Associations between intrapartum death and piglet, placental, and umbilical characteristics

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ABSTRACT: Intrapartum death in multiparous gestations in sows (Sus scrofa) is often caused by hypoxia. There is little information in the literature on the assessment of the placenta in relation to intrapartum death in piglets. The aim of this study was to evaluate the impact of the placental area and weight upon piglet birth characteristics and intrapartum death. Litters from 26 Landrace-Yorkshire sows were monitored during farrowing and the status of each piglet was recorded, including blood parameters of piglets and their umbilical veins. Of 413 piglets born, 6.5% were stillborn. Blood concentrations of glucose, lactate, and CO₂ partial pressure were increased in the stillborn piglets (P < 0.05) and corresponding umbilical veins (P < 0.01) vs. live-born piglets, whereas pH and base excess were decreased (P < 0.001). Time from onset of parturition until birth was increased for piglets born dead vs. live (P < 0.001). Mean birth weight for piglets born dead was not different from live-born piglets (P = 0.631), whereas mean body mass index was reduced (P < 0.001). Mean placental area and placental weight belonging to stillborn piglets were not different from live-born piglets (P = 0.662 and P = 0.253, respectively). Blood concentrations of lactate, hemoglobin, and hematocrit recorded in all piglets pooled were associated with placental area (P < 0.05), but not with placental weight (P > 0.2). Piglet BW was positively correlated with placental area and placental weight (P < 0.001). The risk of being born dead increased with increasing birth order group, and broken umbilical cords explained 71% of the stillbirths (P = 0.001). We conclude that placental area and placental weight are both positively associated with piglet vitality than placental weight. Because umbilical cord rupture and prolonged birth time were associated with being born dead, umbilical cord rupture and placental detachment seem to be probable causes of intrapartum death.

Key words: hypoxia, intrapartum death, piglet, placenta, umbilical cord

INTRODUCTION

Intrapartum death occurs in all mammalian species, with increased risk in multiparous gestations (Luke, 1996; Jainudeen and Hafez, 2000; Walters, 2007). In swine, 6 to 8% of piglets are born dead (USDA, 2008; Agrovision, 2008; Ingris Animalia Norsvin, 2011) and stillbirth is often associated with hypoxia during delivery (Randall, 1972; Herpin et al., 1996; van Dijk et al., 2008). Some degree of fetal hypoxia during birth may be viewed as physiological due to compression of the umbilical cord when the fetus enters the pelvis. This promotes birth further, by stressing the fetus and thus increasing fetal movements, which stimulate the Ferguson reflex (Taverne and Noakes, 2009). Problems occur if hypoxia becomes severe. The percentage of intrapartum death increases with increasing litter size and farrowing time (Randall, 1972; Boulot et al., 2008; Andersen et al., 2011). Causes of severe hypoxia during delivery may have their origin in the uterus, fetal placenta, umbilical cord, or fetus. In humans, placental dysfunction is considered the major cause of late fetal death (VanderWielen et al., 2011). Because the pig placenta is epitheliochorial, the least intimate among placenta types of domesticated animals, the need for an adequately intimate connection between sow and the piglets is met by the large total surface area of the diffuse placenta (Senger, 2003). However, there is a relative paucity of information in the literature on the assessment of the placenta in relation to intrapartum death in piglets.

The aim of this study was to evaluate the impact of area and weight of the fetal placenta upon stillbirth
and piglet characteristics at birth as evaluated by piglet weight, body mass index (BMI), blood chemistry, and hematological variables.

**MATERIALS AND METHODS**

The experimental protocol for this study did not require approval by the Norwegian Animal Research Authority due to an exception for such procedures in the Norwegian regulations for animal testing (FOR 1996-01-15 no. 23, Regulation of animal testing, §2: Scope).

**Animals**

Landrace-Yorkshire sows (n = 26) were selected from a sow pool system as described by Dalin et al. (1997). All sows originated from the same multiplier herd and were housed in a farm in the southeastern part of Norway. The sows were inseminated with heterospermic semen from Landrace-Duroc boars twice by the same technician at sows were housed in a farm in the southeastern part of Norway. The sows originated from the same multiplier herd and were housed in a farm in the southeastern part of Norway. The sows were grouped into 3 categories according to parity number: First and second parity sows were grouped in Parity Group 1, third and fourth parity sows were grouped in Parity Group 2, and fifth to eighth parity sows were grouped in Parity Group 3. Gestation length and litter size were recorded.

Each piglet was recorded as dead or alive. A dead piglet was defined as a piglet born without respiration or palpable heartbeats as assessed by a veterinarian. The gender of the piglet was recorded, and whether it had meconium staining or not. If an intact umbilical cord was present, blood samples were taken from the umbilical vein. The cord was then double ligated with a color code, and cut between the ligations. The piglets were held in dorsal recumbency, and 0.5 mL of blood was evacuated from vena jugularis externa/interna/communis using 2-mL plain syringes with 23-gauge needles. Whole blood from the umbilical veins and piglets was immediately analyzed for concentrations of glucose, lactate, oxygen partial pressure (pO₂), carbon dioxide partial pressure (∆CO₂), pH, base excess (BEₐt), hemoglobin, hematocrit, sodium (Na⁺), potassium (K⁺), and ionized calcium (Ca²⁺) on a hand-held Epoc portable clinical analyzer (Epocal Inc., Ottawa, Canada). Times until birth of each pig from first expulsion of a piglet in the litter were recorded, as well as birth intervals and whether the umbilical cord was ruptured or not. The piglets were weighed on a scale with a 10-g accuracy according to the manufacturer (Premium, EKS International SAS, Wittisheim, France). Body length was measured from the os occipitale to the root of the tail. Weight and length were used for calculation of BMI [BW (kg)/length (m²)]. The piglets were grouped into 3 categories according to birth order: first, middle, or last third of each separate litter. Whether the piglets were born under birth assistance was also recorded, and all piglets of the same litter being born after these were also defined as born under birth assistance. Gross necropsy of the dead piglets was performed within 4 h, and heart, liver lobes, and the middle lung lobes were collected and stored in 4% formalin. Lung tissue samples were later stained with hematoxylin-eosin color by routine protocol and histopathologically examined at the Department of Basic Science and Aquatic Medicine, Norwegian School of Veterinary Science, Oslo, Norway.

The expelled fetal placentas (i.e., the chorioallantois) were kept at room temperature until examination the same or the next day, when they were rinsed in water, each placenta separated, and left to remove excess water for 1 h. Attached amniotic membrane, avascularized necrotic parts of the tip of each chorion, and the umbilical cord at the junction where it joins the placenta and splits into its tributaries, were all removed. Each placenta was spread on solid paper, and the circumference was cut out with a sharp pair of scissors. The paper was numbered, dried, and the area in square centimeters was later recorded by a planimeter (Lasico 42P, B-90899, Los Angeles Scientific Instrument Co., Inc., CA) and multiplied.
by 2 to give the macroscopic surface area. The chorioallantoic sac was then opened at the antimesometrial side and a 5-cm by 5-cm quadrant was placed equidistant between the edge and the center, from the allantoic side. All areolae visible within the quadrant were counted. Further, a 1.5-cm by 1.5-cm tissue sample was removed from the central part of the placenta and put on formalin for histopathology at the Department of Basic Science and Aquatic Medicine, as described previously for the lung samples. The chorioallantoic sac was weighed wet on a digital scale with a 1-g accuracy according to the manufacturer (Z17489, Silvercrest, Milomex Ltd., Bedfordshire, UK). All recorded variables of the placenta, with the exception of those found by the histopathological examination, were performed blindly with regard to piglet identity, but with known sow identity.

Statistical Analyses

Mean level of litter size was calculated using the statistical software JMP 8 (SAS Inst. Inc., Cary, NC). Similarly, means of all 11 blood variables of the piglets and their corresponding umbilical veins were recorded. One-way ANOVA were used for the comparison of blood variables between piglets born dead vs. alive, and for the comparison of blood variables between those born during or after birth assistance, and those that had not been exposed to manual birth assistance. The same statistical procedure was used for the comparison between dead vs. live piglets regarding the time from onset of parturition until birth, birth interval, birth weight, BMI, placental area, and placental weight. Univariate linear regression analyses were used for assessing the association between placental area and blood variables, as recorded in piglet and umbilical vein. The same procedure was used for the evaluation of an association between placental weight and blood variables recorded in the piglet and umbilical vein. Lastly, univariate linear regression was used to evaluate associations between placental area and piglet weight, for the association between placental weight and piglet BW, and for the association between placental area and placental weight. Univariate c² analyses were used to study the association between the state of the umbilical cord and stillbirth. Attributable risk calculation [i.e., (incidence in exposed group) – (incidence in nonexposed group)/(incidence in exposed group)], was used to evaluate the risk of being stillborn if the umbilical cord was ruptured. Univariate c² analyses were also used to study the association between birth order group and stillbirth, and between meconium staining of the piglet and stillbirth.

For the outcome variables birth weight, BMI, placental area, and placental weight, separate multivariable GLM were conducted using the xtreg option in Stata SE11 (StataCorp LP, College Station, TX). The explanatory variables gestation length, parity group, birth assistance, birth order group, gender, and litter size were all simultaneously included in the model. Also, sow was included as a random effect variable to account for clustering at the sow level. A backwards elimination procedure was employed, and explanatory variables with an association to the outcome variable yielding a P-value > 0.10 were omitted from the final models. Dead vs. live-born was forced into all models for the comparison. A similar procedure was also used for the associations between placental area and the blood concentrations of lactate, hemoglobin, and hematocrit in the piglet. The outcome variable lactate was log transformed to approximate normality of residuals. Overall statistical significance of the models was assessed by the type III F-test in Stata SE11. Homoscedasticity and normality of the residuals were assessed using plots of standardized residuals.

RESULTS

Parity number of the sows ranged from 1 to 8, with 7 sows in Parity Group 1, 11 sows in Parity Group 2, and 8 sows in Parity Group 3. Of the 26 sows, 5 required birth assistance with 15 IU oxytocin injected intramuscularly once, and manual birth extraction of 42 piglets, of which 6 were dead. In total, 413 piglets were born, and mean litter size was 15.9 ± 0.59 piglets. Of all piglets, 27 (6.5%) were stillborn; 16 males, 9 females. Gender was not recorded in 2 of the piglets. All 9 mummiﬁed piglets from a total of 7 litters were excluded from the study. Of the 27 stillborn piglets, 4 piglets were not blood sampled due to lack of time, and in 5 piglets there was failure in recording during analyses. Additionally, 12 piglets failed to produce a result from the analysis of blood lactate concentration. Analyses of blood samples from the umbilical veins were unsuccessful in 5 cases due to lack of time, and in an additional 17 cases because no blood was available in the cord.

Recorded blood concentrations of glucose, lactate, and pCO₂ were increased (P < 0.05) in the piglets born dead vs. live-born piglets and their corresponding umbilical veins (P < 0.001), whereas pH and base excess were decreased (P < 0.001; Table 1). Recorded blood concentration of glucose was increased in piglets born during or after birth assistance vs. piglets born naturally (P < 0.001), whereas base excess in the umbilical vein was decreased (P = 0.036; data not shown).

Time until birth from expulsion of the first piglet, and birth interval were increased for stillborn piglets vs. live-born (P < 0.001 and P = 0.037, respectively). Mean birth weight for piglets born dead vs. alive was not different (P = 0.631), whereas BMI was decreased in piglets born dead (P < 0.001). Mean placental area and placental weight belonging to piglets born dead vs. alive were not different (P = 0.662 and P = 0.253, respectively; Table 2). After adjusting for explanatory
variables, BMI remained decreased in piglets born dead vs. alive \((P < 0.001)\), and placental area and placental weight remained not different \((P = 0.386 \text{ and } P = 0.311, \text{ respectively}; \text{Table 3})\). Univariate analyses of piglet BW showed associations with placental area \((n = 179; R^2 \text{ adjusted} = 0.50)\) and placental weight \((n = 180; R^2 \text{ adjusted} = 0.27)\), and placental weight showed a significant association with placental area; \(n = 178; R^2 \text{ adjusted} = 0.53 \text{ \((P < 0.001)\)}\).

Univariate analyses of blood concentrations of lactate, hemoglobin, and hematocrit recorded in the umbilical vein revealed that these variables tended not to be associated with placental area \((P > 0.05)\), whereas the same variables recorded in the piglet were associated with placental area \((P < 0.05)\). Neither the recorded blood variables in the umbilical vein nor in the piglet (except for \(Na^+\)) were associated with placental weight \((P > 0.1; \text{Table 4})\). After adjusting for explanatory variables, lactate
of 24 stillborn piglets vs. in 190 of 351 live-born piglets ($P = 0.013$).

Of the 27 stillborn piglets, 25 were subjected to autopsies. On macroscopic examination, no piglet had anomalies, and all except 1 had a pale or pink color and normal texture of the skin. The 1 exception showed a purple discoloration and had a softer skin texture. Meconium was observed in the throat of 9 piglets, 6 had meconium in the bronchi, 17 had meconium in the stomach, and 20 piglets had meconium in any of these locations. On histopathology of 14 stillborn piglets, meconium was found in the bronchi, bronchioles, or alveoli of all lung samples, except 1. Hearts and liver samples revealed no pathological findings, except 1 liver with cholangitis.

Macroscopic examination of the placentas revealed variations in color from pale to dark pink, dependent on the degree of filling of blood vessels, and edema. Areolae varied within the quadrant from 0 to 30 in numbers, appeared from opaque to white or red, from 1- to 4-mm diameter in size, and from flat to convex in shape. Macroscopic findings of the areolae thus varied too extensively to be categorized. On histopathological examination, placentitis was not observed in either the stillborn or live-born groups with regard to increased amounts of plasma cells or neutrophilic cells in the epithelium or interstitium.

**DISCUSSION**

The overall number of stillborn piglets in this study was in accordance with results from other studies (USDA, 2008; Agrovision, 2008; Ingris Animalia Norsvin, 2011).

Blood glucose is an important nutrient in all cells. In domestic animals, blood concentrations of glucose increase during stress due to glycogenolysis caused by activity in the sympathetic nervous system and by the stress hormone cortisol (Sjaastad et al., 2010a). Fetal piglets store glycogen (Randall, 1987; Fowden et al., 1995), which can be mobilized to yield glucose (Comline et al., 2008; Agrovision, 2008; Ingris Animalia Norsvin, 2011). From the placenta to the piglet, the increased concentrations of lactate and placental area were unaffected by explanatory variables ($P > 0.1$). Parameter estimates were therefore similar as the means; 95% confidence intervals were from opaque to white or red, from 1- to 4-mm diameter in size, and from flat to convex in shape. Macroscopic findings of the areolae thus varied too extensively to be categorized. On histopathological examination, placentitis was not observed in either the stillborn or live-born groups with regard to increased amounts of plasma cells or neutrophilic cells in the epithelium or interstitium.

**Table 4.** Univariate associations between placental area and placental weight, and recorded blood variables in the piglets and corresponding umbilical veins

<table>
<thead>
<tr>
<th>Item</th>
<th>Piglet Placental area</th>
<th>Piglet Placental weight</th>
<th>Umbilical vein Placental area</th>
<th>Umbilical vein Placental weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, mmol/L</td>
<td>151.0, 0.153</td>
<td>79.0, 0.295</td>
<td>151.0, 0.227</td>
<td>81.0, 0.476</td>
</tr>
<tr>
<td>Lactate, mmol/L</td>
<td>129.0, 0.008</td>
<td>77.0, 0.072</td>
<td>129.0, 0.211</td>
<td>78.0, 0.113</td>
</tr>
<tr>
<td>Partial pressure</td>
<td>148.0, 0.206</td>
<td>89.0, 0.832</td>
<td>149.0, 0.220</td>
<td>91.0, 0.954</td>
</tr>
<tr>
<td>O₂, mmHg</td>
<td>147.0, 0.189</td>
<td>88.0, 0.569</td>
<td>149.0, 0.009</td>
<td>90.0, 0.057</td>
</tr>
<tr>
<td>Hematocrit, L/L</td>
<td>145.0, 0.353</td>
<td>86.0, 0.067</td>
<td>146.0, 0.344</td>
<td>88.0, 0.832</td>
</tr>
<tr>
<td>Hemoglobin, g/L</td>
<td>145.0, 0.035</td>
<td>86.0, 0.068</td>
<td>147.0, 0.308</td>
<td>88.0, 0.859</td>
</tr>
<tr>
<td>Na⁺, mmol/L</td>
<td>147.0, 0.189</td>
<td>88.0, 0.569</td>
<td>149.0, 0.009</td>
<td>90.0, 0.057</td>
</tr>
<tr>
<td>K⁺, mmol/L</td>
<td>147.0, 0.851</td>
<td>88.0, 0.533</td>
<td>148.0, 0.468</td>
<td>90.0, 0.401</td>
</tr>
<tr>
<td>Ca²⁺, mmol/L</td>
<td>147.0, 0.511</td>
<td>88.0, 0.421</td>
<td>149.0, 0.852</td>
<td>90.0, 0.904</td>
</tr>
</tbody>
</table>

1Values are n, P-value.

2Parameter estimate = slope of multivariable regression for explanatory variables.

3Logarithmically transformed.

**Table 5.** Associations between piglet blood concentrations of lactate and placental area

<table>
<thead>
<tr>
<th>Item</th>
<th>n</th>
<th>Parameter estimate</th>
<th>SE</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate, mmol/L</td>
<td>129</td>
<td>-0.000</td>
<td>0.000</td>
<td>0.021</td>
</tr>
<tr>
<td>Placental area, cm²</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth Order Group 1</td>
<td></td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Birth Order Group 2</td>
<td></td>
<td>0.279</td>
<td>0.077</td>
<td>&lt;0.000</td>
</tr>
<tr>
<td>Birth Order Group 3</td>
<td></td>
<td>0.323</td>
<td>0.081</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Male piglet</td>
<td></td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Female piglet</td>
<td></td>
<td>0.157</td>
<td>0.067</td>
<td>0.018</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>1.662</td>
<td>1.155</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

1Piglets were grouped into 3 categories according to birth order: first, middle, or last third of each litter.

2Parameter estimate = slope of multivariable regression for explanatory variables.

3Logarithmically transformed.

**Table 6.** Number of stillborn piglets and piglets born with a broken umbilical cord in each birth order group

<table>
<thead>
<tr>
<th>Item</th>
<th>Birth order group 1</th>
<th>Birth order group 2</th>
<th>Birth order group 3</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stillborn piglets in total</td>
<td>4</td>
<td>8</td>
<td>15</td>
<td>0.014</td>
</tr>
<tr>
<td>Stillborn piglets with a broken umbilical cord</td>
<td>3</td>
<td>3</td>
<td>7</td>
<td>0.563</td>
</tr>
<tr>
<td>Piglets (dead or alive) born with a broken umbilical cord</td>
<td>15</td>
<td>34</td>
<td>36</td>
<td>0.002</td>
</tr>
</tbody>
</table>

1There were equally many piglets in each birth order group and comparisons were made with c² analysis in JMP 8 (SAS Inst. Inc., Cary, NC).
trations of glucose recorded in the stillborn piglets and in their umbilical veins in this study seem to be an indicator of fetal stress, as suggested by other studies (Herpin et al., 1996; Trujillo-Ortega et al., 2007). The increased glucose concentration of piglets born during or after birth assistance also supports this. Conclusions should nevertheless be drawn with care, due to very few samples from the umbilical veins. The increased concentrations of glucose in the stillborn piglets in this study could also be due to failure of the fetoplacental tissues of stillborn piglets to metabolize glucose. Fructose, which is converted from glucose, could provide an index of placental function. This variable was not recorded in this study because earlier studies indicate that fructose is of less importance for piglet perinatal survival (Aherne et al., 1969; Leenhouders et al., 2002).

Lactate is produced from glucose in the absence of oxygen and is an indicator of hypoxic stress (Herpin et al., 1996; van Dijk et al., 2008). Lactate concentrations were increased in the stillborn piglets and increased with birth order group. This indicates stress hypoxia during birth and is in accordance with earlier results of Herpin et al. (1996) and van Dijk et al. (2006). The recorded increased concentrations of pCO₂ in the umbilical vein of the stillborn piglets in this study indicate a reduced gas exchange over the placenta, and suggest placental dysfunction or a mechanical cause (i.e., partial or complete placental detachment or a broken umbilical cord). Because histopathology of the placenta did not reveal any abnormalities, a mechanical cause seems most probable. In the extracellular fluid, pH must be in the range of 6.9 to 7.8 to be consistent with life (Sjaastad et al., 2010b). Not surprisingly, the pH of stillborn piglets was below this range. Like pH, BE_{ecf} is also correlated with lactate, but requires a somewhat longer time span to be affected. This is supported by van Dijk et al. (2006), who found that BE_{ecf} was not affected by the condition of the umbilical cord at birth, in contrast to pH. In our study, BE_{ecf} was decreased in the umbilical cord and the corresponding dead piglets, which suggests severe hypoxia for some time. The BE_{ecf} was also increased in piglets born during or after birth assistance (i.e., piglets that had been subjected to a protracted course of delivery).

Unfortunately, several of the dead piglets and their umbilical cords had no recorded blood variables. This was partly due to lack of time, such as when several piglets were born simultaneously and partly by failure of analyses by the Epoc instrument. Failure of analyses especially occurred for the variable lactate, of which 74% happened in stillborn piglets vs. 8% in live-born piglets. This indicates that the results were outside the range of the Epoc analyser, which for lactate was 0.30 to 20 mmol/L. It may also be due to microcoagulation of noncirculating blood in the dead piglet. In addition, blood samples from umbilical cords were limited because two-thirds of the cords were either broken or empty due to tense stretching.

Increased time until birth from expulsion of the first piglet, and increased birth interval was associated with stillbirth and is in accordance with other studies (Friend et al., 1962; Randall, 1972). Whether the increased time was secondary to a dead fetus as suggested by van Dijk et al. (2005), or if the fetus died due to hypoxia as a result of repetitive uterine contractions (Curtis, 1974; Bakker et al., 2007) or a complete placental detachment, cannot be confirmed. Nevertheless, because there were no signs of placentitis, and because no piglet had visible anomalies and only one was discolored, it seems most probable that death was secondary to increased birth time and not vice versa.

The number of piglets born with a ruptured umbilical cord in this study was in accordance with results from van Dijk et al. (2006). The number increased with birth order group, as did the number of stillborn piglets. A broken umbilical cord is harmful due to the abrupt loss of oxygenation. It is furthermore probable that the hazard of the cord breaking, despite its elasticity, increases with increasing birth order group due to a longer anatomical distance through the uterine horn. Hence, it is likely that a ruptured cord is the cause of fetal death and not the other way around, unless the fetus has been dead for a longer time. High concentrations of meconium and low pH of the umbilical cord blood can cause apoptosis of smooth muscle cells, and thus damage the umbilical cord (Altschuler et al., 1992). Piglets are reported to survive for only 5 min after asphyxia (Miller and Miller, 1965), although clamping of the umbilical cord of full-term piglets for 5 to 8 min did not result in severe asphyxia in another study (van Dijk et al., 2008).

Meconium is expelled from the colon descendens when the contractility of the gut increases. Moreover, the anus dilates in stressed fetuses (Curtis, 1974). Hypoxia during parturition is a common cause of acute fetal stress, and meconium staining of the skin is, therefore, associated with hypoxic stress in several species, including humans (Swarnam et al., 2012). Meconium staining may be somewhat more difficult to record in piglets than in uniparous gestations due to discoloring of the birth canal caused by debris from previously passing meconium-stained piglets.

The piglet is a product of the placenta, as supported by this study where piglet BW was positively associated with placental area and placental weight, and piglet blood concentrations of hemoglobin and hematocrit were positively associated with placental area. The area of the elastic fetal placenta is difficult to measure exactly, although easier when the membranes are mostly intact, as in this study, and not cut open along any of the curvatures. Inter-
et al. (1977), placental surface area per fetus was <800 cm² at d 101 of gestation, when the surface area was at its maximum before it slightly decreased towards term. Van Rens et al. (2002), on the other hand, recorded a surface area of about 1,600 cm² at delivery, and Baxter et al. (2008) recorded mean surface area of approximately 1,500 cm². In a study of Knight et al. (1977), placental surface area per fetus was <800 cm² at d 100 of gestation. In this study, the area was also recorded by the same planimeter technique as in ours, but it seems that the given results were of the intact chorioallantoic sacs and that the recorded measurements were not multiplied by 2. Variations in recorded placental area may be explained by different practice of registration, crossbreeds (Wilson et al., 1998; Biensen et al., 1999; Vonnahme et al., 2002), and litter size because increasing litter size results in reduced placental area, as shown in this study. Very interestingly, in the study of Knight et al. (1977), placental area was greater in intact control gilts vs. unilaterally hysterectomized gilts, and they concluded that placental insufficiency was the primary cause of decreased fetal growth of piglets in the unilaterally hysterectomized group.

Body mass index was a better predictor of the outcome (dead or alive) at birth than birth weight, and is in accordance with the results of Baxter et al. (2008). As in this study, they found no association between placental area or placental weight and stillbirth, although these results were not adjusted for clustering at the sow level. It is reasonable that birth weight is not a reliable predictor of stillbirth. The association between fetal size and stillbirth may be confounded by the birth rank of the piglet, because stillbirth often occurs in later-born piglets, which are often larger. Stanton and Carroll (1974) hypothesized that this was due to increased vascularization from the cranial part of the uterus. Regardless, our study supports the notion that larger placental area and placental weight are observed in the last birth-order group. Birth weight was also numerically increased in the last birth-order group. Care should nevertheless be taken not to conclude that being born in the last birth order group is synonymous with a cranial uterine location of the fetus. Trujillo-Ortega et al. (2007) found that heavier piglets were more asphyxic than lighter littermates. This is supported by Leenhouters et al. (2001, 2002), who found that selection for improved piglet survival at birth will lead to slightly smaller piglets, yet in contrast to results of van Dijk et al. (2006), who found that lighter piglets were more acidic than their littermates at birth, independent of birth order or condition of the umbilical cord. In humans, the risk of stillbirth for full-term twin fetuses is not associated with birth weight, even if the smaller twin experiences intrauterine malnutrition (Sonntag et al., 1996). It must be emphatically sized that lack of an association between birth weight and stillbirth, as in this study, is in contrast to postpartum survival, where studies unanimously show that heavier birth weight is positively associated with post-partum survival. In our study, placental area and placental weight were not associated with the incidence of stillbirth. Because placental area and weight were both correlated with piglet birth weight, it would be interesting to study whether these variables are associated with postpartum survival.

In conclusion, this study supports the hypothesis that stillbirth is associated with hypoxia during delivery, but does not indicate that placental area or placental weight is associated with being born dead vs. alive. Nevertheless, placental area was a better predictor of piglet vitality than placental weight. Because umbilical cord rupture and prolonged birth time were associated with being born dead, umbilical cord rupture and placental detachment seem to be probable causes of intrapartum death.

**LITERATURE CITED**


