EFFECT OF ENERGY SOURCE FED TO SOWS DURING LATE GESTATION ON SUBSEQUENT NEONATAL SURVIVAL, ENERGY STORES AND COLOSTRUM COMPOSITION

by

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Preweaning death losses of 10 to 25% (Leman et al., 1972) are one of the major economic losses occurring in the swine industry. The first 4 d post-partum generally account for between 60 to 70% of preweaning deaths (Pomeroy, 1960). Researchers have attempted to identify both nutritional and management factors which may minimize these losses.

In contrast to other neonates, the pig has relatively sparse pelage and only 1-2% total body fat to provide elemental insulation. A lack of brown fat, for non-shivering thermeogensis, contributes to a poorly developed homeostatic process (Bruck, 1970).

These factors require the newborn pig to nurse very soon after birth or to rely on glycogen and fat stores to maintain homeostasis. The pig is born with large amounts of liver glycogen relative to calves and sheep (Shelley, 1961). These stores are nearly depleted within the first 24 hours of life (Hakkarainen, 1975; Elliot and Lodge, 1977) resulting in hypoglycemia.

Researchers have attempted to evaluate methods to nutritionally affect neonatal energy reserves. Feeding fat to sows in late gestation has been reported to increase neonatal glycogen and lipid deposition in addition to increasing survival from birth to weaning (Seerley et al., 1974, 1978b; Boyd et al., 1978a). Other research has evaluated maternal glucose and free fatty acid changes induced by alloxan diabetes (Ezekwe and Martin, 1978, 1980). These researchers reported an increase in neonatal liver glycogen and body fat from alloxan induced diabetic sows. Stahly et al. (1980) and Spence et al. (1985) have induced maternal

ketosis by feeding 1,3-butanediol which resulted in increases in pig survival, liver glycogen and glycogen synthetase activity. Steele et al. (1980) and Thulin (1985) indicated that the fetus may utilize the excess ketone body metabolites for lipogenesis.

Rosebrough et al. (1981) observed that medium chain triglycerides (MCT) were mildly ketogenic when fed as 20% of daily energy to sows during late gestation. They found that when fed alone or in combination with 1,3-butylene glycol, MCT increased fetal brain development and elicted an increase in hepatic glycogen synthetase and phosphorylase. These data would tend to indicate that the mild ketosis produced by MCT addition may have positively influenced neonatal energy stores. One could expect from research showing milkfat and milk volume increases in response to fat fed during late gestation (Seerley et al., 1974; Kruse et al., 1977; Boyd et al., 1978b, 1979, 1981; Okai et al., 1978; Pettigrew 1981; Coffey et al., 1982) that fat added as MCT and soybean oil would also elicit the same response.

The objectives of the study reported here were to evaluate the effects of isocaloric additions of wheat starch, soybean oil and MCT to a basal diet from d 100 post-mating until parturition on neonatal liver and skeletal muscle glycogen, whole body fat, colostrum composition and on survival of pigs to weaning.

Mortality of preweaned pigs

Neonatal pig mortality represents a great economic loss to the swine industry. Of the reported 13 to 25% preweaning death loss (Hutchinson et al., 1954; Bauman et al., 1966; Fahmy and Bernard, 1971; Leman et al., 1972; Stanton and Carroll, 1974) it has been demonstrated that the majority occurs within the first 4 d postpartum (Hutchinson et al., 1954; Pomeroy, 1960; Bauman et al., 1966; Fahmy and Bernard, 1971; Nielson et al., 1974; Glastonbury, 1976).

Of the 16.4% death loss from parturition until 56 d of age reported by Fahmy and Bernard (1971) the majority (26.9%) died of congenital weakness and inanition. Of the 31% preweaning death loss reported by Hutchinson et al. (1954), 42% reportedly died of inanition and/or congenital weakness. Both Hutchinson et al. (1954) and Fahmy and Bernard (1971) reported overlaying by the sow to be the next highest cause of death with a variety of other factors being responsible for the rest of the total.

Low birth weight has often been reported to be the major contributing factor to neonatal death loss (Fahmy and Bernard, 1971; Pomeroy, 1960; Rosebrough, 1981). Newborn pigs weighing less than 900 g do not seem to be able to compete with larger littermates for nourishment from the dam. This lack of competitiveness and subsequent failure to receive proper nourishment from the dam leads to acute hypoglycemia with the most critical period being the first day of life as reported by numerous researchers (Graham et al., 1941; Sampson et

al., 1942; Swiatek et al., 1968; Gentz et al., 1970; Mersmann, 1974). This poor glucose homeostasis and subsequent inanition results in a general weakness of the pig and a predisposition to being overlain by the dam.

Glycogen stores of neonatal pigs

The glycogen stores of the neonate consist mainly of liver and skeletal muscle glycogen. On a wet basis, liver contains a higher percentage of glycogen than does skeletal muscle (211 vs. 180 mg/g; Okai et al., 1978). However, skeletal muscle glycogen represents a greater total amount of glycogen due to mass of tissue (Okai et al., 1978). Only hepatic glycogen may be utilized for blood glucose homeostasis since skeletal muscle does not contain glucose-6-phosphatase necessary for the conversion of glycogen to free glucose (Caraway, 1970). Compared to the adult animal, the neonate contains a greater proportion of total glycogen. This glycogen store is rather transitory and must be supplemented by nourishment from the dam very soon after birth (Okai et al., 1978).

Lipid stores and utilization

Lipid stores of the neonatal pig have consistently been reported to be about 2 percent of total body weight (Manners and McRea, 1963; Seerley and Poole, 1974; Okai et al., 1978; Boyd et al., 1978a; Lodge et al., 1978). Of this 2%, much appears to be structural and unavailable to the pig as an energy source (Mersmann, 1974). This low percentage of fat

allows for very little thermoinsulation (Manners and McRea, 1963). A lack of brown fat necessary for uncoupled oxidative phosphorylation for non-shivering thermogenesis (Bruck, 1970) further complicate the thermo-stability of the young pig.

Feeding of fat or ketogenic substrates

Neonatal fat and blood FFA

Several studies (Seerley et al., 1974; Boyd et al., 1978a; Bishop et al., 1985a) have indicated that neonatal fat may be increased by the dietary inclusion of fat to the dam prior to parturition. In a study comparing the effects of cornstarch (CS) and corn oil (CO) added to a basal diet at 4 kcal · kg body weight^{-1}, Seerley et al. (1974) demonstrated that neonatal fat composition can be altered. Significant increases in total carcass lipid were noted in pigs from dams fed the corn oil group over the cornstarch and control (2.43, 2.04 and 2.01%, respectively). Boyd et al. (1978a) compared the addition of a 9300 kcal day increase in energy intake to a basal ration as either stabilized tallow or corn starch for gestating gilts from day 100 to parturition. Blood FFA was increased from .11 mEQ·L^{-1} to 15 mEQ·L^{-1} for gilts fed cornstarch compared to tallow, respectively. In trials comparing 4 Mcal·d^{-1} addition of soybean oil or corn starch for 9 days pre-partum to a 6 Mcal·d^{-1} basal ration, Bishop et al. (1985a) found that the SO treatment increased carcass fat at birth from 1.25 to 1.33% for control versus SO, respectively. Others have noted that various

dietary lipid additions during late gestation have not increased total carcass lipids of the progeny (Seerley et al., 1978b; Boyd et al., 1981; Bishop et al., 1985b). Boyd et al. (1981) demonstrated that the inclusion of 8% bleachable fancy tallow to the basal diet of multiparous sows resulted in a nonsignificant increase in carcass lipids of 1.50 vs. 1.48% for the basal + cornstarch treatment. Seerley and Poole (1974) reported that total carcass lipids were not affected by the addition of 24 kcal·kg body wt^{-1} of corn oil for at least 4 days prepartum over that found in the basal diet (2.3 vs. 2.1%, respectively). In a recent study by Bishop et al. (1985b) the addition of 4 Mcal·d^{-1} as either starch or soybean oil (SO) to a basal diet supplying 6 Mcal·d^{-1} ME beginning 7 days prepartum resulted in no difference in pig carcass lipid content at birth.

Glycogen stores

While total carcass lipids may indicate energy reserves in the neonatal pig, other factors such as liver glycogen, ability to regulate blood glucose and perinatal thermostability may be of equal or greater importance. Boyd et al. (1979) reported that liver glycogen diminished by 51% during the first 6 hours of life and by 86% during the first 24 hours of life. Bieber et al. (1979) demonstrated that livers of 5 day old pigs lack homeostatic control of either hyperglycemia ($\geq$300 mg·dl^{-1}) or hypoglycemia ($\leq$30 mg·dl^{-1}). The effects of dietary inclusion of fat or ketogenic substrates to the dam on liver glycogen, blood glucose homeostatic control and thermostability of the progeny have been evaluated in numerous experiments. Table 1 presents liver glycogen

contents at birth for pigs in several studies which have attempted to vary energy level and source in the diet of the gestating sow. The lack of consistent results makes interpretation of the data difficult. Several studies (Stahly et al., 1980; Steele et al., 1980; Rosebrough et al., 1981) suggest that either addition of energy as fat or as a ketogenic substrate tends to increase progeny liver glycogen. With the longer carbon chain fat sources fed, only Coffey et al. (1982) reported an increase in progeny liver glycogen. None of the researchers (Boyd et al., 1978a, 1981; Okai et al., 1978; Bishop et al., 1985b) working with tallow or soybean oil demonstrated significant differences in liver glycogen of progeny from control and fat fed dams.

Glucose homeostasis

Hypoglycemia is often implicated in preweaning pig deaths (Swiatek et al., 1968). Work by Bieber et al. (1979) reports that the newborn pig has developed the capacity for gluconeogenesis (glucose production from non-hexose precursors). Although capacity for gluconeogensis may exist at birth, Ballard and Hanson (1967) suggest that it is poorly developed and must undergo a major developmental process.

The addition of fat or ketogenic substrates to the diet of gestating sows has produced inconsistent responses in glucose homeostasis of the progeny. Work by Boyd et al. (1978a, 1981) and Coffey et al. (1982) report significant increases in blood glucose at birth of progeny from fat fed compared to control sows. Okai et al. (1981) and Bishop et al. (1985b) reported no increase in blood glucose at birth. Boyd et al. (1981) and Coffey et al. (1982) demonstrated improvements in

maintenance of blood glucose following a 24 h preweaning fast in progeny of fat fed dams. Okai et al. (1978) and Bishop et al. (1985b) failed to show an increase in blood glucose at 24 h in pigs from sows fed fat. Overall, it would seem that lipid addition to the gestation diet may improve the ability of progeny to maintain blood glucose.

Thermostability

The affect of fat on thermostability of progeny was addressed by Seerley et al. (1974). While no significant treatment effects were noted from cold stressing pigs for 6 or 54 h after birth at 3 C on rectal temperature, trends indicate the progeny of higher energy intake dams (corn oil and corn starch) appear to increase blood glucose more than the low energy progeny. Mersmann (1974), reported cold stress to have the following effects on neonatal pigs; 1) increase in blood glucose, 2) decrease in skeletal muscle glycogen, but not liver glycogen and 3) increase in blood lactate. Mersmann (1974) found that since temperature is maintained predominantly by shivering, muscle glycogen would be expected to decrease as a result of phosphorylase stimulation by increased muscle activity. Increases of blood glucose, as observed by Seerley et al. (1974) and Mersmann (1974) may be assumed to be generated from gluconeogenesis as skeletal muscle does not contain glucose-6-phosphatase.

On colostrum composition

Feeding fat or ketogenic substrates not only affects neonatal composition, but sow colostrum and milk composition also are altered. Kruse et al. (1977) demonstrated that carbon length and proportion of C18:2 of dietary fat fed to a sow can change the resulting distribution of fatty acids in colostrum.

Researchers have evaluated colostrum fat and fatty acid profile when fat has been added to the gestation diet. While the technique and timing of colostrum collection have varied, and extraction techniques for fat are seemingly arbitrary, researchers (Seerley et al., 1974, 1978a; Boyd et al., 1978b, 1981; Okai et al., 1978; Coffey et al., 1982; Bishop et al., 1985b) have demonstrated increases in colostrum fat by dietary fat additions to the gestating sow. In a review of the affects of feeding fat to sows, Pettigrew (1981) concluded that fat fed during late gestation does increase colostrum fat. In addition to increasing colostrum fat, it seems that dietary fat fed during gestation also increases milk production (Kruse et al., 1977; Boyd, 1979; Pettigrew, 1981).

On survivability

Pettigrew (1981) concluded that supplemental fat exceeding 1000 g prior to parturition may increase overall survival by 6.1% in herds with existing survival $\leq$80%. In research conducted since this review, Cieslak et al. (1983) found that supplementation of casein encapsulated white grease increased survivability of all pigs, but especially of pigs

weighing 700-1100 g at birth. However, these results were confounded in that the control diet was formulated to contain 2.8 $kcal \cdot g^{-1}$ and the fat diet 3.8 $kcal \cdot g^{-1}$. These differences in caloric density prevent drawing of conclusions since both groups were fed about 2 $kg \cdot Hd^{-1} \cdot d^{-1}$. Work by Bishop et al. (1985a) using supplemetal soybean oil or cornstarch at 4 $Mcal \cdot d^{-1}$ report no improvement in pig survival from sows fed fat compared to CS (81.2 vs. 85.1%, respectively). Stahly et al. (1980, 1985) isocalorically substituted 1,3-butanediol for CS beginning 9 d prepartum. In both experiments, significant increases in survival of pigs from 1,3-butanediol supplemented sows compared to CS sows (85.3 vs. 92.2%; 78.4 vs. 84.1%, respectively) were reported. Work by Stahly et al. (1980, 1985) indicates that the ketone former 1,3-butanediol may improve survival even in herds with slightly higher than 80% pig survival.

Maternal hormone/glucose effects

In a study designed to evaluate the ability of sows to maintain blood glucose concentrations when infused intraveneously with an exogenous source of glucose (glucose tolerance test), it was observed that sows with less blood glucose homeostatic control gave birth to higher birth weight progeny (Anderson et al., 1971). The exact physiological mechanism of this phenomena is not addressed. In a study by Ezekwe and Martin (1978), alloxan induced diabetes of the dam seems to be responsible for elevated maternal glucose concentrations and a concomitant increase in fetal liver weight and glycogen content. Work by Hausman et al. (1982) attempted to replicate the work of Ezekwe and

Martin (1978) to determine adipose tissue histology changes of the neonate. Progeny of diabetic dams contain adipose tissue that differed in; 1) size and morphology of adipocytes, 2) extent of adipocyte "lipid filling", 3) adipocyte intracellular glycogen stores and 4) number of fat cell clusters. Maternal alloxan induced diabetes or 20 d fasting increased fat cell clusters of the progeny resulting in an increased total progeny lipid compared to controls. Only offspring of diabetic sows exhibit increased size, single lobed conformation, intracellular glycogen and intercellular collogen connection, all of which are suggested to be indicative of more physiologically mature adipose tissue. Kasser et al. (1982) reported that maternal alloxan induced diabetes increased fetal serum concentration of insulin and triodothyronin (T_3). Progeny from diabetic sows had decreased serum growth hormone and glucagon from that of the control progeny. From these data, it would appear that maternal glucose concentrations influence the neonatal energy stores by increasing total lipid and glycogen and improving adipose tissue maturity. The practicality of increasing maternal glucose levels has not been addressed.

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Summary

Two studies utilizing 24 primiparous sows and 34 multiparous sows stratified by weight and parity in a completely randomized design were conducted to evaluate the effects of isocaloric additions of wheat starch (ST), soybean oil (SO) and medium chain triglyceride (MCT) during late gestation (d 100 to parturition) on neonatal energy stores, survival at weaning and on colostrum composition. Diets were formulated to supply 7200 kcal of $ME \cdot sow^{-1} \cdot d^{-1}$. All sows received the same 14% crude protein diet ad libitum during lactation.

Maternal blood samples taken 2 h postprandial on d 110 of gestation were analyzed for insulin, glucagon and glucose. Both insulin and glucagon levels changed ($P<.05$) in response to dietary treatments. Insulin levels decreased in SO dams compared to ST, with a concomitant increase in glucagon for SO dams compared to ST. Medium chain triglyceride treatment resulted in an intermediate level for both insulin and glucagon compared to ST and SO. These hormone changes however, resulted in equal blood glucose levels for all treatments ($P>.10$).

One pig from each litter was killed prior to nursing and analysis of liver glycogen, total eviscerated carcass glycogen and total eviscerated carcass fat were accomplished. No differences ($P>.10$) in amounts of these energy stores were obtained.

Analysis of colostrum composition indicate a numerical trend for SO to increase total colostrum lipid. Fatty acid profiles of colostrum

indicate MCT is equal or closer to ST than SO for all fatty acids with the exception of C18:3. These data support the hypothesis that SO may be preferentially utilized by mammary tissue for milk production. However, it seems that MCT are not preferentially incorporated into milk. Rather, it seems that MCT sows synthesize colostrum lipids as would ST sows.

MCT increased weight gain prefarrowing over that expected for normal fetal growth and mammary development. It is possible that MCT slowed rate of passage thus increasing gastrointestinal fill.

Postfarrowing weight change indicates that MCT sows have the greatest loss ($P<.05$). Soybean oil sows gained weight during lactation.

No treatment effects were noted on percent survival, birth weight or weaning weight.

From the research reported herein, one may conclude that MCT is a poorer choice as a fat source during late gestation to produce more total colostrum fat. It would appear that substantial improvements in energy stores of the neonatal pig are difficult to produce by feeding either SO or MCT to the sow during late gestation.

Introduction

Preweaning death losses of 10 to 25% (Leman et al., 1972) are one of the major economic losses occurring in the swine industry. The first 4 d postpartum generally account for between 60 and 70% of preweaning deaths (Pomeroy, 1960). Low birth weight (Cieslak, 1983), low body reserves of energy (Seerley et al., 1974) and a poorly developed capacity to regulate blood glucose concentrations (Hakkarainen, 1975) may all contribute to these preweaning deaths.

Researchers have attempted to evaluate methods to nutritionally affect neonatal energy reserves. Feeding fat to sows in late gestation has been reported to increase neonatal glycogen and lipid deposition in addition to increasing survival from birth to weaning (Seerley et al., 1974, 1978b; Boyd et al., 1978a). Rosebrough et al. (1981) found that medium chain triglycerides (MCT) fed to sows at 20% of daily energy positively influenced glycogen synthetase and phosphorylase activity. One could expect from research showing milkfat and milk volume increases in response to fat fed during late gestation (Seerley et al., 1974; Kruse et al., 1977; Boyd et al., 1978, 1979, 1981; Okai et al., 1978; Coffey et al., 1982) that fat added as MCT and soybean oil would also elicit the same response.

The objective of this study was to evaluate the effects of isocaloric additives of wheat starch, soybean oil and MCT to a basal diet from d 100 post-mating to parturition on neonatal liver and skeletal muscle glycogen, whole body fat, colostrum composition and on survival of pigs to weaning.

Experimental Procedure

Two experiments where conducted to evaluate the effects of soybean oil (SO) or medium chain triglycerides (MCT) on neonatal carcass parameters and survival.

Trial 1 was conducted in February-March 1985. Twenty-nine primiparous and multiparous crossbred sows were utilized. On d 99 postcoitum of the first bred female, sows were randomly allotted to treatment by weight and parity and were placed in individual gestation stalls. On d 100 postcoitum, each animal was changed from a 14% corn-soy (1.8 $kg \cdot d^{-1}$) gestation diet to the appropriate dietary treatment (Table 3). Sows were fed between 0500-0600 h daily. Starch treatment animals received 2.19 $kg \cdot d^{-1}$ and SO and MCT treated sows received 1.89 $kg \cdot sow^{-1} \cdot d^{-1}$. This provided equal amounts of energy (7200 kcal of $ME \cdot d^{-1}$), protein, lysine and minerals. All animals received a 14% crude protein diet ad libitum during lactation. Blood samples were obtained via jugular vena puncture 2 h postprandial on d 110 postcoitum for subsequent analysis of insulin, glucagon and glucose. Blood was collected and 7 ml portions were immediately transferred into 2 silicone coated glass tubes and into 2 tubes with Na-EDTA[a] and 350 ml trasylol inhibitor[b]. Serum and plasma was harvested after centrifugation at 2400xG for 20 minutes. Samples were stored at -20 C until analysis. On d 113 postcoitum of the first bred female, all sows were washed, disinfected and moved into individual farrowing crates. Weights into the farrowing house were recorded.

[a]Vacutainer-Tube, Bectin-Dickson, Rutherford, N.J.

[b]Cambridge Medical Diagnostics, Cambridge, MA.

All farrowings were attended and the first born gilt weighing between 1.27 and 1.45 kg was dried, electically stunned and killed by exsanguination. Livers were collected, patted dry, weighed, frozen in liquid nitrogen and then maintained at -20 C. Eviscerated pig carcasses were dried of external blood and frozen at -20 C. All pigs were killed less than 30 minutes after birth and prior to suckling. To keep pigs within the desired weight range, in 2 cases boar pigs were substituted for gilts.

Colostrum samples were obtained during parturition (150 ml) by manual expression of several glands until dry. Fresh samples were extracted in duplicate via a modified chloroform, methanol, water extraction (Folch, 1957). The chloroform extract was maintained under nitrogen gas along with whole colostrum at -20 C.

Litters were equalized within treatment for the first 24 h postpartum after which time no further transfers were allowed.

Trial 2 was conducted in March-April 1985. Again, 29 primiparous and multiparous sows were utilized. Sows were allotted, treated and samples were collected as per trial 1. An exception being the killing of either gilt or boar pigs within the 1.27 to 1.45 kg framework.

Plasma samples were analyzed for glucagon by radioimmunoassay kits[C] (Faloona and Unger, 1974). Company supplied performance data indicates recovery of 102% of the theoretical amount (pg/ml) of plasma glucagon. Intra-assay precision data indicate a standard deviation of 33.8 pg/ml with a coefficient of variation of 8.7%. Inter-assay data indicate a standard deviation of 50.2 pg/ml with a C.V. of 11.8%. The calculated

[C]Cambridge Medical Diagnostics, Cambridge, MA.

sensitivity for 20 replicates of a zero standard for this assay is 50 pg/ml (human data). The assay utilized double antibodies, the first of which was rabbit glucagon antiserum (Lot no. 21AFLE1) and the second goat anti-rabbit gamma globulin (Lot no. 96ASLCZ). ^{125}Iodine (2 μCi) labeled porcine glucagon (Lot no. 1208504) was employed as the tracer. Porcine standards of 95, 175, 300, 600, 1500 and 3600 pg/ml and samples were assayed in duplicate. Counts per minute were made using a gamma radiation counter[d]. The standard curve of concentration versus percent bound was plotted on semi-log paper with each sample being interpolated from this curve. A 10% duplicate variation was accepted and the mean used for statistical analysis. Cross reactivity is given in table 2.

Serum samples were analyzed for insulin by radioimmunoassay kits[e] (Goetz and Greenberg, 1961; Morgan and Lazarow, 1963). Company supplied performance data indicate 95% recovery of the theoretical value of serum insulin. Intra-assay data indicate a standard deviation of .75 uU/ml (samples $\leq$ 17.1 μU/ml) with a coefficient of variation of 7.55%. Inter-assay data indicate a standard deviation of 2.5 uU/ml (samples $\leq$ 45.9 uU/ml) with a C.V. of .75%. Cross reactivity is given in table 2.

The assay utilized double antibodies. The first antibody was guinea pig anti-insulin serum (Lot no. 8503014). The second was rabbit anti-guinea pig precipitating complex (Lot no. 841010). ^{125}Iodine (2 μCi) labeled porcine insulin (Lot no. 8520001) was employed as the tracer. Counts per minute were made using a gamma radiation counter as per glucagon assay. Porcine standards of 4, 8, 16, 32 and 100 μU/ml and samples were assayed in duplicate. The standard curve of concentration

[d]Beckman Gamma 4000, Model 8040302, Irvine, CA.

[e]Immuno Nuclear Corporation, Stillwater, MN.

compared to percent bound was plotted on semi-log graph paper and each sample was interpolated from this curve. A 10% sample variation was accepted and the mean used for statistical analysis.

Plasma glucose was determined in duplicate by the automated procedure[f] of Gochman and Schmitz (1972) using glucose oxidase. A 5% sample variation was accepted and the mean used for statistical analysis. Tissue glycogen was determined by a modified technique of Seifter et al. (1949) (Appendix 1). Isolated rabbit liver glycogen[g] was used to produce standard curves with concentrations of .025, .050, .075, .100, .150, .200 and .05, .10, .15, .20, .25 g/500 ml H_2O for analysis of carcass and liver glycogen respectively. Correlation coefficients $\geq$.9800 were accepted for standard curves. All samples were run in duplicate and the average was accepted at $\leq$10% sample variation.

Carcasses were cryogenically ground through a 3.2 mm screen in a Fitz mill[h]. The resulting homogenate was manually mixed and a 200 g subsample was saved for laboratory analysis. Approximately 3 g of this homogenate and 1 g of liver from the left lateral lobe was used for gylcogen determination.

Approximately 30 g of the carcass homogenate was extracted according to the chloroform/methanol/water 1:1:0.5 V/V/V method of Melton et al. (1979) (Appendix 2). A portion of the resulting lipid containing extract was dried by nitrogen gas, then by vacuum oven, to determine the fat content.

[f]Technicon Autoanalyzer II, Tarryton, NY.

[g]Aldrich Chemical Co., Milwaukee, WI. Cat. no. 85-143-4.

[h]Fitz Mill, The Fitzpatrick Co., Elmhurst, IL.

The extracts of colostrum were dried under nitrogen and methylated according to the procedure of Supelco, Inc. - GC Bulletin 721G. Profiles of methylated fatty acids were determined via gas chromotography[i] on a 2mm x 2 M glass column packed with SP-2330 on chromasorb[j] (Appendix 3) to separate C10:0, C12:0, C16:0, C16:1, C18:0, C18:1, C18:2, C18:3 and C20:4. Heptadecanoic acid was used as the internal standard[j] with a standard mix containing the fatty acids to be profiled. Peak areas were determined via an interfacing integrator[k].

Analysis of colostrum for percent protein, ash and dry matter were accomplished according to A.O.A.C. (1975). The Roese-Gottlieb (A.O.A.C., 1975) procedure for dairy products was employed for total colostral fat determination. Gross energy of colostrum was determined by bomb calorimetry. Protein was determined by Kjeldhal procedure.

Statisical analysis was performed to test for treatment differences using the General Linear Model analysis of variance (SAS, 1982). There were no trial by treatment interactions, therefore the data from the two trials were pooled and are presented as such. Model statements for all milk data, sow hormone data, sow weight change, survival data and neonatal composition data included treatment and parity as independent variables. Analysis of survival data included litter number as a covariate. Linear contrasts of ST versus SO, ST versus MCT, and SO versus MCT were constructed for treatment separation.

[i]Hewlett-Packard, Model 5890A.

[j]Supelco, Co., Bellfonte, PA.

[k]Hewlett Packard, Model 3992A.

Results

Sow weight gain (table 4) for the 15 d prepartum period indicate that MCT fed sows gained more ($P<.05$) than SO fed sows. MCT sows lost an average of 5.23 kg during lactation which was more ($P<.05$) than the SO sows which gained 4.21 kg during lactation. The ST sows were intermediate, losing 1.18 kg during lactation.

No differences were noted in number of pigs born alive, birth weight, weaning weight or percent survival at weaning (table 5).

Colostrum fat (table 6) increased ($P<.05$) for SO compared to MCT fed sows. Protein was increased ($P<.05$) for ST compared to SO sows. Dry matter, ash, crude protein and gross energy were similar for all treatments ($P>.10$). Fatty acid profiles (table 7) for MCT and ST colostrum were the same ($P>.10$) and both were different ($P<.05$) than SO in percent C16:0, C16:1, C18:1, C18:2 and C20:4. Percent C14:0 and C18:0 were different ($P<.05$) for all dietary treatments. Only for percent C18:3 were MCT and SO values the same ($P>.10$) and were both less than ($P<.05$) ST colostrum.

No differences ($P>.10$) were noted between treatments for total carcass fat or for carcass glycogen (table 8). While the difference was not significant ($P>.10$), liver glycogen was numerically highest for SO pigs.

Blood insulin, glucagon and glucose are presented in table 9. Insulin values for ST sows were significantly ($P<.05$) greater compared to SO and MCT sows with a concomitant decrease in glucagon for ST sows compared to SO and MCT sows. Blood glucose values were the similar for all treatments ($P>.10$).

Discussion

It appears that MCT is not as effective as SO in changing liver glycogen or colostrum fat or colstrum fatty acid profile from that observed for ST fed sows.

Both ST and SO sows had weight gains normally associated with prepartum fetal growth and mammary maturation. However, MCT sows had weight gains of 9 kg during this 12 d period. This weight gain would generally be most indicative of increases in gastrointestinal content, however rate of passage was not determined in this study. Bach and Babayan (1982) suggested that MCT are hydrolyzed more completely than long chain (>12 carbons) triglycerides and that the resulting FFA are absorbed into the portal venous system at a rate similar to glucose. Therefore, a possible increase in digestibility of MCT over SO may occur, but it would be difficult to attribute a 9.19 kg increase in 12 d to "true" weight gain.

Contrary to results of Nelssen et al. (1985), who reported greater weight loss during lactation for sows fed long chain length fat (tallow) than for starch controls, our data suggests that ST and MCT fed sows lost more weight than SO fed sows. This trait was however, extremely variable within each treatment. Upon examination of litter weaning weight as a gross measure of milk production, no differences are noted to substanstiate a hypothesis that MCT may have stimulated milk production and thus caused greater lactational weight losses.

Examination of number born alive reveals no treatment differences. This should be expected because of only an energy source change during

late gestation. Percent survival at weaning was extremely high for all treatments. Pettigrew (1981) suggested that fat additions may not be beneficial in herds with survival $\geq$80%. Our study supports this obvservation.

Moser (1980) suggests addition of fat to the late gestating sow diet results in increases in total colostrum fat. The results of our trials indicate that additions of fat as SO results in a tendancy for increased colostrum fat. However, contrary to the results obtained by Thulin (1985) which indicate a tendancy for MCT to increase total colostrum fat over SO, MCT did not increase total colostrum fat in these studies. Medium chain triglyceride addition resulted in less ($P<.05$) total colostrum fat than SO addition. Starch and MCT resulted in similar values. Bach and Babayan (1982) suggest that MCT are not metabolized as normal longer chain fatty acids. Rather, MCT are metabolized more completely to ketone bodies which are preferentially utilized by the sow for energy, possibly sparing glucose for de novo synthesis of fat (Allee, 1970). Colostrum fatty acid profiles support the hypothesis that MCT are metabolized sparing glucose. Results indicate that percent C14:0 and C18:0 are different ($P<.05$) for all treatments. Percent C14:0 and C18:0 for MCT was closer to ST than to SO (2.11, 1.37, .28; and 7.37, 6.42, 4.58, respectively). Only in percent C18:3 did MCT values equal those of SO ($P>.05$). For percent C16:0, C16:1, C18:1, C18:2 and C20:4, ST and MCT values were equal ($P<.05$) and were both different ($P>.05$) than SO. These results tend to indicate that SO may be preferentially shunted to milk production as suggested by Nelssen et al. (1985), however, it would also appear that MCT are not preferentially utilized by the mammary glands.

Analysis of crude protein indicates that ST increased content of protein over SO. This however, would not be expected from a mere change in energy source, but may be partially explained by the tendancy for SO to increase total colostrum fat which may dilute relative amounts of other components.

Work by Bishop et al. (1985b) indicating no beneficial effect of SO over ST for glycogen and fat of the neonate were corroborated in this study. Also, no beneficial effects of MCT addition were noted for neonatal glycogen or fat content.

Analysis of maternal blood insulin and glucagon indicate that the treatments did affect circulating hormone levels. Insulin levels decreased in SO dams compared to ST, with a concomitant increase in glucagon for SO dams compared to ST. Medium chain triglyceride treatment resulted in an intermediate level for both insulin and glucagon compared to ST and SO. These hormone changes however, resulted in equal ($P>.10$) blood glucose levels for all treatments.

If MCT are ketogenic as suggested by Bach and Babayan (1982) and Thulin (1985), one could make the hypothesis that excess maternal glucose could be utilized by the fetus to increase energy stores in a manner similar to that utilzed by the fetus of the diabetic sow (Ezekwe and Martin, 1978). This increase in maternal circulating glucose or increase in fetal energy stores does not seem to occur as can be seen from these data. The sow seems to adjust insulin and glucagon levels to maintain blood glucose. Therefore, during mild MCT produced ketosis, the sow does not have elevated blood glucose for fetal use.

TABLE 1. SUMMARY OF RESEARCH INVESTIGATING LIVER GLYCOGEN CHANGES AS A RESULT OF DIETARY FAT ADDITIONS DURING GESTATION

Reference	Days	Dietary addition	% change in liver glycogen[a]
Okai et al., 1978	15	10% tallow	67.4[b]
Boyd et al., 1978	8	5750 kcal tallow	107.4
Boyd et al., 1981	15	8% tallow	102.6
Steele et al., 1980	55	20% ME butylene glycol (BG)	145.7
		20% ME MCT	141.8
		20% MCT/BG	146.2
Rosebrough et al., 1981	45+	20% MCT	133.5[b]
		20% 1,3-BG	150.1[b]
Stahly et al., 1980	8	1700 kcal butanediol	123.9
Coffey et al., 1982	5	Mean of animal fat Trial 1	110.9[b]
	35	Mean of animal fat Trial 1	110.9[b]
Bishop et al., 1985	8	4 Mcal Soybean oil	.8

[a]Calculated as amount or concentration of $\frac{\text{fat treatment}}{\text{control treatment}} \cdot 100$.

[b]Significant difference between treatments ($P<.05$).

TABLE 2. RIA CROSS REACTIVITIES

	Glucagon kit(%)[a]	Insulin kit(%)[b]
Pancreatic glucagon	100	<.01
Gut glucagon-like immunoreactivity (dog)	<3	-
Glucagon fragment (1-26)	<1	-
Jejunum extract	<1	-
Human C-peptide of insulin	-	<.01
Human insulin	-	100
Porcine insulin	-	100
Proinsulin	-	55

[a]Cambridge Medical Diagonistics, Inc. 1984. Cambridge, MA.

[b]Immuno Nuclear Corporation. 1984. Stillwater, MN.

TABLE 3. COMPOSITION OF GESTATION DIETS

	Gestation Diet, %		
Ingredient	Control	Soybean Oil	MCT
Ground sorghum grain (IFN 4-04-444)	49.80	57.83	57.83
Soybean meal (IFN 5-04-604)	19.00	22.10	22.10
Wheat starch	27.40	-	-
Soybean oil	-	15.70	-
Medium-chain triglycerides[a]	-	-	15.70
Calcium carbonate (IFN 6-01-069)	0.81	0.94	0.94
Monocalcium phosphate (IFN 6-01-080)	2.24	2.61	2.61
Sodium chloride	0.36	0.42	0.42
Trace mineral premix[b]	0.10	0.10	0.10
Vitamin premix[c]	0.30	0.30	0.30
Total	100.00	100.00	100.00

[a]Capital City Products Co., Columbus, OH.

[b]Provided the following in mg/kg of complete diet: Mn, 50; Zn, 100; Fe, 100; Cu, 10; I, 3 and Co, 1.

[c]Provided the following per kg of diet: Vitamin A, 8800 USP; Vitamin D_3, 660 USP; Vitamin E 44 IU; riboflavin, 9.9 mg; menadione 3.4 mg; d-pantothenic acid 26.4 mg; niacin, 55 mg; choline chloride, 1014.2 mg; vitamin B_{12}, 48.4 g.

TABLE 4. EFFECT OF ENERGY SOURCE FED DURING LATE GESTATION ON SOW WEIGHT CHANGES[a]

Item	Treatment			SE
	Starch	Soybean Oil	MCT	
No. Sows	16	17	19	
Weight gain, prefarrowing, kg[be]	5.04	3.02	9.19	6.41
Sow weight change, weaning-farrowing, kg[cde]	-1.18	4.21	-5.23	7.20

[a]Values reported as LS means.

[b]Difference based on mean of 12.37 d on experimental diet for trial 1 and 12.08 d for trial 2. Days on trial used as a covariate.

[c]Mean time span of 19.08 d for trial 1 and 25.57 d for trial 2.

[d]Significant contrast ST vs. SO ($P<.05$).

[e]Significant contrast SO vs. MCT ($P<.05$).

TABLE 5. EFFECT OF ENERGY SOURCE FED DURING LATE GESTATION ON LITTER PARAMETERS

Item	Treatments			SE
	Starch	Soybean oil	MCT	
No. born alive	10.04	8.89	9.47	2.40
Mean birth weight, g	1510	1538	1574	266.99
Standardized no. per litter	9.47	8.48	8.62	1.32
Number weaned per litter	9.03	8.24	8.30	1.51
Litter weaning weight, kg	50.53	48.81	48.83	10.53
Percent survival at weaning	97.35	95.17	96.29	7.46

TABLE 6. EFFECT OF LATE GESTATION ENERGY SOURCE ON COLOSTRUM COMPOSITION

Item	Treatment			SE
	Starch	Soybean Oil	MCT	
Dry matter, %	24.44	24.95	24.18	3.34
Ash, % dry matter	2.44	2.22	2.19	.99
Crude protein, % dry matter[ab]	68.79	57.81	63.62	10.17
Fat, % dry matter[c]	20.93	23.01	17.88	5.55
Gross energy, cal/g dry matter	5496	6089	6288	1386

[a]Based on 6.25 multiplication factor.

[b]Significant contrast starch vs. SO ($P<.05$).

[c]Significant contrast SO vs. MCT ($P<.05$).

TABLE 7. EFFECT OF ENERGY SOURCE FED DURING LATE GESTATION ON COLOSTRUM FATTY ACID PROFILE[a]

Item	Treatment			SE
	Starch	Soybean Oil	MCT	
C10:0, %	.00	.01	.36	.89
C12:0, %	.00	.02	.03	.05
C14:0, %[bcd]	1.37	.28	2.11	.97
C16:0, %[bd]	14.50	8.28	14.45	2.87
C16:1, %[bd]	3.04	1.21	2.74	.80
C18:0, %[bcd]	6.42	4.58	7.37	1.11
C18:1, %[bd]	20.61	12.33	19.58	3.65
C18:2, %[bd]	49.25	68.72	47.62	6.75
C18:3, %[bc]	1.68	2.82	2.51	1.09
C20:4, %[bd]	3.13	1.75	3.23	1.01
Total	100.00	100.00	100.00	

[a]Calculated as $\frac{\text{mg of peak} \cdot 100}{\text{mg of total peaks}}$.

[b]Significant contrast ST vs. SO ($P < .05$).

[c]Significant contrast ST vs. MCT ($P < .05$).

[d]Significant contrast SO vs. MCT ($P < .05$).

TABLE 8. EFFECT OF ENERGY SOURCE FED DURING LATE GESTATION ON NEONATAL ENERGY STORES

Item	Treatments			SE
	Starch	Soybean Oil	MCT	
Total body fat, % wet	1.77	1.98	1.87	.39
Homogenized carcass glycogen, % wet	1.80	1.74	1.75	.57
Liver glycogen, % wet	14.01	17.65	13.24	9.38

TABLE 9. EFFECT OF ENERGY SOURCE FED DURING LATE GESTATION ON MATERNAL BLOOD METABOLITES

Item	Treatments			SE
	Starch	Soybean oil	MCT	
Glucagon, $pg \cdot ml^{-1}$[ab]	200.44	304.32	255.68	94.41
Insulin, $\mu\mu l \ ml^{-1}$[ab]	8.07	4.92	5.21	2.22
Glucose, $mg \cdot dl^{-1}$	78.12	79.91	78.99	12.73

[a]Significant contrast ST vs. MCT ($P<.05$).

[b]Significant contrast ST vs. SO ($P<.05$).

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APPENDIX 1

Stardard Operating Procedure for Glycogen Analysis

Method modified from Seifter et al. (1949). The estimation of glycogen with the Anthrone reagent (Arch. Biochem. 25:191).

Sample Preparation

1) Weigh 1 g of neonatal pig liver or 3 g of neonatal pig carcass homogenate (enough tissue to supply approximately .15 g of glycogen). Add to test tube.
2) Add 3 ml KOH and tissue to tubes and seal. Vortex.
3) Submerge tube in H_2O bath at 100 C for 20 minutes.
4) Immerse tube in ice bath. Store at 0 C until analysis.

Stardard Preparation

1) Weigh .625 g isolated mammalian glycogen into 25 ml volumetric flask.
2) Bring to volume with dd H_2O. Stir until glycogen is in solution.
3) Transfer the following volumes to 500 ml volumetric flasks, q.i.d. with dd H_2O: 1; 2; 3; 4; 6; 8 ml. Label flasks = .025*volume added to flask.

Reagent Preparation

1) 95% H_2SO_4:950 ml H_2SO_4 concentrate, 50 ml H_2O.
2) .2% Anthrone:2 g Anthrone reagent, 1 L 95% H_2SO_4.

Analysis

1) Bring sample to ambient temperature, then quantitatively transfer with dd H_2O to 500 ml volumetric flask, q.i.d. with dd H_2O. Mix well.
2) Add 1 ml portions of diluted standards and samples to culture tubes.
3) Add 2 ml of .2% Anthrone in 95% H_2SO_4. Seal. Vortex 30 sec.
4) Heat tubes for 10 min at 100 C.
5) Immerse in ice bath to prevent over development.
6) Vortex well and read absorption at 620 nm. Use .2% Anthrone reagent in reference cell.

Cautions

1) Seal tubes with non-carbohydrate lined acid resistant tops.
2) Vortex well.
3) Reagents are viscous and must be allowed time in cuvette for air bubble dissapation.

APPENDIX 2

Standard Operating Procedure for Total Lipid Extraction from Tissue

Method of Melton, S., R.E. Moyer and G.C. Playford. 1979. Lipids extracted from soy products by different procedures. J. Amer. Oil Chem. Soc. 56:489.

Sample:

1) Tissue amount should be as large as possible, but should have less than 3 g of total fat. For neonatal pig carcass homogenates, approximately 30 g will be adequate.

Procedure:

1) Weigh out 30 g of tissue homogenate onto fat free weigh paper.
2) Place sample along with weigh paper in Waring blender and add 130 ml methanol. Blend for 5 min.
3) Add 65 ml chloroform, reblend 5 min.
4) Add 65 ml chloroform, reblend 20 sec.
5) Add 65 ml dd H_2O containing 1.5 g zinc acetate, reblend 10 sec.
6) Filter through Whatman no. 1 filter paper with suction. Save filtrate (chloroform/methanol/water 1:1:.5 V/V/V).
7) Transfer filtered residue and paper back to Waring blender. Wipe funnel with facial tissue and add to blender.
8) Reblend with 100 ml chloroform for 2.5 min.
9) Filter as step 6 quantitatively transfer blender contents with 75 ml chloroform.
10) Transfer filtrate quantitatively to 500 ml graduate cylinder with 25 ml methanol.
11) Refrigerate at 6 C until sharp interface appears.
12) Record volume of chloroform layer and determine solid content of duplicate 10 ml aliquots.
13) Calculate % lipid = (ml $CHCl_3$ layer)(g solid/10 ml)(100)/sample wt.

APPENDIX 3

References: 1) Supelco, Inc. - GC Bulletin 721G
Esterification and Acylation
2) Supelco, Inc. - Bulletin 746F
Fatty Acid Methyl Esters
3) Folch et al., 1957. J. Biol. Chem. 226:467.

Procedures

1) Total lipids extracted from 1 ml colostrum in duplicate as described by Folch et al. (1957).

2) Methylation of fatty acids, GC Bulletin 721G
- Folch extract ($CHCl_3$ layer) is dried under N_2
- Add 2 ml BF_3-methanol[a]
- Add 2 ml heptane
- Boil on steam bath for 3 minutes
- Remove, add 1 ml dd H_2O to stop rxn and bring phase separation
- Sample heptane layer

3) Separate fatty acids with a flame ionization detector GC[b] on a 2 mm x 3 M glass column packed with SP-2330 on chromasorb.[a] Column should be conditioned overnight at 185 C.
- Program used:

Temperature, C	Time	Rate, C/min
70	2.0	5
135	.5	3
150	2.0	2.5
170	1.0	4.0
185	7.0	

- Attenuation 5.

4) Standards used:[a]
- RM-5
- RM-6
- C17:0 (internal standard)
- C20:4
- Standards methylated as in Step 2.

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[a]Supelco, Inc. Bellfonte, PA.

[b]Hewlett-Packard, Model 5890A.

EFFECT OF ENERGY SOURCE FED TO SOWS DURING LATE GESTATION ON SUBSEQUENT NEONATAL SURVIVAL, ENERGY STORES AND COLOSTRUM COMPOSITION

by

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Abstract

Two studies utilizing 24 primiparous sows and 34 multiparous sows stratified by weight and parity in a completely randomized design were conducted to evaluate the effects of isocaloric additions of wheat starch (ST), soybean oil (SO) and medium chain triglyceride (MCT) during late gestation (d 100 to parturition) on neonatal energy stores, survival at weaning and on colostrum composition. Diets were formulated to supply 7200 kcal of $ME \cdot sow^{-1} \cdot d^{-1}$. All sows received the same 14% crude protein diet ad libitum during lactation.

Maternal blood samples taken 2 h postprandial on d 110 of gestation were analyzed for insulin, glucagon and glucose. Both insulin and glucagon levels changed ($P<.05$) in response to dietary treatments. Insulin levels decreased in SO dams compared to ST, with a concomitant increase in glucagon for SO dams compared to ST. Medium chain triglyceride treatment resulted in an intermediate level for both insulin and glucagon compared to ST and SO. These hormone changes however, resulted in equal blood glucose levels for all treatments ($P>.10$).

One pig from each litter was killed prior to nursing and analysis of liver glycogen, total eviscerated carcass glycogen and total eviscerated carcass fat were accomplished. No differences ($P>.10$) in amounts of these energy stores were obtained.

Analysis of colostrum composition indicate a numerical trend for SO to increase total colostrum lipid. Fatty acid profiles of colostrum

indicate MCT is equal or closer to ST than SO for all fatty acids with the exception of C18:3. These data support the hypothesis that SO may be preferentially utilized by mammary tissue for milk production. However, it seems that MCT are not preferentially incorporated into milk. Rather, it seems that MCT sows synthesize colostrum lipids as would ST sows.

MCT increased weight gain prefarrowing over that expected for normal fetal growth and mammary development. It is possible that MCT slowed rate of passage thus increasing gastrointestinal fill.

Postfarrowing weight change indicates that MCT sows have the greatest loss ($P<.05$). Soybean oil sows gained weight during lactation.

No treatment effects were noted on percent survival, birth weight or weaning weight.

From the research reported herein, one may conclude that MCT is a poorer choice as a fat source during late gestation to produce more total colostrum fat. It would appear that substantial improvements in energy stores of the neonatal pig are difficult to produce by feeding either SO or MCT to the sow during late gestation.