Changes in fetal organ weights during gestation after selection for ovulation rate and uterine capacity in swine

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ABSTRACT: We hypothesized that the ability of the fetus to alter nutrient shunting and organ growth might be associated with uterine capacity. White crossbred gilts from a randomly selected control line, a line selected for ovulation rate, and a line selected for uterine capacity (UC) were unilaterally hysterectomized-ovariectomized at 160 d of age, mated at estrus, and slaughtered at 45, 65, 85, and 105 d of gestation (9 to 18 gilts for each line × day combination). Analysis of the data revealed that heart weights and fetal weights were decreased in the ovulation rate line. No significant differences were obtained in fetal, placental, or fetal organ weights between the control and UC lines. Allometric growth of organs was assessed by examination of the slopes of the relationships between fetal weights and fetal organ weights after natural log transformation. Only the relative growth of the liver differed between selection lines and was greater ($P = 0.01$) in the UC compared with the control line during early pregnancy (d 45 and 65). Allometric growth of the fetal brain, liver, and heart differed with day of gestation. A brain-sparing effect was greater ($P < 0.01$) on d 85 and 105 compared with d 45 and 65. By contrast, a heart-sparing effect was present during early gestation and disappeared in later gestation. Fetal liver weights were hypersensitive to differences in fetal weights on d 45, possibly associated with placental effects on fetal liver weight. Fetal spleen weights were proportional to fetal weights throughout gestation. These results indicate that selection for ovulation rate decreased total fetal and fetal heart weights, and that selection for UC altered the relationship between total fetal and fetal liver weights during early gestation. Results further indicate significant changes in allometric growth of organs during gestation.

Key words: brain, fetus, heart, liver, spleen, uterus

INTRODUCTION

Uterine capacity (UC) and ovulation rate (OR) contribute to litter size in swine (Bennett and Leymaster, 1989). Uterine capacity is a complex trait composed of uterine, placental, and fetal factors that influence the survival of fetuses in a crowded intrauterine environment (Vallet, 2000). One fetal factor that may influence the survival of an individual fetus is the ability to control the growth of various organs during pregnancy. It is known that mechanisms preserving fetal brain growth exist because, whereas intrauterine crowding decreases overall fetal weight, fetal brain weights are less affected, resulting in a brain-sparing effect (Dickerson et al., 1971; Ashworth et al., 2001; Vallet and Christenson, 2004). Little information is available on the ontogeny of this effect during gestation, whether other fetal organs display similar resistance to changes in fetal weights, or whether these sparing effects are associated with increased uterine capacity.

Leymaster and Christenson (2000) recently described lines of gilts selected at random (control, CO), for increased OR, or for increased UC. The OR line displayed an OR of 3.2 ova per estrous cycle greater than the CO line, and the UC line displayed an increase in uterine capacity of about 1 fetus per uterine horn. Gilts from these lines are useful to investigate changes in maternal and fetal physiological mechanisms that contribute to changes in OR and UC or both.

The objective of the current study was to compare the growth of vital fetal organs throughout gestation in the CO, OR, and UC lines. Many organ systems are not vital during gestation; rather, they develop to fulfill vital functions during adult life. Exceptions to this are
Figure 1. Scatterplots of the natural log of brain weight vs. natural log fetal weight for fetuses in this experiment from the control, ovulation rate (OR), and uterine capacity (UC) lines on the different days of gestation. The slopes of the linear relationships did not differ among selected lines and were greatest \((P < 0.01)\) on d 45, decreased on d 65 and 85, and were not different between d 85 and 105.

The fetal liver and spleen, which are major erythropoietic organs (Ducsay et al., 1982; Vallet, 2000), and the fetal heart.

MATERIALS AND METHODS

The care and use of animals in this experiment were approved by the Animal Care and Use Committee at the US Meat Animal Research Center and met USDA guidelines for the use of agricultural animals in research. White crossbred gilts from the randomly selected CO, OR, and UC selected lines available at the US Meat Animal Research Center were unilaterally hysterectomized-ovariectomized (UHO) at 160 ± 3 d of age, allowed to recover, and then were mated at standing estrus (not necessarily first standing estrus). The UHO treatment reduces intrauterine space without altering OR, thus increasing intraterine crowding in the gilts during gestation (Christenson et al., 1987).

Gilts (9 to 18 per line × day combination, distributed over 2 seasons, July to October and January to April) were slaughtered on d 45, 65, 85, and 105 of gestation, and the remaining uterine horn was recovered. The uterine horn was opened, and each fetus and placenta was removed and weighed. For each fetus, the fetal brain, heart, liver, and spleen (at d 45 the spleen was either nonexistent or too small to accurately weigh) were dissected and weighed.

Statistical Analysis

This report focuses on differences in fetal organ weights between the selection lines; a more complete analysis of differences in fetal and placental weights will be described in a subsequent report. Fetal and fetal organ weight data were analyzed by ANOVA after natural log transformation using PROC MIXED (SAS Inst. Inc., Cary, NC) and a model that included day of gestation, line, and the day × line interaction as fixed effects, and season, season × line × day, and gilt (season × line × day) as random effects. The Kenward-Rogers method was used to calculate the appropriate denominator degrees of freedom for \(F\)-tests.

The following individual contrasts were performed to further determine the effects of line: 1) the CO line was
To analyze allometric growth of fetal organs and the whole fetus, ln fetal organ weights were analyzed using PROC MIXED with a model that included the day of gestation, line, line × day interaction, the linear effect of ln fetal weight, the day × linear effect of ln fetal weight interaction, the line × linear effect of ln fetal weight interaction, and the line × day × linear effect of ln fetal weight interaction. Random effects in the model were season, season × day × line, gilt (season × day × line), ln fetal weight × season, ln fetal weight × season × day × line, and ln fetal weight × gilt (season × day × line). According to Huxley (1932), proportional changes in fetal organ weights with changes in fetal weights are reflected by a slope of 1. Deviations from a slope of 1 are indicative of either a sparing effect (slope less than 1; changes in fetal organ weights are resistant to changes in fetal weight) or of a hypersensitivity effect (slope greater than 1; changes in fetal organ weights are enhanced compared with changes in fetal weight).

A second analysis was performed to determine the influence of fetal and placental weights on fetal organ growth. To perform the analysis, the linear effects of ln fetal weight and ln placental weight were fit simultaneously using PROC MIXED. The fixed effects in the model included day, line, the day × line interaction, the linear effect of ln placental weight, the day × linear effect of ln placental weight interaction, the line × linear effect of ln placental weight interaction, and the line × day × linear effect of ln placental weight interaction. Random effects in the model were season, season × day × line, gilt (season × day × line), ln fetal weight × season, ln fetal weight × season × day × line, and ln fetal weight × gilt (season × day × line). According to Huxley (1932), proportional changes in fetal organ weights with changes in fetal weights are reflected by a slope of 1. Deviations from a slope of 1 are indicative of either a sparing effect (slope less than 1; changes in fetal organ weights are resistant to changes in fetal weight) or of a hypersensitivity effect (slope greater than 1; changes in fetal organ weights are enhanced compared with changes in fetal weight).

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fitted were season, line × day × season, gilt (line × day × season), season × linear effect of ln placental weight, line × day × season × linear effect of ln placental weight, gilt (line × day × season) × linear effect of ln placental weight, season × linear effect of ln fetal weight, line × day × season × linear effect of ln fetal weight, and gilt (line × season × day) × linear effect of ln fetal weight.

**RESULTS**

Table 1 indicates the least squares means for fetal, placental, and fetal organ weights, calculated from the means from ln transformed data. As anticipated, all weights dramatically increased with advancing gestation. There were no line × day interactions for any of the weights. Analysis indicated that line effects were present for fetal weights and fetal heart weights, and contrasts indicated that fetal (P < 0.05) and fetal heart weights (P > 0.01) were less in the OR line compared with the CO line. There was no effect of the UC line on any of the weights measured.

Table 2 indicates the slopes of the linear relationships between ln fetal organ weights and ln fetal weights for the different line × day combinations in this experiment. Homogeneity of regression analysis indicated a line × day effect (P < 0.05) on the relationships between ln fetal liver weights and ln fetal weights. Further analysis indicated that this interaction was partially due to differences (P = 0.01) in the slopes on d 45 and 65 compared with d 85 and 105 between the CO and UC lines, indicating that the slopes were greater for the UC line compared with the CO line during early pregnancy. The overall line × day interaction was also partially due to a decreased (P < 0.05) slope in the OR line compared with the CO line on d 85 of gestation. Although this could be a real difference, it seems likely that this result may have occurred due to random variation. No other line or line × day interaction effects were detected on the slopes for the other organ weights. A day effect on the relationships between fetal brain weights and fetal weights was detected. The slopes of the relationships between brain weights and fetal weights were less than 1, decreased (P < 0.05) from d 45 to 65, from d 65 to d 85, and did not change between d 85 and 105 (Figure 1). These results indicate that the previously described brain-sparing effect is less during early gestation and reaches maximum by d 85 of gestation. A day effect on the relationships between fetal heart weights and fetal weights was also present. The slopes of the relationships between fetal heart weights and fetal weights were also less than 1 during early gestation but, in contrast with fetal brain, increased (P < 0.05) steadily during gestation and reached approximately 1 on d 105, indicating that a heart-sparing effect was present during early gestation and gradually disappeared. Furthermore, an overall significant day effect was present on the relationships between fetal liver weights and fetal weights. The slopes of the relation-
ships were greatest ($P < 0.05$) on d 45 and were greater than 1. Slopes approximated 1 on all other days measured. These results suggest that fetal liver weights were hypersensitive to differences in fetal weights on d 45 and were proportional during the rest of gestation. The relationships between fetal spleen weights and fetal weights were similar throughout gestation, and the slope near 1 indicated proportional growth.

One explanation of the hypersensitive relationship between fetal liver weights and fetal weights was some contribution of the placenta to the control of liver growth, prompting a further analysis to examine the additional contribution of placental weights to differences in fetal liver and the other organ weights after adjusting for fetal weights by regression. The slopes resulting from fitting fetal weight and placental weights simultaneously are indicated in Table 3. Examination of the slopes in Table 3 indicated that placental weights had only minor or no effects on brain, spleen, heart, or liver weights for most gestational ages. However, significant linear effects of placental weight on brain (d 85 in the UC line only), liver (d 45 of gestation for all 3 lines), and heart weights (although slopes were near 0, across all days and lines there appeared to be a slight positive slope; all but d 65 in the UC line were positive) were detected (Table 3). These results indicate that the size of the placenta may somehow contribute significantly to growth of the fetal liver on d 45 of gestation.

**DISCUSSION**

This is the first experiment to compare fetal organ growth on individual days of gestation in lines of pigs selected randomly, for OR, or for UC. The allometric analysis is enhanced in this experiment by the applica-

**Table 3.** Linear slopes ± SE for the relationships between the natural log of fetal organ weights and the natural log of fetal and placental weights (top and bottom numbers in each cell, respectively) when fetal and placental weights were fit simultaneously using data from the control (CO), ovulation rate (OR), and uterine capacity (UC) lines for each day of gestation.

<table>
<thead>
<tr>
<th>Line × day of gestation</th>
<th>Brain weight$^1$</th>
<th>Liver weight$^2$</th>
<th>Spleen weight$^3$</th>
<th>Heart weight$^4$</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO d 45</td>
<td>0.68 ± 0.08</td>
<td>0.97 ± 0.09</td>
<td>0.66 ± 0.09</td>
<td></td>
</tr>
<tr>
<td></td>
<td>−0.08 ± 0.03</td>
<td>0.11 ± 0.03</td>
<td>0.06 ± 0.03</td>
<td></td>
</tr>
<tr>
<td>OR d 45</td>
<td>0.65 ± 0.07</td>
<td>0.62 ± 0.07</td>
<td>0.70 ± 0.08</td>
<td></td>
</tr>
<tr>
<td></td>
<td>−0.04 ± 0.03</td>
<td>0.29 ± 0.03</td>
<td>0.02 ± 0.03</td>
<td></td>
</tr>
<tr>
<td>UC d 45</td>
<td>0.69 ± 0.07</td>
<td>0.89 ± 0.06</td>
<td>0.62 ± 0.07</td>
<td></td>
</tr>
<tr>
<td></td>
<td>−0.03 ± 0.02</td>
<td>0.23 ± 0.03</td>
<td>0.12 ± 0.03</td>
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<tr>
<td>CO d 65</td>
<td>0.37 ± 0.07</td>
<td>0.61 ± 0.09</td>
<td>0.83 ± 0.09</td>
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<tr>
<td></td>
<td>−0.01 ± 0.04</td>
<td>0.20 ± 0.05</td>
<td>0.03 ± 0.05</td>
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<tr>
<td>OR d 65</td>
<td>0.35 ± 0.05</td>
<td>0.82 ± 0.06</td>
<td>0.75 ± 0.06</td>
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<tr>
<td></td>
<td>−0.01 ± 0.03</td>
<td>0.08 ± 0.04</td>
<td>0.07 ± 0.04</td>
<td></td>
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<tr>
<td>UC d 65</td>
<td>0.40 ± 0.07</td>
<td>1.00 ± 0.09</td>
<td>0.87 ± 0.09</td>
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<tr>
<td></td>
<td>−0.04 ± 0.03</td>
<td>0.00 ± 0.04</td>
<td>−0.03 ± 0.04</td>
<td></td>
</tr>
<tr>
<td>CO d 85</td>
<td>0.21 ± 0.07</td>
<td>1.10 ± 0.09</td>
<td>0.87 ± 0.09</td>
<td></td>
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<tr>
<td></td>
<td>−0.01 ± 0.04</td>
<td>0.02 ± 0.05</td>
<td>0.04 ± 0.05</td>
<td></td>
</tr>
<tr>
<td>OR d 85</td>
<td>0.39 ± 0.06</td>
<td>0.87 ± 0.07</td>
<td>0.84 ± 0.07</td>
<td></td>
</tr>
<tr>
<td></td>
<td>−0.10 ± 0.04</td>
<td>0.00 ± 0.05</td>
<td>0.03 ± 0.05</td>
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<tr>
<td>UC d 85</td>
<td>0.08 ± 0.07</td>
<td>1.09 ± 0.08</td>
<td>0.90 ± 0.08</td>
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<tr>
<td></td>
<td>0.15 ± 0.05</td>
<td>−0.06 ± 0.06</td>
<td>0.00 ± 0.06</td>
<td></td>
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<tr>
<td>CO d 105</td>
<td>0.30 ± 0.04</td>
<td>1.01 ± 0.05</td>
<td>0.99 ± 0.05</td>
<td></td>
</tr>
<tr>
<td></td>
<td>−0.02 ± 0.04</td>
<td>0.02 ± 0.05</td>
<td>0.03 ± 0.05</td>
<td></td>
</tr>
<tr>
<td>OR d 105</td>
<td>0.25 ± 0.05</td>
<td>1.02 ± 0.07</td>
<td>0.91 ± 0.07</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.03 ± 0.05</td>
<td>−0.05 ± 0.06</td>
<td>0.00 ± 0.06</td>
<td></td>
</tr>
<tr>
<td>UC d 105</td>
<td>0.21 ± 0.05</td>
<td>0.95 ± 0.06</td>
<td>0.90 ± 0.06</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.01 ± 0.04</td>
<td>0.01 ± 0.05</td>
<td>0.06 ± 0.05</td>
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</tbody>
</table>

$^1$There were significant effects of line × day × linear effect of natural log placental weight ($P < 0.01$), natural log fetal weight ($P < 0.01$), and day × linear effect of natural log fetal weight ($P < 0.01$). The significant line × day × linear effect of natural log placental weight appeared to be due primarily to the positive slope for the natural log placenta in the UC line on d 85 compared with a slight negative linear effect of natural log placental weight for all the other combinations.

$^2$Significant effects of line × day × linear effect of natural log placental weight ($P < 0.01$) and line × day × linear effect of natural log fetal weight ($P < 0.01$). These appeared to be due to significant positive linear effects of placental weight, primarily on d 45 of gestation.

$^3$Significant linear effect of natural log fetal weight ($P < 0.01$). Placental weights did not affect spleen weights.

$^4$Significant linear effect of natural log placental weight ($P < 0.01$), linear effect of natural log fetal weight ($P < 0.01$), and day × linear effect of natural log fetal weight ($P < 0.01$). The significant effects of natural log placental weight appeared to be due to a slight positive linear effect overall. However, the magnitude of the slopes suggests only minor effects.
tion of the UHO model, resulting in increased intrauterine crowding and more variability in fetal weights within each litter. Our results indicated reductions of fetal heart weights in the OR line compared with the CO line and alterations in allometric growth of the liver in the UC line during early gestation. Results indicated that the decrease in heart weight in the OR line occurred proportionally with differences in fetal weight between the CO and OR lines; thus, these changes are probably due to reductions in fetal weight in the OR line due to increased crowding, a result that has been reported previously (Christenson and Leymaster, 2002). Overall, clear differences in allometric growth of fetal brain, liver, and heart were detected between the different days of gestation. Collectively, these results indicate that growth relationships with fetal weights change during gestation for these organs and suggest a potential opportunity to alter individual organ weight growth to optimize uterine capacity, especially for fetal liver.

Differential fetal organ growth during pregnancy in relation to other organs and in relation to the fetus is not a new concept (Huxley, 1932; Hammond, 1950). Analysis of this type of data is often done by expressing organ weight as a ratio of some base weight to examine changes in proportionality of growth. Numerous reports use the overall fetal weight for this purpose (Anderson and Wahlstrom, 1970; Dickerson et al., 1971; McMillen et al., 2001; Da Silva-Buttkus et al., 2003; Bauer et al., 2004; Mc Pherson et al., 2004), but Hammond (1950, 1960) recommended the use of some aspect of the fetus that is least variant, such as brain weight, head size, or the length of portions of the developing skeleton. Regardless of what is used in the ratio, the calculation of ratios of weights has several disadvantages. First, because error is involved in organ and total weight measurements, the variance of the ratio is a combination of the error variances of both measurements. Second, the calculation of a ratio between organ weight and total weight assumes that the relationship between them is linear and passes through the origin, which often is not true. Finally, if one wishes to examine proportional growth over a range of fetal weights, one still has to perform a regression analysis between the ratio of organ/fetal weight and fetal weight to determine whether or not the ratio is the same throughout the range of both variables. The interpretation of these regression relationships can be difficult when growth is not proportional, or when growth rates are not linearly related, or if the relationship between the 2 weights does not actually pass through the origin. The equations of Huxley (1932) used in a regression analysis have several advantages over the use of ratios. The natural log transformation necessary so that the slope of the regression becomes the ratio of the 2 growth rates has the added benefit of controlling the scale effect (variance increasing with the mean) that typically exists in weight data. In addition, the error variance in the analysis is only the error variance of the organ weight data, not a combination of both, although increased variance in fetal weight would be expected to increase the standard error of the estimate of the slope (but not the estimate itself) in the regression equation. One does not have to assume a linear relationship between growth rates, and curvilinearity of the relationship is easily tested. However, the major advantage of the Huxley (1932) equations, particularly for this report, is that if the log-transformed data of organ weight and fetal weights are linearly related as they are here, the slope obtained from the equation corresponds to the ratio between the growth of the organ and the whole fetus. This allows an easy interpretation of the relationship between growth of the 2 as proportional with a slope near 1, hypersensitive with a slope greater than 1, or sparing with a slope less than 1.

The effect of selection for UC on fetal liver growth in relation to fetal weight suggests that the differences in allometric growth of the liver may somehow play a role in increasing UC. This possibility is made more likely by the rough temporal coincidence of this effect (d 45 and 65) with a large proportion of the fetal losses that occur due to limitations in UC (d 30 to 40; Vallet, 2000). A further curiosity is the hypersensitivity effect observed in this experiment on d 45, which was greater in the UC line. However, simultaneously fitting the effects of fetal and placental weights suggested that on d 45 of pregnancy, the size of the placenta positively influences fetal liver growth. Ashworth et al. (2001) reported that the placental weight to fetal weight ratios are much greater for normal-sized fetuses compared with small fetuses, especially during early pregnancy; thus the relative contribution of the placenta to total conceptus weight is likely to be greater for larger fetuses. The fetal liver serves 2 main functions, metabolic and erythropoietic. Larger placentas would require greater metabolic activity because of increased transport and greater blood cell synthesis due to the increase in vascular volume. Albumin and α-fetoprotein, markers of liver metabolic function, have both been reported to be less in small fetuses and piglets compared with large piglets (Stone, 1981; Stone and Christenson, 1982), suggesting that the metabolic function of the fetal liver is compromised in small fetuses/piglets. Hepatocytes, which are responsible for the metabolic function and secrete both albumin and α-fetoprotein, are present beginning in early gestation (i.e., by d 24). Thus, one possible consequence of the change in allometric growth of the liver after selection for UC may be an improvement in metabolic function increasing the survival of small piglets. On the other hand, the number of circulating blood cells increases dramatically in the fetus from d 24 to 40 in pigs (Pearson et al., 1998) and, because the spleen is essentially nonexistent during this period, the primary fetal organ responsible for generation of blood cells is likely to be the fetal liver. The increase in early gestation occurs coincidently with residency of blood cell precursors in the fetal liver (Vallet, 2000) and increased erythropoietin receptor
gene expression by the fetal liver (Pearson et al., 2000). The mechanisms controlling the increase in residency of blood cell precursors and the increase in erythropoietin receptor gene expression are not well understood. Erythropoiesis in the fetal liver is maximal around d 60 (Ducsay et al., 1982) and is still present even in late gestation. We have also reported (Vallet et al., 2001) greater hematocrits on d 105 of gestation in the UC line compared with the CO line. Although the differences in allometric growth in this experiment were confined to d 45 and 65, fetal hematocrits have not been compared between the UC and CO lines at these times during pregnancy. Given this information, another possible explanation for the differences in allometric growth may be alterations or improvements in fetal erythropoiesis. Both mechanisms could play a role in subsequent UC, and further work is necessary to determine the role of the fetal liver in the survival of fetuses in a crowded uterine environment and the role of the placenta in controlling fetal liver growth.

The reductions in fetal heart weights in the OR line were probably due to the overall reduction in fetal weights in this line, which was observed previously (Christenson and Leymaster, 2002). The reduction in fetal weights was most likely due to the increased intrauterine crowding that resulted in this line due to increased OR and, therefore, increased number of embryos competing for uterine space during the period of elongation. The reduction in heart weights was consistent with the known effects of intrauterine growth restriction on heart development in other species (Barker, 2000; Robinson and Barker, 2002; Corstius et al., 2005). From previous reports, it is likely that this reduction in heart weight results in permanent changes in the piglets. The postnatal growth of small piglets does not compensate for low birth weight, resulting in a permanent reduction in overall body size at later ages (Widdowson, 1971; Ritacco et al., 1997; Milligan et al., 2002). Thus, the proportional reductions in heart weight in small pigs may contribute to the inability of small piglets to compensate. In addition, it is possible that this might compromise future fertility because cardiac output is a component of overall blood flow to the developing litter in swine. Further experiments will be necessary to truly address these questions.

Even though selection for uterine capacity did not alter the growth of the fetal brain, heart, and spleen, clearly allometric growth of fetal organs differs dramatically on different days of gestation for specific organs. Fetal brain weights were very resistant to differences in fetal weights on d 85 and 105 (brain sparing effect) but were much less resistant on d 65 and were greatly affected by changes in fetal weight on d 45. Ashworth et al. (2001) reported that the size differences between large and small littermates are established even during early pregnancy. Thus, the lack of resistance to changes in fetal weights during early pregnancy could explain some of the known differences in brain structure and function between large and small littermates (Dickerson et al., 1971; Tuchscherer et al., 2000) despite the clear ability of the brain to preserve normal growth during late gestation.

Likewise, the changes in proportionality of fetal heart weight to fetal weight during gestation suggested a slight heart-sparing effect and a proportionately greater need for heart function during early gestation. One might expect this to occur because the heart must actually perfuse both the fetus and the placenta. As previously indicated, the relative contribution of the fetus to the load on the heart would be low during early gestation and would increase with advancing gestation as fetal weight becomes much greater compared with placental weight (Knight et al., 1977). However, simultaneously fitting fetal and placental weights did not support an association between placental weight and fetal heart weight at any time during gestation. Although a significant effect was detected, most of the slopes obtained were near 0, indicating very little additional contribution of placental weights to differences in fetal heart weights. The heart-sparing effect, and the changes in this effect observed here, may be controlled by something other than the load represented by the placenta.

A slope of nearly 1 for the relationship between fetal liver weights and fetal weight after d 65 of gestation at first seems to contrast previous results that indicated that the proportion of fetal weight represented by fetal liver decreased until d 102 of gestation (Pomeroy, 1960; McPherson et al., 2004). Whereas these results are apparently contradictory, the method of analysis used in our study and previous studies differ. Using the means provided in Table 1, similar results to those reported previously can be generated; the proportion of fetal weight represented by fetal liver decreased until d 85 of gestation. Our analysis differs from that of McPherson et al. (2004) in that we compared allometric relationships within each day of gestation, not throughout gestation. Collectively, these 2 studies indicate that the proportion of fetal weight represented by fetal liver weight decreased throughout gestation; but after d 65, if measured on a single day, the slope of the relationship between fetal liver weights and fetal weights is nearly 1. This could only happen if the growth rate of the liver compared with the fetus slows uniformly with gestational age, regardless of the actual weight of the fetus, and provides an example of growth regulation that is gestation dependent, not fetal weight dependent.

**IMPLICATIONS**

These results indicate alterations in fetal liver growth in response to selection for uterine capacity and suggest the presence of mechanisms that control the growth of fetal brain, liver, and heart differentially during gestation. With further research, these mechanisms might be exploited to improve the distribution of nutrients to developing organs, which may result in increased uterine capacity and litter size in pigs.
LITERATURE CITED


