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Studies on Factors Affecting Stored Energy Characteristics of Baby Pigs at Birth and Subsequent Changes During Early Neonatal Life

R. Harry Anderson

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STUDIES ON FACTORS AFFECTING STORED ENERGY CHARACTERISTICS OF **BABY PIGS AT BIRTH AND SUBSEQUENT CHANGES DURING EARLY NEONATAL LIFE**

 9.28

BY

R. HARRY ANDERSON

A thesis subnitted in partial fulfillment of the requirements for the degree Doctor of Philosophy, Major in Animal Science, South Dakota State University

1970

SOUTH DAKOTA STATE UNIVERSITY LUBRARY

STUDIES ON FACTORS AFFECTING STORED ENERGY CHARACTERISTICS OF BABY PIGS AT BIRTH AND SUBSEQUENT CHANGES DURING EARLY NEONATAL LIFE

This thesis is approved as a creditable and independent investigation **by** a candidate for the degree, Doctor of Philosophy, and is acceptable as meeting the thesis requirements for this degree, but without implying that the conclusions reached by the candidate are necessarily the conclusions of the major department.

Thesis Adviser

Date

Head, Animal Science Department

Date

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Arthur Line

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STUDIES ON FACTORS AFFECTING STORED ENERGY CHARACTERISTICS OF **BABY PIGS AT BIRTH Ai�D SUBSEQUENT CHANGES DURING EARLY NEONATAL LIFE** Abstract

R. HARRY ANDERSON

Under the supervision of Professor R. c. Wahlstrom

Studies designed to investigate the factors affecting stored energy levels of baby pigs at birth and changes in energy sources during early neonatal life were conducted over a three year period. During the three years 6C4 newborn pigs representing 85 different litters were partially or completely analyzed chemically at ages ranging from 0 to 72 hours. Various treatments of the dam during gestation and treatment of the newborn pig were utilized. The treatments used in the different trials were: (1) gestation weight gain of the gilts of either 0 to 10 kg or 10 to 20 kg, (2) energy intakes of 5924, 6183 or 6975 Kcal of metabolizable energy (ME) per day for 10 days prior to farrowing, (3) daily energy intake to meet the resting metabolic requirement of the gilt, resting metabolic requirement plus 1000 Kcal ME per day or resting metabolic requirement plus 2000 Kcal ME per day, for 10 days prior to farrowing, (4) feeding 0 or 800 mg of dichlorvos per sow per day for 21 to 30 days prior to farrowing, (5) administration of 2 g sucrose, orally, and 1 IU insulin, subcutaneously, per kg body weight to pigs at birth and (6) sacrifice and tissue sampling of pigs at O, 3, 6, 9, 12, 15, 18, 24, 36, 48 or 72 hours of age. Gilts gaining 10 to 20 kg during gestation produced pigs with higher liver glycogen and total body ash and lower liver fat,

blood reducing sugar levels and total body fat at birth than pigs_ from gilts **gaining** Oto 10 **kg** of body weight. Feeding gilts either 5924, **6183** or **6975** Kcal of ME per day for 10 days prior to farrowing had no effect on liver or muscle glycogen, liver fat or moisture of the neonatal pig. Gilts fed **at a** level **of** 2000 **Kcal** of **ME** more than their resting metabolic requirement per day for 10 days prior to **farrowing** produced pigs with **a slower** growth rate to **72** hr **of age thm** gilts fed either at their resting metabolic requirement or metabolic requirement plus 1000 Kcal per day. Pigs from gilts receiving 800 mg dichlorvos per day for 21 to **30** days prior to farrowing had higher liver glycogen levels and total body ash at birth, lower total body fat and moisture and less body weight gain to 72 hr of age. Administration of sucrose and insulin to the newborn pig produced lower serum glucose levels at 3 and 18.hr of age and higher serum. fructose levels at J, 6, 9, 12, 15 and 18 hr of age. Chemical analyses of liver, muscle and blood samples for the three years showed the following changes during the early neonatal period: (1) liver glycogen levels decreased from birth (11.5 to 14.9%) to 36 hr of age (3 to $4\frac{2}{3}$) and then increased to $5.0%$ at 72 hr of age, (2) muscle glycogen decreased steadily from 8.5 to **9.0%** at birth to **2.8%** at **72** hr of age, (3) liver fat increased from birth $(1.0 \text{ to } 1.6\%)$ to 12 or 18 hr $(2.5 \text{ to } 2.7\%)$ and then decreased to **72** hr· of age **(1.6%), (4)** liver moisture increased from birth **(73%)** to 18 hr **(77** to 78.5%) and then decreased to **76%** at **72** hr of age, (5) liver weight, expressed as a percent of body weight, decreased from **2.7%** at birth to **2.3%** at 18 hr and then increased to **3.2%** at 72 hr of

age, (6) total reducing sugar content of the blood increased from **birth (120 mg per IOD:· mI} to 12 hr (124 mg per 100 ml), decreased to· 118 mg per 100 ml at_ Iff hr· and then increased to 128 mg per 100 ml at 72 hr of age, (7) serum triglycerides increased from 190 mg per 100 ml** at birth to 305 mg per 100 ml at 36 hr of age, (8) serum glucose increased from 50 mg per 100 ml at birth to 122 mg per 100 ml at 36 hr of age, (9) serum fructose decreased rapidly from birth (41 mg per 100 ml) to 24 hr of age (4.0 mg per 100 ml) and then decreased slightly to 3.5% at 36 hr of age, and (10) total urine sugar content of the urine decreased from birth to 36 hr of age. Changes in total **body composition. during_: the first ?2 hr of life were characterized** by: (1) decreased moisture content from 82.87% at birth to 79.56% at **72 hr of age.** (2) increased fat content from birth $(6.73%)$ to 72 hr **of age (20.99%), (3) increased protein content from 54.26% at birth** to 56.47% at 24 hr of age and then decreased protein content to 72 hr of age $(52.53%)$, and (4) decreased ash content from $16.47%$ at **birth to 13. 74% at 7Z: hr:- of age.**

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDIX TABLES

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Hoy all yes for Se line creations positionities to the Index:

INTRODUCTION

Improvanent of reproductive efficiency is one area of swine research that is receiving much interest at the present time. Economically, it is far more important for the producer to increase efficiency of production in terms of pigs marketed per sow farrowed than simply an increase in number of sows farrowed. Such factors as male fertility, ovulation rate, conception rate, fertilization rate and embryonic mortality can affect reproductive efficiency. However, before these factors can exert their maximum infiuence on the reproductive capabilities of swine, the problem of post-natal mortality must be resolved.

At the present time, neonatal death loss does not allow realization of the full advantage from increasing litter size due to improved ovulation rate and fertilization rate. Causes of neonatal death loss and prefarrowing factors affecting livability of pigs are many and complex. The literature is not consistent as to the factors which determine the relative livability of the pig at birth. Dietary factors during gestation have been studied extensively to determine what effects nutrition of the sow have on birth weight and livability of the newborn pig. However, there is no well established feeding regime that will consistently produce optimum productivity in sows.

In most studies dealing with swine productivity such things as birth weight, survival rate and growth rate are of main concern in evaluating the well being of the newborn pig. Recently there has been more interest in the physiological and biochemical well being

or the newborn and how these relate to neonatal mortality. More precisely the energy status at birth and utilization of energy sources immediately following birth have now become important areas of study when considering neonatal mortality. The work reported in this thesis attempted to investigate the aforementioned factors in swine productivity and newborn pig livability. The objectives of the investigation were as follows:

1. To characterize some of the stored energy sources of the pig at birth.

2. To measure the changes in the same stored energy sources at various time intervals following birth.

J. To study the effects of treatment or the sow during gestation on birth weight, litter size, growth rate and stored energy characteristics of the newborn pig.

4. To evaluate the administration of insulin and sucrose at birth and its effect on energy utilization by the newborn pig.

REVIEW OF LITERATURE

Gestation Energy Lavel

Some of the earliest work done in the area of sow nutrition and its effect on reproductive performance was that of Hammond (1921) in which a substantial increase in ovulation rate was accredited to high energy feeding at breeding time. Christian and Nofziger (1952) compared full feeding to 70% of full feeding in prepuberal gilts through first pregnancy and found full feeding produced greater ovulation rate but much greater embryonic mortality. This allowed for a large advantage to the 7� full fed group in litter size. Self, Grummer and Casida (1955) found a high energy intake during the first 25 days of gestation to be detrimental to litter size. King and Young (1957) also studied the relationship between energy intake of the sow during gestation and reproductive performance and found no noticeable difference between feeding 2.27 or 4.54 kg per day of a high energy ration. However, most of the work concerning gestation energy intake has been done during the last seven years and will be of primary concern in the following discussion.

Clawson et al. (1963) studied the effects of two levels of energy intake by the gravid gilt and found no differences in pig survival, number of pigs weaned or litter weight at weaning. The gilts were fed either 1.36 or 2.73 kg of a diet which was adequate or equal in all nutrients except energy at both levels. Gilts fed the high energy treatment gained significantly more weight during gestation than those on the low energy treatment. These authors concluded that the low energy intake was adequate for reproduction. Gilts receiving the higher energy level did farrow heavier pigs; however, no difference was observed at the time of weaning.

Meade et al. (1966) compared the effects of feeding 2.72 kg of feed throughout gestation to feeding an increasing plane of nutrition during gestation. The first increasing feed treatment consisted of feeding 1.82 kg for the first 28 days, 2.27 kg during the second 28 day period and 2.72 kg during the remainder of gestation. The other increasing feed treatment consisted of feeding 1.36, 1.82 and 2.27 kg of feed during the first 28 days, second 28 days and the remaining days of gestation. respectively. These authors found no differences in the number of live pigs per litter, average birth weights, number of pigs per litter at 21 days or 21-day pig weights. This is in agreement with Seerley and Magstadt (1966) who fed either 1.82 or 1.36 kg from 21 to 70 days of gestation, 2.27 or 1.82 kg from 70 to 93 days and 2.27 or 4.09 kg from 93 days to term. Similar results were reported by German, Seerley and Wahlstrom (1967a) under the same feeding regime as Seerley and Magstadt. In these two trials, the total feed intake during gestation was equal between treatments, and only during specific periods of gestation was feed intake different.

Frobish, Speer and Hays (1966) studied the effects of two energy levels on reproductive performance of sows through three reproductive cycles. When sows were fed either 10,800 or 5,400 Kcal or metabolizable energy·per sow per day, they found no differences in number of pigs farrowed alive, birth weight of live pigs or pig

gain from birth to weaning. This would indicate that 5,400 Kcal metabolizable energy is at least adequate for reproduction in sows through more than one reproductive cycle.

Lodge, Elsley and MacPherson $(1966a,b)$ also reported a gestation energy level study conducted over three reproductive cycles of Large White gilts. They used 12 sets of 3 littermate groups and fed them 2.73 kg per day during gestation, 1.36 kg per day during gestation or 1.36 kg per day for 76 days and 2.73 kg per day until parturition. **There** were highly significant differences in body weight gain between treatments. There was also a significant parity interaction in that **sows** receiving 2.73 kg throughout gestation showed a significant decline in net weight gain from parity to parity, whereas sows in the other two groups did not. A significant linear increase was found in numbers of pigs born with successive parities but no significant differences between treatment or sister groups. Mean piglet birth weight was greater for pigs-born to sows fed 2.73 kg during the entire gestation period and from 76 days to parturition than from sows fed 1.36 kg during the entire gestation period. However, there were no significant differences in growth performance of the pigs from birth to weaning due to treatment, sister-group or parity. In a similar **trial** covering three reproductive cycles, 0¹ Grady (1967) fed 1.36 kg for the entire gestation period or J.64 kg during the first and fourth months and 1.82 kg during the second and third months. Results in this trial also showed that litter size increased with parity, treatment had no effect on litter size and birth weight was greatest

on the higher energy intake. However, there were differences in total litter weight at weaning which is in contrast to the work of Lodge et al. $(1966a,b)$.

Mayrose, Speer and Hays (1966) fed either 2. 67 or J.18 kg of feed per day at the time of breeding and/or during the last third of gestation to study the effects on reproductive performance. Sows fed the higher level both at breeding time and during the last third of gestation farrowed fewer pigs than did sows on the other treatments. Sows fed the high level at breeding time farrowed significantly heavier pigs than did those fed the low level. Increasing the level of feed intake during the last third of gestation significantly increased sow weight gains but did not affect birth weight of pigs.

Frobish (1968) used daily energy intakes of either 6, 000 or 3, 200 metabolizable Kcal to study the relationship between energy level and reproductive performance in gravid sows. The treatments used were 3,200 Kcal metabolizable energy (ME) throughout gestation, 6, 000 Kcal ME throughout gestation, 3,200 Kcal during the first half of gestation and 6,000 Kcal during the second half of gestation or 6, 000 Kcal during the first and 3,200 Kcal during the second half of gestation. Significantly fewer pigs were farrowed by gilts receiving 3,200.Kcal ME per day throughout gestation than the other three treatment groups. Significantly more pigs **were** weaned by gilts on the $6,000$ Kcal per day treatment than gilts on the $3,200$ Kcal per day treatment throughout gestation. No significant differences in birth weight or weaning weight of pigs were observed between treatment groups.

Significant differences in number of pigs farrowed, average birth weight and weaning weight were reported by Vermedahl et al. (1968) when comparing daily feed intakes of 1.36 and 2.27 kg during gestation. Sows fed the higher level of feed intake farrowed larger litters with pigs having greater birth and weaning weights. Similarly, Baker et al. (1969) reported that increasing feed intake increased birth weight and weaning weights. However, they found no difference in number of pigs born per litter. Daily feed intakes of 0.9, 1.4, 1.9, 2.4 and J. O kg were used in this trial and weaning weight increased as feed intake increased from 0.9 to 2.4 kg per day. Birth weight plateaued at the 1.9 kg per day feed level and weaning weight plateaued at 2.4 kg feed per day. In contrast to the work of Vermedahl et al., German, Seerley and Wahls rom (1967b) reported that l.J6 kg feed per day during gestation may be adequate when compared to 2.27 kg per day.

Dichlorvos

Dichlorvos (2.2-dichlorovinyl dimethyl phosphate) has recently been shown to be an effective anthelmintic for swine. Jacobs (1968) treated 93 sows with dichlorvos resin pellets $(9%$ active ingredient) and stated that 1.44 g of active ingredient was adequate to control several parasitic species. England et al. (1969) studied the effect of feeding 800 mg dichlorvos per day to nine-month-old gilts on blood sugar level, ovulation rate and embryonic survival. Treatments used were dichlorvos 21 days before and 25 days after breeding, dichlorvos 21 days before breeding, dichlorvos 25 days after breeding or no

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dichlorvos during either period. All gilts were slaughtered at day 25 of gestation. Statistical analysis did not indicate any significant advantage in corpora luten. live or dead embryos or any change in blood glucose of the gilt.

Batte, Robison and Moncol (1968) fed dichlorvos at a rate of 800 mg of the coated resin pellet per sow per day for various lengths **of time ranging from t�e:e: weeks before breeding through gestation and** from 21 to 42 days before parturition. Number of pigs born alive, **birth weights, survivaI. and growth rates to weaning of pigs from dichlorvos treated dams: were significantly greater in comparison to untreated controls. At_ birth . offspring from dichlorvos treated dams had significantly higher- blood glucose, liver and muscle glycogen values in comparison. ta cnntrol offspring. Under conditions of complete starvation for the: first 24 hours of life significantly** higher values were also observed for the same measurements in offspring from dichlorvos treated dams as compared to control dams. Singh, **Perkins and Schooley (lg6.8 :) found similar results in regard to efficacy of dichlorvos- in. :increasing number of pigs born alive, birth and weaning weight and. survival rate to weaning. They fed dichlorvos** for 21 to 30 days prior to farrowing at 400 or 800 mg per day in the **feed or 200 mg per day· m- the drinking water.**

Batte, Robison and Moncol (1969a) reported on feeding levels of **O, 400 and 800 mg dich:1or.vos.:: to- sows. They used 472 sows and fed dichlorvos from three weeks:. prior to breeding through gestation or from 21 to 42 days before parturition. Inditidual pig weights and**

litter weights at farrowing and at weaning were highly significantly greater in dichlorvos treated dams. In another trial Batte, Robison **and Moncol (1969b) fed:. 800 mg of dichlorvos daily from three weeks before breeding throug}l gestation or fran 18 to 56 days before** parturition. Data were from 681 sows representing eight replicates **over a two-year· per.i.od:.. Individual farrowing weights , litter farrowing weights ,. number weaned, individual weaning weights and** litter weaning weights all favored dichlorvos treatment in six of **eight replications.**

Foster (1968) studied the effects of dichlorvos in sows and **gilts when given aa: a.: single large dose compared to low level daily dosages. In the. first. t-rial dichlorvos was given as a single dose of** 1.34 g 14 days prior to farrowing. This treatment resulted in more live pigs and fewer stillborn at birth, heavier litter weights at birth and weaning as well as decreased mortality from birth to weaning. In the second trial dichlorvos was fed at 800 mg per day for the final three to six weeks of gestation. This treatment **resulted in fewer- stillbirths , heavier litter weights at birth and** reduced mortality from birth to four weeks of age. In both trials dichlorvos was of greater advantage when fed to gilts than to sows.

In contrast to the above results, Hays, Overfield and Cromwell (1969) **and** Hollia **(I-9,69) found no significant advantages in litter siz e, birth weight:,**1 **wemiing weight or survival rate when feeding** dichlorvos at 800 mg per day for 21 to 42 days before farrowing. **Bazer, Robison and Ulberg** (1969) **fed dichlorvos at 800· mg per day**

beginning 21 days before breeding and found no significant effect on number of pigs born alive, litter weight or pig weight at birth. They also injected one group with **1, 500 IU** pregnant mares serum **(FMS)** on day **16** of treatment. Both **FMS** and dichlorvos increased corpora lutea count at 25 days postbreeding when compared to control gilts but had no effect when the treatments were combined.

The Newborn Pig

The ability of the newborn pig to utilize dietary sources of certain carbohydrates is limited. Becker et al. (1954) compared the feeding of sucrose, D-glucose, D-fructose and an invert sugar to young pigs and found them unable to utilize sucrose and D-fructose for energy. D-glucose was found to be well utilized and produced normal gains. Severe diarrhea occurred when pigs were fed sucrose or Dfructose but did not occur when glucose was fed. The invert sugar was utilized at an intermediate rate between D-glucose and sucrose or D-fructose. It produced some diarrhea but not as severe as with sucrose and D-fructose. However, there was no obvious relationship between utilization and severity of diarrhea. Cunningham and Brisson $(1957b)$ studied the rate of digestion of maltose by newborn pigs through the use of digestion trial and intestinal loop technique. **Orally** ingested maltose was 97.4% digested by 2- to 5-day-old pigs. The rate of digestion was 0.72 g per kg of body weight per hour. Maltose injected into tied-off small intestines of 0- to 4-day-old pigs was digested at the rate of **0.-62** g per kg of body weight per hour. Cunningham (1959) also canpared carbohydrate utilization by

the newborn pig. Raw corn starch was compared to maltose and glucose. both of which are degradation products of starch. Glucose, maltose and a soluble starch solution were all digested more rapidly than raw starch. When raw starch was introduced directly into the intestine, the addition of pancreatic amylase did not improve the utilization. These findings are in agreement with Cunningham. and Brisson (1957a) where the addition of pancreatic amylase had no beneficial effect on the digestion of raw or cooked starch in 2-day-old pigs. It was summarized from these trials that initial rupture of the starch granule is the limiting factor in the utilization of raw starch by newborn pigs. Hudman et al. (1955) compared three sources of sugar (all cane, half cane-half corn and all corn) at three different levels (15, 25 and 35 percent of diet) in one- to three-week-old pigs and found no differences in gain or feed efficiency.

In an attempt to delineate the causes of variable utilization of carbohydrates by the newborn pig, Dahlquist (1961) studied the intestinal carbohydrase activity in the newborn pig and found no invertase activity and only small amounts of maltase and amylase. Lactase was found to be present in the highest concentrations at birth. This would explain part of the differences in utilization or carbohydrates by the pig as described above. When Sprague et al. (1963) studied intestinal lactase levels in fetal and newborn pigs, it was found that there is a significantly greater amount of lactase present at birth than in fetal pigs at JO, 51. 72 or 93 days post breeding.

Glucose is an important component of baby pigs' blood as well as being efficiently assimilated from a dietary source. According to Goodwin (1957) the metabolism of the newborn pig is governed completely by the concentration of circulating glucose. Starvation in a newborn pig produces hypoglycemia, which can result in a decline in general metabolism if severe enough. This is evidenced by a reduction of the heart and respiratory rates and a lower body temperature. If the bI.oDd: glucose concentration or the newborn pig is reduced with insulin, a syndrome of complete collapse develops which is indistin-�hable from the state that is seen during starvation. As the baby pig gets older, it becomes more refractory to starvation with hypoglycemia being much harder to produce.

Plasma glucose level is low at birth in the pig, but there is a significant increase after the first nursing (Bengtsson et al., 1969). Following first nursing, a similar level of plasma glucose persists throughout the first few weeks of life. Swiatek et al. (1968), when studying starvation hypoglycemia, described a similar change in blood glucose levels immediately following birth. Blood glucose levels in ho::tn-. the fed and fasted groups increased from approximately *50* mg per IOD ml blood at birth to approximately 80 mg per 100 ml at six hours of' ag_e.l By 24 hours of age the fasted pigs had lowered blood glucose Levels similar to those at birth, while blood glucose of the fed pigs increased to 110 mg per 100 ml. In the fed pigs this level persisted through three weeks of age.

A HOLE EAST

Curtis, Heidenreich and Foley (1966) fasted newborn pigs from birth to eight hourss of: age and found an increase in blood glucose from 80 mg per 100 ml at birth to 140 mg per 100 ml at eight hours of age. During the same period of time blood fructose decreased from 70 mg per lOO_ nrI at: birth to JO mg per **100** ml at eight hours of age. During the treatment: period rectal temperature decreased from 39° to 36.5° C. indicating the deleterious effect of fasting on metabolism of the newborn pig. Also, when these pigs were cold stressed from 7.5 to 8.0 hours after birth, the blood glucose levels increased more rapidly and rectal temperature decreased more rapidly than when maintained at the same ambient temperatures.

While studying the relationship between birth weight and blood sugar concentrations: at various intervals after farrowing, Frobish (1969) reported an increase in plasma glucose and a decrease in plasma fructose from birth: to: 24 hours of age. Plasma glucose concentration was found to increase from 91.5 mg per 100 ml at birth to 111.2 mg per 100 ml at 24 hours, while plasma fructose decreased from 30.3 mg per 100 ml to 5.8 mg per 100 ml during the same time period. Within litters, the correlation coefficient for fructose concentration and birth weight was 0.03 , while for glucose concentration and birth weight it was 0.11. It was also reported that for each one mg increase in glucose concentration there was a 0.11 mg increase in fructose concentration. Similar changes in blood sugar concentrations following birth were reported by Aherne et al. (1969) while studying glucose and fructose concentrations in the blood of fetal and newborn

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pigs. Glucose levels:were found to increase from 82 mg per 100 ml **, at birth to 102 mg_- pe . IO0� ml at 12 hours and decrease again to 89 mg per 100 ml at. 48- h�, Fructose levels decreased from 48.0 mg per 100 ml at birth ta- 4-.B� mg- per 100 ml at 48 hours. Urinary fructose** was also measured and it was found that fructose excretion over the first 48 hours of life was considerably greater than the apparent loss from the blood.

The fact that the newborn pig has a low inherent ability to assimilate and utilize certain carbohydrate sources has been established. This is an important factor in explaining the newborn pig's inability to adapt rapidly to a lower than optimum environmental temperature. Pomeroy⁽¹⁹⁵³⁾ described the following changes that take place shortly after birth in rectal temperature of the pig. A **newborn pig may· experⁱenc e: a decline in rectal temperature from 40. 0 to 40.5° C at birth. to: JT.8 ° C in the first few minutes of life. The** rectal temperature will continue to fall until the pig begins to suckle at which time a temporary increase is seen. When the pig rests, the temperature drops again until the time of next suckling. Their rectal temperature does not return to normal until about 12 hours after birth. If deprived of milk for any reason, the baby pig will **experience a steady- :f.a.ll. in rectal temperature and become lethargic** and apathetic. When the rectal temperature reaches 35^o C, they become comatose and diesifimilk is not given. The rate and extent of rectal temperature decline is dependent upon the external environmental **temperature (Mount, 1968) and the amount of milk available. This**

phenomenon is in contrast to the domestic calf which shows a remarkable independence of the environment (Roy, Huffman and Reineke, 1957) and the lamb which has a body temperature close to the mature level approximately an hour after birth (Alexander and McCance, 1958).

The only major source of stored carbohydrate that the newborn **pig can utilize:: toc maintain blood glucose levels and ultimately its** vital body functions during a time of starvation is the glycogen stores in the liver and muscle tissues. Liver glycogen stores in **fetal pigs were:- observed by Itoh and Hansard (1965) anJ found to be** at maximum levels: by 112 days postbreeding. Breed of sow and fetus, fetal age and feed intake were all shown to have a direct effect upon total fetal glycogen storage in livers of developing swine. Swiatek et al. (1968) reported relatively high levels of liver **glycogen at.. birth·. (14. 8%) and a rapid decrease to 18 hours of age (3.7�) where it r-emained relatively constant to 72 hours of age. In** contrast, pigs fasted: from birth had liver glycogen levels of 0.2% **at 18 hours and: r.emained at that level when fasted until 72 hours of age.**

One of the most important mechanisms of carbohydrate metabolism control in the body is insulin secretion by the pancreas. Any change **in glucose concentration in the blood from normal levels will con**currently change the pancreatic output of insulin (Grodsky et al., 1963). Insuling release increases with increases in glucose concen**tration and decreases� with decreases in glucose concentration.** Fructose is also capable of stimulating insulin release by the

P.ancr.eas (Rieser, 1967). Also, insulin adm�istration has a direct effect on the carbohydrate metabolism of the body. As insulin levels in: the blood increase, glucose levels decrease (Asplund, Grummer and **ffiillips**, 1962), hepatic glycogenolysis is decreased (Steele et al., 1965) and hepatic glycogenesis is increased (Bishop et al., 1965). Insulin has further effects of increasing triglyceride synthesis in adipose tissue as well as enhancing amino acid transport into the **ceDJ. (Rieser , 1967).**

At birth, the pig has a very low level of blood insulin (Swiatek et al., 1968) which does not reach normal levels until six **hour.s: o'f. age . This increase in insulin level during the first six hours.: of. life corresponded to the period of most rapid increase in 1no:od: gl.ucose levels and liver glycogen loss reported by the same** authors. Bowie, Mulligan and Schwartz (1963) reported that human infants also have very low levels of insulin present at birth and **theor.iz·ed it to be the reason for the infant• s inability to efficiently utilize glucose during the first few hours of life.** Machlin et al. (1968) reported that even at 50 kg of body weight pigs **have. a· - Iowered ability to adapt to exogenous glucose sources compared to: other species as evidenced by a smaller increase in plasma insulin** levels.

EXPERTMENTAL PROCEDURE

The research reported here consisted of three separate trials extending over a period of three years. Each trial was similar in design and objective to the other two, yet each was different in scope and treatments used. In designing the second trial, an attempt was made to expand upon and utilize the results of the first trial, and in turn the third trial was an attempt to use what was learned from trials 1 and 2.

Trial l

Eighteen, 8-month-old, primiparous gilts were used in this trial. Nine Hampshire gilts were bred to a Duroc boar and nine crossbred gilts were bred to a Hampshire boar. During gestation, all gilts were maintained in a dirt lot with uninsulated wooden sheds for shelter and fed a corn-soybean ration ad libitum (table 1) until day 104 of the gestation period at which time they were placed in the farrowing house and fed 1.82 kg per day of one of the three experimental rations. The three rations consisted of (1) com-soybean basal. (2) basal + 15 percent sucrose and (3) basal + 15 percent prime yellow grease. When fed at the level of 2 kg per day, these rations furnished 6510, 6794 and 7666 Kcal of metabolizable energy per day, respectively. At the time of farrowing, each pig was earnotched to identify it by time of birth and order of birth within the litter and randomly assigned to one of four time treatments. Two or three pigs, depending upon whether the gilt had more than eight

Ingredient	% of ration
Ground yellow shelled corn	65.5
Ground cats	10.0
Dehydrated alfalfa meal (17%)	10.0
Soybean oil meal (44%)	12.0
Dicalcium phosphate	1.8
T.M. salt.	0.5
Vitamin mix ^a	

TABLE 1. RATION CCMPOSITIW

&. **Pr.ovided the following per kilogram of diet : 4994 IU vitamin A, 492..8 IlL vitamin D, 8.8 mg riboflavin , 17.6 mg pantothenic acid , 39.6 mg_niacin, 44.0 mg_choline chloride and 14.5 ug_vitamin B₁₂.**

or more than twelve pigs, were sacrificed at 0 hr. 12 hr. 24 hr or 36 hr. after birth and liver and muscle samples were collected. The collection procedure was as follows: the pig was jugulated and as rapidly as possible the liver was removed and a 3 to 5 g sample was **placed. :i.ir.** a;, **tared test tube containing 25 ml of 30% potassium hydroxida., The remainder of the liver was placed in a bottle and** frozen. A 3 to 5 g sample of semitendinosus muscle was also removed **as rapifily· as possible and was placed in a tared test tube containing 25 mI. o^f3-0'% potassium hydroxide. The liver and muscle samples in the.. po.tassium hydroxide solutions were quantitatively analyzed for** glycogen content by the isolation method of Cowgill and Pardee (1957) immediately following collection. The remainder of the liver was **stored iir: a: conventional deep freeze for fat analysis at a later date**

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(A.O.A. C., 1965). Least squares analysis was performed on the data collected and F values were used to test for significance (Harvey, 1960).

Trial 2

Fifty-two, 10-month-old, 175 kg, crossbred, primiparous gilts were randomly allotted to four groups of 13 gilts per group. All gilts were estrous synchronized by groups with ICI-33828 **(Aimax)** to allow a 2-week interval between farrowing of each group. Each gilt was randomly mated to one of nine Yorkshire boars 12 hours after the beginning of standing estrus. Each group of 13 gilts was randomly **assigned** to one or the treatments presented in table 2. All gilts were maintained in dirt lots with uninsulated wooden sheds for shelter and group fed 1 to 2 kg of a high fiber alfalfa and oats diet formulated to meet the National Research Council recommendations **(N** .R.C. , 1968) when fed at 1.5 kg per gilt per day (table 3). This diet was calculated to contain 2202 Kcal of metabolizable energy per kg. Individual weekly weights were used to determine the level of feeding to be used during the following week to keep the weight gain within the designed weight gain range.

On day 104 of gestation, each gilt was weighed, placed in a clean farrowing pen and randomly assigned to one of three prefarrowing energy level treatments. The prefarrowing energy level rations were designed to vary the energy intake without altering the daily intake of feed and were formulated as follows: (A) gestation diet, (B) gestation diet containing 15% added sucrose and (C) gestation diet

TABLE 2. GESTATION SOW TREATMENTS

a **Period** from **breeding through day** 104 of **gestation�**

b Fed for 21 **to JO** days **before farrowing.**

TABLE 3. RATION COMPOSITION

186.5 g **crude** protein/kg

a Provided the following per kilogram. of diet : 5993 IU vitamin A, 44o IU vitamin D, 13.2 mg riboflavin, 24.J mg pantothenic acid, 58.4 **mg** niacin and 22 ug vitamin B12.

containing; IS� **added lard. Diet A was fed at a rate calculated to** meet the resting metabolic requirement of the gilt (Brody, 1945), diet B was fed: to provide an additional 1000 Kcal of metabolizable energy **(ME)** per day and diet C an additional 2000 Kcal ME per day. All diets were fed at levels of 0.85 to 1.0 kg per day according to the resting **metaboli� ��anent of the gilt. Each gilt** was **weighed within 6 hours of farrowin� and: again immediately after birth of the last pig. As** each pig was born, it was weighed and earnotched to identify it by **litter and: or.der� o£ birth within the litter. The time of birth for** each pig was recorded and it was randomly assigned to one of eight **time treatment�... The time treatments indicated the hour after birth that the pig; was: to. be euthanized and samples taken. The time treatments wer� � follows: (1) O, (2) 6, (3) 12, (4) 18. (5) 24, (6) 36, (?) 48.. and:. (8:))** *7Z-***hours after birth.**

Flach: pig; was· euthanized at the assigned time by the following procedure... The pig was weighed and placed in a holder designed to **secure it** m·. **such . a position as to present the ventral. side to the experimentor.· with . each leg held fast with a heavy rubber b and. A 10 ml blood sampie�. was obtained by cardiac puncture and stored for later blood sugar�ana.Iysis. The pig was euthanized by in jecting sodium** pentobarbital into the heart. As rapidly as possible, the liver was removed in toto and a 3 to 5 g sample was placed in a 5 ml polyolefin container which was immediately capped, labeled and frozen in liquid nitrogen for future liver glycogen analysis. After removing the gall bladder and common bile duct, the remainder of the liver was placed

in a 60 ml plastic: bottle and frozen for use in liver moisture and fat determinations. Al2to₂ g sample of semitendinosus muscle was also placed in a 5 ml polyolefin container, labeled and frozen in liquid **nitrogen to be: uaed: f.or muscle glycogen analysis. Liver and muscle** glycogen were measured by the method of Cowgill and Pardee (1957). Official methods of analysis (A.O.A.C., 1965) were used for liver moisture and fat. Total reducing sugar content of the blood was **measured color.iine�y using the method of Folin and Wu (Bausch and Lomb).**

Following removal of the blood. liver and muscle samples, the entire carcass: was placed in a plastic bag and frozen for whole body analysis. This was done only with pigs on time treatments 1, 5, 7 and 8. When time permitted, the frozen carcasses were ground intact through a meat grinder with 3.175 mm diameter holes. A sample was then taken of the ground material and analyzed for moisture, fat, ash and nitrogen content. The entire sample was dried to constant weight in teflon pans to obtain moisture content. The dried material remaining was then ground in a large mortar to a powder and stored for ash, fat and nitrogen determinations. Official methods of analysis **(A.O.A.C., 1965)** were then used to determine ash, fat and nitrogen content of the dried samples.

The data were analyzed by the least squares analysis method **of Harvey (1960).** *c*

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Twenty-eight first and second litter' gilts and **sows ,** weighing approximately 100 kg and 165 kg, respectively, were bred by artificial insemination according to the following scheme. Two Duroc sows were bred to a Yorkshire boar, six Yorkshire sows were bred to a Duroc boar and twenty. crossbred gilts were bred to a Yorkshire boar. All gilts and sows were fed in individual feeding stalls during gestation and received the high fiber alfalfa and oats diet described in table 3. All animals were maintained on pasture or dirt lot with uninsulated wooden sheds for shelter and received 0.5 to 1.5 kg of ration daily to control weight gain and allow a total gain of 10 to 20 kg during gestation. Weekly weights were used to determine level of feeding for the following week. On day 109 of gestation, each gilt or sow was washed, placed in a clean farrowing pen and randomly assigned to one of two litter treatment groups. Allotment was conducted to allow for breed groups and littermates. At this time, all gilts and sows were fed the high fiber diet at a level to maintain body weight until parturition.

Within six hours of parturition each sow was weighed and her rectal temperature was measured and recorded. Rectal temperature was again measured and recorded during active parturition and the sow was weighed. immediately after discharging her placenta and before any feed or water was allowed. As each pig was born, it was weighed and earnotched to identify it by litter and order of birth within the litter and the rectal temperature was recorded. The time of birth was

recorded and each of the first nine pigs born was randomly assigned to one of nine time treatments. The time treatment indicated the hour after birth that the pig was to be euthanized and samples collected. The time treatments were as follows: (1) 0, (2) 3, (3) 6, (4) 9, (5) 12, (6) 15, (7) 18, (8) 24 and (9) 36 hours after birth. Pigs born to the seven gilts and three sows in group 1 served as controls and received no treatment. Pigs born to the eight gilts and four **sows** in group two received the following treatment at birth: 1 IU per kg of body weight of an insulin mixture containing 0.5 IU regular insulin, 0.25 IU NPH insulin and 0.25 IU Protamine zinc insulin in a 4 unit per ml solution given subcutaneously and 2ρ of sucrose per kg body weight given orally in a 13.5% solution. One Yorkshire sow and five crossbred gilts did not farrow.

Each pig was euthanized at the assigned time and samples collected using the same procedure as in trial 2. However, in addition to muscle, liver and blood samples, the pancreas and adrenal glands , were removed and weighed. A urine sample was collected and frozen for use in determining the presence of sugar. Immediately following collection, the blood sample, which was allowed to clot, was centrifuged and the serum. removed. A sample of the serum was then deproteinized by diluting 1:10 with 3� trichloroacetic acid and centrifuging to precipitate the protein. The protein-free sample was frozen for glucose and fructose analyses. The remaining serum was frozen for serum triglyceride analysis.

Liver and muscle glycogen were measured by the method of Cowgill and Pardee (1957). Official methods of analysis (A. O. A.C.,

1965) were used for liver moisture and fat. Serum triglyceride levels were measured by the method of Stern and Shapiro (1953). Serum glucose was measured by the glucose-oxidose method. The following modification or the method outlined by Roe (1934) was used in determining serum fructose. Serum was deproteinized by mixing 1:10 with 3� trichloroacetic acid rather than mixing whole blood 1 :7:1:1 with H20, 10% ZnS04• H20 and *0.5* N NaOH, respectively. Following reaction of the deproteinized serum with 30% HCl and 0.1% alcoholic resorcinol in an 80° C water bath for 8 minutes, they were read in a spectrophotometer at 510 my and compared to a 10 or 25 mg per 100 ml standard, depending on which standard was the closest to the unknown. The presence of sugar and glucose in the urine was detenmined by the use of Clinitest R and Clinistix R . Pooled litter data were analyzed statistically by the method of least squares analysis (Harvey, 1960). Within time treatment, data were analyzed by analysis of variance (Steel and Torrie, 1960).

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RESULTS AND DISCUSSION

$Trial 1 (1967)$

Treatment averages and the averages of pigs in all treatments for each time interval are presented in table 4. Altering energy intake of the gilt for ten days prior to farrowing did not significantly affect liver glycogen, muscle glycogen, liver fat or liver moisture content of the pigs at birth, 12, 24 or 36 hours after birth. There was a large variation between treatment groups for liver glycogen at each time interval. Pigs from gilts on the lowest energy level had the highest liver glycogen levels at birth (15.83%) but the lowest at 36 hr after birth (2.06%) . Pigs from gilts on the intermediate energy level had the lowest liver glycogen levels at birth (14.00%) and were intermediate between the other two treatment groups by 36 hr of age $(2.63%)$. Pigs from gilts on the highest energy level had the intermediate liver glycogen levels at birth (15.10%) and reached the highest level by 36 hr after birth (3.91%). The levels at 12 and 24 hr after birth also indicated that no one treatment group consistently had higher or lower liver glycogen levels than the other two groups. Muscle glycogen levels were similar between treatment groups at all time intervals. At birth, gilts on the low, intermediate and high energy intakes produced pigs with muscle glycogen levels of 9.21, *_ 9.05* and 9.01%, respectively, and at 36 hr of age the levels were $4.76.$ 4.80 and 4.40% for the same groups. respectively. A large amount of variation between 1itters was found within treatment and breed groups for liver and muscle glycogen.

TABLE b . TREATMENT MEANS AT VARIOUS TIME INTERVALS AFTER BIRTH FOR TISSUE DATA

^a All differences due to age of the pig were highly significant $(P < 0.005)$. Least squares analysis of variance presented in appendix tables 1 and 2.

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 \overline{b} H = Hampshire, D = Duroc and C = Crossbred. **^Cp** *<. 005.* her of lane, transactivelers

This was evidenced by highly significant $(P < .005)$ differences for the interaction of litter within treatment of the gilt times breed. Although not significant, pigs from crossbred gilts bred to a Hampshire boar produced pigs with higher liver glycogen (15.75 vs. **· 14. JO%) and: lower. · muscle glycogen levels** (8.81 **vs.** 9. 30%) **at birth than Hampsh� gilts bred to a Duroc boar. By 36 hr after birth** higher levels of liver glycogen were found in pigs from Hampshire **gilts bred· . to:- a;_Duroc boar than pigs from the crossbred gilts bred to a** Hampshire boar $(3.32 \text{ vs. } 2.23\%)$. However, pigs from the crossbred **gilts had. muscl:e- glycogen levels of** 6.68, **5. 42 and 4. 01% at 12, 24 ·** and 36 hr after birth, respectively, which were consistently lower **than the 7.69�1** 7� 03 **and** *5.15%* **for pigs from Hampshire gilts at the** same respective time intervals.

The_ data: in table 4 show a great amount of variation in liver fat content within gilt treatment groups as well as within time . intervals. No: consistent difference existed between treatment groups for liver fat. However, there was a highly significant difference **(P <.005) irr. liYer fat due to breed of the pig. Pi gs from crossbred** gilts and sired by a Hampshire boar had liver fat values of 1.64 , 3.15, **J. 15 and: Z. 79% at birth, 12, 24 and 36 hr of age, respectively,** which were significantly higher than the 1.52, 2.18, 1.93 and 2.02% **at the same� �espective time intervals for pigs fran Hampshire gilts and sir� by: aa. Duroc boar. It is possible that the breed difference in liver fat: content is an indicator of hybrid vigor reported for crossbred dams. A higher level of stored fat in the liver may be**

associated with increased vitality and growth rate of crossbred pigs reared by crossbred dams.

Figure 1 shows the levels of liver and muscle glycogen and liver fat and moisture at birth, 12 , 24 and 36 hr after birth. The values used in constructing the figure are the overall averages, regardless of treatment. Liver glycogen decreased progressively from birth to 36 hr. Levels were 14.92, 6.40, *3.56* and *2.85%* at birth, 12, 24 and 36 hr, respectively. These changes are in close agreement with the work of Swiatek et al. (1968), who described changes of 14.8% at birth to 3.7% at 18 hr and 3.3% at 48 hr after birth. These changes in liver glycogen indicate the importance of glycogen stores at birth as a readily available source of glucose to maintain blood glucose levels during the first few hours of life in the pig before a dietary source of energy is available. Muscle glycogen also decreased progressively from 9.09% at birth to the lowest level of 4.66% at 36 hr after birth. Even though muscle glycogen decreased in a manner similar to liver glycogen, the rate is not nearly as rapid (approximately an 80% decrease in liver glycogen compared to *50%* for muscle glycogen). This would indicate that liver glycogen is the immediate and rapidly available source of glucose at birth, while muscle glycogen is utilized less rapidly and represents a more sustained source of glucose. Also, when the relative amounts of liver and muscle glycogen available are considered, muscle glycogen would supply the larger total amount of glucose and may be the more important of the two sources. Liver fat

Figure 1. Changes in liver and muscle glycogen and liver moisture and fat levels of pigs from birth to 36 hours of age.

and moisture levels changed in the opposite direction from the glycogen levels. Liver fat increased from 1.56% at birth to 2.54% at 12 hr of age and then decreased slightly to 2.40% at 24 hr and 2.32� at 36 hr of age. Liver moisture also increased from birth (72.64%) to 12 hr of age (76.10%). However, it continued to increase to 24 hr of age (76.89�) and remained relatively unchanged to 36 hr of age (76.83%).

Trial 2 (1968)

Treatment averages for growth and production data are presented in tables *5* and 6.

During gestation, gilts in the lower gain treatment gained 3.97 kg while those on the higher gain treatment gained 12.44 kg. During the 10 days prefarrowing when energy level was varied, weight gain of the gilts varied with energy intake. Gains were 8.ll, 7.69 and 6.90 kg for the high. intermediate and low energy intakes, respectively. Those gilts which gained the most during the first 104 days of gestation gained the least weight during the prefarrowing energy level treatment and those that gained the least during the first 104 days gained the most during the prefarrowing energy level treatment.

The only significant ($P \le 0.025$) effect on gestation length was due to the interaction between gestation gain, dichlorvos and energy level, indicating that these three factors in combination can alter gestation length.

There were no significant main effect differences in total pigs born or number of pigs born alive per litter. However, the interaction between gestation gain and dichlorvos was significant $(P<.05)$.

TABLE *5 .* MEANS OF GESTATION WEIGHT GAIN, DICHLORVOS AND PREFARROWING ENERGY LEVEL FOR PRODUCTION PERFORMANCE⁸

a Values presented in this table are the arithmetic means of the treatments. Least squares analysis of variance presented in appendix tables 3 and 4 .

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TABLE 5 CONTINUED

TABLE 6. MEANS OF GESTATION WEIGHT GAIN, DICHLORVOS AND PREFARROWING ENERGY LEVEL FOR PRODUCTION PERFORMANCE²

a Values presented in this table are the arithmetic means of the treatments. Least squares analysis of variance presented in appendix tables 3 and 4.

In the group of gilts that did not receive dichlorvos, more total and live pigs were farrowed by those gilts that gained the least during gestation, while of those gilts fed dichlorvos more total and live pigs were farrowed by gilts having the greater gestation gains. Within the lower gestation gain group more total and live pigs were farrowed by the gilts that did not receive dichlorvos. In contrast, within the group of gilts that had the higher gestation gain more total and live pigs were farrowed by those gilts receiving dichlorvos.

There were no significant differences in birth weight of pigs due to any treatments or their interactions. These results agree with Frobish <u>et al</u>. (1966) and Meade <u>et al</u>. (1966) but are in contrast to the results of Clawson et al. (1963) , Lodge et al. $(1966a)$, $0'$ Grady (1967) and Baker et al. (1969) who reported that increasing energy intake during gestation increased pig birth weight. However, the weight gain differences of the gilts in this trial were not as great as those reported by the above authors. The lack of a significant effect of dichlorvos on average birth weight of pigs in this experiment is in agreement with Foster (1968) and Hays $\underline{\text{et}}$ al. (1969) but does not agree with Batte et al. (1968) or Singh et al. (1968) who reported an increase in birth weight due to dichlorvos treatment of the dam. These authors fed dichlorvos at the same level and for a similar length of time as in the present trial.

Growth of pigs from birth to 72 hours of age showed highly significant ($P \leq 0.005$) differences due to dichlorvos treatment, energy

level treatment and time treatment. Pigs from gilts that did not receive dichlorvos gained highly significantly ($P \le 0.005$) more weight by 72 hours of age than did pigs fran gilts receiving dichlorvos (34 vs. 26%). Comparing prefarrowing energy level treatments, pigs from gilts receiving the basal diet plus sugar, diet B, gained 31.9% , those from gilts fed the basal diet A gained 30. 9% and those from gilts fed the basal diet plus fat, diet C, gained 29.1%. As would be expected, weight gain of the pigs increased with each time interval following birth. Significant interactions were found between gestation gain and prefarrowing energy level treatment $(P < .05)$, dichlorvos x prefarrowing energy level treatment ($P \leq 0.005$) and gestation gain x dichlorvos x prefarrowing energy level treatment x time $(P < 005)$. Figure 2 shows the growth of the pigs during the first 72 hours following birth with the increase in body weight expressed as a percent of the birth weight.

Treatment averages for liver as a percent of body weight, liver moisture, liver fat, liver glycogen, muscle glycogen and blood reducing sugar are presented in tables 7 and 8.

Treatment of the gilts had no significant effect on liver weight of the newborn pigs expressed as a percent of body weight. There were two significant interaction effects, those being gestation gain x dichlorvos x time ($P \le 05$) and prefarrowing energy level treatment x time $(P < .05)$. A highly significant $(P < .005)$ difference was present due to time treatment alone. Figure 3 shows that liver weight as a percent of body weight decreased from 2.6% at birth to 2.3% at

TABLE 7. MEANS OF GESTATION WEIGHT GAIN, DICHLORVOS AND PREFARROWING ENERGY LEVEL FOR TISSUE DATA AT BIRTH

a Values presented in this table are the arithmetic means of the treatment. Least squares analysis of variance presented in appendix table 4.

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TABLE 8. MEANS OF GESTATION WEIGHT GAIN, DICHLORVOS AND PREFARROWING ENERGY LEVEL FOR TISSUE DATA AT BIRTH^a

a Values presented in this table are the arithmetic means of the treatments. Least squares analysis of variance presented in appendix table 4.

Figur� J. **Changes in liver and muscle glycogen, liver fat and liver as a percent of body weight of pigs from birth to 72 hours of age.**

18 hours of age and then increased to J.2% at 72 hours of age. The only sign�ficant. difference in water content of the liver was due to the time treatment $(P < 0.05)$. Liver moisture increased from birth $(73.3%)$ to 18 hours of age $(78.4%)$ and then decreased steadily from 18 to 72 hours of age to 76.1% (figure 4).

A significant **(P<.05)** difference in liver fat content of the newborn pigs was present for the interaction of gestation gain x dichlorvos x prefarrowing energy level. Highly significant (P<.005) differences in liver fat content were present due to time treatment following birth. As seen in figure 2, liver fat content increased from **1.00%** at birth to 2.66% at 24 hours and then decreased to **1.57%** at 72 hours of age. 'Ihe interaction of gestation gain x dichlorvos x prefarrowing energy level treatment x time treatment was also significant $(P \leq 01)$, indicating the complexity of the mechanism that controls liver fat content.

Glycogen content of the liver was significantly (P<.025) affected by the interaction between gestation gain and dichlorvos. Gilts in the higher gestation gain treatment produced pigs having higher liver glycogen levels at birth than pigs from gilts in the lower gestation gain treatment (12.12 vs. 11.00%). Pigs from gilts receiving dichlorvos had liver glycogen levels of 12.J6% which was higher than the 10. 71% liver glycogen of pigs from those gilts that did not receive dichlorvos. By 24 hours of age, however, pigs from dichlorvos treated gilts had only slightly higher liver glycogen levels (3. 97%) than pigs from control gilts (J.88%). This is in

contrast to the work of Batte et al. (1968) , who reported nearly equal gl�ogen levels at birth with pigs ,from dichlorvos treated sows having much greater levels at 24 hours of age. The interaction effect showed that pigs from gilts in the higher gestation gain group receiving dichlorvos had the highest liver glycogen levels. Gilts in the lower gestation gain group receiving dichlorvos produced pigs having higher liver glycogen levels than pigs from gilts in either of the gestation gain groups that did not receive dichlorvos. Also, when dichlorvos was not fed, gilts in the higher gestation gain group produced pigs with higher liver glycogen levels than gilts in the lower gestation gain group. The interaction between gestation gain and prefarrowing energy level treatment was also significant **(P <. 05).** Pigs from gilts on the higher gestation gain treatment and basal energy level treatment (A) had the highest levels of liver glycogen $(13.46%)$ and pigs from gilts on the lower gestation gain treatment and the basal energy level treatment (A) had the lowest level (8.11�). Liver glycogen levels also differed significantly **(P <. 005)** from birth to 72 hours of age. Data presented in figure 3 show that liver glycogen levels decreased rapidly from ll.5� at birth to 2.83% at 18 hours and increased to 5.11% at 72 hours of age. These results are in close agreement with those of trial 1.

The interaction between gestation gain treatment of the gilt and dichlorvos treatment produced a significant $(P₀01)$ difference in glycogen content of the semitendinosus muscle at birth. Pigs from gilts on the higher gestation gain treatment and no dichlorvos

had the highest muscle glycogen levels followed by pigs from gilts on the same gestation gain treatment but receiving dichlorvos. Pigs from gilts on the lower gestation gain treatment had the lowest muscle glycogen levels regardless of dichlorvos treatment. The lack of significance due to dichlorvos treatment is in contrast to the work of Batte et al. (1968) who found significant differences in muscle glycogen at birth and 24 hours of age. Highly significant $(P < .005)$ differences in muscle glycogen levels were observed in pigs from birth to 72 hours of age. Figure J shows that muscle glycogen levels decreased steadily from birth $(8,54%)$ to 72 hours of age $(2.77%)$. The interaction of prefarrowing energy level treatment and time treatment produced highly significant (P_e005) differences in muscle glycogen levels. Pigs fran gilts on the highest prefarrowing energy level (diet C) had higher muscle glycogen levels at birth than pigs fran gilts on the other two energy levels. However, the low energy level (diet A) produced the highest muscle glycogen levels at J6 hours of age and maintained higher levels to 72 hours of age.

A highly significant $(P<.005)$ difference in total reducing sugar levels of pigs at birth was noted between gestation gain treatments. Pigs from gilts on the lower gestation gain treatment had blood sugar levels at birth of 128.2 mg per 100 ml compared to a level of 111.2 mg per 100 ml for pigs from gilts on the higher gestation gain treatment. Highly significant $(P<.005)$ differences in blood sugar levels at birth were found due to the following interactions: gestation gain and dichlorvos, gestation gain and

prefarrowing energy level treatment as well as dichlorvos and prefarrowing energy level treatment. Within the higher gestation gain treatment, pigs from gilts that did not receive dichlorvos had higher blood sugar levels at birth than pigs from gilts receiving dichlorvos. Within gestation gain treatment I, the low and intermediate energy level treatments produced higher blood sugar levels at birth than did the high energy level. Within dichlorvos treatment the intermediate prefarrowing energy level treatment produced the highest blood sugar levels in the treated group and the high energy level produced the lowest blood sugar levels. However, in the control group (no dichlorvos), the low prefarrowing energy level treatment produced the highest blood sugar levels and the intermediate energy level produced the lowest blood sugar levels. Time treatment also significantly affected blood sugar levels. Figure 4 shows that the total reducing sugar in the blood remained relatively constant from birth $(120 \text{ mg per } 100 \text{ mL})$ to 48 hours $(122 \text{ mg per } 100 \text{ mL})$ and then increased slightly to 72 hours of age (128.6 mg per 100 ml).

Some interrelationships that exist in the chemical changes of the pig following birth are apparent in figures 3 and 4 . The most striking relationship is that of liver glycogen and liver moisture. As glycogen in the liver is broken down, the water content of the liver increases by a similar, but opposite, amount. However, during the first 18 hours of life the pig gains practically no body weight, yet the liver weight expressed as a percent of body weight is decreasing. This would indicate that the total liver weight is

decreasing and that the loss of liver glycogen is not completely offset by an equal gain in water content. During the period of most rapid glycogen loss from the liver, blood sugar levels remained relatively unchanged, indicating that the pigs were utilizing the glucose as rapidly as it was being produced. Curtis et al. (1966) and Frobish (1969) have s�own that following birth blood fructose levels decrease rapidly while blovd glucose levels increase. Therefore, it is possible that a greater fluctuation in blood sugar levels due to time treatment was not seen because the rise in blood glucose, caused by rapid liver glycogen breakdown, was offset by a reduction in blood fructose levels.

From 18 to 72 hours after birth the changes that take place in the newborn pig are less rapid. These changes are marked by increases in body weight, liver as a percent of body weight, liver glycogen and reducing sugar levels in the blood. These are accompanied by simultaneous decreases in liver water, liver fat and muscle glycogen levels.

Means for total body moisture, fat, protein and ash for each age group and treatment are presented in table 9. Weight gain of the gilt had highly significant ($P < .005$) effects on fat and ash content of the pigs. Those pigs from gilts in the 0 to 10 kg gain group had higher average fat levels $(16.42 \text{ vs. } 13.13\%)$ and a lower average ash content $(13.11 \text{ vs. } 16.98\%)$ than pigs from gilts in the 10 to 20 kg weight gain group. Fat content increased in a similar manner from birth to 72 hr *of* age in both groups, but fat content of pigs from

TABLE 9. TREATMENT MEANS FOR TOTAL BODY COMPOSITION OF NEWBORN PIGS FROM BIRTH TO 72 HOURS OF AGE⁸

TABLE 9 CONTINUED

^a Least squares analysis of variance presented in appendix table 5.
 b $P < .025$.
 c $P < .005$.

the lower gaining gilts increased at a faster rate to 16.42% by ?2 hr of age. Ash content of pigs fran gilts in the lower gaining group was considerably lower at birth and decreased to a greater extent by 72 hr of age, while pigs from gilts in the higher gaining group had nearly the same levels from birth (17.88%) to 48 hr (17.38%) and then decreased to 15.53% at 72 hr of age.

Dichlorvos treatment of the gilts for 21 to JO days prior to farrowing at a rate of 800 mg per gilt per day also produced highly significant ($P \leq 0.005$) differences in total body ash and fat content from birth to 72 hr of age. Pigs from treated gilts had lower average fat levels (12.86 vs. $16.68\%)$ and higher average ash values (17.34 vs. 12. 76%) than pigs from control gilts (table 9). Average fat content was 7.34 and 6.12% at birth and 24.01 and 17.97% at 72 hr for pigs from control and dichlorvos fed gilts, respectively. Ash content decreased from 15.37 to $11.37%$ from birth to 72 hr of age in pigs from control gilts, which was more than the decrease of 17.58 to 16.11% for pigs from treated gilts.

Significant $(P < .025)$ differences were also present in moisture content between pigs from dichlorvos treated and control gilts. Pigs from control gilts had higher moisture contents than pigs from gilts receiving dichlorvos in a1l four age groups. The decrease in moisture from birth to 72 hr was very similar between groups.

Data for body moisture, fat, protein and ash of pigs at O, 24, 48 and 72 hr are presented in table 10. Age had a highly significant (P <.005) effect on total body moisture, fat and protein and a

TABLE 10. TOTAL BODY MOISTURE, FAT, PROTEIN AND ASH CONTENT **OF NEWBORN PIGS AT VARIOUS AGES EXPRESSED .AS MOISTURE-FREE AND WET-WEIGHT**

a Changes in moisture, fat and protein content highly significant $(P<.005)$ and in ash content significant $(P<.025)$ due to age of the pig. Least squares analysis of variance presented in appendix table *5.*

significant $(P < 0.025)$ effect on total body ash. Moisture content of the pigs at birth was 82.87% , which was higher than the 80.2% reported by Curtis, Heidenreich and Martin (1967) using total carcass composition and the 74.5% reported by Brooks et al. (1964) using skinned, unboned carcasses. However, this moisture level was lower than the 85% found by Spray and Widdowson (1950) using total body values. Following birth, moisture content decreased to 79.56% at 72 hr of age. Brooks et al. (1964) and Curtis et al. (1967) found fat levels of pigs to be 5.49 and 3.03% at birth, which are lower than the 6.73% found in the present trial. From birth to 72 hr of age, a 312% increase was seen, reaching a level of 20.99% of body weight at this time. Protein content of the pig body at birth was 54.26% , which was lower than the 55.05% found by Curtis et al. (1967) but higher than the 45.86% reported by Brooks et al. (1964). Body protein increased to 56.47% at 24 hr of age and then decreased to 52.53% by 72 hr of age. Ash content of the pig at birth was reported to be 20% by Curtis <u>et al</u>. (1967) as well as Brooks <u>et al</u>. (1964). However, this is considerably greater than the 16.47% seen in table 10. A 22.6% decrease in body ash content was found from birth to 72 hr of age, at which time a level of 13.74% of the moisture-free body weight was present.

Trial *l* (1969)

Although nonsignificant, rectal temperature of the sows increased from 38. 70 C just prior. to farrowing to -39.20 C during active labor. This increase in rectal temperature could be due to

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the increase in activity and physical work during labor. Treatment averages for rectal temperature of the pigs at birth and time of sacrifice, pancreas and adrenal gland weights and body weight gain at the various time intervals after birth are presented in table 11. Rectal temperatures of the pigs from birth to the time of sacrifice were not significantly affected by treatment with insulin and sucrose. However, an average of all pigs in this trial showed that rectal temperature was similar at all time intervals until 24 hr of age when an increase of 0.48° C was noted. The difference in rectal temperature was similar at 36 hr (0.45°C). The absence of a significant decrease in rectal temperature during the first few hours of life would indicate that the pigs were obtaining adequate milk from the sow, since a fall in rectal temperature is an early sign of starvation hypoglycemia in the newborn pig. At 24 hr of age the average rectal temperature of pigs in both groups was 39.1° C, which is slightly higher than the 38.7° C for the sows just prior to farrowing. This would be expected, since the smaller body weight of the newborn pig should produce a higher metabolic rate and thus a higher body temperature than the larger mature animal.

Insulin and sucrose treatment of the pig at birth had no significant effect on adrenal gland or pancreas weight expressed as a percent of body weight. Table 11 shows that the weight of these glands in relation to body weight increased from birth to 36 hr of age. **There was** a great variation within treatment groups as well as between treatment groups. However, these glands were very difficult to remove

TABLE 11. EFFECT OF INSULIN AND SUCROSE ON RECTAL TEMPERATURE, ADRENAL GLAND AND PANCREAS WEIGHT AND BODY WEIGHT GAINS OF PIGS FROM BIRTH TO 36 HOURS OF AGE^a

a Least squares analysis of variance presented in appendix table 6.

 b Treated with 1 IU of an insulin mixture subcutaneously and 2 g of sucrose orally per kg of body weight at birth.

c Control pigs receiving no special treatment.

d Changes due to age of the pig for rectal temperature at sacrifice, adrenal gland and pancreas weight and body weight gain were highly significant $(P < .005)$. No significant differences in rectal temperature at birth.

in a precise, quantitative manner and variations in technique could have been a factor in some of the variation in results.

Growth rate of the pigs from birth to J6 hr of age expressed as a percent increase of birth weight is shown in figure *5.* There was no significant treatment effect. However, control pigs had a greater increase in birth weight at each time interval after birth except 9 hr after birth at which time the treated pigs had a greater gain $(4.33 \text{ vs. } 2.26\%$, table 9). At 36 hr of age, control pigs had an increase in body weight equal to 18.22% of their birth weight, while treated pigs had an increase in body weight equal to 14.20% of their birth weight. The reduction in weight gain due to treatment with insulin and sucrose may have been due to the higher incidence of diarrhea and dehydration of pigs in that group. At autopsy some diarrhea was observed in many of the treated pigs. A similar condition was reported by Becker et al. (1954) who found a high incidence of diarrhea in newborn pigs fed sucrose. However, pigs in both the treated and control groups in this trial had greater increases in birth weight by 36 hr of age than the 12.21% average increase for pigs of the same age in trial 2.

Although nonsignificant, pigs treated with insulin and sucrose had lower serum triglyceride levels at 3 hr (table 12) and 18 hr after birth. Figure 6 shows that in general serum triglycerides increased from birth to 36 hr of age. These differences were highly significant $(P < .005)$. There was a great variation in serum triglyceride levels within treatment at each time interval. This

TABLE 12. EFFECT OF INSULIN AND SUCROSE ON LIVER AND MUSCLE GLYCOGEN, SERUM GLUCOSE,
FRUCTOSE AND TRIGLYCERIDES, LIVER FAT AND MOISTURE, TOTAL URINE SUGAR
AND URINE GLUCOSE LEVELS OF PIGS FROM BIRTH TO 36 HOURS OF AGE^a

	Age of pig in hours ^b								
	0		6	Ω	12	15	18	24	36
					Total urine sugar				
T	2.00	1.56	1.00	1.13	1.33	1.10	0.75	0.70	0.11
$\mathbf C$	2.00	2.22	1.25	1.50	1.75	1.25	0.88	0.33	0.33
					Urine glucose				
$\mathbf T$	$\mathbf 0$	0.11	0	$\mathbf 0$	0.11	0.40	0.38	0.90	0.89
$\mathbf C$	$\mathbf 0$	0	0.25	0.38	C _v 50	0.43	1.50	0.86	0.83

TABLE 12 CONTINUED

a Least squares analysis of variance presented in appendix tables 6 and 7. bChanges due to age of the pig for liver and muscle glycogen, serum glucose, fructose and triglycerides, liver moisture, urine glucose and total urine sugar highly significant (P .005) and for liver fat significant $(P < .05)$.

 c Treated with 1 IU of an insulin mixture subcutaneously and 2 g sucrose orally per kg of body weight at birth.

d Control pigs receiving no special treatment.
e $P < 025$.

f $P < .01$.

g P< *. 005 .*

leads to the speculation that a factor such as time of sampling in relation to time of suckling may have caused some of the differences *seen* between treatment groups, since a temporary rise in serum triglycerides could very well take place during the period of most rapid absorption from the intestine.

Pigs treated with insulin and sucrose at birth had highly significantly $(P<.005)$ lower serum glucose levels at 3 hr of age $(68.8 \text{ vs. } 112.6 \text{ mg per } 100 \text{ ml})$ and significantly $(P < .025)$ lower serum glucose levels at 18 hr of age (91.7 vs. 125.9 mg per 100 ml) than control pigs (table 12). There was a significant $(P<025)$ treatment effect on serum glucose level when data of all time treat- . ments are combined. Serum glucose increased most rapidly from birth to J and 6 hr after birth for the control and treated pigs, respectively, and then changed very little to 36 hr of age. The highly significant ($P \le 0.005$) changes in serum glucose due to age of the pig are presented in figure 7. The large difference in serum glucose levels 3 hr after birth can be explained by the *rate* and duration of action of the *0.5* IU of regular insulin per kg of body weight. Regular insulin injection is the most rapid acting component of the insulin mixture used, reaching its peak of activity at 2 to J hr after administration and having a duration of action of 6 to 8 hr. Since the peak of action for regular insulin is 2 to 3 hr, the decrease in serum glucose seen at J hr after birth was probably due to the effects of the regular insulin on glucose utilization. By 6 hr after birth the body had adjusted to the insulin while the effects

of the insulin were decreasing. The decrease in serum glucose levels of the treated pigs at 18 hr after birth was in all probability due to the combined effects of the 0.25 IU of Neutral Protamine Hagedorn (NPH) insulin and the 0.25 IU of Protamine Zinc insulin. NPH insulin has a peak of action of 8 to 12 hr and a duration of action of 12 to 24 hr after administratio�, while Protamine Zinc insulin has a peak of action of 12 to 16 hr after administration and a duration of action of 16 to 36 hr after administration. Therefore, during the period of 12 to 24 hr after birth there should have been an effect similar to that seen with the *0.5* IU of regular insulin. However, the pigs were probably absorbing enough glucose from the sucrose treatment during the 12 to 15 hr after birth interval to mask the full effect of the insulin. At 18 hr after birth the absorption of glucose into the system was probably slowing down while the effects of the two insulins were still rather high and the decrease in glucose levels were . observed.· By 24 *hr* after birth the action of the NPH insulin should have decreased to the point where only the 0.25 IU of Protamine Zinc insulin was present. Apparently, this was not enough to significantly reduce serum glucose levels but did result in slightly reduced levels until 36 hr after birth.

The highly significant $(P<.005)$ changes in serum fructose levels due to age of the pig as well as the differences due to treatment are presented graphically in figure 7. Serum fructose changed most rapidly from birth to 3 hr of age. Serum fructose levels in the control pigs decreased while in the treated pigs they increased. From 3 to 24

hr of age serum fructose decreased in both groups, reaching equal levels by 24 hr. Frau 24 to 36 hr after birth both groups of pigs had similar serum fructose levels.

Highly significant differences $(P<.005)$ in serum fructose levels were found due to treatment with insulin and sucrose at birth. Treated pigs had highly significantly $(P<.005)$ greater levels of serum fructose at 3 hr (62.17 vs. 22.81 mg per 100 ml), 6 hr (50.85 vs. 13.88 mg per 100 ml), 9 hr (37.19 vs. 9.03 mg per 100 ml), 12 hr (19.58 vs. *5.78* mg per 100 ml) and 15 hr after birth (10. 84 vs. 5.18 mg per 100 ml) and significantly $(P < 01)$ higher levels at 18 hr after birth *(7.95* vs. 4. JO mg per 100 ml, table 12). Fran 24 to 36 hr after birth both treated and control pigs had nearly equal levels of serum fructose.

No significant differences were present in urine glucose levels due to treatment or time after birth. Urine glucose levels based on a scale from O to 3, also according to a color code chart, were zero at birth for both treated and control pigs. This would suggest that all of the sugar present in the urine was fructose. However, total urine sugar levels were highly significantly $(P < 0.005)$ different due to time after birth. Based on a scale of 0 to 4 , according to a color code chart, at birth both groups of pigs had values of 2.0 (table 12), and it decreased to 0.11 for treated pigs and 0.33 for control pigs by J6 hr of age. Control pigs had slightly higher total urine sugar levels at each time interval, beginning at 3 hr of age, than treated pigs. Furthermore, since urine glucose

remained at about zero or slightly greater up to 12 hr of age and this was a period of rapid decline in serum fructose levels, it could be suggested that the decline in serum fructose was due in part to excretion in the urine rather than due to utilization by the pigs. This would agree with Aherne et al. (1969), who stated that the fetal and newborn pig is unable to utilize fructose as an energy source. If this is true, there is no physiological basis for the very high levels of fructose found in the fetal and newborn pig.

Treatment of the newborn pig with insulin and sucrose at birth did not cause any significant differences in liver glycogen, muscle glycogen, liver fat or liver moisture (table 12). However, there were highly significant differences for all four parameters due to age of the pigs. The changes in liver glycogen from birth to 36 hr are presented graphically in figure 8. The rapid decrease in liver glycogen is in close agreement with the changes seen in trials 1 and 2. The-decrease in liver glycogen content from birth to J hr of age corresponds to the changes seen in serum glucose in that treated pigs had a relatively small decrease in liver glycogen (12.83 to 12.17%) and small increase in serum glucose $(50.01$ to 68.83 mg per 100 ml), while control pigs had a much greater decrease in liver glycogen (13.58 to 11.l�) and greater increase in serum glucose (4J.42 to 112.60 mg per 100 ml). This is a further indication that liver glycogen is an important source of glucose to the newborn pig for the first few hours after birth. There was relatively little difference in muscle glycogen between treated and control pigs at

any time interval. Muscle glycogen levels declined from 8.9% at birth to 3. 8� at 36 hr of age. The changes seen in liver fat and liver moisture as well as muscle glycogen from birth to 36 hr of age (figure 9) are similar to those seen in trials 1 and 2. Liver moisture content increased frau birth (72.9�) to 14 hr (78.06%) and then remained relatively unchanged to 36 hr of age (77.87%) . Liver fat content also increased from 1.15% at birth to 2.27% at 15 hr but then decreased to *1.55%* at 36 hr of age.

SUMMARY

Three trials were conducted over three years' time to study the stored energy present in neonatal pigs at birth and the changes that take place in these same stored energy sources during the first 36 to 72 hr after birth. A total of 664 pigs representing *85* different litters were sacrificed at various ages ranging from birth to 72 hr of age. Blood and tissue samples were collected and analyzed chemically.

In the first trial gilts were self-fed a com-soybean diet during the first 104 days of gestation and then 5924, 6183 or 6975 Kcal metabolizable energy per day to farrowing. Additions of 15% sugar or fat were used to increase the energy content of the basal ration.

In the second trial, gilts **were** divided into two groups and \blacksquare . In the contract of t fed to gain 0 to 10 kg or 10 to 20 kg during gestation. One-half of , each weight gain group was fed 800 mg of dichlorvos per day for 21 to JO days before farrowing, while the other one-half served as controls. Three different rations were again fed from day 104 of gestation to farrowing. These diets were calculated to fumish O, 1000 or 2000 Kcal of ME above the resting metabolic requirement of the individual gilt.

In the third experiment, all gilts were treated equally during gestation and allowed to gain 10 to 20 kg of body weight. At birth, half of the litters served as controls and pigs from the other litters each were given 2 g of sucrose orally in a 13.5� solution and 1 IU of

an insulin mixture containing *0.5* IU regular insulin, o. 25 IU Neutral Protamine Hagedorn insulin and o. 25 IU Protamine Zinc insulin subcutaneously per kg of body weight. The ages of pigs analyzed in **each** trial were O, 12, 24 and 36 hr in the first trial; O, 6, 12, 18, 24, 36, 48 and 72 hr in the second trial and O, J, 6, 9, 12, 15, 18, 24 and 36 hr in the third trial.

In the first trial highly significant $(P < .005)$ differences were present in liver and muscle glycogen and liver fat and moisture due to age of the pig. Liver glycogen decreased from 14.92% at birth to 2.85% at 36 hr of age. Similarly, muscle glycogen decreased steadily from birth to 36 hr of age $(9.09$ to $4.66%)$. Liver fat content increased from birth (1.56%) to 12 hr (2.54%) and then decreased to 36 hr of age (2.32%) . Liver moisture also increased changing from 72. 64% at birth to 76.83% at 36 hr *of* age. The only highly significant effect was a breed difference in liver fat content. Pigs from crossbred gilts sired by a Hampshire boar had higher liver fat levels at each age.

In the second trial the changes due to age of the pig were also highly significant (P **<. 005).** Liver weight, expressed as a percent of body weight, decreased from 2.6% at birth to 2.3% at 18 hr of age and then increased to 3.2% at 72 hr of age. Moisture content of the liver increased from birth $(73.3%)$ to 18 hr of age $(78.4%)$ and then decreased to 72 hr of age $(76.1%)$. Liver fat content also increased following birth from 1. 00% at birth to a level of 2.66% at 24 hr of age and then decreased to 1.57% at 72 hr of age.

Liver and muscle glycogen both decreased as the age of the pig increased. Liver glycogen level was the highest at birth $(11.5%)$ and the lowest at 18 hr of age $(2.83%)$, after which time it increased to 5.11% at 72 hr of age. Muscle glrcogen decreased progressively from birth (8.54) to 72 hr of age (2.77) . Total reducing sugar level in the blood changed more slowly following birth. It increased only slightly from 120 mg per 100 ml at birth to 122 mg per 100 ml at 18 hr of age followed by a more rapid increase to l28 mg per 100 ml by 72 hr of age.

No significant differences were noted in gestation length, total or live pigs per litter, birth weight, liver weight as a percent of body weight and liver fat or moisture content due to treatment of the gilt. Pigs fran gilts that received dichlorvos gained highly significantly ($P < .005$) less weight, expressed as a percent increase in birth weight, from birth to 72 hr of age (26 vs. 34%). Significant differences (P<.025) were present in liver glycogen due to weight gain of the gilt and dichlorvos treatment. Gilts in the 10 to 20 kg gain group produced pigs with 12.12% liver glycogen at birth as compared to 11.00% for pigs from the lower gaining gilts. Pigs fran the gilts receiving 800 mg of dichlorvos per day for 21 to 30 days prior to farrowing had liver glycogen levels of 12.36%, which was greater than the 10.71% for pigs from gilts not receiving dichlorvos. No significant main effects were present for muscle glycogen or total reducing sugar content of the blood.

Several significant interaction effects were present and are discussed.

Proximate analysis of pigs at 0, 24, 48 and 72 hr of age in this trial showed that pigs from gilts receiving dichlorvos had significantly $(P < 0.025)$ less moisture $(80.57 \text{ vs. } 81.78\%)$, highly significantly $(P < 0.005)$ less fat $(12.86 \text{ vs. } 16.68\%)$ and more ash (17.34 vs. 12.76%) at each age than pigs from gilts not receiving dichlorvos. Gilts that gained 10 to 20 kg during gestation produced pigs having highly significantly $(P < .005)$ less fat $(13.3 \text{ vs. } 16.42\%)$ and more ash $(16.98 \text{ vs. } 13.11\%)$ than gilts that gained 0 to 10 kg during gestation. Highly significant $(P < .005)$ changes in total body moisture, fat and protein and significant ($P < 0.025$) changes in total body ash were seen due to age of the pig. Moisture content decreased from 82.87% at birth to 79.56% at 72 hr of age. Ash content also decreased from birth to 72 hr of age $(16.47$ to $13.74%$). There was a temporary increase in total protein content from 54.26% at birth to 56.47% at 24 hr of age and a subsequent decrease to 52.53% at 72 hr of age. Conversely, total body fat increased from birth to 72 hr of age (6.73) to 20.99%).

In the third trial treatment of the newborn pig with $2 g$ of sucrose orally and 1 IU of insulin subcutaneously per kg of body weight had no significant effects on rectal temperature, pancreas or adrenal gland weights, growth rate, liver or muscle glycogen, liver fat or moisture, serum triglycerides, total urine sugar or urine glucose. However, treated pigs were found to have highly

significanUy (P < . 005) lower serum glucose levels at 3 hr or age, significantly (P<.025) lower glucose levels at 18 hr of age and highly significantly (P<.005) higher serum fructose levels at 3, 6, 9, **12,** 15 **and 18 hr or age than control pigs. Highly significant (P< . 005) changes from birth to 36 hr of age were characterized by** increased rectal temperature, pancreas and adrenal gland weights, **growth rate, liver moisture and serum glucose and triglycerides and decreased liver and muscle glycogen, serum fructose and total urine** sugar. Liver fat level increased significantly (P<.05) from birth **to** 15 **hr of age and then decreased slightly to** 36 **hr or age.**

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APPENDIX

TABLE 1. LEAST SQUARES ANALYSIS OF VARIANCE FOR LIVER AND MUSCLE GLYCOGEN (TRIAL 1)^a

a Mean square values.

b Prefarrowing energy level treatment.

c Breed •

d Litter within treatment.

e Time treatment following birth.

 $P < .005.$

a Mean square values .

b Prefarrowing energy level treatment.

^c Breed.

d Time treatment following birth.

^e**P** <.005.

TABLE 3. LEAST SQUARES ANALYSIS OF VARIANCE FOR VARIOUS TISSUE CHARACTERISTICS AND PERCENT INCREASE IN BIRTH WEIGHT^a

a Number in parenthesis by error mean square indicates **degrees** of freedom.. Numbers represent mean square values.

- b Weight gain treatment of gilt.
- � 2,2 dichlorovinyl dimethyl phosphate treatment.
- d Prefarrowing energy level treatment.
- ^e Time treatment following birth.
- *^P <.05.*
- $g P < .025$.
- $h P < .01.$
- i $P < 0.005$.

Source	df	Liver glycogen	Muscle glycogen	Blood glucose	% wt. gain from birth
WGD		29.1366B	2.1803	63.0850 ¹	57.2699
DDVPC		20.5603E	2.1795.	0.1437	1054.9500 ¹
WG X DDVP		18.82488	6.2107^{h}	41.90401	169.5481
ELd	2	6.6238	0.6168	3.5276	420.1539 ^g
WG X EL		13.7108 ^I	0.5584	50.3345 ¹	209.09265
DDVP X EL	2	1.3928	1.2190	18.22461	259.50301
WG X DDVP X EL		0.3348	3.2625 ^I	4.0903	398.84101
T ^e		328.3777 ¹	148.2244 ¹	41.13121	4568.80901 (6)
WG X T		3.3387	0.6924	5.8709	26.6542 (6)
DDVP X T		3.6857	0.5222	6.8318	(6) 89.9701
WG X DDVP X T		3.9922	0.5721	0.7156	(6) 15.3805
EL X T	14	3.8822	3.3529 ¹	6.4322	266.32561 (12)
WG X EL X T	14	3.0205	1.0430	5.2606	(12) 58.7555
DDVP X EL X T	14	1.5753	1.4480	1.5576	1.2687 (12)
WG X DDVP X EL X T	14	2.2381	0.2549	7.4397	(12) 20.3537
Error		3.4227 (250)	0.8740(248)	5.6554 (237)	44.9585 (222)

TABLE 3 CONTINUED

CONT

TABLE 4. LEAST SQUARES ANALYSIS OF VARIANCE FOR VARIOUS GILT WEIGHT GAIN AND PRODUCTION PERFORMANCE CHARACTERISTICS^a

^a Mean square values.

^b Weight gain treatment of gilt.

^c 2,2 dichlorovinyl dimethyl phosphate treatment.

^d Prefarrowing energy level treatment.

- $P < .05$
f $P < .01$.
-

 $B P < .005.$

TABLE 5. LEAST SQUARES ANALYSIS OF VARIANCE FOR TOTAL BODY MOISTURE, FAT, PROTEIN AND ASH (TRIAL 2)^a

^a Mean square values.

b Weight gain treatment of gilt.

c 2,2 dichlorovinyl dimethyl phosphate treatment.

d Prefarrowing energy level treatment.

e Time treatment following birth.

- $f_{P<.05}$
-
- $R P < .025$
h $P < .005$.

TABLE 6. LEAST SQUARES ANALYSIS OF VARIANCE FOR RECTAL TEMPERATURE AT BIRTH AND TIME OF SACRIFICE, ADRENAL GLAND AND PANCREAS WEIGHTS, BODY WEIGHT GAIN, **URINE GLOCOSE AND TOTAL URINE SUGAR (TRIAL 3) a**

a Mean square values . b Insulin and sucrose treatment at birth.

� Time treatment following birth.

P<. 005.

TABLE 7. LEAST SQUARES ANALYSIS OF VARIANCE FOR LIVER AND MUSCLE GLYCOGEN,
SERUM TRIGLYCERIDES, GLUCOSE AND FRUCTOSE AND
LIVER FAT AND MOISTURE (TRIAL 3)²

a Mean square values.

b Insulin and sucrose treatment at birth.

c Time treatment following birth.
d $P < 0.05$.

 $P < 005$