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RELATIONSHIPS AMONG MATERNAL BACKFAT DEPTH, PLASMA
ADIPOKINES AND THE BIRTHWEIGHT OF PIGLETS

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Abstract:	A potentially important role into the mechanisms controlling intrauterine growth is provided by adipose tissue. The adipose tissue acts as a dynamic organ, with differentiated adipocytes functioning in an endocrine manner to secrete adipokines, as leptin and adiponectin, involved in modulating energy homeostasis. With the aim of assessing the relationships among maternal adiposity, leptin and adiponectin-pregnancy trend and foetal growth, 48 sows Large White x Landrace were recruited. On basis of backfat (BF) depth at the end of lactation phase, a Low Fat (LF, 11.60 mm; n=24) and a High Fat (HF, 18.20 mm; n=24) groups were formed. During the study, both groups received the same feeding level. At mating and at days 28, 85 and 113 of gestation, the BF was recorded and blood samples were collected to assess the leptin and adiponectin concentrations. At farrowing (BW0) and after 24 h (BW24), piglets were weighed and temperature was recorded. Body mass index (BMI) and ponderal index (PI) were calculated. Before suckling colostrum, blood samples were collected and leptin, adiponectin and IGF-I plasma levels were analysed. The BF depth change during pregnancy was the same in both groups ($P > 0.05$); the BF depth differences between LF and HF groups observed at mating persist during pregnancy as well ($P < 0.05$). A decrease in adiponectin and an increase in leptin plasma levels occur in both groups. Plasma adiponectin was lower in HF group compared to LF group ($P \leq 0.05$), while plasma leptin was not different between groups ($P > 0.05$). Consequently, the adiponectin-leptin ratio resulted lower in HF than in LF group ($P < 0.05$). Compared to HF group, LF sows give birth to lighter piglets ($P < 0.05$). In addition, 14% of the piglets in the LF group and 7% in the HF group reveal a weight that falls in the 25th percentile (≤ 1 kg) ($P < 0.05$). Piglet's anthropometric parameters and thermoregulatory ability were unaffected by maternal adiposity ($P > 0.05$). Piglet's mortality at 24 h from birth was 13% and 3%, in LF and HF groups, respectively ($P < 0.05$). Haematological differences were found only in the case of IGF-I level, which was lower in piglets born from LF than in those from HF sows ($P < 0.05$). Our findings suggest that differences in

	maternal adiposity, due to individual factors and not to feeding levels, influence the offspring weight by acting on the secretion of adipokines.
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1 **RELATIONSHIP AMONG MATERNAL BACKFAT DEPTH, PLASMA ADIPOKINES**
2 **AND BIRTHWEIGHT OF PIGLETS**

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11 Short title: Maternal backfat depth and birthweight of piglets

12

13 **Abstract** A potentially important role into the mechanisms controlling intrauterine
14 growth is provided by adipose tissue. The adipose tissue acts as a dynamic organ,
15 with differentiated adipocytes functioning in an endocrine manner to secrete
16 adipokines, as leptin and adiponectin, involved in modulating energy homeostasis.
17 With the aim of assessing the relationships among maternal adiposity, leptin and
18 adiponectin-pregnancy trend and foetal growth, 48 sows Large White x Landrace were
19 recruited. On basis of backfat (**BF**) depth at the end of lactation phase, a Low Fat (**LF**,
20 11.60 mm; n=24) and a High Fat (**HF**, 18.20 mm; n=24) groups were formed. During
21 the study, both groups received the same feeding level. At mating and at days 28, 85
22 and 113 of gestation, the BF was recorded and blood samples were collected to assess
23 the leptin and adiponectin concentrations. At farrowing (**BW0**) and after 24 h (**BW24**),
24 piglets were weighed and temperature was recorded. Body mass index (**BMI**) and

25 ponderal index (**PI**) were calculated. Before suckling colostrum, blood samples were
26 collected and leptin, adiponectin and IGF-I plasma levels were analysed. The BF depth
27 change during pregnancy was the same in both groups ($P >0.05$); the BF depth
28 differences between LF and HF groups observed at mating persist during pregnancy
29 as well ($P <0.05$). A decrease in adiponectin and an increase in leptin plasma levels
30 occur in both groups. Plasma adiponectin was lower in HF group compared to LF group
31 ($P \leq 0.05$), while plasma leptin was not different between groups ($P >0.05$).
32 Consequently, the adiponectin-leptin ratio resulted lower in HF than in LF group (P
33 <0.05). Compared to HF group, LF sows give birth to lighter piglets ($P <0.05$). In
34 addition, 14% of the piglets in the LF group and 7% in the HF group reveal a weight
35 that falls in the 25th percentile (≤ 1 kg) ($P <0.05$). Piglet's anthropometric parameters
36 and thermoregulatory ability were unaffected by maternal adiposity ($P >0.05$). Piglet's
37 mortality at 24 h from birth was 13% and 3%, in LF and HF groups, respectively (P
38 <0.05). Haematological differences were found only in the case of IGF-I level, which
39 was lower in piglets born from LF than in those from HF sows ($P <0.05$). Our findings
40 suggest that differences in maternal adiposity, due to individual factors and not to
41 feeding levels, influence the offspring weight by acting on the secretion of adipokines.

42

43 **Keywords:** pregnant sows; backfat depth; adipokines; piglets; birth weight

44

45 **Implications.** Birthweight represents a challenge in the pig industry. The adipose
46 tissue, by secreting leptin and adiponectin, which are important in modulating glucose
47 and lipid homeostasis, is a potentially regulator of the mechanisms that link maternal
48 adiposity and intrauterine growth. It is likely, that leptin's role in foetal growth, untied
49 by maternal fatness, is only permissive. Its increase during pregnancy may be the

50 signal to enhance the mobilization of maternal fat storage and the transplacental
51 transfer of lipids to the foetus. Conversely, reduced levels of adiponectin may represent
52 a mechanism by which a high maternal fat mass leads to an increased foetal weight.

53

54 **Introduction**

55 Birthweight is considered to be a critical indicator of postnatal performance. Growth
56 and development of the foetus are the result of a balance between the foetal demand
57 and the maternal substrate availability (Jansson and Powell, 2006). To ensure
58 nutrients to foetus, the sow undergoes to an alteration in the regulation of glucose
59 metabolism leading to a state of relative insulin resistance, attributed to the effect of
60 placental hormones, as pregnancy progresses (Père *et al.*, 2000). The development
61 of an insulin-resistant state, increasing hepatic gluconeogenesis and reducing glucose
62 uptake in maternal skeletal muscle and adipose tissue, promotes lipolysis in adipose
63 tissue, thereby making glucose and lipids available to the foetus. This complex system
64 is monitored by a multitude of factors, including the body condition of the mother, the
65 utero-placental blood flow and the expression and function of trophoblast nutrient
66 transporters (Aye *et al.*, 2013). Maternal adiposity, as reflection of its nutritional status,
67 is one of the main extrinsic factors programming nutrient partitioning and growth,
68 development and function of the major foetal organ systems (Redmer *et al.*, 2004;
69 Amdi *et al.*, 2014). Adipose tissue is a specialised endocrine and paracrine organ that
70 modulates energy metabolism via secretion of circulating adipokines (such as leptin
71 and adiponectin), which are involved in the regulation of body fatness and energy
72 expenditure (Ahlsson *et al.*, 2013). Adiponectin and leptin are key modulators of insulin
73 action and glucose metabolism; thus, they can be considered potential candidates for
74 regulation of intrauterine foetal development. During pregnancy, leptin and adiponectin

75 participate in the process of insulin resistance with antagonistic actions: leptin
76 increases insulin resistance whereas adiponectin increases insulin sensitivity. Serum
77 leptin concentrations have been demonstrated to be positively correlated with backfat
78 depth in sows (De Rensis *et al.*, 2005). High leptin levels from the end of mid pregnancy
79 to farrowing are reported in breeding sows where gestational hyperphagia is managed
80 through dietary strategies (Saleri *et al.*, 2015). The increase of maternal leptin levels
81 during pregnancy may also be a consequence of placental production. The expression
82 of the short leptin receptor form in the sows' placental tissue at farrowing has been
83 observed (Saleri *et al.*, 2015).

84 Inverse correlations between adiponectin and fat mass and between adiponectin and
85 body mass index have been reported (Skvarca *et al.*, 2013). It has been shown that in
86 pregnant women longitudinal changes in serum adiponectin concentrations occurs;
87 lowest levels were seen in late pregnancy when insulin levels rise (Fuglsang *et al.*,
88 2006). An inverse correlation between levels of maternal adiponectin and insulin
89 resistance as well as rates of endogenous glucose production has been observed
90 (Ahlsson *et al.*, 2013).

91 The purpose of this study was to assess the trend of leptin and adiponectin plasma
92 concentrations in pregnant sows with different backfat depth at mating-time. We
93 hypothesised that the interaction between maternal backfat and adipokines constitutes
94 one of the mechanisms behind the offspring growth.

95

96 **Material and Methods**

97 The study was carried out in compliance with Italian law (Lgs. D. 26/2014) on animal
98 experiments and all animals were housed in accordance with EC Directive 2010/63/UE
99 on animal husbandry.

100

101 *Animals*

102 Based on backfat (**BF**) depth at the end of lactation and parity order, 48 sows Large
103 White x Landrace were recruited and Low Fat (**LF**, 11.60 ± 1.51 mm; n=24) and High
104 Fat (**HF**, 18.20 ± 2.05 mm; n=24) groups were formed. Backfat depth was measured
105 by a single operator on both left and right side on standing sows using an ultrasound
106 scanner and the mean value was utilised (Lean-meater, Renco Corporation,
107 Minneapolis, Minnesota, USA) (Maes *et al.*, 2004). Parity order was 4.0 ± 2.1 and 3.6
108 ± 1.2 for LF and HF groups, respectively. Sows were identified and moved in individual
109 pens in the same gestation room. At onset of standing oestrus (4.75 ± 0.95 days after
110 the end of lactation), they were artificially inseminated two times, at day 0 of gestation
111 and again 24 h later using, in both cases, semen pooled from five Large White x Duroc
112 boars. During gestation and transition period (from day 110 of gestation to day 3 after
113 farrowing) all sows received the same standard diets offered twice a day at 0800 h and
114 1600 h (Table 1). From 0 to day 10 after mating sows were fed 1.5 kg per day of the
115 gestation diet. The same diet was administered at a dose of 2 kg per day until day 28
116 when the sows were evaluated with ultrasound examination (Aloka SSD 500®, Hitachi
117 Medical System SpA, Milano, Italy; linear probe 5.0 MHz). One of the sows belonging
118 to the LF group was not pregnant and thus, it was removed from the study. From day
119 29 until day 109 of gestation, sows were housed in a group housing system and fed
120 2.5 kg/sow per day of the gestation diet. At day 110 of gestation, sows were moved to
121 individual farrowing pens and fed 2.5 kg per day of the transition diet until three days
122 after farrowing. Parturition was induced on day 113 of gestation by deep intramuscular
123 injection of 0.7 ml per head of cloprostenol sodium (PGF VEYX ®, Veyx-Pharma
124 GmbH, Söhreweg, Germany). The farrowing took place on day 114 of gestation for all

125 sows. Floor heating and an infrared lamp were used to create a microclimate for the
126 piglets. After 24 h from birth, litter sizes were standardised to 12 ± 2 pigs by cross-
127 fostering within groups. Access to water was always available on an *ad libitum* basis.
128 Gestation took place in a temperature-controlled room with an average ambient
129 temperature of 20°C. The day of farrowing and in the next 72 hours the ambient
130 temperature was raised up to 23°C.

131

132 *Measurements*

133 The BF was evaluated at days 0, 28, 85 and 113 of gestation. In the same time-points,
134 blood samples from sows were collected using jugular venepuncture in 10-ml
135 vacutainer tubes with lithium heparin before the morning meal. Samples were
136 immediately centrifuged ($1327 \times g$ for 10 min) and plasma was collected and stored at
137 -20°C until analysis.

138 Number of total born, born alive, dead within the first 24 hours of life and gender were
139 recorded. Piglets were individually weighed (electronic dynamometer; Wunder Sa. Bi.
140 srl, Trezzo sull' Adda -MI, Italy) within the first 2 hours of life (**BW0**) and 24 h after birth
141 of first born piglet (**BW24**). Body mass index (**BMI**) and ponderal index (**PI**) of all piglets
142 were calculated from the crown-rump length and birthweight (Baxter *et al.*, 2008). Body
143 temperature was assessed at birth and at 24 h using an infrared ear thermometer
144 (accuracy 0.2°C) (GIMA, Gessate- MI, Italy). Before suckling colostrum, six piglets
145 born alive per litter - three males and three females- were held in dorsal recumbency
146 and 5 ml of blood from external jugular vein into vacutainer tubes with lithium heparin
147 were taken. The blood samples were processed in the same way as above mentioned
148 for sows.

149

150 *Diets and blood analyses*

151 Proximate analyses of diets were performed according to the Commission Regulation
152 (EC) 152/2009 laying down the methods of sampling and analysis for the official control
153 of feed (Annex III).

154 Adiponectin plasma concentration, was determined using a species specific
155 commercial kit (Porcine Adiponectin ELISA, BioVendor, Brno, Czech Republic). The
156 sensitivity of the method was 0.03 ng/ml and the variability coefficients within and
157 among samples were 6.7% and 8.2%, respectively. Leptin plasma concentration was
158 assessed using a commercial kit (Multispecies Leptin RIA- Linco Research, St. Louis,
159 MO, USA). The sensitivity of the method was 100 pg/ml and the variability coefficients
160 within and among samples were 4.7% and 9.1%, respectively. Plasma IGF-I content
161 was evaluated using a multispecies IGF-I ELISA (Alpco Diagnostic, Salem NH, USA),
162 according to manufacturer's instructions. The intra- and interassay coefficients of
163 variation were 7.8% and 5.3 %, respectively. The minimal detection limit was 30 pg/ml.

164

165 *Statistical analyses*

166 Data on litter size were processed by ANOVA (SAS 9.4, SAS Inst. Inc., Cary, NC,
167 2013), according to a model including the group of sows (LF, HF) as a fixed factor and
168 the parity order as a covariate. Data on piglets were processed by ANOVA, according
169 to a model that included the fixed factors of the group of sows (LF, HF) and sex (M, F),
170 and the random factor of sow within the group. The data recorded on sows were
171 analysed according to a repeated measure model, including the group (LF, HF) as a
172 fixed factor and, as a covariate, the parity order. Time was included as the repeated
173 statement, and interaction between time and group was considered. Residual were
174 checked for normality by means of PROC UNIVARIATE. When not normally

175 distributed, data were log transformed. Based on birthweight, piglets were divided in
176 four percentiles classes (25th, 50th, 75th and 95th) by means of PROC UNIVARIATE.
177 The chi square test was applied to the observed and expected frequencies of piglets,
178 as categorised in percentiles weight classes, in relation to the adiposity of sows.
179 Mortality per litter was evaluated applying the chi square test. The significance level
180 was set at $P \leq 0.05$.

181

182 **Results**

183

184 *Sows parameters*

185 Data on maternal adiposity changes during gestation are reported in Table 2. The
186 differences in backfat depth observed at mating between LF and HF groups persist for
187 the entire pregnancy ($P < 0.05$). During pregnancy, no differences in backfat depth gain
188 were observed between groups ($P > 0.05$). Adiponectin and leptin plasma
189 concentrations and adiponectin-leptin ratio are summarised in Figure 1. Adiponectin
190 concentration showed a decrease from 0 to 113 days of gestation ($P < 0.05$): at these
191 point times, mean values (\pm SD) were 195.85 ± 21.5 and 61.67 ± 7.4 $\mu\text{g/ml}$ for LF group
192 and 98.19 ± 10.5 and 19.87 ± 2.4 $\mu\text{g/ml}$ for HF group, respectively (Figure 1A). As
193 compared to LF group, plasma adiponectin was lower in HF group at mating, at day
194 85 of gestation ($P < 0.05$) and at farrowing ($P = 0.05$) (Figure 1A). After the first month
195 of gestation plasma leptin increased in both groups to reach the highest concentration
196 at farrowing ($P < 0.05$). No differences between groups were observed in plasma leptin
197 throughout the pregnancy ($P > 0.05$). At conception, 85 and 113 days of gestation, the
198 adiponectin-leptin ratio was lower in HF than in LF group ($P < 0.05$) (Figure 1B).

199

200 *Newborn piglet's performance and hormone plasma levels*

201 The effects of maternal BF depth on piglet's performance are reported in Table 3. No
202 differences were observed in respect to total born and born alive number between
203 groups ($P > 0.05$). Conversely, maternal adiposity affected foetal growth; piglets born
204 from HF sows showed a higher BW0 (+ 7%; $P < 0.014$) and BW24 (+ 4%; $P < 0.001$)
205 than those from LF sows. In addition, significant differences in percentile weight
206 grouping was observed ($P < 0.001$). On total born per group, 14% of piglets in the LF
207 and 7% in the HF group revealed a weight falling in the 25th percentile (≤ 1 kg).
208 Anthropometric parameters (BMI, PI) were not affected by maternal BF depth (P
209 > 0.05). In the first 24 h of life, the thermoregulatory ability was the same in both groups
210 ($P > 0.05$). Piglet's mortality from 0 to 24 h of life was 13% and 3% for LF and HF
211 groups, respectively ($P = 0.035$). Plasma adiponectin and leptin levels were not affected
212 by maternal adiposity ($P > 0.05$) (Table 4). Higher IGF-I plasma levels were recorded
213 in piglets born from HF than LF sows ($P < 0.001$).

214

215 **Discussion**

216 Pregnancy is a dynamic anabolic state during which nutritional needs increase due to
217 the growing foetus and the development of associated maternal tissues. Pregnancy-
218 induced changes in body weight and fat deposition are physiological events of
219 maternal adaptation, linked to the hormonal pregnancy changes (i.e. progesterone,
220 prolactin and cortisol) (Saleri *et al.*, 2015). A previous research reported that maternal
221 backfat depth at mating had a greater influence on offspring growth, than feed level
222 during gestation (Amdi *et al.*, 2014). In this study, we focused on the endocrine role of
223 maternal adipose tissue during pregnancy, to investigate how a different adiposity at
224 mating could influence the evolution of pregnancy both in terms of maternal hormone

225 levels and weight and size of the newborn piglets. Firstly, the increase in maternal fat
226 stores during pregnancy was the same in both groups, but sows with the lower backfat
227 depth at mating gave birth to lighter piglets. The percentage of piglets weighing less
228 than 1 kg was double in LF than HF group. This result could suggest an intrauterine
229 growth restriction in piglets born from LF sows, although the thermoregulatory capacity
230 and anthropometric parameters were found to be adequate in all piglets (Amdi *et al.*,
231 2013). It is known that foetal glucose/insulin/insulin-like growth factor axis is the main
232 driver of growth (Hellström *et al.*, 2016). In the foetus, growth is directly modulated by
233 IGF system and it is not regulated by growth hormone. Insulin-like growth factor-I and
234 -II are expressed in foetal tissue only until birth. Insulin-like growth factor-II expression
235 is more extensive in foetal tissues than IGF-I from mid to late gestation in rodents and
236 humans (Randhawa and Cohen, 2005). It has been shown that a poor maternal body
237 condition at conception and in early pregnancy is signaled to the foetus which reacts
238 by promoting peripheral glucose utilization, essential for brain and heart, and by
239 reducing the demand for amino acids for growth, thereby to maintain energy supply at
240 the expense of growth (Bloomfield *et al.*, 2013). After birth, GH stimulates IGF-I
241 secretion with a quick increase of IGF-I blood levels. In our study, lower IGF-I values
242 were observed in piglets born from LF compared to HF group. Energy balance is
243 modulated also by adipose tissue hormones, i.e adipokines, which are acting to
244 regulate insulin sensitivity, appetite and lipid metabolism (Ahlsson *et al.*, 2013). In both
245 groups, the most abundant adipokines produced by adipose tissue, leptin and
246 adiponectin, showed an inverse trend during pregnancy. Physiologically, decreased
247 adiponectin and increased leptin concentrations are associated with insulin-resistant
248 state (Ahlsson *et al.*, 2013). However, it is known that the regulation of maternal
249 adiponectin and leptin during pregnancy becomes very complex. The high levels of

250 leptin in pregnancy are in agreement with a mechanism of resistance to central leptin
251 action previously reported in swine (Saleri *et al.*, 2015), in humans and mice (Trujillo
252 *et al.*, 2011). Production and regulation by non-adipose tissue, i.e placenta, contribute
253 to this increase in pregnant sows. Probably, the increase in maternal leptin enhances
254 the mobilization of maternal fat stores to increase the transplacental transfer of lipids
255 to the foetus during the latter stages of pregnancy (Walsh *et al.*, 2014). In isolated rat
256 adipocytes, leptin inhibits accumulation of lipids by increasing the turnover of
257 triglycerides, inhibiting basal and insulin-stimulated *de novo* lipogenesis, but
258 stimulating oxidation of glucose and free fatty acids (Harris, 2014). In humans,
259 maternal leptin concentration was not associated with infant birthweight (Misra *et al.*,
260 2013). Similarly, in our study, leptin levels in sows and piglets do not differ between
261 groups, even though differences in birthweight of piglets were observed. These results
262 support that leptin may play a permissive role in foetal growth. In addition, it has been
263 shown that leptin deficiency in women is not associated with major abnormalities
264 (Christou *et al.*, 2002). To clarify the leptin role, it is important to take into account also
265 the adiponectin role. The strict link between adiponectin and leptin is underlined by the
266 plasma adiponectin to leptin ratio (A/L ratio) which has been proposed as a better
267 marker for insulin resistance in pregnancy than leptin or adiponectin alone (Skvarca *et*
268 *al.*, 2013). Our data showed that the A/L ratio is lower in HF than LF group, as evidence
269 that HF sows could have a better ability to provide adequate carbohydrate availability
270 for the fast-growing foetus. The lower adiponectin levels detected in HF than LF group
271 match the higher birthweight of offspring. Therefore, adiponectin may be an important
272 player in the mechanism by which maternal energy is stored in fat mass leading the
273 pattern of foetal growth (Zhang *et al.*, 2016). Since we detected the same trend in the
274 adiponectin release in both groups, we hypothesised that adiponectin in pregnant sows

275 is involved in the management of mother's energy resources, regardless their backfat
276 depth. In the concept of energy balance, we can see a complex of mechanisms
277 involving nutrient availability, growth and immunity. In this sense, adiponectin role is
278 also connected to the success of pregnancy: a crucial aspect is the maternal immune
279 tolerance of the foetus. Maternal immune responses regulate the early key events of
280 pregnancy (i.e., implantation, angiogenesis, and vascular remodeling) and
281 abnormalities in these early events are associated with foetuses' intrauterine growth
282 restriction and survival (Morelli *et al.*, 2015). It is known that intrauterine growth
283 restriction results in significant perinatal and long-term complications, including
284 increased neonatal mortality and morbidity. We may hypothesise that also in sow,
285 adiponectin might play an active role in this mechanism. In support of this hypothesis,
286 recently the presence of adiponectin and its receptors was demonstrated in the porcine
287 uteri, conceptuses, and trophoblasts during early pregnancy (Smolinska *et al.*, 2012).
288 In *vitro* studies, it was showed that adiponectin arouses pro-inflammatory cytokine
289 production in human trophoblasts (i.e., IL-1 β and IL-8) and placenta (i.e., IL-1 β , IL-6
290 and TNF α) and thus it may be detrimental to the initiation and progression of pregnancy
291 (Lappas *et al.*, 2005; McDonald and Wolfe, 2011). In our study, the maternal immune
292 responses did not influence the litter number. However, the different plasma levels of
293 adiponectin observed between the groups of sows was associated with different weight
294 of the piglets at birth. All together, these findings suggest a critical role of adipokines,
295 i.e leptin and adiponectin, in modulating maternal energy balance in different stage of
296 pregnancy. Keeping an eye on the new findings on the endocrine function of adipose
297 tissue, we propose that the adiposity at mating time can be used as an effective
298 indicator to optimise the reproduction efficiency of sow. Endocrinology of maternal

299 adipose tissue and its influence on the offspring request further studies to elucidate the
300 pathways involved in foetus growth and its adaptation to intrauterine environment.

301

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304 declare that there are no conflicts of interest.

305

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380 **Table 1** *Ingredients and chemical analysis of diets (on air dry basis g/kg as fed)*

	Gestation diet (g/kg)	Transition diet (g/kg)
Ingredients:		
Wheat bran	240	--
Barley	230	267
Corn	180	310
Unmolassed sugar-beet pulp	147	150
Sunflower meal	137	--
Soybean meal	--	135
Fish meal	--	46
Soybean protein	--	10
Soybean oil	15	20
Dextrose	--	40
Rapeseed meal	20	--
Ground limestone	15	--
Dicalcium phosphate	10	15
Sodium chloride	4	4
Lysine HCl	0.5	1
Vitamins and minerals ¹	1.5	2.0
Chemical analysis:		
Dry matter (g/kg)	878	872
CP (g/kg)	141.2	153.6
Crude fibre (g/kg)	82.2	58.4
Crude fat (g/kg)	38.2	42.1
Digestible energy (MJ/kg) ²	12.9	13.5

381 ¹ Supplied per for kg of diet : Cu: 40 mg, Zn: 80 mg Fe: 150 mg, Se: 0.2 mg, I: 0.6 mg, Mn: 50 mg,
382 vitamin A: 12,000 IU, vitamin D3: 1,000 IU, vitamin E: 100 IU, vitamin K: 20 mg, vitamin B12: 55 µg,
383 vitamin B1: 2 mg, vitamin B2: 5 mg, vitamin B6: 1.5 mg, nicotinic acid: 12 mg, pantothenic acid: 10 mg,
384 folic acid: 5 mg, choline chloride: 500 mg, biotin: 200 µg.

385 ² According to the equation proposed by Noblet and Perez (1993).

386

387 **Table 2** *Pregnancy changes in backfat depth in sows with Low Fat or High Fat at the*
 388 *end of the previous lactation (Least squares mean values \pm SEM).*

	Groups		SEM	P - value
	LF	HF		
BF depth (mm)				
day of mating	11.9	17.1	1.69	<0.001
day 28 of gestation	12.4	17.3	1.77	0.003
day 85 of gestation	13.9	18.9	1.47	0.013
day 113 of gestation	14.6	19.7	1.47	<0.001
Δ BF (mating – day 113 of gestation) (mm)	2.7	2.5	0.48	0.358

389 LF (Low Fat; 11.6 \pm 1.5 mm) and HF (High Fat; 18.2 \pm 2.0 mm); BF= backfat

390

391 **Table 3** *Effects of maternal backfat depth on anthropometric parameters and growth*
 392 *performance of piglets (Least squares mean values \pm SEM).*

	Groups		SEM	P – value
	LF	HF		
Total born (n.)	17.2	15.7	3.16	0.327
Born alive (n.)	15.1	14.1	1.94	0.182
BW0 (g)	1244	1334	278	0.014
BW24 (g)	1310	1360	85	<0.001
BMI (kg/m ²)	20.87	20.63	2.091	0.384
PI (kg/m ³)	83.98	84.44	6.739	0.599
Body temperature (°C)				
at birth	37.92	37.78	0.947	0.267
at 24 h	38.75	38.71	0.495	0.608

393 LF (low fat: 11.6 \pm 1.5 mm) and HF (high fat: 18.2 \pm 2.0 mm) are referred to backfat depth recorded by
 394 sows at the end of the previous lactation; BW0= Birthweight; BW24= Body weight at 24 h of life; BMI=
 395 body mass index; PI= ponderal index

396

397 **Table 4** Plasma adiponectin, leptin and IGF-I in newborn piglets in relation to backfat depth of
 398 sows (Least squares mean values \pm SEM).

	Groups		SEM	<i>P</i> – value
	LF	HF		
Adiponectin ($\mu\text{g/ml}$)	14.3	14.9	1.66	0.339
Leptin (ng/ml)	3.0	3.3	0.57	0.096
IGF-I (pg/ml)	4.4	5.6	1.00	< 0.001

399 LF (low fat: 11.6 ± 1.5 mm) and HF (high fat: 18.2 ± 2.0 mm) are referred to backfat depth recorded by
 400 sows at the end of the previous lactation

401

402 **Figure caption**

403 **Figure 1** Trend of adiponectin and leptin (A) and adiponectin/leptin ratio (B) during
404 gestation, in relation to backfat depth of sows. Blood sampling period started at the
405 day of mating (0) and ended at the day 113 of gestation. Error bars indicate standard
406 deviation. Differences between groups within sampling time are labelled * ($P < 0.05$).
407 Differences among sampling time within groups are labelled a,b,c, ($P < 0.05$).

408

