate/growth yield (i.e. micromoles of substrate used/unit cell/unit time). However, when glycolysis or the Entner-Doudoroff pathways are also available, only a fraction of total metabolism uses the shunt. We attempted to determine this fraction from the specific activity of a pyruvate derivative after growth on [1-14C]carbon source. Gluconate metabolism by the Entner-Doudoroff pathway or glucose metabolism by glycolysis would give radioactive pyruvate, while metabolism via the 6-phosphogluconate dehydrogenase reaction would give unlabeled products. Thus, one may derive (see "Experimental Procedures") an estimate of the fraction of metabolism by the two pathways from the specific activity of pyruvate. Alanine from protein is a convenient pyruvate derivative, and we have shown in earlier experiments that its radioactivity is in the carboxyl group when coming from 1-labeled substrate by the Entner-Doudoroff pathway and not in the carboxyl group when derived by glycolysis (13). A similar protocol has recently been used to indicate pathways of fructose metabolism in pseudomonads (30).

Table V shows the relative specific activities of alanine after growth on the 1-labeled substrates. The expected values for a strain not using the shunt would be 0.5, and this expectation was fairly well met: DF663, $gnd^-$, and, presumably using the Entner-Doudoroff pathway, specific activity was 0.51 on gluconate; DF655, $zwf^-$, and, presumably using glycolysis, specific activity was 0.47 on glucose and 0.52 on glucose-6-P. The other control was a strain presumably using only the shunt (DF654, $pgi^-$ $edd^-$): metabolites such as pyruvate should be unlabeled, which was the case for growth on 1-labeled gluconate (specific activity, 0.007); the value from cells grown on 1-labeled glucose-6-P was 0.04, also low, but somewhat higher than expected (as found previously (13)).

The values for the strains where both the shunt and the Entner-Doudoroff pathway should be functioning (DF662 or both the shunt and the glycolytic pathway (DF567)) were all approximately 0.43 in this experiment, as expected for minor use (approximately 20%) of the shunt in each case (see Table VI).

Table VI gives the rates of in vivo use of the 6-phosphogluconate dehydrogenase reaction as determined from the total rate of substrate utilization corrected for fractional use of the shunt. It also shows (last column) the rates which would be predicted from the mearured amount of enzyme were it to be functioning in vivo as it does in vitro with the actual concentrations of substrates and effectors. Predicted rates ranged between 16 and 98% of the estimated actual uses of the reaction.

**DISCUSSION**

There was generally poor agreement between the estimated rates of actual use of the 6-phosphogluconate dehydrogenase reaction in vivo and the rates predicted from studies of the enzyme in vitro. The amount of enzyme assayed with saturating substrates and without inhibitors was somewhat lower than the estimated rates of in vivo use of the reaction, and lower yet using the in vivo substrate and inhibitor concentrations. Hence, the predicted rates ranged between 16 and 98% of the actual rates. There is considerable uncertainty in both sets of values, and the following problems may be cited.

(i) The physical state and environment of the enzyme in the cell is not known—there is even ignorance about the internal phosphate concentration—and any number of other ions might have importance. It is also not known whether the enzyme properties in dilute solution accurately reflect the in vivo situation. We have not observed substantial kinetic differences using cells made permeable to substrates with toluene, but further studies of this type might be useful.

(ii) Even if the physical situation or ionic conditions were not critical, we may have not measured the right metabolites. There still might exist an unidentified activator. One modification of the experiments would be to include the reaction products ribulose-5-P, NADPH, and bicarbonate. An energy charge study would also be of interest.

(iii) Assay of metabolite concentrations is difficult. There may be degradation during harvest, or incomplete recoveries. Systematic errors of cell water content, etc., might not greatly affect conclusions about relative metabolite concentrations.
but could substantially affect the rates determined on the enzyme in vitro. Our measured metabolite levels are in general agreement with literature values (e.g. ATP measured with luciferase (31), fructose-1,6-P₂ (32), NADP⁺ (5), and several metabolites measured by Lowry et al. (19)) but still might be too inaccurate for the intended purpose.

(iv) Another impediment to the evaluation of possible roles of ATP, fructose-1,6-P₂, or other effectors is that there are no mutants in 6-phosphogluconate dehydrogenase altered allosterically but active catalytically. It is also not yet possible to change at will the level of the enzyme and observe the effect of ATP, fructose-1,6-P₂, or other effectors is that there are no mutants in 6-phosphogluconate dehydrogenase altered allosterically but active catalytically. It is also not yet possible to change at will the level of the enzyme and observe the effect on metabolism. Strains of these types probably could be obtained by appropriate mutagenesis and genetic construction.

(v) Determination of in vivo use of the 6-phosphogluconate dehydrogenase reaction is only easy for the trivial case when all metabolism is by the shunt (Table VI, last two lines). For the other cases, where another and quantitatively more important pathway is also available, a correction was made according to the specific activity of a pyruvate derivative, alanine. Such calculations depend on many assumptions (see Refs. 1 and 2). In particular, biosynthetic use of metabolites from the shunt was not taken into account. Thus, if the 6-phosphogluconate dehydrogenase reaction were the source of all ribose and aromatic amino acids, approximately 20% of all assimilation and 7% of metabolism might use the shunt without affecting the specific activity of alanine; the in vivo estimates in the first three cases of Table VI, therefore, might be low by approximately 30%. It should also be noted that it is not even certain that alanine derives exclusively from pyruvate (33).

Accordingly, although the present work has given useful data, it does not allow any strong conclusions to be drawn about control of metabolism in the hexose monophosphate shunt, let alone about the particular roles of fructose-1,6-P₂ and ATP as inhibitors of the 6-phosphogluconate dehydrogenase reaction. Even for the single situation where there is strong indication that the shunt must be inhibited, namely, anaerobic growth (see Ref. 4), the substrates were present in adequate amount and the potential inhibitor, fructose-1,6-P₂, was in lower concentration than aerobically (see Table III).

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