UTILIZATION OF MEDIUM-CHAIN TRIGLYCERIDES
BY NEONATAL PIGLETS: I. EFFECTS ON MILK
CONSUMPTION AND BODY FUEL UTILIZATION

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ABSTRACT

Two experiments were conducted utilizing neonatal piglets. In the first experiment, 18 piglets were used to determine the effect of an oral supplement of 0, 12 or 24 ml of a medium-chain triglyceride (MCT) product on subsequent milk consumption. Results from the weigh-suckle-weigh experiment showed that force-feeding 24 ml of the MCT decreased (P < .05) milk intake but 12 ml did not. In the second experiment, two trials (each with 24 piglets) were used to investigate the effect of 12 ml of MCT or 12 ml of MCT plus .6 mmol of L-carnitine on the concentration of blood glucose, ammonium N and urea N at 0, 12 or 24 h and liver and biceps femoris glycogen at 24 h post-treatment. Blood urea N decreased (P < .05) in piglets receiving the MCT. Blood ammonium N and glucose concentrations were not different (P > .10) among treatments. In Trial 1, the predicted loss of liver glycogen was less (P < .05) in pigs given the MCT treatments, but this response was not repeated in the second trial. In general, supplemental carnitine provided no added benefit over the MCT treatment alone. The results from this study indicate that MCT is utilized as a fuel by the newborn piglet and that its use may spare critical fuels, glycogen and protein, that were stored in the piglet prior to birth.

(Key Words: Newborn Animals, Piglets, Medium-Chain Triglyceride, Blood Urea, Energy Metabolism.)

Introduction

Of the pigs born alive, from 15 to 25% do not survive through weaning (Fahmy and Bernard, 1971; Bereskin et al., 1973; English and Smith, 1975). One of the factors that could contribute to this problem is low body fat (1 g/100 g body weight) at birth (Girard, 1981). Pettigrew (1981) stated that the "notable energy stores in the piglet at birth are glycogen in the liver and skeletal muscles, and fat in the adipose tissue." One important additional source of energy, protein, should be mentioned. Protein (i.e., amino acids) can be a fuel in starvation or in other instances in which energy intake is less than energy loss (Kinney et al., 1973). Little attention has been given to protein loss in neonatal pigs.

One strategy is to provide an exogenous energy source to the newborn piglet with the hope of decreasing the depletion of stored fuels such as liver and muscle glycogen and body protein. Our work with a special lipid (Steinman and Benevenga, 1985; Benevenga et al., 1986) and that of Pettigrew et al. (1986) focused on the immediate postpartum supplementation of the piglet. We utilized triglycerides containing medium-chain-length (C6 to C12) fatty acids because of their rapid digestion and metabolism (Bach and Babayan, 1982).

Direct supplementation of the newborn piglet with this energy-rich supplement has at least two advantages. First, only a small amount of fat is required because the sow is
not supplemented, and second, only the small, weak piglets that could benefit from the additional energy need to be treated. We report here the use of a medium-chain triglyceride (MCT) product that is readily available commercially.

**Materials and Methods**

Experiments were conducted to determine the effect of an oral supplement of a MCT product\(^4\) on voluntary milk consumption, blood metabolites (glucose, ammonium N and urea N) and concentration of glycogen in the liver and the biceps femoris muscle in neonatal piglets.

**Experiment 1**

This experiment was designed to determine whether the level of MCT, required to meet the piglets' energy need, would suppress voluntary milk consumption. Milk consumption was of concern because the piglet obtains its passive immunity from colostrum within the first 12 h of life (Payne and Marsh, 1962; Bourne, 1969).

Eighteen crossbred (Yorkshire x Hampshire x Duroc) piglets 24 to 48 h old from two litters were assigned randomly within litter in blocks of three to receive a 0-, 12- or 24-ml oral supplement of the MCT product. These levels were based on a basal metabolic rate of 100 kcal/(kg·d) (Mount, 1968). A 12-ml supplement should provide 94 kcal GE assuming a combustion value of 8.3 kcal/g and a specific gravity of .943 g/ml. The MCT product is a reconstituted coconut oil. The triglyceride is liquid at room temperature and has a fatty acid composition of 1 to 2% C 6:0, 65 to 75% C 8:0, 25 to 35% C 10:0 and 1 to 2% C 12:0 (personal communication, P. J. Reilly, Capital City Products, Columbus, OH). A 405-mm x 4.8-mm o.d. x 3.2-mm i.d. piece of flexible tygon tubing attached to a 30-cc syringe was used to force-feed the MCT. Piglets were held from behind the head, letting their bodies hang while the tubing was inserted until it was in the stomach. No difficulties in restraint, administration or acceptance of the product by the pig were encountered with this procedure.

Voluntary milk consumption was determined using the weigh-suckle-weigh procedure over a 10-h period. Rather than establish an artificial suckling pattern (i.e., 30 or 60 min), the litter was removed from the farrowing crate as a group immediately prior to suckling and each piglet was weighed individually to the nearest gram. The piglets were returned to the sow as a group and allowed to suckle. After the suckling bout was completed, the piglets were removed as a group and weighed individually. The difference between the initial and final weight of each piglet is assumed to equal the amount of milk consumed. This procedure was followed for each suckling bout over the 10-h experiment. Although this technique has been criticized for inaccuracies (Pettigrew et al., 1985), it was considered sufficiently accurate to detect a major difference in milk consumption.

**Experiment 2**

A total of 48 newborn crossbred piglets (Landrace x Large White x Duroc) were used in two trials to determine whether 12 ml of an oral supplement of the MCT product or the MCT product plus L-carnitine would provide sufficient fuel to decrease the use of glycogen and protein as sources of energy. The plan showing the treatment groups, sampling times and specific analyses is presented in Table 1. A level of 12 ml MCT was chosen because this level did not decrease voluntary milk consumption (Table 2). Carnitine was included in one treatment because some studies indicated that carnitine increased medium-chain fatty acid oxidation (Groot and Hulsmann, 1973; Shipp et al., 1982; Stanley et al., 1983), whereas others, based on enzyme distribution patterns, indicated that carnitine was not required for medium-chain fatty acid oxidation (McGarry and Foster, 1971, 1974). The level of carnitine (.6 mmol) was chosen based on estimates of colostrum intake and its carnitine content (Kerner et al., 1984).

Piglets were removed from the sow immediately after birth to prevent them from suckling. They were assigned randomly within blocks to one of the four treatments: control (killed at 0 h), starved (killed at 24 h), force-fed 12 ml MCT (killed at 24 h) or 12 ml MCT plus .6 mmol of L-carnitine (killed at 24 h). Piglets were kept in separate cages. The room was maintained at 20 to 30°C in Trial 1.

\(^4\)Captex 300, Capital City Products Company, Columbus, OH.
TABLE 1. EXPERIMENTAL PLAN FOR THE STUDY OF SUPPLEMENTATION OF NEWBORN PIGLETS WITH A MEDIUM-CHAIN TRIGLYCERIDE (TRIALS 1 AND 2)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Killed, h</th>
<th>Sample, h</th>
<th>Analysis</th>
<th>Samples, h</th>
<th>Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0</td>
<td>0</td>
<td>packed cell volume, glucose, ammonium N, urea N</td>
<td>0</td>
<td>glycogen</td>
</tr>
<tr>
<td>Starved</td>
<td>24</td>
<td>0,12,24</td>
<td>packed cell volume, glucose, ammonium N, urea N</td>
<td>24</td>
<td>glycogen</td>
</tr>
<tr>
<td>12 ml MCT</td>
<td>24</td>
<td>0,12,24</td>
<td>packed cell volume, glucose, ammonium N, urea N</td>
<td>24</td>
<td>glycogen</td>
</tr>
<tr>
<td>12 ml MCT+.6 mmol L-carnitine</td>
<td>24</td>
<td>0,12,24</td>
<td>packed cell volume, glucose, ammonium N, urea N</td>
<td>24</td>
<td>glycogen</td>
</tr>
</tbody>
</table>

Piglets were removed from the sow at birth and assigned randomly in groups of four to treatment groups. N = 6 per treatment for Trial 1 and Trial 2.

(winter) and at approximately 30°C in Trial 2 (spring) and water was available free choice. Blood samples were taken at 0 h, 12 h and 24 h. Blood was analyzed for packed cell volume, glucose, ammonium N and urea N. At 24 h the piglets were killed and the glycogen concentration in the liver and biceps femoris muscle was determined.

**Blood Sampling and Preparation.** Blood was collected from the heart with a 20-gauge, 3.8-cm needle fitted to a 5-ml heparinized syringe. Approximately 1 ml of blood was collected at 0 h from all pigs and at 12 and 24 h from the pigs killed at 24 h. The packed cell volume was determined with a Micro-Hematocrit centrifuge. For the determination of glucose, ammonium N and urea N, .25 ml of the heparinized blood was added to 2.5 ml of .16 M phosphate buffer (pH 7.0), vortexed and let stand for 10 min before addition of 1 ml of .6 N perchloric acid followed by 1.25 ml of .3 M potassium hydroxide. After centrifugation at 800 x g, the supernatant fluid was stored frozen. Blood glucose was determined with glucose oxidase (EC 1.1.3.4) type II5 (20.5 units/assay), peroxidase (EC 1.11.1.7) type II5 (.33 units/assay) and ABTS (2,2'-Azinobis [3-ethylbenzthiazolesulphonic acid])5 as the chromophore (Bergmeyer and Bernt, 1974). The standard curve (0 to .15 μmol) was linear. Heparin had no effect on the color yield. Recoveries ranged from 99 to 100%.

The sum of blood ammonium and urea N was determined in the neutralized supernatant fluid treated with jack bean urease (EC 3.5.1.5) type IX5, (130 units/assay) for 30 min at 37°C in a shaking waterbath. Nitrogen was determined utilizing the colorimetric method described by Chaney and Marbach (1962) using ammonium sulfate as the standard. Ammonium N was determined on the same sample but without pretreatment with urease. Urea N was calculated as the difference. The standard curve for ammonium N was linear from 0 to 2.62 μg N. Heparin did not affect the assay. Recoveries of urea N ranged from 98 to 100%.

**Tissue Glycogen Analysis.** Piglets were anesthetized by injecting 2 ml of 4.0% solution of thiamylal sodium5 into the heart. After the piglets were unconscious, the carotid and jugular vessels were severed. The liver and right-side biceps femoris muscle were removed, weighed to the nearest gram and frozen in liquid N. Samples were kept at -22°C

![Image](https://via.placeholder.com/150)

**TABLE 2. EFFECT OF A SINGLE ORAL SUPPLEMENT OF A MEDIUM-CHAIN TRIGLYCERIDE ON VOLUNTARY MILK CONSUMPTION OF NEONATAL PIGS OVER 10 HOURS**

<table>
<thead>
<tr>
<th>Item</th>
<th>Medium-chain triglyceride, ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>163 ± 46</td>
</tr>
<tr>
<td>12</td>
<td>167 ± 41</td>
</tr>
<tr>
<td>24</td>
<td>49 ± 26</td>
</tr>
</tbody>
</table>

Means ± SE. Each mean represents the total milk intake for each of six piglets per treatment over the 10-h period.

Milk consumption differed between 0 and 12 vs 24 ml medium-chain triglyceride levels (P < .05).
until they were prepared for analysis. The tissue samples were powdered in liquid N using a Waring Blender. One gram (liver) or .5 g (biceps femoris) of the powdered sample was homogenized in 20 ml of .15 N perchloric acid. The homogenate was centrifuged at 800 × g for 20 min and the supernatant fluid was diluted 1:6 and stored in the refrigerator. Release of glucose from glycogen was accomplished by adding .025 ml of the diluted supernatant fluid to .23 ml of an acetate buffer (2 M; pH 4.8)-amyloglucosidase (EC 3.2.1.3)5 (18 units/assay) mixture followed by incubation at 37°C for 30 min in a shaking water bath (Keppler and Decker, 1974). The glucose released from standard glycogen (Type IX)5 was determined using the glucose-oxidase assay described above. Free glucose in the tissue was estimated by incubating the sample without amyloglucosidase. This value was used to correct the glycogen estimate.

Statistical Analysis. In both experiments, data were analyzed for litter and treatment effects, utilizing analysis of variance procedures (Snedecor and Cochran, 1980) appropriate for a randomized block design. Orthogonal contrasts were utilized to detect differences among treatments. Contrasts included the effect of age (control, killed at h 0, vs treatment groups killed at h 24), the effect of supplementation (supplemented vs starved) and the effect of carnitine (MCT with vs MCT without added carnitine). Liver or biceps femoris weight from pigs killed at 0 h were used to predict, by regression analysis, the organ weight at the zero time for pigs killed at 24 h. The calculated organ weight and average glycogen concentration of 0 h was used to estimate the total glycogen at 0 h in the piglets killed at 24 h. Body weight was used as the independent variable in the regression analysis. The calculated 24-h weight or glycogen loss was determined by subtracting the predicted 0-h value from the measured 24-h value.

Results

The results from force-feeding 12 or 24 ml of the MCT product (Exp. 1) on voluntary milk consumption of 2-d-old piglets are shown in Table 2. Force-feeding 24 ml of MCT decreased (P < .05) total milk intake per pig over the 10-h experiment (49 g vs 163 g or 167 g). Milk intake was not different (P > .10) between piglets given 0 or 12 ml MCT (163 g vs 167 g). In piglets given the 24-ml MCT dose, a trace of material resembling MCT was excreted within 1 h after force-feeding. This was unexpected, because similar volumes of milk can be consumed without apparent abnormal effects. We decided from this limited study to use 12 ml of the MCT product, because a decrease in colostrum consumption would be inconsistent with the purpose of the project, which was to enhance the survival of newborn piglets. A more intense study of milk consumption in response to MCT supplementation is required before a specific volume of supplement is selected as optimal.

The results from the two 24-h trials on blood metabolites and liver and muscle glycogen levels (Exp. 2) are shown in Tables 3 through 6. Except where treatment × trial interactions were significant, the results from the trials were pooled.

The average packed cell volume varied from 33% to 36% at all sampling times in piglets killed at 24 h. The packed cell volume in control piglets killed at birth was 30% and lower (P < .05) than the three other groups, but these differences were not considered to be biologically significant. The values reported are similar to those found by Ramirez et al. (1963).

The average whole blood glucose levels ranged from 2.9 to 3.6 mM at birth and remained within this range irrespective of treatment over the 24 h (P > .05). Although not decreased (P > .05), the value for the piglets starved for 24 h was 84% of that measured in piglets that received the supplements. The variation in these values is similar to that reported in the literature for piglets over the first 24 h of life (Pegorier et al., 1981). The average total blood ammonium N levels ranged from 39 to 44 µM at birth and were not altered (P > .05) with treatment. Blood urea N levels varied from 9.6 to 10.0 mM at birth (Table 3) and were not different among the treatment groups. The urea N levels in piglets starved for 24 h were 9.7 mM at birth, 11.5 mM at 12 h and 11.2 mM at the 24-h sampling. The levels in piglets supplemented with MCT or MCT plus carnitine were lower (P < .01) than levels in piglets starved from birth (11 vs 6 to 8 mM) at both the 12- and 24-h samplings. New steady state concentrations appeared to be achieved early in all groups because levels at 12 and 24 h were similar.
The suggestion that supplemental MCT spared body fuel reserves is supported in part by the lower calculated weight loss of the liver shown in Table 4. The liver weights of the piglets starved for 24 h were less (P < .05) than the liver weights of piglets receiving the supplemental MCT product. Calculation of the weight loss of the liver and biceps femoris required estimation of the weight of these organs at birth. These weights were estimated by prediction equations developed from regressing the weight (g) of the liver or biceps femoris at birth against body weight (g). The loss of liver weight in piglets starved for 24 h was greater (P < .05) than in piglets given either MCT or MCT plus carnitine. No difference was seen in weight loss of the biceps femoris. These estimates of weight loss could be in error because of the dynamic nature of body substance movement between organs; hence, the weight (or metabolite) lost from one organ may be recovered in another.

The concentration of liver glycogen was higher (P < .02) at birth than at 24 h (Table 5). Relative to piglets that were starved, MCT supplementation resulted in a greater concentration of liver glycogen (P < .02) only in piglets from Trial 1. No differences (P > .05) in biceps femoris glycogen concentration were observed in piglets from Trial 1, but those from Trial 2 had more (P < .05) glycogen at birth than at 24 h. Utilizing the approach taken to estimate the liver weight loss, the approximate loss of liver glycogen was calculated by subtracting the amount measured at 24 h from the amount predicted to be in the liver at birth. The concentration of glycogen in the liver of pigs killed at 0 h (Table 5) was used to calculate total liver glycogen, and total glycogen was used to develop the prediction equation using total liver glycogen and body weight. The results of the glycogen determinations and calculations are shown in Table 6. The results from the two trials could not be pooled because of a significant treatment × trial interaction (P < .05). In Trial 1, piglets supplemented with the MCT products had significantly less glycogen disappearance from the liver. This difference was not detected in Trial 2.
TABLE 5. TISSUE GLYCOGEN CONCENTRATION AT BIRTH AND AFTER 24 HOURS WITHOUT AND WITH MEDIUM-CHAIN TRIGLYCERIDE SUPPLEMENTATION

<table>
<thead>
<tr>
<th>Treatmenta</th>
<th>Trial 1</th>
<th>Trial 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Killed, h</td>
<td>Liverb&lt;sup&gt;bc&lt;/sup&gt; femoris</td>
</tr>
<tr>
<td>Control</td>
<td>0</td>
<td>98</td>
</tr>
<tr>
<td>Starved</td>
<td>24</td>
<td>30</td>
</tr>
<tr>
<td>12 ml MCT</td>
<td>24</td>
<td>75</td>
</tr>
<tr>
<td>12 ml MCT + .6 mmol L-carnitine</td>
<td>24</td>
<td>53</td>
</tr>
<tr>
<td>SE</td>
<td>11.2</td>
<td>5.0</td>
</tr>
</tbody>
</table>

<sup>a</sup>Treatment x trial interaction (P < .05). N = 6 per treatment group for Trials 1 and 2.
<sup>b</sup>Control vs starved, MCT and MCT plus carnitine treatment groups differ (P < .01).
<sup>c</sup>Starved vs MCT and MCT plus carnitine treatment groups differ (P < .02).

**Discussion**

The results suggest that MCT can be used as a fuel by the neonatal pig. The decreased concentration of blood urea N in piglets given 12 ml of MCT (Table 3) supports the idea that this exogenous fuel decreased the use of body protein as a fuel. The suppression in the loss of liver weight (Table 4) is also consistent with this idea. But the lack of a major effect on body weight loss (Table 4) is not supportive. Supplemental energy decreased disappearance of liver glycogen in Trial 1 (Table 6) but not in Trial 2. Thus, although the results from this work are encouraging, more work is required before a strong conclusion on the use of MCT as a fuel for neonatal pigs can be reached.

The advantage of an exogenous fuel, such as the MCT used in this work, is for sparing the fuels stored in the neonate prior to birth. From the body composition of newborn piglets reported by Elliot and Lodge (1977), the amount of energy stored in various body components of the neonate can be calculated (Table 7). The major stored fuel is body protein, followed by carbohydrate and then fat. The amount of time required for these fuels to be totally consumed can be calculated based on an expected rate of heat production. By using the oxygen consumption estimates of 18.9 ml-min<sup>-1</sup>-kg<sup>-1</sup> for newborn piglets at thermal neutrality (Noblet and Le Dividich,

TABLE 6. TOTAL LIVER GLYCOGEN AT BIRTH AND EFFECT OF MEDIUM-CHAIN TRIGLYCERIDE SUPPLEMENTATION ON THE LOSS OF LIVER GLYCOGEN IN PIGLETS<sup>a</sup>

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Trial 1</th>
<th>Trial 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Killed, h</td>
<td>Measured&lt;sup&gt;bc&lt;/sup&gt; at birth</td>
</tr>
<tr>
<td>Control</td>
<td>0</td>
<td>3.09</td>
</tr>
<tr>
<td>Starved</td>
<td>24</td>
<td>.65</td>
</tr>
<tr>
<td>12 ml MCT</td>
<td>24</td>
<td>2.03</td>
</tr>
<tr>
<td>12 ml MCT + .6 mmol L-carnitine</td>
<td>24</td>
<td>1.57</td>
</tr>
<tr>
<td>SE</td>
<td>.45</td>
<td>.38</td>
</tr>
</tbody>
</table>

<sup>a</sup>Treatment x trial interaction (P < .05). N = 6 per treatment group for Trials 1 and 2. Total liver glycogen was determined at birth in control piglets. The total liver glycogen at birth in piglets killed at 24 h was estimated from a regression equation developed from the control piglets. The equation for predicting liver glycogen was \( y = .0033X - .92, R^2 = .60 \) where \( y \) = predicted total liver glycogen at birth in grams and \( X \) = weight in grams of pigs at birth.
<sup>b</sup>Control vs starved, MCT and MCT plus carnitine treatment groups differ (P < .01).
<sup>c</sup>Starved vs MCT and MCT plus carnitine treatment groups differ (P < .05).
<sup>d</sup>MCT vs MCT plus carnitine treatment groups differ (P < .05).
TABLE 7. CALCULATED ENERGY CONTENT IN VARIOUS BODY COMPONENTS OF NEONATAL PIGS.

<table>
<thead>
<tr>
<th>Component</th>
<th>Amount (g/kg)</th>
<th>Fuel value (kcal/g)</th>
<th>Total energy (kcal)</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crude protein</td>
<td>90.5</td>
<td>4.8^a</td>
<td>608</td>
<td>72.1</td>
</tr>
<tr>
<td>Ether extract</td>
<td>6.1</td>
<td>9.5</td>
<td>81</td>
<td>9.6</td>
</tr>
<tr>
<td>Nitrogen-free extract</td>
<td>27.6</td>
<td>4.0</td>
<td>154</td>
<td>18.3</td>
</tr>
</tbody>
</table>

^aTotal energy content in a 1,400-g pig.
^bThe fuel value for crude protein is corrected only for the energy content of the urea produced.

1981) and a caloric value of 4.8 kcal/liter oxygen consumed, a heat production estimate of 130 kcal·24 h⁻¹·kg⁻¹ would be expected, which, for a 1,400-g piglet, totals 182 kcal·24 h. This estimate would be high for the current application because heat production would be expected to decrease as the piglets are starved over the 24 h of the experiment (Mount et al., 1963; Waterlow, 1986). If heat production was maintained at 182 kcal·24 h⁻¹·1,400 g⁻¹, it would take 10.7 h to consume all of the body fat or 20.3 h to consume all of the stored carbohydrate. It is unlikely that these fuels burn sequentially or are totally consumed; thus, some protein also must be catabolized in order to meet the fuel demands of the fasting piglet.

If the 94 kcal of energy provided by the 12 ml of MCT is totally absorbed, it could provide slightly more than 50% of the energy required by the 1,400-g piglet. If the MCT becomes the primary fuel, as expected, it should decrease the rate of utilization of glycogen and protein. Protein would not be expected to become a primary fuel until the piglet had fasted for more than 18 h. A recent report (Odle et al., 1987) showed that newborn piglets starved for 24 h excrete 147 mg of urinary N·24 h⁻¹·kg⁻¹, whereas those receiving an MCT supplement excreted 117 mg of urinary N·24 h⁻¹·kg⁻¹. This suggests that protein can account for only 3.4% of the heat produced by the starving piglet over the first 24 h of life (147 mg·24 h⁻¹·kg⁻¹ × 1.4 kg = 206 mg N; 206 mg N × 6.25 g CP·g N⁻¹ × 4.8 kcal·g CP⁻¹ = 6.2 kcal or 6.2 kcal·182⁻¹·100 = 3.4%). As glycogen reserves are depleted, protein will make up the bulk of the fuel so that urinary N values of 4.3 g N/kg would be expected (130 kcal/24 h + 4.8 kcal/g CP = 27.1 g CP; 27.1 g CP·16 g N·g CP⁻¹ = 4.3 g N·24 h⁻¹·kg⁻¹). As pointed out earlier, this probably is an overestimate, because with prolonged starvation, heat production/kg body wt would decline from the expected 130 kcal·24 h⁻¹·kg⁻¹.

Thus, the strategy used in providing a fuel of the newborn should be to spare the stored carbohydrate and protein fuels, and by doing this to decrease gluconeogenesis (Bieber et al., 1979) as an important element in the metabolism of the newborn. The carbon for this process comes from protein and thus further weakens the piglet.

Supplemental carnitine was not of apparent benefit in that no consistent differences were noted between piglets given MCT and those given MCT along with .6 mmol of L-carnitine. As part of this study, we (Mares-Perlman et al., 1985) showed that this level of carnitine supplementation increased the liver carnitine fourfold (fasted control = 117 nmol/g wet weight) and muscle carnitine 1.7-fold (fasted control = 500 nmol/g wet weight) that of the fasted control. The concentrations for the fasted piglets were similar to those reported by Kerner et al. (1984).

The high tissue concentrations of medium-chain fatty acids expected from the amount of MCT used in this experiment should create the strong possibility that part of the medium-chain fatty acids would enter the mitochondrial matrix as octanoyl-carnitine or decanoyl-carnitine rather than as the free acids. Their high concentration in the cell and the lack of a single substrate, single Kₘ relationship with fatty acids of different chain length by the fatty acid activating enzymes (Aas, 1971) should give rise to both octanoyl-CoA and decanoyl-CoA and hence to a need for carnitine. Groot and Hulsmann (1973) reported a threefold increase in oxygen consumption and a 40-fold increase in octanoate ¹⁴C conversion to CO₂ when muscle mitochondria were supplied with L-carnitine. More detailed substrate utilization-product catabolism studies will have to be conducted before carnitine supplementation can be dropped from consideration.
In future studies, more attention will have to be devoted to the study of the development in the capacity to oxidize fatty acids because of the reports of Shipp et al. (1982) and Stanley et al. (1983), who showed 3- to 12-fold increases in fatty acid oxidation over the 1st d of life using guinea pig liver preparations. Observations of this nature are critical to future work on the piglet because differences in the time of development in the ability to utilize fatty acids may dictate the most appropriate time to give piglets the supplement, and whether or not additional co-factors may be required. Additionally, attention must be directed toward the small piglet (<900 g) at birth with regard to the maturity of its metabolic development in relation to that of the average piglets (1,200 to 1,500 g) because the smaller piglet has a lower probability of survival (Pettigrew, 1981; Cieslak et al., 1983).

The original suggestion that MCT could be considered as a potential oral source of supplemental energy for newborn piglets appears to be supported and warrants further intensive study of the ability of newborn piglets to use medium-chain fatty acids as a source of energy to conserve the on-board fuels, glycogen and protein.

Implications

Small, weak piglets have the greatest risk of death. These piglets are most likely to be born with limited body energy reserves. The use of an oral high-energy supplement that can be given at the time the litter is processed (usually within 12 h of birth) should enhance the probability of survival of these piglets. The supplemental energy should help conserve body energy reserves and provide a source of energy for the maintenance of body temperature. The supplemental energy should help the piglet establish itself as a viable member of the litter.

Literature Cited


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