Biosynthesis in Escherichia coli of sn-Glycerol 3-Phosphate, a Precursor of Phospholipid

PALMITOYL-CoA INHIBITION OF THE BIOSYNTHETIC sn-GLYCEROL-3-PHOSPHATE DEHYDROGENASE*

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The biosynthesis of sn-glycerol 3-phosphate (glycerol-P), which is required for phospholipid biosynthesis in Escherichia coli, occurs in a single step from the glycolytic intermediate, dihydroxyacetone phosphate (dihydroxyacetone-P). The enzyme which catalyzes the NAD(P)H-dependent reduction of dihydroxyacetone-P to produce glycerol-P is the biosynthetic glycerol-P dehydrogenase (EC 1.1.1.8). The biosynthetic role for the enzyme was established by the isolation of glycerol (1) and glycerol-P (2) auxotrophs deficient in glycerol-P dehydrogenase activity. Since glycerol-P was a potent inhibitor of the glycerol-P dehydrogenase in vitro, it was suggested that glycerol-P might be a feedback inhibitor of glycerol-P biosynthesis in vitro (3). The physiological significance of the inhibition by glycerol-P was established by the isolation of strains bearing a mutation within the structural gene for the glycerol-P dehydrogenase, gpsA (4). Crude extracts of the mutant strains contained glycerol-P dehydrogenase activity about 10-fold less sensitive to glycerol-P inhibition than the parental activity (4). Glycerol-P dehydrogenase has been purified to homogeneity from the parental and feedback-resistant strains (5). Comparison of the kinetic properties of the homogeneous enzymes demonstrated that the property of feedback resistance was inherent to the mutant dehydrogenase and indicated that regulation of the enzyme by glycerol-P occurred at an allosteric site (6).

Although the regulation of glycerol-P dehydrogenase by glycerol-P has been extensively investigated in vitro (3, 5, 6) and its physiological significance has been established (4), little information exists regarding other mechanisms which may regulate glycerol-P biosynthesis. Kito and Pizer reported that glycerol-P dehydrogenase activity present in crude extracts and in partially purified preparations (3, 7) was potently inhibited by palmitoyl-coenzyme A. Numerous mammalian (8-12) and microbial enzymes (3, 13) are inhibited in vitro by palmitoyl-CoA, but the physiological significance of this inhibition has been questioned because of the inherent detergent properties of this compound (14, 15) and its irreversible inhibition of some enzymes (8). These criticisms are especially relevant for enzymes which exhibit inhibition at concentrations of palmitoyl-CoA in excess of its critical micellar concentration. Another criticism has stemmed from the absence of teleonomic rationales for the regulation of some enzymes which are strongly inhibited in vitro by this compound (8). The physiological significance of inhibition by palmitoyl-CoA remains to be established.

In this paper, we report investigations on the inhibition by palmitoyl-CoA of homogeneous, biosynthetic glycerol-P dehydrogenase from E. coli. In an attempt to evaluate the physiological significance of such inhibition with respect to glycerol-P synthesis, we have investigated the concentration dependencies, mode of inhibition, and reversibility of inhibition by palmitoyl-CoA. The effects of palmitoyl-CoA on the sedimentation coefficient of glycerol-P dehydrogenase and on the inhibition by glycerol-P were investigated. The specificity of inhibition was investigated by examining the effects of other acyl-CoA thioesters, palmitoyl-ACP, palmitoyl carnitine, palmitate, and detergents on glycerol-P dehydrogenase activity.

EXPERIMENTAL PROCEDURES

Materials—Dihydroxyacetone-P dimethylketal, sn-glycerol (1)-3-
phosphate, palmityl-L-carnitine chloride, taurocholic acid, sodium deoxycholate, Triton X-100, catalase, yeast alcohol dehydrogenase, ovalbumin, bovine serum albumin, and cyclodextrin were purchased from Sigma. NADPH, dihydrothreitol, and acyl-CoA derivatives were bought from P-L Biochemicals. Sodium dodecyl sulfate and palmitic acid were purchased from Schwarz/Mann and the Hormel Institute, respectively.

Dihydroxyacetone-P dimethylketal was converted to dihydroxyacetone-P according to the manufacturer's directions and quantitated as described previously (5).

E. coli acyl carrier protein acylated with palmitic acid was the generous gift of Drs. C. O. Rock and J. E. Cronan, Jr. of Yale University. Palmityl-(1,6,-etheno)CoA was the generous gift of Dr. Gary Powell of Clemson University.

The concentrations of CoA derivatives were quantitated by the millimolar extinction coefficient of 15.4 at 259 nm. The concentration of palmityl-CoA was quantitated by the millimolar extinction coefficient of 5.7 at 275 nm.

Glycerol-P Dehydrogenase Assays—Glycerol-P dehydrogenase was assayed by monitoring dihydroxyacetone-P-dependent consumption of NADPH (5). Assays were carried out at 23°C in 100 mM Tris (pH 7.4), 5 mM dihydrothreitol containing variable concentrations of substrates, dihydroxyacetone-P, NADPH, and compounds tested as inhibitors.

Protein Determination—Protein was determined by the procedure of Lowry et al. (15) using bovine serum albumin as standard.

Glycerol-P Dehydrogenase Purification—Glycerol-P dehydrogenase was purified from Escherichia coli strain 8 of Lin and co-workers (17) to apparent homogeneity as reported previously (5). The specific activities of the preparations employed ranged from 60 to 70 μmol/min/mg.

Preparation of Phospholipid Vesicles—Phospholipid vesicles were prepared from E. coli phospholipids by the method of Huang (18) and quantitated by phosphate analysis (19).

Biological Activity of Palmityl-ACP—To establish the biological activity of palmityl-ACP, the concentration dependency of palmityl-ACP was compared to that of palmityl-CoA as an acyl donor for glycerol-P acyltransferase activity. Crude membranes were prepared from strain 8 and assayed by the procedures used previously to compare the two acyl donors (20). The assays contained 30 μg of membrane protein, 1.25 mM [2-14C]glycerol-P (1.1 mCi/mmol), and variable concentrations of palmityl-ACP or palmityl-CoA (5 to 25 μM). Lineeweaver-Burk (21) plots of the data were reduced by least squares analysis (correlation coefficients greater than 0.99). For palmityl-ACP and palmityl-CoA, the respective apparent Kₜ values were 42 and 46 μM, and the respective apparent Vₘax values were 0.2 and 0.7 nmol/min/mg.

Sedimentation in Sucrose Density Gradients—The sₘₐₓ of glycerol-P dehydrogenase activity was determined by a modification of the method of Martin and Ames (22) in 5.0 ml of a 10 to 30% sucrose gradient containing 50 mM Tris-HCl (pH 7.4), 0.2 M NaCl, 1 mM dithiothreitol. The standards employed were: catalese (11.3 S), alcohol dehydrogenase (7.4 S), and ovalbumin (3.6 S).

Analysis of Kinetic Data—The kinetics of dead-end inhibition conformed to the following relationship expressed in the nomenclature of Cleland (23):

$$
\frac{1}{v} = \left(1 + \frac{I}{K_i}\right) \frac{1}{V} + \left(1 + \frac{I}{K_s}\right) \frac{K_s}{V} S
$$

Kᵢ and Kₛ are inhibitor constants, and Kᵥ and V are apparent kinetic constants. While I and S are concentrations of inhibitor and varied substrate and v represents the initial velocity. The concentration of one substrate was fixed in these studies; v was plotted against S at several fixed levels of inhibitor. The data were reduced by least squares analysis, and in all cases, correlation coefficients were 0.99 or greater. Either the slopes or 1/v intercepts derived from least squares analysis varied with I, depending on whether inhibition was competitive, noncompetitive, or uncompetitive. Replots of these slopes and intercepts against I were used to determine Kᵢ and Kₛ.

RESULTS

Inhibition of Glycerol-P Dehydrogenase by Palmityl-CoA—The inhibition of homogeneous glycerol-P dehydrogenase by palmityl-CoA is shown in Fig. 1. When glycerol-P dehydrogenase activity was assayed at nearly saturating levels of both substrates, NADPH (0.1 mM) and dihydroxyacetone-P (1.0 mM), the presence of 1.0 μM palmityl-CoA caused 50% inhibition of enzyme activity. With various enzyme preparations and solutions of palmityl-CoA, the concentration of palmityl-CoA required for 50% inhibition varied from 0.8 to 1.5 μM. Complete inhibition occurred at a palmityl-CoA concentration greater than 10 μM. The concentration dependency of the palmityl-CoA inhibition appeared rectilinear and was not cooperative. The inhibition by palmityl-CoA was

![Fig. 1. Inhibition of glycerol-P dehydrogenase by palmityl-CoA](http://www.jbc.org/)

![Fig. 2. Kinetics of palmityl-CoA inhibition. A, the initial rates of dihydroxyacetone-P reduction catalyzed by 35 ng of enzyme were determined as dihydroxyacetone-P was varied and NADPH was fixed at 0.1 mM. Several concentrations of palmityl-CoA were added: 0, □; 1 μM, □; 2 μM, □; and 4 μM, □. The data are presented as double reciprocal plots. B, the intercepts (●●●) and slopes (▲▲▲) of the plots in A are replotted against palmityl-CoA concentration in order to derive the inhibition constants, Kᵢ and Kₛ, as described under "Experimental Procedures." C, experiments were conducted and data were plotted as described under A, but NADPH was varied and dihydroxyacetone-P was fixed at 0.4 mM. D, replots of the slopes and intercepts of C were used to derive Kᵢ and Kₛ, as described under B.)
The presence of 20 PM palmitoyl-CoA, a 100-fold dilution into glycerol-P dehydrogenase was incubated at 4°C for 1 min in the presence of either substrate or with the enzyme. When glycerol-P dehydrogenase activity after 100-fold dilution into assay mixtures. Glycerol-P dehydrogenase activity is plotted along with the positions of markers: catalase, A; yeast alcohol dehydrogenase, B; and ovalbumin, C.

immediate. In the absence and presence of 1.0 µM palmitoyl-CoA, constant activity was observed for at least 8 min, and the initial rate was proportional to the amount of enzyme employed over the range 0 to 100 ng. The amount of inhibition observed was independent of whether the reaction was initiated by addition of either substrate or with the enzyme. When glycerol-P dehydrogenase was incubated at 4°C for 1 min in the presence of 20 µM palmitoyl-CoA, a 100-fold dilution into the assay mix resulted in activity equal to that observed when an equivalent amount of enzyme was assayed with 0.2 µM palmitoyl-CoA. Other preincubations of enzyme at 0 or 23°C for 5 min and with 2, 4, or 5 µM palmitoyl-CoA established that inhibition was fully reversible by 100-fold dilution.

The inhibition of glycerol-P dehydrogenase by several concentrations of palmitoyl-CoA was investigated by varying the concentration of one substrate and keeping the concentration of the other fixed. At saturating NADPH (0.1 mM), palmitoyl-CoA was a noncompetitive inhibitor with respect to dihydroxyacetone-P (Fig. 2A). The replots of the slopes or intercepts derived from these data were linear functions of palmitoyl-CoA concentration (Fig. 2B). The inhibitor constants, K_i and K_m, derived from these replots were 1.1 and 0.7 µM, respectively. At 0.4 mM dihydroxyacetone-P, palmitoyl-CoA was a noncompetitive inhibitor with respect to NADPH (Fig. 2C). The replots of the slopes or intercepts derived from these data were linear functions of palmitoyl-CoA concentration (Fig. 2D). The K_i and K_m derived from these replots were 1.5 µM and 0.2 µM, respectively. Since palmitoyl-CoA was a noncompetitive inhibitor with respect to either substrate, the inhibitor appears to be able to bind all enzyme-substrate intermediates and free enzyme. Previous kinetic data indicated the kinetic mechanism required NADPH to bind prior to dihydroxyacetone-P (6). Since the K_m with respect to NADPH was considerably lower than the other K_m and the two K_i values, the free enzyme appears to have higher affinity for palmitoyl-CoA than the enzyme-substrate intermediates.

Since several enzymes subject to inhibition by palmitoyl-CoA have been demonstrated to dissociate in its presence (8, 12, 13), the effect of palmitoyl-CoA upon the s_{20w} of glycerol-P dehydrogenase was investigated by centrifugation in sucrose gradients. The s_{20w} in the absence of palmitoyl-CoA was 4.2 S (Fig. 3), in agreement with the previously reported value (5). The presence of 20 µM palmitoyl-CoA did not alter the s_{20w} (Fig. 3). Hence, the addition of palmitoyl-CoA did not cause dissociation or aggregation of the enzyme. The recovery of glycerol-P dehydrogenase activity after 20 h at 4°C was approximately 75% from the gradient containing 20 µM palmitoyl-CoA and from the control. Irreversible inhibition of the enzyme by palmitoyl-CoA did not occur. The similar recoveries of activity indicate that palmitoyl-CoA did not produce an inactive species with an altered sedimentation coefficient.
Inhibition of Glycerol-P Dehydrogenase by Other Acyl-CoA Thioesters and Other Amphiphilic Molecules—The effect of other long chain acyl-CoA thioesters on glycerol-P dehydrogenase is shown in Fig. 4. C₁₂-CoA, C₁₄-CoA, C₁₆-CoA, and C₁₈-CoA caused substantial inhibition at levels less than 5 μM. Glycerol-P dehydrogenase activity was quantitatively inhibited at 20 μM concentrations of each of these acyl-CoA derivatives except C₁₂-CoA. In all cases, the concentration dependence of the inhibition was noncooperative and appeared rectilinear. The potency of the long chain acyl-CoA thioesters employed appeared to be inversely related to their critical micellar concentrations (Table I). This generalization was also valid for palmityl-CoA (Fig. 1). When the concentration dependence of the inhibition of palmityl-CoA inhibition was investigated in assays containing 10% methanol or 0.2 mM potassium phosphate (pH 7.4) and 10 mM potassium phosphate (pH 7.4), the concentration dependence of palmityl-CoA inhibition was essentially unchanged (Fig. 1; Table I). When assays were conducted in buffers of lower ionic strength, 10 mM Tris-HCl (pH 7.4) and 10 mM potassium phosphate (pH 7.4), the concentration dependence of palmityl-CoA inhibition was unaltered.

CoA, C₂-CoA, C₄-CoA, C₆-CoA, and C₈-CoA did not inhibit the glycerol-P dehydrogenase at concentrations up to 20 μM, while 20 μM C₁₀-CoA inhibited 30%. The combination of 20 μM CoA and 20 μM palmitate was not inhibitory.

Other amphiphilic molecules were tested to define the specificity of the inhibition by acyl-CoA thioesters. The effect of palmitoyl-ACP was investigated, because it is a principal product of de novo fatty acid biosynthesis in E. coli (26) and serves as an acyl donor in vitro for the biosynthesis of phospholipids (20, 26). Neither palmitoyl-ACP nor ACP at concentrations up to 50 μM (Table I) had any effect on glycerol-P dehydrogenase activity. Palmitate produced detectable inhibition at levels greater than 50 μM (Fig. 5); 200 μM palmitate caused 50% inhibition. The anionic detergents, deoxycholate and dodecyl sulfate, were potent inhibitors of glycerol-P dehydrogenase activity (Fig. 5). Fifty percent inhibition occurred at 20 μM dodecyl sulfate and at 600 μM deoxycholate while nearly total inhibition occurred at 45 μM and 4 mM, respectively. When enzyme was preincubated at 4 or 23°C for 2 min in the presence of 45 μM dodecyl sulfate, greater than 95% of the glycerol-P activity was recovered after a 100-fold dilution. Palmitoyl-carnitine inhibited glycerol-P dehydrogenase activity 50% at 30 μM (Fig. 5). Substantial inhibition by deoxycholate and dodecyl sulfate occurred below the reported critical micellar concentrations of these detergents (Table I).

This was not the case for nonionic detergents like Triton X-100 (Fig. 5), Tween-20, or Tween-80 (Table I).

Effect of Phospholipid Vesicles on Palmitoyl-CoA Inhibition of Glycerol-P Dehydrogenase—Since long chain acyl-CoA compounds associate spontaneously with membranes or phospholipid vesicles in vitro (27, 28), significant amounts of these compounds would be expected to be associated with membranes in vivo. In order to simulate the conditions likely to exist in vivo, the effect of phospholipid vesicles on the inhibition of glycerol-P dehydrogenase by palmitoyl-CoA was examined. The addition of phospholipid vesicles prepared from E. coli phospholipids neither stimulated nor inhibited glycerol-P dehydrogenase activity when added up to 100 μM. At 2 μM palmitoyl-CoA, glycerol-P dehydrogenase activity was inhibited 67%. The addition of phospholipid vesicles lessened the inhibition by 2 μM palmitoyl-CoA (Fig. 6A). The addition of 8 μM phospholipid lessened this inhibition by 50%, and 100 μM completely relieved the inhibition by 2 μM.
and genetic data indicate that glycerol-P synthesis is regulated by palmitoyl-CoA. At palmitoyl-CoA levels of 10 and 20 μM, 50% of the enzyme activity (data not shown). Phospholipid vesicles associated with palmitoyl-CoA were a less potent inhibitor of glycerol-P dehydrogenase than palmitoyl-CoA. The concentration dependency of the inhibitory effect was undertaken. Unfortunately, both of these inhibitors, glycerol-P and palmitoyl-CoA, appeared to act independently.

**DISCUSSION**

While the mechanisms which regulate the biosynthesis of membrane phospholipids in Escherichia coli remain incompletely understood, significant progress has been made recently by combined biochemical, genetic, and physiological approaches (29). Such investigations have established the biosynthetic role of the glycerol-P dehydrogenase (2) and the physiological significance of feedback inhibition by glycerol-P (4-9). The objective of the present investigations was to evaluate by kinetic studies of homogeneous glycerol-P dehydrogenase whether palmitoyl-CoA, another biosynthetic precursor of phospholipid, may function as a modulator of glycerol-P synthesis. Recent investigations have suggested that palmitoyl-CoA may regulate the activities of several lipogenic enzymes in both microbial (13) and mammalian systems (9-11). At present, the physiological significance of palmitoyl-CoA inhibition has not been established for any of the enzymes investigated.

Homogeneous glycerol-P dehydrogenase is potently inhibited by palmitoyl-CoA and other long chain acyl-CoA thioesters (Figs. 1 and 4). This demonstrates that the inhibition, noted by previous workers for activity in crude extracts (7) and in partially purified preparations (3), is an inherent property of the enzyme. Long chain fatty acyl-CoAs by virtue of their inherent detergent properties have the potential to perturb enzyme structure nonspecifically. The general mechanism of inhibition by detergents occurs through cooperative binding at concentrations equal to or greater than the critical micellar concentration. Since the concentrations of acyl-CoA thioesters which cause significant inhibition of glycerol-P dehydrogenase activity are less than their respective critical micellar concentrations (Table I), inhibition may be mediated by specific binding. The concentration dependencies of inhibition by all acyl-CoA thioesters tested were not cooperative (Figs. 1 and 4), when both NADPH and dihydroxyacetone-P were saturating. The conclusion that palmitoyl-CoA inhibition was noncooperative can be extended to include various concentrations of NADPH and dihydroxyacetone-P (Fig. 2). The findings that the inhibition by palmitoyl-CoA is fully reversible and consistent with physiological regulation by long chain acyl-CoA thioesters. These data indicate that inhibition by palmitoyl-CoA and other acyl-CoA thioesters may be mediated by specific binding. The binding of palmitoyl-CoA to glycerol-P dehydrogenase was not investigated due to the difficulty in obtaining a sufficient quantity of purified enzyme. The binding of a few moles of palmitoyl-CoA per mol of enzyme in the range of concentrations at which inhibition occurs might suggest interaction with a specific, regulatory site, but the binding of many moles per mol of enzyme could not rule out the presence of such a site (9).

### Note

*The binding of palmitoyl-CoA to glycerol-P dehydrogenase was not investigated due to the difficulty in obtaining a sufficient quantity of purified enzyme. The binding of a few moles of palmitoyl-CoA per mol of enzyme in the range of concentrations at which inhibition occurs might suggest interaction with a specific, regulatory site, but the binding of many moles per mol of enzyme could not rule out the presence of such a site.*
ple, function physiologically to regulate glycerol-P dehydro-
genase activity by a mechanism independent of that afforded
by glycerol-P (Fig. 7). Any physiological correlate for this
inhibition must take into account that the presence of phos-
pholipid vesicles lessened the inhibition by palmitoyl-CoA
(Fig. 6) and that palmitoyl-ACP was not inhibitory at levels
up to 50 μM.

If long chain acyl-CoA thioesters are to function as physi-
ological regulators of glycerol-P synthesis in E. coli, their
levels would be expected to vary with physiological conditions,
as noted in mammalian tissues (30). Unfortunately, the levels
of acyl-CoA thioesters in Escherichia coli have not been
quantitated under any conditions. Acyl-CoA thioesters are
intermediates in the uptake of exogenous fatty acids (31), can
undergo ω oxidation (32), and can serve as acyl donors in the
biosynthesis phospholipids (31). For strains growing in the
presence of fatty acids, the inhibition of glycerol-P synthesis
by long chain acyl-CoA derivatives might serve to limit phos-
pholipid synthesis. In addition, long chain acyl-CoA thioesters
may regulate de novo fatty acid biosynthesis by inhibition of
acyetyl-CoA carboxylase (3).

The physiological significance in E. coli of the inhibition of
the biosynthetic glycerol-P dehydrogenase by acyl-CoA
thioesters remains to be demonstrated. The significance of
this inhibition might be established, if mutations in the struc-
tural gene of the glycerol-P dehydrogenase altered the sensi-
tivity of the enzyme to acyl-CoA thioesters and were expressed
phenotypically.

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