



# **Effect of pre- and post-mating dietary restriction on embryo survival of group housed gilts**

By

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## Declaration

I declare that this thesis is a record of original work and contains no material which has been accepted for the award of any other degree or diploma in any university. To the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text.

Patricia Condous

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# Literature Analysis

## **Introduction**

Reproduction has a major impact on the efficiency and productivity of pig production systems (Almeida *et al.* 2000). Sow reproduction efficiency is determined by the number of viable piglets produced per sow per year, which is a function of number of farrowings per year and piglets born per farrowing (litter size), and can vary markedly between sows within a facility (Almeida *et al.* 2000). The reproductive performance of individual sows is affected by a number of, often interrelated, factors; including environment, genetics, health and nutrition. These factors influence ovulation rate and oocyte quality, as well as embryo survival and implantation, which in turn affect pregnancy maintenance and litter size of the sow (Soede *et al.* 2011). The relationship between nutritional status and reproduction has been extensively researched and the focus of a number of reviews (Prunier and Quesnel 2000). However, due to current, and ongoing, changes within the pig industry, in particular the transition from individual to group housing of sows during gestation, there are also new nutritional problems arising which may possibly influence reproductive performance (Kongsted 2005). It is, therefore, essential that optimal feeding strategies are developed which maximise the reproductive performance, and thus productivity, of group housed sows. This review will discuss the changes in gestating sow housing, with particular focus on how these changes may impact reproductive performance.

## **Gestating sow housing: recent developments and problems**

Until recently, the main form of sow housing during gestation was in individual stalls. Individual stalls have been largely adopted in the Australian pig industry as they prevent bullying and aggression between the sows and provide a controlled feeding environment, leading to improved reproductive performance (Barnett *et al.* 2001). Surveys by Patterson (1997) found that approximately 62% of sows in Australia were housed individually at some

stage during pregnancy (Barnett *et al.* 2001). However due to increasing demand by consumers, retailers and scientists for better welfare conditions for gestating sows, individual stalls are gradually being phased out and replaced by group housing systems (Kongsted 2005). Group housing of non-lactating sows has become widespread in European countries due to changed legislation in response to welfare concerns, and more countries are starting to adopt similar actions (Kongsted 2005). The Australian Pork Industry has pursued the voluntary phase-out of individual gestation stalls by 2017 and therefore group housing will soon become standard management for non-lactating sows in Australia.

Although group housing improves sow welfare due to freedom of movement and increased capacity to express natural behaviour, there are concerns that group housing systems will have negative impacts on sow reproductive performance (Andersen *et al.* 1999). Group housing of gestating sows is associated with sow to sow aggression, resulting either from the desire to establish a social hierarchy or competition over limited resources, in particular feed (Barnett *et al.* 2001). High variation in feed intake between sows is evident in a group housing system, with low ranking sows thought to receive less food than their higher ranking counterparts (Andersen *et al.* 1999). The social status of the sows (rank index and rank place) has been shown to increase with increasing parity and live weight gain (Hoy *et al.* 2009). It was concluded that parity one sows were the low ranking sows when grouped with older and heavier sows (Hoy *et al.* 2009). Therefore, during early gestation when maintenance requirements are already so close to feed supply, it would be expected that the energy intake of the low ranking sows would fall below maintenance and lead to reduced reproductive performance (Hoving *et al.* 2011).

Different types of feeding systems are used in group housing, which include floor feeding, individual access pens and electronic sow feeding (Kongsted 2005). The level of competition and distribution of feed between the sows varies between the systems (Andersen *et al.* 1999). Electronic sow feeding and individual access pens allow the sows to feed in individual pens,

while in floor feeding systems, the food is placed on the floor and so there is no form of individual rationing (Andersen *et al.* 1999). Therefore it might be expected that group housing systems with floor feeding would have the largest effect on unequal feed intake and therefore possibly reproductive performance. Brouns and Edwards (1994) showed the effect of dominance in a floor feeding system with low ranking sows, sows dominated by 75% or more of the group, having a significantly lower live weight gain compared to the high ranking sows.

The effect of feed intake on reproductive performance has mainly been investigated in individually fed sows, with only a few studies investigating this effect in group housed systems. However in a review, Kongsted (2006) concluded that the unintentional variation of feed intake that occurs in a group housing system may be sufficient to exert a negative effect on pregnancy rate and possibly litter size. On-farm studies reviewed by Kongsted (2006) have also shown that group housing non-lactating sows impaired litter size, with group housing resulting in 0.3-0.6 fewer piglets born per litter compared to individual housing. The effects of restricted feed intake in early gestation are also believed to be greater in parity one sows. Parity one sows were found to have lower farrowing rates, compared to gilts and parity two sows, when fed a low feed intake for the first month of gestation (Graph 1) (Hughes and van Wettere unpublished data).

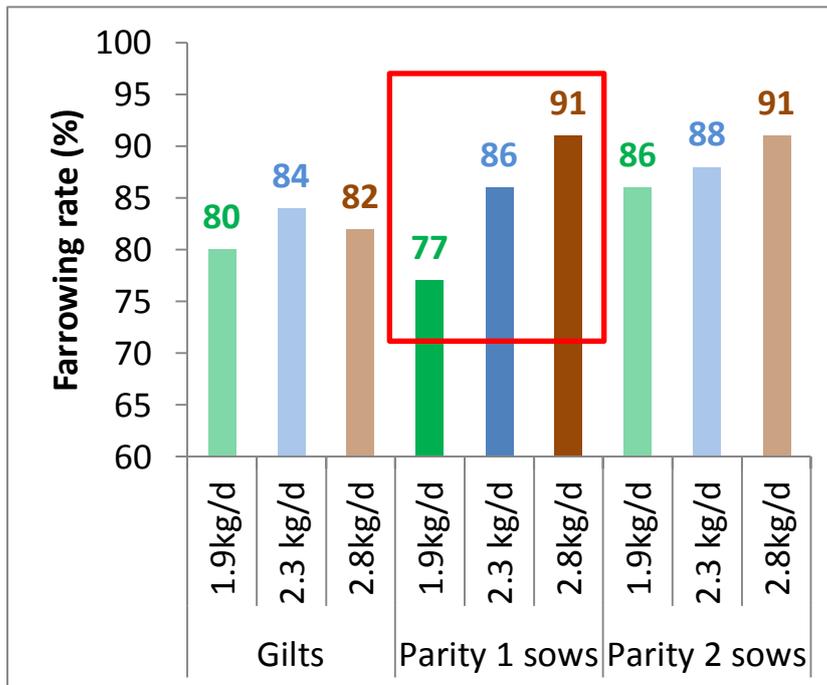


Figure1. Effect of feed intake during the first 28 days of gestation on farrowing rates of sows in groups and fed using electronic sow feeding systems (Hughes and van Wettere unpublished data).

### Reproduction and nutrition in the female pig

It is widely accepted that nutrition profoundly affects the reproductive performance of female pigs (Prunier and Quesnel 2000). The reproductive cycle is controlled by the hypothalamic-pituitary-ovarian axis (Prunier and Quesnel 2000). Gonadotrophin releasing hormone stimulates the release of the gonadotrophins, luteinising hormone (LH) and follicle stimulating hormone (FSH), which in turn regulate follicle development, intra-follicular steroidogenesis and ovulation (Prunier and Quesnel 2000). Changes in feed intake can affect reproductive function directly via the effect of metabolic hormones on ovarian function and gonadotrophin release, or indirectly, via changes in the pattern and concentration of gonadotrophin release (van Wettere and Hughes, 2007).

In the pig, the oestrous cycle lasts for approximately 21 days and consists of a luteal phase and follicular phase (Soede *et al.* 2011). During the follicular phase, LH and FSH support the

development and growth of ovarian follicles, with the largest and most developed follicles avoiding atresia and developing through to ovulation (Soede *et al.* 2011). Ovulation is followed by the luteal phase, during which the corpora lutea (CL) secrete progesterone, which maintains the pregnancy and supports embryo survival (Soede *et al.* 2011). The increase in progesterone suppresses LH and FSH secretion and causes follicle development to be largely suppressed (Prunier and Quesnel 2000). If the pregnancy is not maintained, the corpora lutea will regress, causing a decline in progesterone and the cycle will start again (Soede *et al.* 2011).

Similar to the luteal phase, LH release and follicle development are impaired during lactation; however, it is the suckling stimulus, rather than progesterone which is responsible (Soede *et al.* 2011). During early lactation, LH pulse frequency and amplitude are low, but increase during lactation to support follicle development (Soede *et al.* 2011). While the suckling stimulus is known to be the predominant cause of LH suppression during lactation, the level of LH suppression is also related to the negative energy balance of the sow (Zak *et al.* 1997a). Although early parity sows, in particular those in their first lactation, have a lower appetite than multiparous sows, they produce similar volumes of milk. As a result, parity one sows generally experience a more severe negative energy balance during lactation (Prunier and Quesnel 2000), and are more likely to exhibit impaired reproductive performance post-weaning (Hoving *et al.* 2011).

## **Influence of feed intake prior to mating**

### *Pre-mating nutrition and embryo survival*

Feed restriction prior to mating has been shown to decrease embryo survival and litter size (Almeida *et al.* 2000; Zak *et al.* 1997a). Restricted feed intake of sows during their first lactation reduced ovulation rate by 4.5 and extended the weaning to oestrus interval by 33.6

hours, when compared to sows fed ad libitum throughout gestation (Zak *et al.* 1997a). The data of Zak *et al.* (1997a) suggests that nutritional intake during the last week of lactation exerts the greatest impact on subsequent litter size (Table 1), with embryo survival on day 28 significantly lower following dietary restriction during the last week of lactation compared to the first three weeks of lactation. Using a cycling gilt model, Almeida *et al.* (2000) supported this by demonstrating that embryo survival was significantly lower in gilts subjected to feed restriction during the second week of oestrus compared to feed restriction during the first week or a high feeding level throughout oestrus (Table 1). It is believed that the nutritional influence on embryonic survival is caused by effects on follicle development, oocyte quality and luteal function (Almeida *et al.* 2000; Novak *et al.* 2003).

Table 1. Summary of studies investigating pre-mating feed intake on embryo survival.

| Source                | Stage of production cycle | Period of feed restriction                 |   |                           | Embryo survival % |
|-----------------------|---------------------------|--|---|---------------------------|-------------------|
|                       |                           | Day 0 to 7 of oestrous cycle/<br>lactation | Day 8 to 14 of oestrous cycle/<br>lactation | Day 15 to 28 of lactation |                   |
| Zak et al. (1997a)    | Lactating sow             |  | high  | restricted                | 64.4 <sup>b</sup> |
|                       |                           |  | high  | high                      | 87.5 <sup>c</sup> |
| Almeida et al. (2000) | Cycling gilt              | high                                       | restricted                                  |                           | 68.3 <sup>b</sup> |
|                       |                           | restricted                                 | high  |                           | 81.7 <sup>a</sup> |
|                       |                           | high                                       | high  |                           | 83.6 <sup>a</sup> |

#### *Pre-mating nutrition and oocyte quality*

It has been suggested that the beneficial effects of high feeding levels on embryo survival prior to mating may be due to increased oocyte developmental competence, thus promoting

the capacity of the oocyte to be fertilised, develop into a functional embryo and survive to farrowing (Zak *et al.* 1997b; van Wettere and Hughes, 2007). Sows fed to appetite during lactation had larger follicles and the oocytes were more developed after *in vitro* maturation compared to sows fed a restricted diet during the last week of lactation (Zak *et al.* 1997b). It was also shown that the rate of maturation of randomly selected oocytes *in vitro* was greater in follicular fluid from sows fed to appetite than that from restricted fed sows (Zak *et al.* 1997b). The decreases in follicle development and oocyte quality are thought to be mediated through decreased frequency of LH pulsing (Hazeleger *et al.* 2005). After weaning, a pre-ovulatory LH surge initiates the maturation of the oocyte, and while periods of feed restriction suppress LH release in lactating sows, LH pulse frequency resumed when sows were fed to appetite (Zak *et al.* 1997a) . It is probable that suppressed LH would impair follicle development and oocyte maturation (Hazeleger *et al.* 2005), resulting in reduced ovulation rate and embryo survival (Ashworth and Antipatis 1999).

Insulin and insulin-like growth factor-1 (IGF-1) have been identified as probable mediators of the effect of feed restriction on ovulation rate due to their effects on follicle and oocyte development (Wientjes *et al.* 2012). Early studies by Cox (1987) found that administration of insulin increased ovulation rate in gilts and that the increase was greater in gilts fed a higher energy diet of 2.3 times maintenance. This increased ovulation rate was found without the presence of significant changes in LH, suggesting that insulin could have a direct effect on the ovary (Cox *et al.* 1987). Further studies supported these results showing that treatment with insulin in feed restricted gilts during the late luteal phase increased ovulation rate (Almeida *et al.* 2001). It was again shown that LH concentrations were not affected by feed restriction and concluded that gonadotrophin secretion during feed restriction did not mediate the effects of feed intake on reproductive performance (Almeida *et al.* 2001).

In contrast, Quesnel *et al.* (2000) demonstrated that insulin did not increase the number of large follicles in gilts fed a high feeding level. This study did find that gilts treated with insulin on a restricted diet had significantly lower levels of plasma IGF-1 compared to gilts fed high feed levels. Earlier studies by Quesnel *et al.* (1998) also found that follicular fluid IGF-1 concentrations were lower in feed restricted sows compared to high fed sows prior to mating. Therefore lower levels of insulin from under nutrition may lead to lower IGF-1 concentrations in the plasma and follicles (Quesnel *et al.* 1998). This decrease in IGF-1 could lead to impaired follicular development and in turn lower ovulation rate (Quesnel *et al.* 1998; Quesnel *et al.* 2000). Therefore while the evidence is contradicting it is possible that insulin and or IGF-I mediate nutritional effects by altering follicle development and ovulation rate (Quesnel *et al.* 1998).

#### *Pre-mating nutrition and CL function*

The maintenance of pregnancy is dependent on the presence of the functional corpora lutea and production of progesterone (Soede *et al.* 2011). Luteolytic factors, such as prostaglandin F<sub>2α</sub>, cause the regression of the corpora lutea and the subsequent decline in progesterone production, which leads to termination of pregnancy (Galeati *et al.* 2007). Therefore, factors which impair luteal function and progesterone concentrations may lead to lower embryo survival.

The sensitivity of the corpora lutea to luteolytic factors has also been found to be greater in gilts fed a restricted diet prior to mating (Galeati *et al.* 2007). Gilts fed a restricted diet during the oestrus cycle had an increase in prostaglandin receptors compared to control animals on day 12 of the oestrous cycle, thereby making them more susceptible to luteolysis (Galeati *et al.* 2007). Studies by Almeida *et al.* (2000) found that feed restriction in gilts during the oestrous cycle reduced progesterone concentrations in early pregnancy. It was found that

progesterone concentrations in early pregnancy were lower in gilts fed a restricted feed intake in the second week of the oestrous cycle compared to the first week (Almeida *et al.* 2000). The feed restriction prior to mating possibly impaired luteal function, resulting in the lower progesterone concentrations in early pregnancy (Prunier and Quesnel 2000). Further studies conducted by Almeida *et al.* (2001) again found that feed restriction during the second week of the oestrous cycle resulted in a slower rise in progesterone after the LH surge, further supporting the concept that feed restriction during the late luteal phase has a greater effect on progesterone concentration, possibly through impaired ovarian function.

### **Influence of feed intake post-mating**

#### *Post-mating nutrition, embryo survival and pregnancy maintenance*

It is estimated that up to 40% of embryos do not survive throughout gestation, with the greatest loss in the first month of pregnancy (De *et al.* 2009). Progesterone stimulates uterine function and environment, which in turn provides the optimal conditions for the embryo to survive and develop (van Wettere and Hughes, 2007). Changes in feed intake post-mating can alter progesterone concentrations and affect critical stages of embryo development (Mburu *et al.* 1998).

There is increasing evidence that a change in feed intake after mating could have a significant effect on embryo survival in pigs. A number of studies have demonstrated that medium feed allowances are the most beneficial to embryo survival (De *et al.* 2009; Jindal *et al.* 1997). Gilts fed 1.5 times maintenance (medium feeding level) compared to 2 times maintenance (high feeding level) post-mating had higher embryo survival on day 5 of pregnancy: 86.5% vs 74.2% (Table 2) (Jindal *et al.* 1997). In the same study, the post LH peak rise in peripheral progesterone was delayed by 10h in gilts fed a high feed allowance, indicating that nutritional changes during the periovulatory period can affect timing of the progesterone rise (Jindal *et*

*al.* 1997). This could then affect the timing of changes in the uterine environment and possibly in turn the synchrony between the uterus and the embryos. Therefore, this suggests that nutritionally mediated changes in plasma progesterone during early pregnancy can affect early development and survival of the embryo (Jindal *et al.* 1997).

A study by De *et al.* (2009) found that gilts allocated to a medium feed allowance (1.2 times maintenance) had significantly higher embryo survival of 80.2% on day 25 of pregnancy compared to gilts allocated to a high (2 times maintenance) and low (0.6 times maintenance) feed allowance with an embryo survival of 65.2% and 62.3% respectively (Table 2). It was again found that gilts fed a high feed allowance had lower progesterone concentrations compared to gilts fed medium and low feed allowance at day 12 of pregnancy, which may have led to the decreased embryo survival (De *et al.* 2009). There was no significant difference in the progesterone concentrations between the low and medium feeding levels (De *et al.* 2009). However, other studies have found that food deprivation after ovulation increased progesterone concentrations (Mburu *et al.* 1998). As the oviduct environment is regulated by steroid hormones, the change in progesterone concentrations may lead to changes in the oviduct environment (Mburu *et al.* 1998). These changes may lead to lower embryo cleavage rates and decrease the rate of transport of ova along oviduct (Mburu *et al.* 1998; Mwanza *et al.* 2000). Therefore, possibly affecting embryo survival as it may lead to the embryos being out of synchrony with the uterine environment (Mwanza *et al.* 2000). However these studies only looked at food deprivation 48 hours after ovulation and no measures of embryo survival were determined. Therefore, apart from the study by De *et al.* (2009) there have been limited studies investigating the effect of feed restriction below maintenance on embryo survival for the first critical month of pregnancy. Therefore it is not yet clear as to the effect a restricted feed intake post-mating may have on embryo survival and reproductive performance.

### *Interaction between pre- and post-mating nutrition and reproduction*

The effect of feed intake before and after mating has been investigated and it was found that gilts fed a low feed intake (maintenance) before and after mating had the lowest embryo survival (Table 2) (Ashworth *et al.* 1999). However, it was found that the post-mating feed intake did not have a significant effect on embryo survival and therefore that the pre-mating feed intake has a greater impact on embryo survival at day 12 (Ashworth *et al.* 1999). This contradicts the results by De *et al.* (2009) and Jindal *et al.* (1997) showing that the post-mating feed intake did affect embryo survival. However, this study only looked at embryo survival to day 12, while it is known that the majority of embryo mortality occurs during the implantation period (days 12 – 18 of gestation). The study of Ashworth also used meishan pigs, with only 6 pigs per treatment, whereas the study by De *et al.* (2009) used 18 landrace cross large white gilts per treatment and looked at embryo survival by day 25 of pregnancy. Therefore the differences between the designs of the studies may have accounted for the contradicting results. However apart from the study by Ashworth *et al.* (1999), there are no other studies looking at the effect of feed intake before and after mating. Therefore it is still unclear as to the effect that the interaction of feed intake before and after mating may have on embryo survival.

Table 2. Summary of studies investigating feed intake on embryo survival pre- and post-mating.

| Source                 | Feed Intake Pre-mating | Feed Intake Post-mating | Embryo survival %  |
|------------------------|------------------------|-------------------------|--------------------|
| De et al. (2009)       |                        | 2 times maintenance     | 65.2 <sup>a</sup>  |
|                        |                        | 1.2 times maintenance   | 80.23 <sup>b</sup> |
|                        |                        | 0.6 times maintenance   | 62.38 <sup>a</sup> |
| Jindal et al. (1997)   |                        | 1.5 times maintenance   | 86                 |
|                        |                        | 2 times maintenance     | 74                 |
| Ashworth et al. (1999) | 3 times maintenance    | 1times maintenance      | 99                 |
|                        | 3 times maintenance    | 3 times maintenance     | 89                 |
|                        | 1 times maintenance    | 3 times maintenance     | 75                 |
|                        | 1 times maintenance    | 1 times maintenance     | 73                 |

It is suggested, that sows that are already metabolically challenged at mating may be more sensitive to negative effects of feed restriction during early gestation (Hazeleger *et al.* 2005), as would be the case for parity one sows. As mentioned earlier a severe negative energy balance can lead to impaired luteal function and oocyte quality prior to mating (Zak *et al.* 1997b; Almeida *et al.* 2001). Therefore, it is suggested that poorer quality oocytes and an impaired corpus luteum may be more sensitive to the possible negative effects of restricted feeding during early gestation and this will in turn lead to lower embryo survival.

## Conclusion

The move towards group housing systems of sows during gestation has led to concerns as to how this change may affect feed intake and subsequently reproductive performance of the

sows. As nutrition has a significant influence on reproduction it is expected that in group housing systems, where there is uncontrolled feed intake, this may lead to impaired reproductive performance (Kongsted 2005).

It is widely accepted that a high feeding level prior to mating increases embryo survival (Almeida *et al.* 2000; Zak *et al.* 1997a) . Therefore sows that receive a restricted feed intake prior to mating, such as sows experiencing a negative energy balance from lactation, may have decreased embryo survival (Almeida *et al.* 2000).

However, it is not yet clear as to the effect feed intake after mating may have on reproductive performance. While few studies have found that the post-mating feed intake has little effect on reproductive performance, other studies have found that high and low feed intakes post-mating impair reproductive performance (Ashworth *et al.* 1999; De *et al.* 2009). However, much of the contradicting evidence may be due to different genotypes, parity, numbers of animals used and differences in the length of treatment applied between the studies. Therefore it is possible that sows receiving a restricted feed intake post-mating may experience impaired reproductive performance. This effect may be evident in low ranking sows, such as parity one sows, in a group housing system where competition exists for the feed (Andersen *et al.* 1999; Hoving *et al.* 2011).

Furthermore, the effect of post-mating feed intake in response to pre-mating feed intake on reproductive performance remains unclear, due to limited studies on this interaction. Therefore it is unknown if the effect of restricted feed intake post-mating experienced by low ranking parity one sows in a group housing system is likely to be greater in these sows that are already metabolically challenged from lactation (Kongsted 2005).

Therefore, this study aims to determine if pre- and post-mating feed intake interact to affect embryo survival and pregnancy maintenance in the pig, using a gilt model.

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Thesis

Effect of pre- and post-mating dietary  
restriction on embryo survival of  
group housed gilts

## **Abstract**

The aims of this study were to determine if pre- and post-mating feeding levels interact to affect embryo survival, and to determine whether feeding to maintenance requirements would impair embryo survival. Gilts were allocated to a pre-mating treatment of 1 times (prehigh) or 0.8 times (prelow) maintenance from d 1 to 14 of the oestrous cycle prior to mating. From day 15 to mating all gilts were group housed and fed ad-lib. All gilts were artificially inseminated at the third oestrus. The day after mating gilts were group housed and allocated to the post-mating treatment of 1.5 times (posthigh) or 1 times (postlow) maintenance. Gilts were slaughtered on day  $25.5 \pm 0.22$  post-insemination and reproductive tracts collected. Prew gilts lost significantly more weight than prehigh gilts over the pre-mating treatment ( $3.7 \pm 0.71$  versus  $6.7 \pm 0.84$  kg). From mating to slaughter, gilts in postlow treatment lost  $0.5 \pm 1.02$  kg liveweight, while those in the post-high group gained  $5.7 \pm 0.90$  kg liveweight ( $P > 0.05$ ). Pre-mating had no effect on any of the reproductive measures. Embryo survival was higher ( $P < 0.05$ ) in the posthigh compared to postlow treatment groups ( $88.4 \pm 2.52$  versus  $77.8 \pm 3.98$  %), resulting in more ( $P < 0.05$ ) embryos being present ( $14.0 \pm 0.63$  versus  $11.7 \pm 0.68$ ). There was no interaction between the pre-mating and post-mating feed intake on any reproductive measures. Therefore, these data demonstrated that reducing post-mating feed intakes to maintenance levels impaired embryo development and thus survival.

## Introduction

The Australian pork industry has responded to consumer and thus retailer pressure to improve sow welfare by reducing confinement during gestation. This had led to the widespread adoption of group housing systems (Kongsted 2005). Group housing improves sow welfare by allowing freedom of movement, increased socialisation and increased capacity to express natural behaviours. However there are a number of negative aspects associated with group housing systems, including competition for resources and a resultant variation in feed intake (Kongsted 2005). High variation in feed intake between sows, due to competition for food, is evident in group housing systems with low ranking sows thought to receive less food than their higher ranking counterparts (Andersen *et al.* 1999). An association between reduced feed intake during early gestation in group housed systems and impaired reproduction has been proposed, with the likelihood of pregnancy loss or low litter sizes increased with decreased backfat gain between weaning and day 21 post-mating (Kongsted 2006).

Changes in feed intake affect ovarian function directly via the effect of metabolic hormones on ovarian follicle growth, or indirectly via changes in the pattern and concentration of gonadotrophin release (van Wettere and Hughes 2007). Feed restriction prior to mating decreased embryo survival in gilts and lactating sows, mostly likely due to impaired follicle development, oocyte quality and luteal function (Zak *et al.* 1997b; Almeida *et al.* 2000). Feed restriction during the late luteal phase (days 8 – 15) of the oestrous cycle had the largest negative effect on embryo survival, with an embryo survival of 68.3 % (Almeida *et al.* 2000). These results suggest the negative energy balance suffered by sows, particularly parity one sows, due to energy demands from milk production and decreased appetite, may lead to impaired embryo survival (Zak *et al.* 1997a).

Although litter size is similar between sows fed at a moderate or high level post-mating (Quesnel *et al.* 2010), more severe feed restriction, such as below maintenance, has been

shown to be detrimental to embryo survival (De *et al.* 2009). A moderate feeding level of 1.2 times maintenance during early pregnancy resulted in significantly higher embryo survival rates compared to that of a more severe feed restriction of 0.6 times maintenance in gilts (De *et al.* 2009). It is believed that the positive effects of moderate feed restriction post-mating are due to changes in progesterone concentrations, resulting in improved synchrony between the embryos and the uterine environment, and consequently improved development and increased survival (Jindal *et al.* 1997). It is, therefore, suggested that sows experiencing a restricted feed intake post mating may experience impaired reproductive performance. This effect may be seen in low ranking sows, such as parity one sows, in a group housing system where competition for feed exists (Kongsted 2005).

Interestingly, the interaction between feed intake pre- mating and post-mating on embryo survival and litter size has received little attention. Ashworth *et al.* (1999) observed lower embryo survival rates in gilts receiving a maintenance feed intake before and after mating compared to those fed at three times maintenance. However this study only looked at embryo survival to day 12, and it is known that the majority of embryo mortality occurs during the implantation period (days 12 – 18 of gestation) (Geisert and Schmitt 2002). Therefore it is not yet well understood if the effect on embryo survival of a restricted feed intake post mating experienced by a group housed low ranking parity one sow is likely to be greater in these sows that are already metabolically challenged from lactation.

The aims of this study were to use an established gilt model (Almeida *et al.* 2000; Chen *et al.* 2012) for a parity one sow to determine whether there is an interaction between the pre-mating and post-mating feed intake on embryo survival and to determine if a restricted feed intake after mating will negatively affect embryo.

## **Materials and Methods**

This experiment was conducted at the University of Adelaide's piggery, South Australia, with approval from the Animal Ethics Committee of the University of Adelaide. The experiment was a two x two factorial design incorporating two feeding levels from day 1 to day 14 of the second oestrous cycle (0.8 x maintenance (PreLow, n = 23) versus 1 x maintenance (PreHigh, n = 23)) and two feeding levels from insemination until slaughter (1 x maintenance (PostLow, n = 23) versus 1.5 x maintenance (PostHigh, n = 23)). This study was conducted in two blocks. Block one ran from May until July 2012 with the second block conducted from July until September 2012.

### *Experimental Design*

Forty-nine Large White x Landrace gilts were used in this study. At 171 days of age, puberty was stimulated using the combination of a single intra muscular injection of PG600 (400IU of Pregnant Mare Serum Gonadotrophin and 200 IU of human Chorionic Gonadotrophin; Intervet, Australia) and 15 minutes of daily, physical contact with a mature boar (Bartlett et al. 2009). Oestrus was defined as the exhibition of a standing reflex, either in response to the manual application of pressure to the gilts back (the back pressure test), or mounting by the boar. From 12 days after detection of the first oestrus until second oestrus, gilts received 15 minutes of daily, fenceline contact with a mature boar in order to maintain oestrous cyclicity and enable oestrus detection.

Following the completion of one oestrous cycle of normal length ( $20.8 \pm 0.14$  days), gilts were stratified according to liveweight and were randomly allocated to the PreLow or PreHigh treatment groups. From day one to day 14 of the second oestrous cycle (day 0 corresponds to the first day of behavioural oestrus), gilts were fed 0.8 x maintenance (PreLow; mean intake of  $1.05 \pm 0.07$  kg feed;  $13.8 \pm 0.18$  MJ/day;  $158.5 \pm 2.08$ g protein; 7.3

$\pm 0.10\text{g}$  available lysine) or 1.0 x maintenance (PreHigh; mean intake of  $1.32 \pm 0.1$  kg feed;  $17.2 \pm 0.27$  MJ;  $198.5 \pm 3.07\text{g}$  protein;  $9.25 \pm 0.13\text{g}$  available lysine) of a gilt developer diet (Table 1). From day 15 of the second oestrous cycle until exhibition of their third oestrus, gilts were fed the gilt developer diet ad-libitum. From 12 days after detection of the second oestrus until third oestrus, gilts received 15 minutes of daily, fenceline contact with a mature boar in order to maintain oestrous cyclicity and enable oestrus detection.

Gilts were artificially inseminated at detection of their third oestrus, stratified according to weight within the pre-insemination treatments and randomly allocated to either the low (PostLow) or high (PostHigh) feeding level post insemination. From day one post insemination until slaughter at  $25 \pm 0.22$  days of gestation, gilts were fed 1 x maintenance (PostLow; mean intake of  $1.37 \pm 0.09$  kg feed;  $17.8 \pm 0.25$  MJ;  $205.1 \pm 2.89\text{g}$  protein;  $9.6 \pm 0.13$  available lysine) or 1.5 x maintenance (PostHigh; mean intake of  $2.06 \pm 0.14$  kg feed;  $26.8 \pm 0.36$  MJ;  $309.4 \pm 4.17$  available lysine) of a gestation diet (Table 1). Gilts were weighed weekly during the pre- and post-mating nutritional treatment periods and the daily feed intake required was adjusted for each gilt. Maintenance energy intake was calculated using the following formula ( $0.444 \times \text{LW}^{0.75}$ ; Feeding Standards for Australian Livestock Pigs). A diagrammatic scheme of the experimental design is presented in Fig. 1.

### *Housing and Management*

From selection until their second oestrus gilts were group housed in grower sheds. From the start of their pre-insemination dietary treatment (day one of their second oestrus until day 14 of their second oestrous cycle) gilts were housed individually in stalls. From day 15 of the second oestrous cycle through till slaughter on day 25 post-insemination, gilts were housed in fixed groups of 6 in a grower shed, with a space allowance of 1.25m per pig. From insemination at their third oestrus until slaughter, gilts were moved daily to individual stalls

where they received post-insemination feed intake, and once this was consumed they were returned to the grower sheds.

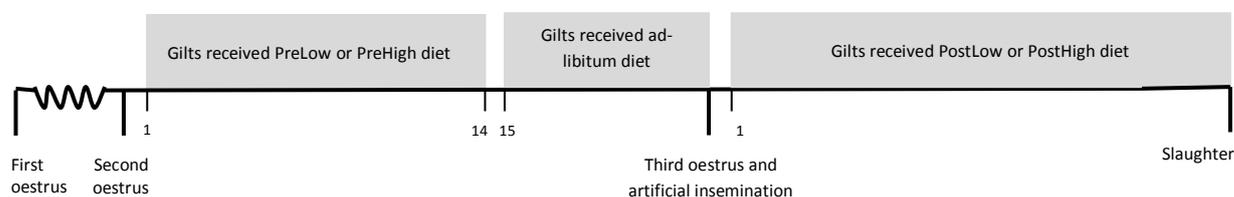


Figure 1. Timing of dietary manipulation relative to mating for each of the treatments. The pre-mating treatment ran from day one to day 14 of the second oestrous cycle in which, gilts received 0.8 times maintenance (PreLow) or 1.0 times maintenance (PreHigh). The post-mating treatment ran from day one post artificial insemination until slaughter at  $d 25.5 \pm 0.22$  of gestation in which, gilts received 1.0 times maintenance (PostLow) or 1.5 times maintenance (PostHigh).

### *Artificial insemination*

All gilts were artificially inseminated at detection of the third oestrus and again 24h later if they still displayed oestrus. All inseminations took place in a detection mating area, with fenceline boar contact. Inseminations were performed using disposable spirette catheters, with each insemination consisting of an 80 ml dose of fresh terminal mix, extended semen ( $3 \times 10^9$  spermatozoa per inseminate). Semen used for this experiment was purchased from a commercial artificial insemination collection centre (SABOR Pty. Ltd, Clare, South Australia).

### *Gilt liveweight and backfat measurements*

Gilts were weighed at onset of the second oestrus, day seven and 14 of the second oestrous cycle, insemination, day seven, 14 and 21 post insemination, and day of slaughter. P2 backfat measurements were taken at detection of second oestrus, insemination and day of slaughter. P2 backfat was measured over the last rib 65mm down from the vertebrae, using a 3.5 MHz linear-array transducer (Ausonics Impact, Kimberly, WI, USA).

### *Collection of Reproductive Tracts*

Gilts were slaughtered at the abattoir on day  $25.5 \pm 0.22$  of pregnancy. After slaughter the reproductive tracts were collected. The left and right ovaries were removed, weighed and the number of corpora lutea on both of the ovaries were counted indicating ovulation rate. The left and right horns of the uterus were separated and cut longitudinally. The embryos and placentas of each horn were removed and weighed. The uterine horns were weighed before and after embryo and placenta removal. Embryo survival was determined as the percentage of corpora lutea represented by a viable embryo. Any embryos that were more than two standard deviations below the average embryo weight for each gilt were recorded as non-viable (Jindal *et al.* 1996).

### *Statistical Analysis*

Data are expressed as means  $\pm$  standard error of the mean (SEM). A general linear model, with slaughter weight and weight and backfat changes as co-variates, and block as a fixed effect, was used to study the effects of the pre- and post-mating diet and their interaction on ovulation rate, embryo number and embryo survival. Embryo weight and placenta weight were analysed using a general linear model, with slaughter weight and day of gestation at slaughter as co-variates, and block and treatment as fixed effects. Statistical significance

between treatments was determined using least significant difference. Results were considered significant at  $P < 0.05$ . All analyses were performed using SPSS, version 19 (IBM).

Table 1. Ingredients and chemical analysis of the gilt developer and dry sow diet fed to gilts.

|                          | Gilt developer | Dry sow diet |
|--------------------------|----------------|--------------|
| <b>Ingredients (%)</b>   |                |              |
| Barley                   | 17.33          | 19.25        |
| Wheat                    | 44.94          | 42.33        |
| Millrun                  | 25             | 30           |
| Canola expeller          | 6.1            | 2.1          |
| Meatmeal                 | 3.57           | 3            |
| Tallow                   | 0.5            | 0.5          |
| Choline chloride         | 0.007          | 0.067        |
| Limestone                | 1.33           | 1.45         |
| Biofos                   | 0.07           | 0.23         |
| Sodium chloride          | 0.25           | 0.35         |
| Lysine sulphate          | 0.45           | 0.25         |
| Threonine                | 0.08           | 0.01         |
| Alimet                   | 0.02           | 0.003        |
| Phyzyme XP 5000 liquid   | 0.01           | 0.01         |
| Breeder + bioplex PMX    | 0.25           | 0.25         |
| Biofix select            | 0.1            | 0.2          |
| <b>Chemical analysis</b> |                |              |
| Digestible energy (MJ)   | 13.20          | 12.99        |
| Protein (%)              | 15.13          | 13.82        |
| Fat (%)                  | 3.65           | 3.44         |
| Fibre (%)                | 5.00           | 5.00         |
| Lysine (%)               | 0.69           | 0.55         |

## Results

### *Growth characteristics*

Gilt liveweight and P2 backfat were not different at detection of second oestrus for all treatment groups. During the pre-mating treatment period, gilts in the prelow group lost significantly more weight than those in the prehigh group ( $6.7 \pm 0.84$  versus  $3.7 \pm 0.71$  kg). Liveweight change from day 14 of the second oestrous cycle to AI was similar between all treatment groups, with all treatments gaining weight. During this period there was a significant pre- and post-mating interaction, with the prelow\*postlow gaining more backfat than the prehigh\*postlow ( $2.6 \pm 1.13$  versus  $1.7 \pm 0.65$  mm). During the post-mating treatment period, gilts in the posthigh treatment group gained significantly more weight than those in the postlow group ( $5.7 \pm 0.90$  versus  $-0.5 \pm 1.02$  kg). During this period, there was a significant pre- and post-mating interaction, with the prelow\*posthigh treatment gaining more backfat than the prelow\*postlow treatment. At slaughter, gilts receiving the posthigh treatment were significantly heavier ( $142.6 \pm 2.82$  kg) compared to those in the postlow treatment ( $137.8 \pm 2.90$  kg).

Table 2. Liveweight, liveweight change and backfat change of gilts fed 1 times maintenance (prehigh) or 0.8 times maintenance (prelow) before mating and 1.5 times maintenance (posthigh) or 1 times maintenance (postlow) after mating.

|                                | PreHigh              |                     | PreLow               |                     | Significance |            |            |
|--------------------------------|----------------------|---------------------|----------------------|---------------------|--------------|------------|------------|
|                                | PostHigh<br>(n = 11) | PostLow<br>(n = 12) | PostHigh<br>(n = 12) | PostLow<br>(n = 11) | PreM         | PostM      | PreM*PostM |
| <b>Live weight (kg)</b>        |                      |                     |                      |                     |              |            |            |
| Prior to pre-mating diet       | 131.8 ± 3.71         | 134.0 ± 3.74        | 131.6 ± 3.07         | 134.9 ± 2.94        | ns           | ns         | ns         |
| End of pre-mating diet (d 14)  | 128.1 ± 4.13         | 130.3 ± 4.39        | 123.9 ± 3.59         | 129.2 ± 3.24        | ns           | ns         | ns         |
| AI                             | 139.2 ± 3.81         | 138.0 ± 3.91        | 134.9 ± 2.94         | 138.7 ± 3.46        | ns           | ns         | ns         |
| slaughter                      | 145.0 ± 4.48         | 137.6 ± 4.71        | 140.5 ± 3.60         | 138.1 ± 3.46        | ns           | P = 0.014  | ns         |
| <b>Live weight change (kg)</b> |                      |                     |                      |                     |              |            |            |
| During pre-mating diet         | -3.7 ± 1.13          | -3.7 ± 0.92         | -7.6 ± 1.04          | -5.7 ± 1.32         | P = 0.0004   | ns         | P = 0.002  |
| d 14 – AI                      | 11.0 ± 1.28          | 7.7 ± 1.58          | 10.9 ± 1.13          | 9.5 ± 1.21          | ns           | ns         | ns         |
| During post-mating diet        | 5.8 ± 1.61           | -0.5 ± 1.66         | 5.6 ± 0.98           | -0.5 ± 1.22         | ns           | P = 0.0001 | ns         |
| <b>Backfat change (mm)</b>     |                      |                     |                      |                     |              |            |            |
| During pre-mating diet         | -1.1 ± 1.42          | -2.6 ± 1.68         | -2.4 ± 1.26          | -2.7 ± 1.90         | ns           | ns         | ns         |
| d 14 – AI                      | 2.0 ± 0.97           | 1.7 ± 0.65          | 1.9 ± 0.83           | 2.6 ± 1.13          | ns           | ns         | P = 0.021  |
| During post-mating diet        | -0.7 ± 1.65          | 1.2 ± 0.96          | 4.02 ± 1.65          | -1.3 ± 1.18         | ns           | ns         | P = 0.004  |

Values are means ± SEM

ns: not significant at P > 0.05

### *Reproductive characteristics*

The data described in Table 2 demonstrates that pre-mating feed intake did not affect ovary weight and ovulation rate. There was no effect of pre-mating treatment on the interval from day 14 of the oestrous cycle until mating ( $6.7 \pm 0.15$  days). There was no effect of the pre-mating diet on uterine weight, placenta weight and embryo weight ( $P > 0.05$ ).

Pregnancy rates for the prehighposthigh, prehighpostlow, prelowposthigh and prelowpostlow groups were 92, 100, 100 and 92% respectively. There was no effect of the post-mating feed intake on ovulation rate, embryo weight, placenta weight or embryo weight ( $P > 0.05$ ). Uterine weight tended to be larger in the posthigh compared to the postlow ( $P < 0.10$ ). Embryo survival ( $77.8 \pm 3.98$  versus  $88.4 \pm 2.52$ ) and embryo number ( $11.7 \pm 0.68$  versus  $14.0 \pm 0.63$ ) were lower ( $P < 0.05$ ) for the gilts in the postlow compared to the posthigh group, respectively.

Table 3. Reproductive characteristics on day  $25.5 \pm .22$  of pregnancy in gilts fed 1 times maintenance (prehigh) or 0.8 times maintenance (prelow) before mating and 1.5 times maintenance (posthigh) or 1 times maintenance (postlow) after mating.

|                     | PreHigh              |                     | PreLow               |                     | Significance |           |            |
|---------------------|----------------------|---------------------|----------------------|---------------------|--------------|-----------|------------|
|                     | PostHigh<br>(n = 11) | PostLow<br>(n = 12) | PostHigh<br>(n = 12) | PostLow<br>(n = 11) | PreM         | PostM     | PreM*PostM |
| Ovary weight (g)    | 15.1 ± 0.49          | 15.2 ± 0.71         | 14.0 ± 0.43          | 14.8 ± 0.66         | ns           | ns        | ns         |
| Ovulation rate      | 15.1 ± 0.48          | 14.9 ± 0.53         | 16.4 ± 0.99          | 15.0 ± 0.63         | ns           | ns        | ns         |
| Uterine weight (g)  | 1019.3 ± 32.06       | 1054.6 ± 45.72      | 1019.1 ± 49.49       | 958.6 ± 35.46       | ns           | P = 0.095 | ns         |
| Placenta weight (g) | 16.5 ± 1.89          | 16.8 ± 2.26         | 15.2 ± 2.47          | 15.9 ± 2.66         | ns           | ns        | ns         |
| Embryo weight (g)   | 0.90 ± 0.09          | 0.94 ± 0.09         | 0.76 ± 0.08          | 0.81 ± 0.10         | ns           | ns        | ns         |
| Number of Embryos   | 13.2 ± 0.72          | 11.7 ± 0.88         | 14.8 ± 0.99          | 11.8 ± 1.09         | ns           | P = 0.006 | ns         |
| Embryo survival (%) | 86.6 ± 4.19          | 78.1 ± 5.64         | 90.0 ± 3.04          | 77.6 ± 5.87         | ns           | P = 0.026 | ns         |

Values are means ± SEM  
 ns: not significant at  $P > 0.05$

### *Correlations between reproductive and growth measurements*

Embryo survival was positively correlated to backfat thickness at day of slaughter (Table 3). Embryo number was positively correlated with liveweight at d 0, 14, mating, slaughter and with backfat thickness at day of slaughter. Embryo number was also positively correlated with weight change over the post-mating treatment and backfat thickness change over the pre-mating treatment. Ovulation rate was positively correlated with weight at d 0, 14 and mating, as well as backfat thickness at d 14 and mating. Ovulation rate was also positively correlated with weight change over the pre-mating treatment. Liveweight at d 0 was positively correlated with liveweight at d 14 ( $r = 0.956$ ,  $P = 0.000$ ) and mating ( $r = 0.913$ ,  $P = 0.000$ ).

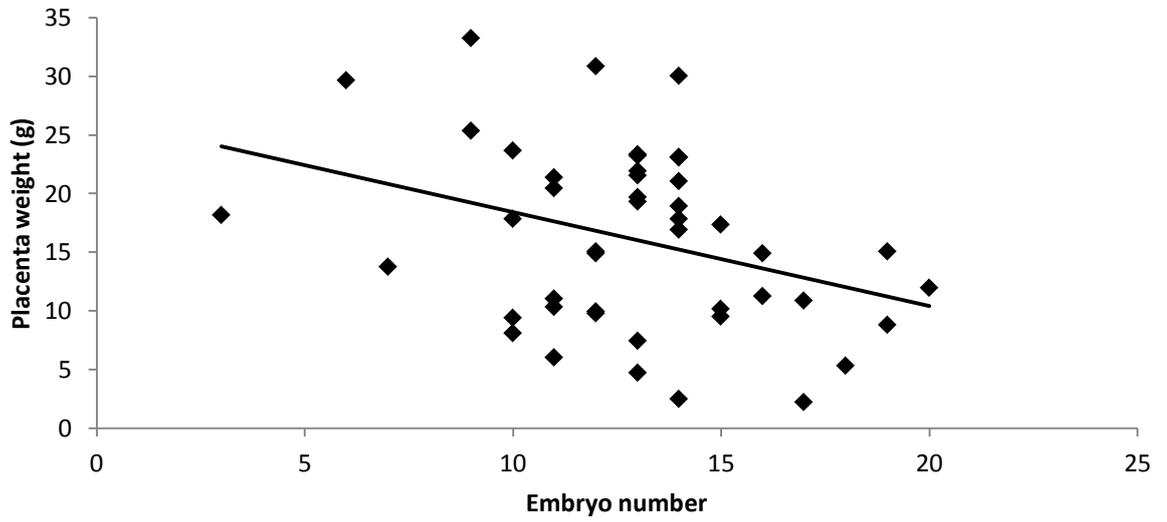
### *Correlations between reproductive traits*

As expected embryo number was positively correlated with ovulation rate ( $r = 0.624$ ,  $P = 0.000$ ) and embryo survival ( $r = 0.781$ ,  $P = 0.000$ ). Embryo number was negatively correlated with placenta weight ( $r = -0.343$ ,  $P = 0.019$ ) (Figure 1). Embryo weight was positively correlated with placenta weight ( $r = 0.874$ ,  $P = 0.000$ ) (Figure 2).

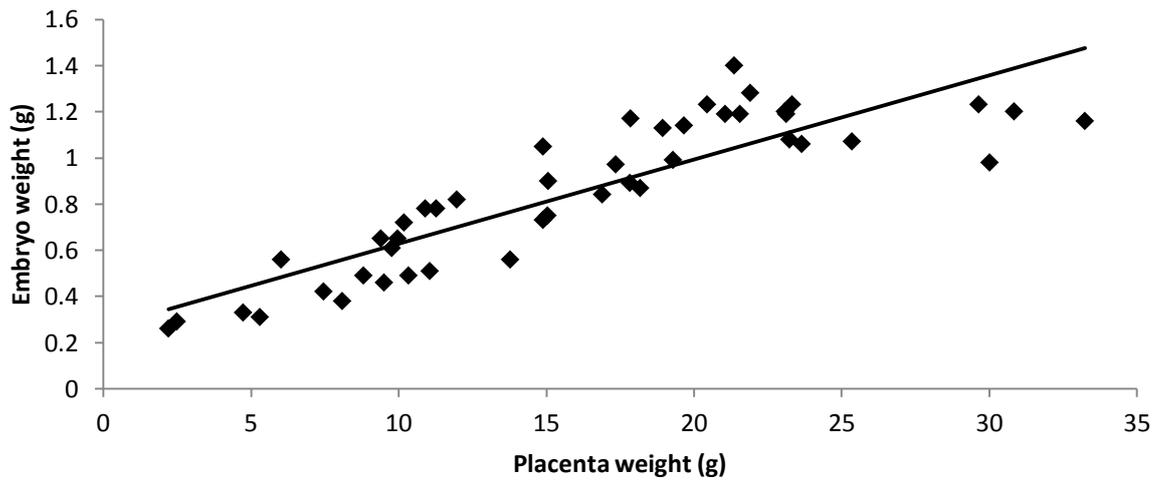
Table 4. Relationship between reproductive and growth characteristics (Pearson correlation coefficients (r) and P-value)

|                                | Embryo survival (%) |         | Embryo number |         | Embryo weight (g) |         | Ovulation rate |         |
|--------------------------------|---------------------|---------|---------------|---------|-------------------|---------|----------------|---------|
|                                | r                   | P-value | r             | P-value | r                 | P-value | r              | P-value |
| <b>Live weight (kg)</b>        |                     |         |               |         |                   |         |                |         |
| Prior to pre-mating diet       | 0.106               | ns      | 0.337         | 0.022   | 0.009             | ns      | 0.402          | 0.006   |
| End of pre-mating diet         | 0.077               | ns      | 0.305         | 0.039   | 0.098             | ns      | 0.396          | 0.006   |
| AI                             | 0.148               | ns      | 0.347         | 0.018   | -0.029            | ns      | 0.379          | 0.009   |
| Slaughter                      | 0.204               | ns      | 0.434         | 0.003   | 0.107             | ns      | 0.465          | 0.001   |
| <b>Backfat (mm)</b>            |                     |         |               |         |                   |         |                |         |
| Prior to pre-mating diet       | -0.286              | ns      | -0.303        | 0.040   | 0.251             | ns      | -0.154         | ns      |
| End of pre-mating diet         | 0.135               | ns      | 0.287         | ns      | 0.296             | 0.046   | 0.349          | 0.017   |
| AI                             | 0.103               | ns      | 0.289         | ns      | 0.260             | ns      | 0.346          | 0.019   |
| Slaughter                      | 0.369               | 0.012   | 0.486         | 0.001   | 0.113             | ns      | 0.308          | 0.037   |
| <b>Live weight change (kg)</b> |                     |         |               |         |                   |         |                |         |
| During pre-mating diet         | -0.047              | ns      | 0.045         | ns      | 0.295             | 0.046   | 0.158          | ns      |
| d 14 – AI                      | 0.161               | ns      | 0.027         | ns      | -0.355            | 0.015   | -0.150         | ns      |
| During post-mating diet        | 0.194               | ns      | 0.343         | 0.020   | 0.328             | 0.026   | 0.353          | 0.016   |
| <b>Backfat change (mm)</b>     |                     |         |               |         |                   |         |                |         |
| During pre-mating diet         | 0.280               | ns      | 0.391         | 0.007   | 0.024             | ns      | 0.330          | 0.025   |
| d 14 – AI                      | 0.012               | ns      | 0.133         | ns      | 0.083             | ns      | 0.160          | ns      |
| During post-mating diet        | 0.191               | ns      | 0.106         | ns      | -0.161            | ns      | -0.090         | ns      |

ns: not significant at  $P > 0.05$



**Figure 2.** Relationship between placenta weight and embryo number ( $r = -0.343$ ,  $P = 0.019$ ).



**Figure 3.** Relationship between placenta weight and embryo weight ( $r = 0.874$ ,  $P = 0.000$ ).

## Discussion

Overall, the current investigation demonstrated that there was no interaction between the pre-mating and post-mating feed intake on embryo survival, and that the post-mating feed intake had a larger effect on embryo survival than the pre-mating feed intake. Although data on the interaction between pre- and post-mating feed intake as a determinant of potential litter size is limited, Ashworth *et al.* (1999) also reported no effect of the interaction between the pre-mating and post-mating feed intake on embryo survival on day 12 of gestation, agreeing with the present findings. However, in contrast to the present data, Ashworth *et al.* (1999) demonstrated that pre-mating feed intake exerted a greater effect on embryo survival than post-mating feed intake (Ashworth *et al.* 1999). In support of recent findings (De *et al.* 2009), the current data demonstrated that reducing post-mating feed intake to below maintenance levels decreased embryo survival, emphasising the importance of optimising post-mating feed intake to achieve maximum reproductive performance in pigs.

While the larger effect of the post-mating diet compared to the pre-mating diet in the current study conflicts with the findings of Ashworth *et al.* (1999), this could be due to the large number of differences between the experimental designs. Firstly, Ashworth *et al.* (1999) only looked at embryo survival at day 12 of gestation, before the implantation period. While the current study measured embryo survival at day 25 of gestation, after the implantation period. It is well understood that a majority of embryo mortality occurs during the implantation period and therefore a measure of embryo survival after this period may better reflect the effect of feed intake post-mating on embryo survival. The pre-mating treatment of the Ashworth *et al.* (1999) study was also imposed for the whole of the oestrous cycle prior to mating, while the gilts in the current study only received the pre-mating diet for the first two weeks of the cycle. The longer period of the pre-mating treatment and the fact that it was imposed right up until mating may have had a larger influence on ovulation rate and embryo survival compared to the current study. Finally, Ashworth *et al.* (1999) used feeding levels of

1 x maintenance and 3 x maintenance, while it was earlier shown that higher and lower feeding levels post-mating impaired embryo survival compared to that of a moderate feed intake (1.5x maintenance). Therefore it might be possible that these feeding levels post-mating would have both impaired embryo survival. This could have caused the insignificant post-mating effect, compared to this study and previous studies that did find a significant post-mating feed intake effect on embryo survival when a moderate post-mating feed intake was included.

Although the beneficial effects of moderate feed restriction post-mating (1.2 to 1.5 times maintenance) on embryo survival have previously been demonstrated (Jindal *et al.* 1997; De *et al.* 2009), the effect of a severe feed restriction (maintenance levels or below) on embryo survival has received little attention. The present study found that feeding gilts at maintenance levels post-mating decreased embryo survival. De *et al.* (2009) found similar results, with a low feeding level of 0.6 times maintenance significantly decreasing embryo survival compared to a moderate post-mating feed level of 1.2 times maintenance, with an embryo survival of 62% and 80% respectively. In the current study the embryo survival of the low and high post-mating feed intake was 78% and 88% respectively, which is much higher than the embryo survival found in the study by De *et al.* (2009). This may be due to the more severe feed restriction imposed in the De *et al.* (2009) study compared to the current study, which suggests that the more severe the feed restriction, the more embryo survival is impaired.

The positive effects of reducing post-mating feed intake from 2 to 1.5 times maintenance on embryo survival have been related to increased circulating progesterone levels (Jindal *et al.* 1997). However, severe feed restriction post-mating decreased plasma progesterone, which resulted in a lower embryo cleavage rate and a decrease in the transport rate of ova along the oviduct, affecting the development of the embryo and its synchrony with the oviductal environment (Mburu *et al.* 1998; Mwanza *et al.* 2000). Furthermore it has been shown that

asynchrony, in which the uterine is further advanced than the embryo, resulted in lighter embryos and placentas (Wilson *et al.* 2001). Based on the literature, the embryos and placentas of the low feed intake group should have been less developed due to asynchrony between the uterine environment and embryo. However the current data demonstrated that gilts fed a low post-mating feed intake, with a decreased embryo survival, had heavier placentas and embryos than the high feed intake group, with a higher embryo survival. Furthermore neither embryo weight nor placenta weight was significantly correlated with embryo survival. This is in contrast with previous studies who found that the treatment group of gilts with the highest embryo survival had significantly heavier embryos (De *et al.* 2009) and a larger crown rump length (Jindal *et al.* 1997). However, similar to the current study, Almeida *et al.* (2000) observed no reduction in embryo or placental weights in gilts with low embryo survival rates. The current data demonstrated a negative correlation between placenta weight and embryo number, suggesting that limitation in uterine capacity may have restricted placental growth and subsequent embryo development when embryo survival rates were high (Wilson *et al.* 2001; van der Waaij *et al.* 2010). Alternatively the lighter, less developed embryos could have died in the postlow gilts, increasing the mean weight of the embryos for these gilts. As part of the implantation process, there is competition between the embryos for uterine space and nutrients, and consequently the less developed embryos are likely to be out competed by the more developed embryos and eventually die (Geisert and Schmitt 2002).

The level of feed restriction imposed in the current study is likely to be similar to the intake achieved in low ranking sows in a group housing system. Low ranking sows experience increased aggression over competition for feed, possibly resulting in a lower feed intake (Andersen *et al.* 1999). While it has been found that unintentional variation of feed intake in a group housing situation decreases the chance of pregnancy (Kongsted 2006), it has not yet been investigated if the unintentional variation of feed intake in group housing has an effect on embryo survival. Furthermore it is currently unknown how much feed intake in a group

housing system does vary between sows. But if it does vary so that low ranking sows receive a feed intake of maintenance or below, both the current data and that of De *et al.* (1999) suggests that impaired reproductive performance, due to reduced embryo survival, is a likely consequence. These data indicate the need to modify feeding strategies for group housed sows so that maximum reproductive performance is achieved.

However apart from just unequal feed intake, it is possible that aggression and stress may also negatively affect reproductive performance of sows in a group housing situation. While there is little current research on the effect of aggression and stress from a group housing situation on reproduction, it has been previously found that aggression during early pregnancy has a negative effect on embryo survival. As the gilts in the current study were group housed during gestation, it is possible that aggression and stress had an effect on embryo survival. However no indicators of aggression or stress were measured in this study, so further research in determining if aggression and stress in a group housing system further effect embryo survival on top of a restricted feed intake would be beneficial in improving reproductive performance in a group housing system.

The effect of feed intake prior to mating on reproductive performance has been widely studied in pigs, with high feed intake increasing ovulation rate and embryo survival (Zak *et al.* 1997a; Almeida *et al.* 2000). However during lactation sows particularly parity one sows, generally suffer a negative energy balance, due to increased energy demands from milk production and decreased appetite, resulting in weight and backfat loss (Quesnel *et al.* 1998). This excessive body condition loss negatively affects reproductive performance, with ovulation rate and embryo survival decreased in sows that were restrictively fed during lactation (Zak *et al.* 1997a) and cycling gilts (Almeida *et al.* 2000). While there was no effect of restricted feed intake prior to mating in the present study on ovulation rate or embryo survival, this could be due to the timing of pre-mating feed restriction used in the current study. The period of pre-mating feed restriction ended the week before mating in the current study, which caused

increased growth rates in all of the gilts in the week prior to mating. This could have allowed for an increase in follicle development and subsequently improved embryo survival. Hazelenger *et al.* (2005) found similar results, demonstrating that a period of increased feeding during the follicular phase prior to mating counteracted the negative effects of previous feed restriction. Conversely, previous studies (Chen *et al.* 2012; Almeida *et al.* 2000) indicate that increased feeding during the follicular phase prior to mating, following a period of restrictive feeding resulted in reduced ovulation rates, embryo survival and embryo number. However the increase in feed intake and growth rate throughout the week prior to mating were much larger in the current study compared to the gilts in the Almeida *et al.* (2000) study. This could suggest that a period of increased feeding over the follicular phase prior to mating may counteract the negative effects of previous feed restriction, but this effect may only be shown when the increase in feed intake and growth is large enough.

The length of the ad-lib feeding period may also have had an influence in counteracting the negative effects of previous feed restriction. Previous studies have reported an increase in the weaning to oestrus interval when sows were subjected to feed restriction during lactation. Zak *et al.* (1997a) found that restricted fed sows during lactation had only a marginally longer weaning to oestrus interval, suggesting this could have had an effect on the ovulation rate. While the period of ad-libitum feed intake prior to mating (equivalent to weaning-to-oestrus interval in the sow) in the current study was not different between treatment groups, it was higher compared to previous studies with significantly different results (Zak *et al.* 1997a). Furthermore it has been suggested that a period of 5 days or more of refeeding after feed restriction allows recovery at the ovarian level through increased follicular growth and oestradiol synthesis and subsequent improved embryo survival (Baidoo *et al.* 1992). Therefore a period of almost seven days of ad-lib feeding in this study may have allowed the gilts to recover from the previous feed restriction (Hazeleger *et al.* 2005).

While there was no treatment effect on ovulation rate in the current study, ovulation rate was positively correlated with liveweight prior to the experimental treatment period. Furthermore liveweight at d 0 was positively correlated with liveweight at d 14 and mating, therefore the heavier gilts at the start of the trial stayed heavier throughout the pre-mating period, which could explain the lack of an effect of the imposed dietary treatments. As growth rate and liveweight have been shown to influence ovarian development (Prunier *et al.* 1987) and follicle development (van Wettere *et al.* 2011), it is possible that the heavier gilts may have been more sexual mature prior to the pre-mating treatment, potentially explaining their increased ovulation rate.

In conclusion, this study has identified that there is no significant interaction between the pre-mating and post-mating feed intake on embryo survival. The pre-mating feed intake had little effect on embryo survival, however low feeding levels post-mating decreased embryo survival when compared to higher post-mating feeding levels. This demonstrates the importance of ensuring sows receive adequate, post-mating nutrition, and may require optimisation of feeding regimes in group housing systems throughout this stage of gestation. This study has provided further insight into the effect of a restricted feed intake during early gestation on embryo survival and into the interaction between the pre-mating and post-mating feed intake.

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