Influence of colostrum intake on piglet survival and immunity

N. Devillers¹,², J. Le Dividich¹,² and A. Prunier¹,²

¹INRA, UMR1079 Systèmes d’Élevage Nutrition Animale et Humaine, F-35590 Saint-Gilles, France; ²Agrocampus Ouest, UMR1079 Systèmes d’Élevage Nutrition Animale et Humaine, F-35000 Rennes, France

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Colostrum intake from birth to 24 h after the onset of parturition (T₂₄) was estimated for 526 piglets from 40 litters. Plasma concentrations of immunoglobulin G (IgG), lactate, glucose and cortisol were determined at T₂₄ for six piglets per litter. Plasma IgG concentration was also assayed at weaning (28 days) on the same piglets. Rectal temperature was measured at T₂₄ on all piglets. Mortality was recorded until weaning and comparisons were made between piglets that died before weaning and those that were still alive at weaning. The piglets that died before weaning had lower birth weight, lower colostrum intake, lower weight gain between birth and T₂₄, and had a lower rectal temperature, higher plasma cortisol concentration and lower plasma IgG and glucose concentrations at T₂₄ than piglets still alive at weaning. In addition, a higher proportion of piglets that died before weaning had difficulty taking their first breath after birth and were affected by splayleg. Considering all piglets, colostrum intake was positively related to rectal temperature and plasma glucose concentration and negatively related to plasma cortisol concentration at T₂₄. Plasma IgG concentration at T₂₄ was explained by colostrum intake, IgG concentration in the ingested colostrum, birth weight and birth rank (P < 0.0001). Plasma IgG concentration at weaning was related to plasma IgG concentration at T₂₄ (r = 0.54; P < 0.0001) and to colostrum intake (r = 0.32; P < 0.0001). Finally, body weight was explained by colostrum intake, birth weight and age until 6 weeks of age (P < 0.0001). These results show that colostrum intake is the main determinant of piglet survival through provision of energy and immune protection and has potential long-term effects on piglet growth and immunity.

Keywords: piglet, colostrum intake, mortality, birth weight, immunoglobulin G

Implications

Piglet mortality remains a major problem in pig production, with the pre-weaning mortality rate stabilized at around 20%. Selection based on prolificacy has increased the number of live-born piglets to as many as 16 in hyper-prolific sows, resulting in greater heterogeneity of piglet birth weight and a higher number of weak piglets at birth. Providing satisfactory colostrum intake to all piglets is a major challenge, considering the variations in colostrum production between sows and in colostrum intake between piglets. This study aims to determine the minimum amount of colostrum that suckling piglets should ingest to ensure their survival and optimum performance.

Introduction

Insufficient colostrum intake has been identified as one of the major causes of neonatal mortality in pig production (Edwards, 2002). Indeed, 50% of pre-weaning mortality occurs within 3 days after birth (Tuchscherer et al., 2000) and mainly affects piglets characterized by low birth weight and low weight gain, which can be related to low colostrum intake (de Passillé and Ruschen, 1989; Milligan et al., 2002). Colostrum intake plays a major role in piglet development by providing energy for thermoregulation (Le Dividich et al., 2005), enabling immune transfer from the sow (Rooke and Bland, 2002) and stimulating intestinal development (Xu et al., 2000). The amount of colostrum ingested is highly variable between piglets and depends on both the sow’s ability to produce enough colostrum for the whole litter and the piglet’s ability to extract colostrum from the teats. Since colostrum production is independent of litter size (Devillers et al., 2007), factors influencing a piglet’s colostrum intake, such as low birth weight and vitality (Herpin et al., 1996; Tuchscherer et al., 2000; Devillers et al., 2007), are crucial for its survival. Most of the studies looking at the role of the colostrum in piglet physiology and immunity were conducted in experimentally controlled conditions using force- or bottle-feeding. Studies in naturally suckling piglets are more scarce, because measuring colostrum intake is more difficult under those conditions and necessitates the use of techniques.
such as the weigh-suckle-weigh method, which influences piglets’ nursing behavior (Pettigrew et al., 1985). This study used a colostrum intake estimation method based on piglet growth over 24 h (Devillers et al., 2004b) to study the impacts of colostrum intake on the survival, physiology and immunity of piglets in conditions close to commercial conditions.

Material and methods

Animals and measurements
A total of 526 piglets from 40 Landrace × Large White sows (22 primiparous and 18 multiparous), which were inseminated with Piétrain semen, were housed in individual farrowing crates (2 × 2.5 m) with straw bedding. Thirty-seven percent of the parturitions occurred naturally, and the other 63% were induced at 114 days of gestation with 1 ml of prostaglandins (Alfabedyl; CEVA Santé Animale, Libourne, France). Parturitions were watched, but observers interfered as little as possible in the farrowing process. Piglets born in placental membranes were extracted and resuscitated if necessary. No additional help or care was given. The births of the first and last piglets, respectively, were considered to be the beginning (T0) and end of parturition. At birth, the piglets were weighed and vitality traits, namely umbilical cord already ruptured before birth, difficulty in breathing (more than 5 s until first attempt to breathe after birth) and piglet still inside placental membranes at birth, were assessed. At 24 h after the onset of parturition (T24), the piglets were weighed again, and rectal temperature and the presence of splayleg were recorded. No fostering was done before T24. Thereafter, the piglets were weighed every week until 42 days of age.

Each piglet’s colostrum intake between birth and T24 was estimated according to a previously described equation (Devillers et al., 2004b) with the mean interval between birth and first suckling estimated to be 30 min as previously reported (Devillers et al., 2007) and where the piglet’s colostrum intake (CI, in g) is calculated from birth weight (BWb, in kg), weight gain (BW−BWb, in kg) and age (t, in min), as follows:

\[
CI = -217.4 + 0.217 \times t + 1861019 \times BW/t + BW_b \\
\times (54.14 - 1838592/t)
\]  
(1)

Sampling and analyses
A 100-ml colostrum sample was collected manually from each sow across all teats at T0 and 2, 4 and 24 h later and was filtered through gauze. Each colostrum sample was analyzed for nutritional composition and immunoglobulin G (IgG) concentration. Nitrogen (N) content was determined according to the Dumas method (Association of Official Analytical Chemists 7 024, 1831) based on sample pyrolysis and direct determination of N2 using an automatic device (LECO FP-428; LECO Corporation, St. Joseph, MI, USA). Crude proteins were estimated as N × 6.38 (Gordon and Whittier, 1965), and total lipids were measured according to the Gerber method (Association of Official Analytical Chemists, 1990). Lactose was assayed using an enzymatic method (Lactose/o-galactose test combination, ref. 0176303; R-Biopharm – Roche, Darmstadt, Germany).

A 5-ml blood sample was collected from the jugular vein of six piglets per litter (two with low birth rank, two with intermediate birth rank and two with high birth rank) at T24 (n = 246) and at weaning (n = 220). The blood samples were collected in heparinized tubes, placed immediately on ice and centrifuged for 10 min at 3000 × g and 4°C. Hemocrit was also measured. All samples were stored at −20°C until analysis. The plasma samples taken at T24 were analyzed for glucose, lactate and cortisol. Lactate and glucose were assayed with commercial enzymatic kits (l-lactic acid test combination, ref. 139084, and o-glucose/o-fructose test combination, ref. 139106; Boehringer, Mannheim, Germany). Cortisol was assayed with a commercial radioimmunoassay (RIA) kit (ref. 2466; IMMUNOTECH, Marseille, France). The assay sensitivity was 1.9 ng/ml. The plasma samples collected at T24 and at weaning and the colostrum samples were analyzed for IgG concentration using a previously described ELISA method (Devillers et al., 2004a) with a sensitivity of 23 ng/ml.

Statistics and data analyses
Statistical analyses were done with the Statistical Analysis Systems software program (SAS, 2002). Differences between categories based on mortality were tested with ANOVA using the MIXED procedure (birth weight, colostrum intake, weight gain, rectal temperature and plasma cortisol, IgG, glucose and lactate concentrations) and the GLIMMIX procedure with a logit transformation (birth rank, interval from previous birth and time of birth), or with \( \chi^2 \) tests (vitality variables). Relationships between colostrum intake, colostrum composition and physiological variables were described using principal component analysis (PCA; PRINCOMP procedure). Further reference to the colostrum concentration of any component corresponds to the estimate of the average concentration of the component \([C_{1a}]\) in the colostrum ingested for each piglet, which was calculated from the colostrum composition measured at T0, T2, T4 and T24 (\([C_{1a}], [C_{2a}], [C_{4a}] \) and \([C_{24a}]\), respectively) and adjusted to the piglet’s time of birth (TB in h) according to the following equation:

\[
\text{If } T_B < 2, \text{Then } [C_{1a}] = ((2 - T_B) \times ([C_{1a}] + [C_{2a}]) / 2 + 2 \times ([C_{2a}] + [C_{4a}]) / 2 + 20 \times ([C_{4a}] + [C_{24a}]) / 2) / (24 - T_B);
\]

\[
\text{ELSE IF } T_B < 4, \text{Then } [C_{1a}] = ((4 - T_B) \times ([C_{1a}] + [C_{2a}]) / 2 + 20 \times ([C_{4a}] + [C_{24a}]) / 2) / (24 - T_B);
\]

\[
\text{ELSE}[C_{1a}] = (([C_{1a}] + [C_{2a}]) / 2)
\]  
(2)

The average colostrum composition for all sows was published previously (Devillers et al., 2007). Regression
analyses and Pearson’s or Spearman’s correlations (REG and CORR procedures) were used to further describe the specific relationship between the two variables. Multiple regression analysis was also used to determine significant factors contributing to variations in plasma IgG concentration at T24. Synthetic variables were also calculated and analyzed in order to estimate IgG absorption. Such variables included the estimated quantity of IgG ingested between birth and T24, calculated by multiplying the piglet’s colostrum intake until T24 and the average IgG concentration of the colostrum ingested as calculated in equation (1). In addition, total amounts of IgG present in plasma were calculated, in the manner used by Bland et al. (2003), from piglet plasma IgG concentration, live weight and hematocrit at T24, assuming an average blood volume of 95 g/kg live weight. Piglet weight between T24 and 6 weeks of age was analyzed for piglets distributed in five categories of colostrum intake according to the 20th, 40th, 60th and 80th percentiles and using the mixed model procedure, with repeated-measures analyses including the factor day, and with colostrum intake, birth weight, litter and the colostrum intake \times day interaction as the main factors. Data in the text are means ± s.e.m., or least square means (CI).

Results

Results relative to the influence of piglet and sow characteristics on colostrum intake were published previously (Devillers et al., 2007). This study focused on the consequences of colostrum intake for piglet survival and immunity.

Colostrum intake and mortality

Table 1 describes the live-born piglets’ characteristics and their performances and physiology at T24 according to the piglets’ age at death in comparison to piglets still alive at weaning. In comparison to live-born piglets, stillborn piglets were characterized by a higher birth rank (10.2 [7.8–12.5]; \(P < 0.05\)) and a longer interval from the previous birth (49.8 [31.9–74.4]; \(P < 0.05\)). A large proportion of them had their umbilical cord already ruptured at birth (42.9%). The piglets that died before weaning were characterized by lower birth weight, a higher incidence of difficulty taking their first breath after birth and a higher occurrence of splayleg than piglets still alive at weaning, indicating a direct relationship between low vitality at birth and early mortality. After birth, piglets that died before weaning also had a lower colostrum intake, lower weight gain (or even weight loss), lower rectal temperature, higher plasma cortisol concentration and lower plasma IgG and glucose concentrations than piglets still alive at weaning. The principal causes of death for the live-born piglets were weakness (49.5%) and crushing by the sow (34.7%).

Colostrum intake and piglet physiology and immunology

The relationships between colostrum intake and the composition of the colostrum ingested and physiological variables measured at T24, as determined by PCA, are presented in Figure 1. Many of the variables, including birth weight, rectal temperature and plasma glucose and IgG concentrations, were positively related to colostrum intake, which was negatively related to plasma cortisol concentration. In contrast,

Table 1 Characteristics at birth, growth performance and physiological state of piglets born alive

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Between birth and 3 days of age</th>
<th>Between 4 days of age and weaning</th>
<th>Piglets alive at weaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>At birth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n (total)</td>
<td>65</td>
<td>36</td>
<td>411</td>
</tr>
<tr>
<td>% of total piglets born</td>
<td>12.4%</td>
<td>6.8%</td>
<td>78.1%</td>
</tr>
<tr>
<td>Birth weight (g)</td>
<td>1078 ± 45a</td>
<td>1259 ± 54b</td>
<td>1407 ± 33c</td>
</tr>
<tr>
<td>Birth rank</td>
<td>7.7 (6.6–8.8)</td>
<td>8.6 (7.1–10.0)</td>
<td>7.2 (6.8–7.6)</td>
</tr>
<tr>
<td>Interval from previous birth (min)</td>
<td>13.7 (9.1–20.5)</td>
<td>11.4 (6.2–20.7)</td>
<td>13.8 (11.7–16.2)</td>
</tr>
<tr>
<td>Time of birth (min)</td>
<td>98 (80–120)</td>
<td>122 (95–154)</td>
<td>96 (88–104)</td>
</tr>
<tr>
<td>In placental membranes</td>
<td>3.1%</td>
<td>2.8%</td>
<td>2.2%</td>
</tr>
<tr>
<td>Ruptured cord</td>
<td>20.0%</td>
<td>19.4%</td>
<td>12.7%</td>
</tr>
<tr>
<td>Difficulty breathing***</td>
<td>12.7%</td>
<td>0.0%</td>
<td>1.7%</td>
</tr>
<tr>
<td>Splayleg***</td>
<td>30.4%</td>
<td>14.3%</td>
<td>8.5%</td>
</tr>
<tr>
<td>At T24</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colostrum intake (g)</td>
<td>147 ± 21a</td>
<td>255 ± 22b</td>
<td>333 ± 14c</td>
</tr>
<tr>
<td>Weight gain (g)</td>
<td>–18 ± 14a</td>
<td>59 ± 15b</td>
<td>104 ± 9c</td>
</tr>
<tr>
<td>Rectal temperature (°C)</td>
<td>36.6 ± 0.15a</td>
<td>37.6 ± 0.15b</td>
<td>37.9 ± 0.09c</td>
</tr>
<tr>
<td>Cortisol (ng/ml)</td>
<td>484 ± 44a</td>
<td>273 ± 45b</td>
<td>275 ± 19b</td>
</tr>
<tr>
<td>IgG (mg/ml)</td>
<td>16.9 ± 1.8a</td>
<td>21.1 ± 1.9ab</td>
<td>24.3 ± 0.9b</td>
</tr>
<tr>
<td>Glucose (mg/l)</td>
<td>758 ± 61a</td>
<td>1016 ± 64b</td>
<td>1048 ± 28b</td>
</tr>
<tr>
<td>Lactate (μmol/l)</td>
<td>6405 ± 471a</td>
<td>6218 ± 488a</td>
<td>5147 ± 181b</td>
</tr>
<tr>
<td>n (blood sampled)</td>
<td>14</td>
<td>13</td>
<td>219</td>
</tr>
</tbody>
</table>

Data represent the percentage of piglets within the category, least square means ± s.e.m., or least square means (CI). Numerals followed by different lowercase letters differ significantly at \(P < 0.05\); *** \(x^2\) test, \(P < 0.001\).
plasma IgG concentration was positively related to both colostrum intake and colostrum IgG concentration and negatively related to birth rank and colostrum lactose concentration. Figure 2 shows the significant relationships between colostrum intake and rectal temperature (Figure 2a), plasma glucose concentration (Figure 2b) and plasma cortisol concentration (Figure 2c). The specific relationship between colostrum intake and plasma IgG concentration at T24 is also presented in Figure 3a. Multiple regression analysis of plasma IgG concentration at T24 revealed that colostrum intake ($P < 0.0001$), the IgG concentration in the colostrum ingested ($P < 0.0001$), birth weight ($P = 0.016$) and birth rank ($P < 0.0001$) were significant contributors. The estimated quantity of IgG ingested, calculated from colostrum intake and colostrum IgG concentration, was also negatively related to birth rank ($r = -0.145; P < 0.001$). The relationship between estimated IgG intake and the total amount of IgG present in plasma at T24 is presented in Figure 3b. The ratio between the total amount of IgG present in plasma and the amount of IgG ingested with the colostrum was also calculated to estimate IgG absorption and was found to average $0.22 \pm 0.008$. This ratio was significantly correlated with colostrum intake ($r = -0.37; P < 0.0001$) and plasma cortisol concentration ($r = 0.34; P < 0.0001$) but
was not correlated with time of birth ($r = -0.04; P = 0.52$) or birth rank ($r = -0.1; P = 0.13$).

Long-term effects of colostrum intake

Plasma IgG concentration at weaning averaged 7.2 ± 0.2 mg/ml and was found to be significantly related to plasma IgG concentration at $T_{24}$ ($r = 0.54; P < 0.0001$), to colostrum intake ($r = 0.32; P < 0.0001$) and to weight gain between $T_{24}$ and weaning ($r = 0.30; P < 0.0001$). A positive linear relationship between the total amount of IgG in plasma at $T_{24}$ and the total amount at weaning is shown in Figure 4. The impact of colostrum intake on piglet growth up to 6 weeks of age is presented in Figure 5. Average body weight was 7.19 ± 0.08 kg at weaning and 11.58 ± 0.11 kg at 42 days of age. Growth was significantly affected by colostrum intake, birth weight and age ($P < 0.0001$). From 21 days of age, the piglets could be divided into three categories of colostrum intake: <290 g, between 290 and 440 g and ≥440 g. Between those categories, growth significantly differed. The piglets that ingested >290 g colostrum ($n = 247$) weighed 12.34 ± 0.13 kg on average at 42 days of age. In comparison, the piglets that consumed <290 g of colostrum ($n = 162$) weighed 10.45 ± 0.17 kg at the same age.

Discussion

The overall mortality rate was 21.9%, which is close to the French national average of 21.0% (Gourmelen and Marouby, 2007). The stillbirth rate was especially low at 2.7%, compared to the national mean of 8%. Characteristics of the stillborn piglets in this study are in agreement with the results of previous studies (Randall, 1972; Zaleski and Hacker, 1993; Le Cozler et al., 2002). The low stillbirth rate observed in this study was probably due to the supervision of the farrowings; however, it did not influence total mortality, because the 19.2% pre-weaning mortality rate in the live-born piglets was high in comparison to the national mean of 14.4%. Therefore, weak piglets that would usually have died...
just after birth and been categorized as stillborn in commercial conditions probably died a few hours or days later in this study.

Of the live-born piglets that died before weaning, 56.5% and 72.2% died within 3 and 7 days of birth, respectively. In agreement with the results of other studies (Gardner et al., 1989; Rydmer, 1992; Quiniou et al., 2002), the piglets from this study, which died within 3 days of birth, had lower vitality because of lower birth weight, a higher incidence of difficulty in breathing at birth and a higher occurrence of splayleg, a condition that impairs a piglet's ability to reach the udder as well as escape being crushed by the dam. Indeed, death resulted mainly from weakness or crushing, the udder as well as escape being crushed by the dam.

Splayleg, a condition that impairs a piglet's ability to reach the udder, is also a sign of hypoxia during the birth process, which leads to metabolic acidosis characterized by high plasma lactate concentration and low rectal temperature (Herpin et al., 2001). After birth, those weak piglets which died before weaning consumed less colostrum and therefore lost weight as also observed in other studies (Thompson and Fraser, 1988; de Passillé and Rushen, 1989). The same piglets also exhibited hypoglycemia, high cortisol concentration and lower IgG intake, which often characterize piglets that die before weaning as already demonstrated (Tyler et al., 1990; Herpin et al., 1996; Tuchscherer et al., 2000). Therefore, the present findings support previous observations that weak piglets, whatever the cause of their weakness (low birth weight, hypoxia or malformation), are disadvantaged for colostrum intake (Devillers et al., 2007) and enter a vicious cycle where low colostrum intake leads to hypoglycemia and hyperthermia (Herpin et al., 2002) and beyond to high plasma cortisol and lactate concentrations, which are characteristic of glycogen reserve mobilization and high gluconeogenesis (Lepine et al., 1989). It is therefore all the more important to identify piglets at risk as early as possible and to give them assistance to increase their chance of getting a proper amount of colostrum. Indeed, only 7.1% of the piglets that consumed at least 200 g of colostrum died before weaning, whereas the rate of death before weaning rose to 43.4% in the piglets that ingested <200 g.

When all the piglets are considered, the relationships between colostrum intake and rectal temperature (Figure 2a), glycemia (Figure 2b), cortisol concentration (Figure 2c) and passive immunity (Figure 3a) confirm the important role of the colostrum in thermoregulation (Le Dividich and Noblet, 1981), glucose homeostasis (Herpin et al., 1992) and immune transfer (Klobasa et al., 1981). Plasma IgG concentration is logically related to colostrum intake, colostrum IgG concentration and birth rank. Indeed, birth weight is a major determinant of colostrum intake and vitality (Devillers et al., 2007), and colostrum IgG concentration decreases quickly after the onset of farrowing (Klobasa et al., 1987). Therefore, the piglets born later consumed colostrum with a lower IgG concentration, as demonstrated by Bland et al. (2003) in piglets with delayed access to the udder. In this study, however, 92% of the piglets were born within 4 h of the onset of parurition, whereas the major drop in colostrum IgG concentration usually happens between 4 and 12 h after farrowing begins (Bland et al., 2003; Devillers et al., 2007). Thus, most of the piglets probably ingested sufficient amounts of IgG before gut closure. As a consequence, the relationship between colostrum intake and plasma IgG concentration is not very close; after a certain amount of colostrum ingested, about 200 g, plasma IgG concentration does not increase anymore (Figure 3a), as already suggested by Rooke and Bland (2002). Therefore, this quantity of colostrum ingested seems adequate to reach the plateau of IgG absorption through the intestinal barrier described by Jensen et al. (2001). Moreover, the ratio between the estimated amount of IgG present in plasma and the estimated amount of IgG ingested with colostrum is not significantly correlated with time of birth or birth rank, suggesting that the percentage of IgG absorbed at $T_{50}$ is independent of the duration of the period piglets had for ingesting colostrum. It also suggests that gut closure happened for most of the piglets after the ingestion of a sufficient amount of IgG.

Indeed, the relationship between the estimated amount of IgG present in plasma and the estimated amount of IgG ingested with colostrum is highly significant (Figure 3b) and suggests a plateau starting around 15 g of ingested IgG. This relationship between IgG absorbed and IgG ingested is consistent with the findings by Werhahn et al. (1981) that the amount of IgG absorbed through the intestines is dose-dependent with the amount of IgG ingested with the colostrum, but ingestion must occur before gut closure. Furthermore, the negative correlation between colostrum intake and the absorption ratio of IgG is in accordance with a mechanism of gut closure induced by the amounts of nutrient ingested (Rooke and Bland, 2002). The ratio was also positively related to plasma cortisol concentration, which is consistent with the stimulatory action of cortisol on IgG uptake (Bate and Hacker, 1985; Sangild et al., 2000). However, it has to be noted that these synthetic variables only approximately estimate IgG absorption. Indeed, colostrum intake and colostrum composition change over time, whereas the synthetic variables estimate the IgG absorption from average data over the first day of lactation. Despite this approximation, the results are consistent with the existing knowledge on the IgG absorption mechanisms.

The results of this study also show the long-term effects of colostrum intake. Klobasa et al. (1981) proposed that elevated absorption of IgG after birth delays the synthesis of the piglet's own IgG. In contrast, this study showed a positive relationship between the IgG concentration in the piglets' blood early in life and that concentration 4 weeks later, as also reported by Rooke et al. (2003). This discrepancy is likely due to the extreme treatments tested by Klobasa et al. (1981), who prevented piglets from naturally consuming colostrum for up to 24 h or artificially fed them with bovine colostrum and milk with different amounts of porcine IgG, whereas this study and the one by Rooke et al. (2003) were done on nursing piglets without modification of their suckling behavior and the colostrum composition. Moreover, Klobasa et al. (1981) found an inverse relationship after
would promote long-term health. On the basis of the results ensured by provision of energy, whereas immune transfer achieve satisfactory growth. Early survival is most likely that the acquisition of good passive immunity via the consumption of colostrum is positively related to the development of the piglet’s active immunity until weaning, but it would be interesting to study if this relationship persists after weaning. Moreover, the amounts of IgG present in the piglets’ blood at T24 and at weaning were almost the same and were linearly related (Figure 4). It remains difficult to determine the proportion of the IgG present in plasma at weaning that comes from the colostrum and that depends on IgG half-life. The half-life of IgG in piglet blood was estimated to average 10 to 15 days of age (Curtis and Bourne, 1973; Frenyó et al., 1981; Klobasa et al., 1981). However, those studies did not take into account the variation in blood volume occurring with piglet growth and probably underestimated the IgG half-life value (Rooke and Bland, 2002). Therefore, a significant proportion of the IgG present in plasma at weaning may still come from the colostrum as reported by Rooke et al. (2003). Furthermore, independently of the rate of disappearance of the IgG absorbed from the colostrum, the amount of IgG synthesized by the piglet should be proportional to the amount absorbed from the colostrum to maintain a quantity of IgG present in the piglet’s blood at weaning equivalent to the one observed at T24, as shown in Figure 4. Despite this relationship, it is still unclear whether the high uptake of immunoglobulins with the colostrum directly stimulates the development of active immunity. The colostrum also contains maternal lymphoid cells, which participate in the active immunity of the piglets (Tuboly et al., 1988), and many immune modulators, such as cytokines, which may participate in the maturation of the neonate immune system (Salmon et al., 2009). Therefore, although the mechanisms still have to be determined, it is most likely that the acquisition of good passive immunity via the consumption of colostrum stimulates the development of the piglet’s active immunity.

Colostrum intake also showed long-term effects on piglet growth, with the piglets that consumed >290 g of colostrum gaining about 2 kg more live weight by 6 weeks of age. These results show that variability in colostrum intake leads to variability in growth and, below a certain amount of colostrum ingested, growth is impaired in the long term through a series of events where a piglet with a low colostrum intake has a lower viability and is disadvantaged for the rest of the lactation. Pre-weaning growth was also positively related to plasma IgG concentration at weaning. However, it remains difficult to untie if the higher IgG concentration is just a marker of a higher colostrum intake and is therefore related to growth, or if higher passive immunity transfer can play a positive role on growth.

In conclusion, ensuring that piglets ingest a minimum amount of colostrum is the best way to guarantee that they survive, receive a satisfactory transfer of immunity and achieve satisfactory growth. Early survival is most likely ensured by provision of energy, whereas immune transfer would promote long-term health. On the basis of the results presented, a minimum colostrum intake of 200 g is recommended. However, given that the colostrum production of sows is highly variable and can range from 1.9 to 5.3 kg (Devillers et al., 2007), it can be difficult for many sows to adequately nurse more than 10 piglets. Therefore, genetic selection on sow prolificacy should be adjusted to the colostrum production capacity. Otherwise, measures should be taken to collect and store colostrum from some sows and bottle-feed piglets at risk of insufficient intake. This study showed that colostrum intake is very important for the survival and development of naturally nursing piglets and has potential long-term effects on their growth and immunity. However, the mechanisms underlying a stimulatory impact of passive immunity on the development of active immunity have still to be demonstrated.

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