Effects of High-Fat Diet and Fasting on Levels of Acyl-Coenzyme A Binding Protein in Liver, Kidney, and Heart of Rat

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Acyl-coenzyme A (CoA) binding protein (ACBP) is a 10-kd protein that binds acyl-CoA moieties and stimulates medium-chain fatty acid synthesis by goat mammary gland fatty acid synthetase. Its exact role in intermediary lipid metabolism has not been fully elucidated. It is hypothesized that ACBP is directly involved in the metabolism of lipid. In the present study, purified rat liver ACBP was used to generate a polyclonal antisera for radioimmunoassay of ACBP in tissue specimens isolated from fasted rats and rats fed normal rat chow and a high-fat diet. In addition, purified ACBP was used to examine its effect on the activity of mitochondrial outer membrane (OM) carnitine palmitoyltransferase (CPT_0). Fasting for 24 hours significantly decreased tissue levels of ACBP in the liver (69.0 ± 7.2 *v* 46.7 ± 5.0 pg/ng DNA), whereas feeding of a high-fat diet for 48 hours caused ACBP levels to increase (69.0 ± 7.2 *v* 103.9 ± 18.0). Hepatic levels of this protein continued to increase and remained elevated with prolonged exposure to the high-fat diet (28 days). A similar pattern of change was observed in the kidney, but the magnitude of change was less. Heart ACBP did not respond acutely to the high-fat diet, but did increase after prolonged exposure (28 days). Fasting had no effect on ACBP levels in kidney and heart. Addition of ACBP to an in vitro assay system significantly increased the activity of CPT_0 (from 5.2 ± 0.8 to 72.1 ± 5.3 nmol palmitoylcarnitine formed · min^{-1} · mg^{-1} protein) when measured under inhibiting concentrations of palmitoyl-CoA (40 μmol/L). These data indicate that liver ACBP is capable of responding acutely to changes in diet, suggesting that this protein may serve a regulatory role in cellular lipid metabolism.

AN ACYL-COENZYME A (CoA) binding protein (ACBP) was isolated and purified from bovine liver in 1987 by Mogensen et al.[1] This 10-kd protein stimulated the synthesis of medium-chain fatty acids by goat mammary gland fatty acid synthetase and was subsequently shown to be coded for by the same gene, and therefore shown to have an identical amino acid sequence to diazepam-binding inhibitor[2] or endozepine.[3] Earlier studies with brain isolates of diazepam-binding inhibitor indicated that this protein may function as a neurotransmitter through interaction at a benzodiazepine-binding site on the gamma-aminobutyric acid receptor complex,[2] regulate insulin release,[4] and mediate corticotropin-dependent adrenal steroidogenesis.[5] Later studies suggested that ACBP may function as a housekeeping protein by maintaining intracellular acyl-CoA pool size and transporting acyl-CoAs within different subcellular membranes.[6] In support of this concept, ACBP has recently been shown to stimulate the synthesis of long-chain acyl-CoA esters by reversing acyl-CoA–mediated inhibition of mitochondrial long-chain acyl-CoA synthetase.[7]

Moreover, the promoter region of the yeast ACBP gene contains two essential elements—one is the inositol-choline regulatory element, which is necessary for expression of the yeast fatty acid synthase genes, FAS1 and FAS2. These inositol-choline regulatory element motifs are present in the upstream regions of all known genes involved in fatty acid biosynthesis.[8] The other element contains sequences corresponding to motifs known as the β-oxidation boxes. These boxes play an essential role in regulating genes that are involved in β-oxidation of fatty acids.[9] The presence of such elements in the promoter region of the yeast ACBP gene supports the notion that ACBP plays an important role in the regulation of lipid metabolism. If ACBP is directly involved in synthesis and oxidation of lipid, tissue levels of this protein would be expected to respond to changes in nutritional status such as fasting and a high-fat diet. According to Hansen et al,[10] there was a threefold to 10-fold induction of ACBP production in 3T3-L1 fibroblasts during differentiation of these cells from preadipocytes to fully developed lipid-producing adipocytes.

In the present studies, we therefore measured the effect of fasting and a high-fat diet on ACBP in liver, kidney, and heart of rat and the effect of this protein on mitochondrial outer membrane (OM) carnitine palmitoyltransferase (CPT_0), the rate-limiting step in mitochondrial fatty acid oxidation. These studies were undertaken to elucidate further the metabolic role of ACBP in intermediary lipid metabolism.

MATERIALS AND METHODS

Animals

Male Wistar rats (200 to 250 g) were used. Seven groups of animals (n = 6 per group) were studied. Two groups were fasted for 24 and 48 hours, whereas the other five groups were fed a high-fat diet for 12 hours and 1, 2, 7, and 28 days. Control animals received Purina rat chow (St Louis, MO) as the diet, which contained approximately 4% fat. The high-fat diet (soybean oil) was formulated to provide 22% of total calories as fat. The prepared diet was supplemented with essential nutrients and vitamins. Animals were housed in individual metabolic cages, allowed free access to water and fresh diet (except when fasted), and maintained on a 12-hour light-dark cycle throughout the study.

At death, serum was collected and tissues (liver, heart, kidney, and skeletal muscle) were immediately harvested into liquid nitrogen and stored at −70°C until analyzed.

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Submitted September 6, 1994; accepted November 28, 1994.

Supported by a research grant from the British Columbia Health Research Foundation.

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0026-0495/95/4409-0014$03.00/0

Analyses

Serum levels of free fatty acids were determined as described previously.[11] Net serum triglycerides were determined by routine methods[12] on a Beckman CX7 analyzer (Fullerton, CA). DNA was determined according to the method reported by Labarca and Paigen.[13]

Preparation of Subcellular Fractions From Rat Liver

Mitochondria were isolated by gradient centrifugation according to the procedure reported by Hovius et al.[14] OM vesicles (OMV) were prepared according to the method reported by Murthy and Pande.[15]

Isolation, Purification, and Separation of ACBP From Fatty Acid–Binding Protein

Fatty acid–binding protein (FABP) and ACBP were isolated from rat liver homogenates as described previously,[16,17] with modifications.[18,19] Briefly, the procedure involved acid and heat treatments, and ultracentrifugation followed by Sephadex G-100 and G-50 gel filtrations. G-50 isolates were lyophilized and then dissolved in 0.1% (vol/vol) trifluoroacetic acid in water (buffer A) and centrifuged at 10,000 × *g* for 5 minutes at 4°C before fractionation. The isolates were fractionated by reverse-phase high-performance liquid chromatography using a dual-gradient system and a semi-prep Delta Pak C18-100Å column (7.8 mm × 30 cm, 15 μm, spherical; Waters, Tokyo, Japan). The column was first equilibrated in 20% (vol/vol) buffer B (0.1% vol/vol trifluoroacetic acid in 50% vol/vol isopropanol) in buffer A with a flow rate of 4.8 mL/min. ACBP and FABP were resolved using the following gradient of buffer B in buffer A: linear gradient 20% to 40%, 10 minutes; linear gradient 40% to 60%, 10 minutes; linear gradient 60% to 70%, 10 minutes; linear gradient 70% to 80%, 10 minutes; isocratic 80%, 5 minutes; linear gradient 80% to 100%, 10 minutes; linear gradient 100% to 20%, 5 minutes. ACBP and FABP peaks were eluted at 20 and 31 minutes, respectively.[19] Purity of these fractions was confirmed by urea sodium dodecyl sulfate–polyacrylamide gel electrophoresis.[19]

CPT_o Assay

Assay of CPT_0 in the forward direction (palmitoyl-CoA to palmitoylcarnitine) was performed at 30°C in 125 μL assay volume with 5 mmol/L carnitine and 40 or 10 μmol/L [1-^{14}C]palmitoyl-CoA as substrate as reported previously.[20,21] Where present, malonyl-CoA, a competitive inhibitor of CPT_o, was 0 to 100 μmol/L; other details are given in figure legends. [1-^{14}C]palmitoyl-CoA was synthesized and purified according to previously reported methods.[22]

Preparation of Antibody

ACBP (10 kd) was coupled to thyroglobulin and injected into rabbits (200 μg ACBP with Freund's complete adjuvant). Subsequent injections consisted of 100 μg ACBP per rabbit plus Freund's incomplete adjuvant. After the fourth booster, the antisera titer had reached approximately 12,500. Cross-reactivity studies using this antisera indicated negligible cross-reactivity with purified rat liver FABP, bovine serum albumin, or rat serum.

Development of Radioimmunoassay for ACBP

The radioimmunoassay buffer consisted of 0.1% bovine serum albumin, 0.1% Triton X-100, 0.3% NaCl, 0.1 mol/L potassium phosphate buffer, pH 7.4, and 0.01% NaN_3. ^{125}I-ACBP was prepared according to the method reported by Greenwood and Hunter.[23] Standards and samples were incubated overnight at 4°C in the presence of antisera (1:10,000 dilution) and tracer. Free and bound fractions were separated using goat antirabbit IgG in 1% serum. After addition of 5% polyethylene glycol (average molecular weight, 8,000), the tubes were centrifuged, the supernatant was aspirated, and radioactivity in the precipitate was determined using a gamma counter equipped with a data-reduction station (Iso-Data, 20/20 Series, Rolling Meadows, IL). Intraassay and interassay coefficients of variation for this assay were 2% and 8.2%, respectively, at an ACBP concentration of 3.33 ng/mL. A typical radioimmunoassay standard curve using this antisera is shown in Fig 1. This assay was subsequently used to measure tissue levels of ACBP in liver, heart, and kidney, together with its response to dietary manipulations.

Statistical Analysis

Student's *t* test for unpaired data was used. Differences were considered significant at *P* less than .05 for a two-tailed test.

RESULTS

ACBP and FABP were purified as outlined earlier. Separation was achieved in a single assay as described earlier.[19] When assayed on urea sodium dodecyl sulfate–polyacrylamide gel electrophoresis, two distinct bands with molecular weights of approximately 10 and 14.5 kd, respectively, were identified as demonstrated elsewhere.[19] It is evident that ACBP and FABP are two distinct proteins with markedly different binding affinities to long-chain acyl-CoA.[19,24] Our purified ACBP and FABP demonstrated these differing binding characteristics. The binding of palmitoyl-CoA to FABP in our hands was so low that a meaningful binding constant could not be calculated. A similar situation occurred with the binding of oleic acid to ACBP, whereas the binding of palmitoyl-CoA to ACBP was high (data not shown).

Effect of ACBP on CPT_o

Studies were undertaken to extend the observations made by Rasmussen et al[7] that suggest ACBP can act as an intracellular acyl-CoA transporter for enzymes that use long-chain acyl-CoAs as substrates and protect acyl-CoA pools from the hydrolyzing action of intracellular acyl-CoA hydrolase. Thus, the effects of ACBP on CPT_o activity were studied as outlined earlier. As shown in the dose-response curve (Fig 2), activity of CPT_o, the malonyl-CoA–inhibitable enzyme of mitochondrial OM, increased several-fold with the addition of ACBP to the assay system.

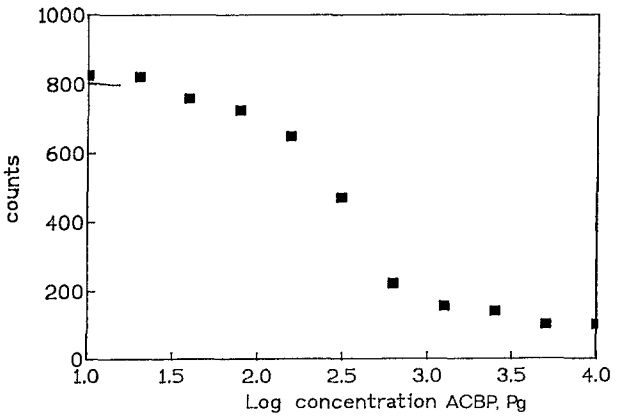

Fig 1. Competitive standard curve—ACBP by ACBP antibody.

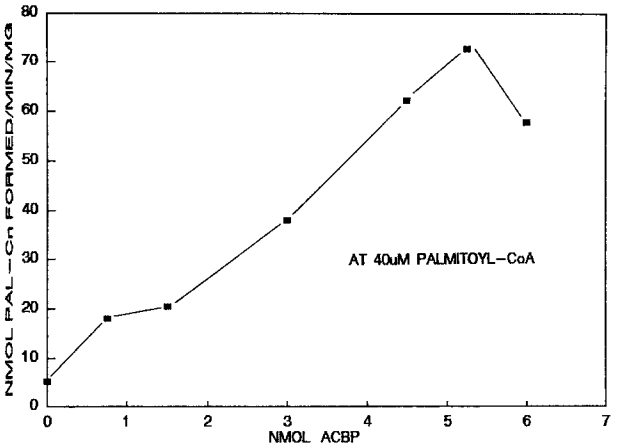

Fig 2. Effect of rat liver ACBP on CPT_o activity. Assays were performed in 125 μL of medium containing 220 mmol/L sucrose, 40 mmol/L KCl, 10 mmol/L Tris hydrochloride, pH 7.4, 1 mmol/L EGTA, 5 mmol/L carnitine, 40 μmol/L [1-^{14}C]palmitoyl-CoA, and purified rat liver ACBP as indicated. Reactions were started with 1 μg OMV protein as a source of CPT_o. After a 3-minute incubation at 30°C, reactions were stopped by adding methanol (80% vol/vol). [1-^{14}C]palmitoylcarnitine (PAL-Cn) was separated and measured as described previously.[18] Results are representative of four experiments.

Although palmitoyl-CoA is the preferred substrate for CPT_o, higher concentrations of palmitoyl-CoA (40 μmol/L), inhibited this activity.[20,25] This inhibition is reversed in that CPT_o activity is increased from 5.2 ± 0.8 to 72.1 ± 5.3 nmol palmitoylcarnitine formed · min^{-1} · mg^{-1} protein (n = 3) by the addition of purified ACBP (Fig 2). When palmitoyl-CoA in the assay system was at subinhibitory concentrations (10 μmol/L), addition of ACBP decreased activity of CPT_o and increased its sensitivity to malonyl-CoA inhibition (Fig 3). Typically, at this concentration of

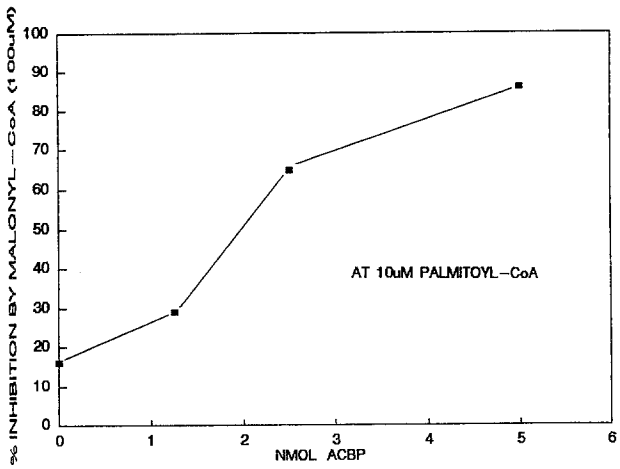

Fig 3. Effect of rat liver ACBP on malonyl-CoA inhibition of CPT_o. Assays were performed in 125 μL of medium containing 220 mmol/L sucrose, 40 mmol/L KCl, 10 mmol/L Tris hydrochloride, pH 7.4, 1 mmol/L EGTA, 5 mmol/L carnitine, 10 μmol/L [1-^{14}C]palmitoyl-CoA, and purified rat liver ACBP as indicated. Where present, malonyl-CoA was 100 μmol/L. Reactions were started with 1 μg OMV protein as a source of CPT_o. After a 3-minute incubation at 30°C, reactions were stopped by adding methanol (80% vol/vol). [1-^{14}C]palmitoylcarnitine was separated and measured as described previously.[18] Results are representative of three experiments.

Table 1. Effect of Diet on Plasma Levels of FFA and Triglycerides in the Rat

Diet	Days of Treatment	FFA (mmol/L)	Triglycerides (mmol/L)
Control	—	0.86 ± 0.16	0.68 ± 0.13
Fasting	1	—	0.39 ± 0.08*
	2	1.53 ± 0.18*	Below detection limit
High-fat	1	—	2.77 ± 0.55*
	2	—	1.83 ± 0.44*
	7	1.13 ± 0.09*	1.71 ± 0.25*
	28	1.20 ± 0.05*	1.68 ± 0.30*

NOTE. Mean ± SD (n = 6).
Abbreviation: FFA, free fatty acids.
*$P < .05$, diet *v* controls.

palmitoyl-CoA substrate, malonyl-CoA does not inhibit CPT_o.[25]

Effect of Fasting and High-Fat Diet on ACBP

Animals tolerated the diets well, with serum lipid levels responding as predicted, which confirmed the nutritional state of the animals (Table 1). Fasting for 24 and 48 hours significantly decreased liver levels of ACBP (69.0 to 46.7 and 66.5 to 39.5 pg/ng DNA, respectively). In contrast, fasting had no effect on ACBP levels in heart and kidney (Table 2).

On the other hand, feeding a high-fat diet significantly increased ACBP levels in the liver (Table 3). This increase required 48 hours of diet exposure to become manifest. The levels remained elevated with prolonged exposure to the diet (28 days). ACBP levels in kidney and heart also increased significantly on the high-fat diet, but did so only after prolonged exposure to the diet. This increase was first evident in the kidney at 7 days, whereas in the heart it required 28 days of high-fat feeding (Table 3).

DISCUSSION

Palmitoyl-CoA is a substrate and an inhibitor of CPT_o.[26] The studies reported here support the concept that ACBP acts both to sequester long-chain acyl-CoAs and to facilitate their transport to the catalytic site of selected enzymes as suggested by Rasmussen et al.[7] Under the conditions used in our study, palmitoyl-CoA should inhibit activity of CPT_o.[25] Instead, addition of equimolar amounts of ACBP with palmitoyl-CoA to the incubation media increased activity of CPT_o several-fold (Fig 2). Furthermore, addition of a molar excess of ACBP decreased activity of CPT_o (Fig 2). This may be due to substrate limitation secondary to the

Table 2. Effect of Fasting on Tissue Levels of ACBP (pg/ng DNA)

Diet	Tissue Levels		
	Liver	Kidney	Heart
24-hour			
Control	69.0 ± 7.2	—	—
Fasting	46.7 ± 5.0*		
48-hour			
Control	66.5 ± 13.1	25.0 ± 4.4	12.5 ± 1.6
Fasting	39.5 ± 6.2*	21.7 ± 8.7	11.2 ± 1.8

NOTE. Mean ± SD (n = 6).
*$P < .05$, fasting *v* control.

Table 3. Effect of High-Fat Diet on Tissue Levels of ACBP (pg/ng DNA)

Days	Liver		Kidney		Heart	
	Control	High-Fat	Control	High-Fat	Control	High-Fat
1	69.0 ± 7.2	69.9 ± 10.4	—	—	—	—
2	69.0 ± 7.2	103.9 ± 18.0*	27.8 ± 9.8	27.1 ± 11.0	—	—
7	66.5 ± 13.1	105.7 ± 24.4*	25.0 ± 4.4	31.9 ± 5.0*	12.5 ± 1.6	11.4 ± 1.1
28	72.4 ± 8.6	120.5 ± 11.7*	30.8 ± 10.0	36.3 ± 6.7*	14.8 ± 1.2	17.0 ± 0.8*

NOTE. Mean ± SD (n = 6).

*$P < .05$, high-fat diet *v* control.

sequestration of palmitoyl-CoA (substrate) by ACBP. The mechanism underlying this increase in activity was not determined. Conceivably, though, it could have been due to binding of excess palmitoyl-CoA to ACBP, thereby relieving inhibition of CPT_o while at the same time delivering optimal amounts of palmitoyl-CoA to CPT_o. This explanation is more likely in that the addition of ACBP at lower substrate concentrations (10 μmol/L) caused activity of CPT_o to decrease, while at the same time it increased sensitivity of this enzyme to malonyl-CoA inhibition (Fig 3). Malonyl-CoA is a competitive inhibitor of CPT_o, and therefore, whether the binding of limited amounts of substrate to ACBP facilitated malonyl-CoA inhibition remains to be determined. The mechanism by which ACBP delivers acyl-CoA to the catalytic site of CPT_o, the site located on the inner side of the OM,[15] remains to be elucidated. It is unlikely that acyl-CoA–bound ACBP can pass through the OM.[27] There is a possibility that the OM might contain a specific acyl-CoA translocator that could deliver acyl-CoA from the ACBP complex directly to the catalytic site of CPT_o. The existence of such a translocator in the OM was first considered by Murthy and Pande[15] and subsequently supported by others.[27,28]

Since ACBP concentration has the potential to regulate effects of palmitoyl-CoA on the activity of CPT_o, we elected to study ACBP levels in animals in whom fatty acid metabolism had been perturbed by fasting and feeding of a high-fat diet. Our data clearly indicate that tissue levels of ACBP can respond to changes in diet (Tables 2 and 3). We surprisingly observed a decrease in liver levels of ACBP within 24 hours of fasting (Table 2). We repeated this study to confirm this observation. This was further confirmed in recent studies where fasting (24 and 48 hours) decreased ACBP levels while it increased the concentration of long-chain acyl-CoA in rat liver.[29] The time course of this response suggests to us that ACBP may play a broader regulatory role in the cell other than general housekeeping.[6] Conceivably, in the liver there may be two pools of ACBP—one of which responds rapidly to acute changes in diet, and the other which responds more slowly with long-term high-fat diet exposure. The latter would be expected to function in a housekeeping role, whereas the former would be more important in the short-term buffering of long-chain acyl-CoA moieties and immediate regulation of intermediary metabolism. However, to confirm this postulation, further study is needed. Feeding of a high-fat diet significantly increases the flux of long-chain fatty acids into the liver. Within 2 days of exposure, liver levels of ACBP had increased significantly (Table 3). Levels of this protein also increased in the kidney and heart, but only after prolonged exposure to the diet. It is also reported that rat liver FABP was increased to a similar extent as ACBP by feeding a high-fat diet.[30] The exact mechanism by which fasting and the high-fat diet changed tissue levels of ACBP was not determined.

ACBP might function to solubilize acyl-CoAs being formed at one membrane surface, so that they can be redistributed from the site of synthesis to the site of utilization at another membrane surface.[27] More specifically, as more and more free fatty acids are activated to acyl-CoA, they could well accumulate at the outer surface of the OM (eg, with feeding of a high-fat diet). Under these circumstances, excess acyl-CoAs could be solubilized and redistributed by ACBP from the OM surface to the endoplasmic reticulum for synthesis of triglyceride. ACBP levels would therefore be expected to correlate positively with the synthesis rate of triglycerides in the liver under conditions of positive energy balance and high-fat diet, and to correlate inversely under conditions of negative energy balance and fasting. In fasting, there is an increased demand for mitochondrial fatty acid oxidation and an increased requirement for acyl-CoA esters. A decrease in ACBP concentration under these conditions would be advantageous in that it would indirectly increase the availability of free long-chain acyl-CoAs, optimizing the rate of β-oxidation.

The inductive lag time on the high-fat diet that was observed in kidney and heart may reflect a protective response on the part of these tissues with prolonged exposure to a high-fat diet. With prolonged exposure to high fat levels, these tissues could metabolically become overwhelmed by the influx of fatty acids. As fatty acyl-CoAs accumulate in the cell, they could eventually become toxic to the cell. ACBP abundancy would facilitate solubilization of these species and serve to protect the cell against their toxic effects.

In summary, we have shown that ACBP increased the activity of one of the important enzymes of lipid metabolism, CPT_o. Cellular ACBP levels increased with a high-fat diet and decreased with fasting. These changes occurred within a short period. Further studies are necessary to elucidate the reciprocal effects of fasting and a high-fat diet on ACBP concentration, as well as the regulatory role of ACBP in intermediary lipid metabolism.

ACKNOWLEDGMENT

We gratefully acknowledge the technical help of Dr Wong and his laboratory staff in iodination of ACBP and generation of ACBP antisera.

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