Determining piglet survival

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The sow, piglet and their environment interact together to determine whether or not a piglet will survive to weaning. The physiology of the mother and offspring, as well as the synchronized expression of their appropriate behaviours, is integral to piglet survival and both are governed by genetic and environmental components. This review discusses the multifaceted nature of piglet survival, concentrating on the environmental factors that pre-dispose prenatal (stillborn) and postnatal death and the progress made towards understanding and decreasing piglet mortality.

Introduction

A certain amount of piglet mortality might be considered an inevitable event. A percentage of piglets are expected to die before weaning (10-20%, Edwards 2002) and may reflect a form of natural selection implemented by the sow whereby only the fittest offspring survive in challenging conditions. The evolutionary strategy adopted by the sow is one of over-producing; a form of parental optimism where production of numerous neonates allows replacement offspring in the event of members of the litter dying (Mock & Forbes 1995; Forbes & Mock 1998) and prepares for an unpredictable lactation phase, where resources may be plentiful or sparse. The often disproportionate provision of resources (i.e. milk) to the litter, further results in intense sibling rivalry, likened to avian facultative siblicide (Fraser *et al.* 1995), and increased mortality. Over-supplying offspring and providing limited resources is evident long before birth; with high ovulation rates but 30-50% of released ova not surviving gestation in the finite uterine space (Pope 1994; Geisert & Schmitt 2002).

In a polytocous species, such as the pig, embryo mortality, parental optimism and "siblicide" are considered normal. The limited success of attempts to reduce piglet mortality under domestic conditions may reflect these hard-wired evolutionary strategies. Continued selection pressure for hyper-prolificacy has further increased the challenge of piglet survival. Understanding the causes of mortality and the risk factors that predispose different types of death is crucial to focus solutions on both environmental and biological elements.

What are the main causes of piglet mortality?

Pre-weaning mortality (PWM) of live-born piglets is, on average, 11.5% (BPEX 2011), which varies between countries (Figure 1). With the inclusion of those piglets that are born dead (approximately 8% - Leenhouwers *et al.* 1999), total mortality averages 16-20% (Knol *et al.* 2002a; BPEX 2012).

Attempts to decrease piglet mortality have mainly been directed at the farrowing environment and husbandry procedures. The farrowing crate was introduced in the 1960s (Robertson *et al.* 1966) to decrease piglet over-lays by restricting sow movements, and to improve ease of



Fig. 1 Relationship between numbers born alive and pre-weaning mortality (percentage of piglets that are born alive but die pre-weaning) for selected countries in the European Union, Canada and Brazil. Source – Interpig 2010 (BPEX 2011).

management (Edwards & Fraser 1997). However, despite efforts to better understand and reduce piglet mortality, average losses have not changed significantly in recent years and, lately, there is a trend for increases (Rutherford *et al.* 2013), most likely as a result of increased prolificacy (Figure 1). There have been a number of comprehensive reviews articles relating to piglet mortality (English & Smith 1975; Dyck & Swierstra 1987; Edwards *et al.* 1994; English & Edwards 1996; Marchant *et al.* 2000; Edwards 2002; Lay *et al.* 2002; Mellor & Stafford 2004), with a great deal of consistency regarding the main causes of death and implicating the first 72 hours of life as the critical period for survival. Dyck and Swierstra (1987) identified eight specific causes of death, but the main three are stillbirths, crushing by the sow and starvation.

What pre-disposes stillborn mortality?

The prenatal period includes the period before farrowing and the parturition process itself. Mortality *in utero* during the embryo elongation phase can be followed by post implantation losses in crowded uterine conditions (Foxcroft *et al.* 2006). For the fetuses that survive to full term, there remain risks that can lead to stillbirth. There are two main types of stillbirth; type 1 includes the fully or partly mummified piglets that have died pre-partum and are generally attributed to intrauterine infection (Alonso-Spilsbury *et al.* 2005) as well as intrauterine crowding (Vallet *et al.* 2002). Type 2 stillbirths are more common, with non-infectious aetiologies (summarised in Figure 2). The major influencing factors are prolonged duration of farrowing, delivery in the last third of the birth order, premature rupture of the umbilical cord, sow behavioural and physiological characteristics, including parity and blood haemoglobin concentration of less than 9 g/100 ml (Randall 1972a,b; Fahmy & Friend 1981; Zaleski & Hacker 1993; van Rens & van der Lende 2004; van Djik *et al.* 2001; Alonso-Spilsbury *et al.* 2005;



Fig. 2 Pre-disposing events of still-born mortality

Mota-Rojas et *al.* 2005; van Dijk *et al.* 2008), or a less viable piglet with poor survival chances post-partum. The predisposing factors are not independent, for example prolonged farrowing duration is influenced by increased litter size (van Rens & van der Lende 2004; Andersen *et al.* 2011), sow stress associated with fatigue (van Kempen 2007; Vallet *et al.* 2010), restrictive farrowing environments (Oliverio *et al.* 2006) and high ambient temperatures (Vanderhaeghe *et al.* 2010).

The pharmacological induction of parturition, to synchronise and supervise farrowings and therefore reduce PWM (Černe & Jöchle 1981), can be counter-productive because of increased birthing complications and stillbirths (Mota-Rojas et al. 2002). The timing of induction is critical, since late fetal development and maturation is a predisposing factor in survival (Randall 1972b; van der Lende et al. 2001) and the general consensus is not to induce before d113 of gestation (see Kirkden et al. 2013 for review) and not in gilts as service dates are rarely accurate. In the days preceding farrowing, the fetus experiences an increase in growth rate (Biensen et al. 1998) and development, with final physiological preparations for extra-uterine life, particularly lung maturation. Premature induction of birth may therefore result in a compromised neonate. Oxytocin administration, designed to aid farrowing progress in situations of fatigue, can increase fetal asphyxia by increasing the uterine muscle contractions, impeding gaseous exchange between mother and fetus and increasing the chances of umbilical occlusion (Alonso-Splisbury et al. 2004, 2005; Mota-Rojas et al. 2002, 2006). Administration of oxytocin in these studies resulted in increased piglet bradycardia and meconium staining, with severe acidosis and compromised survival. Thus the mis-use of drugs designed to improve farrowing outcomes is an important risk factor in stillborn mortality.

Maternal condition - genotype, nutrition, parity and history

The mother plays a crucial role in fetal survival during the gestation phase, mediated through uterine space and placental quality. Van Dijk *et al.* (2005) reported breed effects on stillborn

mortality and found that an increased duration of expulsion was significantly associated with increased litter size, increased number of stillborns per litter and decreased length of gestation (independent of litter size).

Feeding during gestation requires a fine balance, with both under- and over-nutrition affecting many developmental processes. Over-feeding of the sow during the last third of pregnancy can indirectly compromise piglets by affecting the mother's farrowing ability; if piglets become too big, the inter-birth interval will increase, as will the potential for umbilical cord occlusion and stillbirth (Leenhouwers *et al.* 2001). The immediate impact on the fetus if the sow is under-fed during gestation involves negative effects on placental size, fetal growth and the deposition of fetal fat reserves for use after birth (Wu *et al.* 2004). Malnutrition in gestation can lead to impaired mammary gland development (Head & Williams 1992; Kim *et al.* 1999), poor lactational output and may affect the future reproductive capabilities of the offspring (Kerr & Cameron 1995; O'Dowd *et al.* 1997). There are even considered to be intergenerational influences (Gluckman & Hanson 2004), as maternal metabolic function may itself have been programmed when the mother was a fetus.

Young sows of lean-genotype are more likely to suffer excessive depletion of body protein reserves during lactation which will affect neonatal growth rates, as well as the subsequent, developing litter (Edwards 1998). Sows of advanced parity give birth to a higher incidence of stillborn piglets (Randall & Penny 1970; Leenhouwers *et al.* 2003) and farmers report that sows giving birth to dead piglets often consistently produce stillborn piglets in subsequent litters, thus highlighting a genetic component to this trait (Roehe *et al.* 2010).

Placental traits and piglet growth characteristics

Suidae have a non-invasive epitheliochorial placentation, which is considered to be relatively inefficient and diffuse. Placental efficiency (birth weight/placental weight (PE)) was studied in Meishans (Wilson *et al.* 1998) which are able to support a greater litter size to term than their occidental counterparts (Large White, Yorkshire, Landrace - Lee *et al.* 1995). Wilson *et al.* (1998) reported that this was due to the decreased pre-implantation growth rate and oestrogen biosynthetic activity of the Meishan conceptus compared with occidental breeds, which allowed more conceptuses to survive beyond day 18 of gestation. Furthermore, it is hypothesised that Meishan conceptuses are smaller and remain small throughout gestation, and that their placentas are smaller and more efficient. Both large and small fetuses can develop on either a small or large placenta, with more efficient placentas being generally smaller and more vascularised.

Placental insufficiency can result in a chain of events that will compromise fetal and neonatal survival. It is a major cause of intrauterine growth restriction/retardation (IUGR) and, since piglet birth weight is often heralded as the most important survival indicator (e.g. Kerr & Cameron 1995; Roehe & Kalm 2000), such prenatal insults can result in a stillborn piglet or a live-born piglet with low viability and compromised survival. Recently the importance of birth weight *per se* with regard to prenatal survival has been questioned; Baxter *et al.* (2008) found that stillborn piglets were disproportionately long and thin, with lower ponderal index (PI) and body mass index (BMI) (indicative of IUGR) compared with surviving littermates, and found that PI and BMI were more predictive of whether or not a piglet would be stillborn than birth weight alone. These results have since been replicated (Pedersen *et al.* 2011a; Rootwelt *et al.* 2012). The quadratic relationship between birth weight and stillborn mortality (Canario *et al.* 2006) shows that, at least for prenatal survival, birth weight is not a simple predictor of stillbirth. Very small piglets and "giant" piglets are equally at risk, with heavier piglets often being more hypoxic than lighter littermates as a result of birthing difficulties (Trujillo-Ortega *et al.* 2007).

Baxter et al. (2008) associated their gross measure of IUGR (i.e. BMI and PI) with lower total placental areolae number and areolae density, placental traits key in the transfer of nutrients,

particularly protein, between mother and fetus. They found a further relationship between areolae traits and piglet vitality at birth, and Rootwelt *et al.* (2012) also associated placental area with piglet vitality, thus emphasising the influence of placental quality on postnatal viability.

What predisposes live-born mortality?

Survival of the newborn piglet depends on its ability to overcome the physiological challenges associated with the extra-uterine environment. These include behavioural and physiological adaptations such as the ability to adequately thermoregulate, the ability to find and defend a functional teat, suckle and absorb vital nutrients from the mother's colostrum as well as respond to maternal cues. The multifactorial nature of live-born piglet mortality is summarised in Figure 3.



Fig 3. Pre-disposing events of live-born mortality and the interactive events occurring in the chilling-starvation-overlying-disease complex. Adapted from Edwards 2002

Chilling/Hypothermia

Hypothermia is considered, directly or indirectly, to be responsible for more deaths than crushing, starvation, disease or low viability (Curtis 1970). However, without continuous monitoring of rectal temperatures it cannot be determined, and thus the most easily discernible cause of death, crushing by the sow, is often over recorded (Vaillancourt *et al.* 1990; Christensen & Svensmark 1997).

From the moment the piglet is born it suffers from a reduction of ambient temperature (approximately a 15-20°C drop – Curtis 1970; Herpin *et al.* 2002), such that its lower critical temperature (approximately 34°C – Mount 1968), is not achieved. The newborn piglet therefore rapidly loses heat via different processes (reviewed by Curtis 1970). These heat loss processes are affected by physical, behavioural and environmental factors. The size of the piglet affects

the rate of heat loss, with smaller piglets having a proportionally larger surface area from which heat is transferred via convection (Curtis 1970; Herpin et al. 2002). Higher air velocity and a larger temperature gradient will also increase convective heat loss (Mount 1964). Early work on conductive heat loss (Mount 1967; reviewed by Curtis 1970) showed that piglets in contact with a concrete floor lost 40% more heat than those in contact with bedding material (i.e. 2.5 cm straw). Investigations in "natural" farrowing conditions show that nests are robust to climatic extremes as a result of reduction in losses by conduction, convection and radiation (Algers & lensen 1990; Baxter et al. 2009). Radiative heat loss is affected by the temperature gradient, the surface area of the surrounding surfaces and the distance between the piglet and these surfaces, and will not be over-come by increasing ambient temperature. Finally, evaporative heat loss is particularly problematic, because it drains energy to dissipate placental fluids as water vapour. All piglets will experience this rapid cooling since the sow will not remove placental membranes by licking, as do other livestock species. Again, low birth weight will increase relative evaporation losses via the increased relative surface area, as will increased air velocity (e.g. drafts from ventilation systems - Curtis 1972) and low ambient air pressure. Once the placental fluids have evaporated or been physically removed by stockperson intervention or friction with other piglets or bedding material, evaporative heat loss is still experienced from mucosal surfaces via respiration.

The metabolic capability of the neonate affects the extent to which heat loss associated with the extra-uterine temperature drop impacts upon the piglet. If environmental conditions are inadequate to prevent the ambient temperature dropping below 34°C, then secondary defences are deployed via an increase in metabolic rate and shivering thermogenesis. Both these process are likely, given that heating the environment to 34°C would impact on the sow's thermal comfort zone (Mount 1968), which ranges from 12-22°C (Black et al. 1993), and thus is markedly different from that of the newborn piglet. Initiation and sustainability of the thermogenic response to cold (i.e. a continuing increase in metabolic rate) is dependent on intake and metabolism of colostrum (Herpin et al. 1994). However, other factors also contribute, directly or indirectly, to hypothermia risk. If a fetus has a suboptimal placenta this will cause chronic fetal hypoxaemia (Rees et al. 1998). The observed elevated lactate is associated with anaerobic metabolism and a decreased haematocrit reflects the decreased percentage of red blood cells in the plasma resulting from the reduced oxygen supply. Acute asphyxia at birth may also cause metabolic acidosis, hyperlactaemia, hypoxia and meconium aspiration syndrome (Herpin et al. 1996; Alonso-Spilsbury et al. 2005). Herpin et al. (1996) found a relationship between asphyxia at birth and reduced neonatal vitality; piglets were slower to reach the udder and had a lower rectal temperature 24 hours after birth, as well as reduced growth rate and survival over the first 10 days post-partum. Restriction of the oxygen supply during fetal development has possible consequences for central nervous system (CNS) function. Damage to the fetal CNS can impair sucking and locomotor activities in pigs (Herpin et al. 1996) and impairs thermoregulation in the neonate (Stanton et al. 1973).

The typical, domestic piglet is born with very little adipose tissue, no brown fat (Herpin et al. 2002) and little pelage with which to assist thermoregulation. It must take in colostrum as soon as possible to prevent catabolism of its skeletal muscle to fuel heat production. Thermoregulation requires the co-ordinated maturation of various organs and functions that were unnecessary for survival in utero (Herpin & Le Dividich 1995). If a piglet is unable to produce heat, it may be a result of an immature thermogenic mechanism arising from, for example, premature birth and/or reduced thyroidal and adrenocorticol support for postnatal metabolic activity (Mellor and Stafford 2004). Intensive genetic selection for lean tissue growth rate has resulted in piglets with reduced physiological maturity for their size at birth, and thus poor thermoregulatory abilities (Herpin et al. 1993).

Starvation

Starvation occurs when a piglet fails to ingest or metabolise colostrum, and subsequently milk, from the sow. Post-mortem analysis of dead piglets, presumed crushed, often shows the stomach to be empty, indicating that starvation is part of the mortality complex involving hypothermia and crushing (Edwards 2002; Pedersen et al. 2011b). Colostrum is important for both energy balance and immune protection (see later section). Lactational output of the sow is not the only factor determining how much milk the piglets receive. Piglets will fight to gain teat access and to maintain teat fidelity and, if they are unable to perform optimal udder massaging and suckling behaviours, teat function may be impaired. Piglets failing to establish teat fidelity grow more slowly (De Passillé et al. 1988) and get-by on opportunistic suckling or often starve. De Passillé and Rushen (1989) found that heavier piglets, born earlier in the birth order, won more teat disputes, established teat fidelity quicker, suckled more frequently and ultimately were at a distinct advantage over less vigourous littermates. However piglet vigour is not necessarily correlated with weight; using a sophisticated measure of vigour based on the strength and persistence with which the newborn piglet manipulated an artificial teat linked to a computer registration system, Baxter et al (2008) demonstrated that a small yet vigourous piglet could survive equally well as its larger conspecifics. However, even if a piglet possesses such vigour, a large litter size, where piglets outnumber functional teats, will require managerial intervention to prevent starvation (Baxter et al. 2013). Sow physiology and behaviour will also influence the efficiency with which piglets suckle a functional teat; reduced lactational output, poor teat quality (Fraser and Lin 1984) and poor maternal behaviour (Andersen et al. 2005) and udder exposure (Pedersen et al. 2011c) will impact negatively on the nutritional status of the piglet.

Crushing

Crushing is thought to account for the majority of neonatal deaths and, given its importance, is a well-researched area of the hypothermia-starvation-crushing complex. There is a trade-off between the innate need of the piglet to be at the warm udder, gaining valuable colostrum and establishing teat fidelity, against the risk of being crushed by the sow. Weary *et al.* (1996) concluded that crushings are partly the result of the nutritional challenge facing piglets; a piglet with slow weight gain spent more time in risky areas underneath its sitting or standing mother. If the piglet's energy reserves are low, it will also be too weak to escape a moving sow. Sow behaviours influencing crushing risk include lack of piglet directed pre-lying behaviour, the frequency and nature of posture changes and failure to rise in response to a trapped piglet (Marchant *et al.* 2001; Thodberg *et al.* 2002; Andersen *et al.* 2005). The latter aspect will be affected by the sow's condition, which may be linked with parity, as well as her inherent responsiveness and her farrowing environment. Older parity sows may experience increased leg weakness impeding posture changes (Damm *et al.* 2005; Pedersen *et al.* 2006), whilst the enforced restraint of a farrowing crate will limit the effectiveness of response to a crushed piglet.

The farrowing environment, to a certain extent, dictates the interaction between mother and young. In a loose-housed system, piglet-directed pre-lying behaviour is associated with good maternal ability. Sows will paw the ground or substrate, or make nose-to-piglet contact before lying down (Wechsler & Weber 2007). In choice experiments (Herskin *et al.* 1998), provision of bedding and substrate improved maternal behaviour by reducing the number of posture changes and therefore reduced crushing risk.

The sow's temperament is also influential (Marchant et al. 2001). There is individual variation regarding maternal ability and sows who are deemed "crushers" behave differently to "noncrushers" (Andersen et al. 2005; Jarvis et al. 2005). The consistency of behavioural patterns within individuals, and the high variation within populations, suggests a possibility of selecting for "non-crushers" (Grandison et al. 2003). The piglet's behaviour that influence crushing often occur as a result of physiological challenges such as hypoxia, starvation or hypothermia, which may increase lethargy. Since reduction of heat loss at birth is mainly achieved by behavioural adjustments, including huddling and postural adaptations (Hrupka et al. 2000), where a piglet chooses to lie in the nest site may be crucial for its survival. For a piglet with low viability, facing demanding physiological challenges, it will be just as dangerous to lie away from the udder and siblings, since although the risk of crushing is reduced; the risks of starvation and hypothermia become much greater.

Disease, infection and injury

Neonatal diseases result from the interaction of a multitude of factors (Martineau *et al.* 1995). The epitheliochorial nature of the porcine placenta means that the newborn piglet must acquire maternal immunoglobulins from ingesting colostrum for passive immune protection (Rooke & Bland 2002). The immune system of the piglet itself does not become fully developed until at least weaning (Gaskins & Kelly 1995). Failure to ingest sufficient colostrum soon after birth results in sub-optimal transfer of maternal immunoglobulins to the neonate, and increased susceptibility to disease (Gaskin & Kelly 1995; Rooke & Bland 2002). It has been suggested that colostrum intake below 200g per piglet in the first 24 hours of life is a significant risk factor for piglet mortality (Devillers *et al* 2011). Issues to do with piglet colostrum intake have been thoroughly reviewed by Quesnel and colleagues (2012).

The physiological maturity of the newborn will affect its ability to maintain health and survive. Immature organ development will impact upon the piglet's ability to process any milk it obtains and there is a finite amount of time before gut closure commences (approximately 48 h) when it is important for the piglet to obtain and process colostrum (Cranwell 1995). Getting to the udder, commanding a functional teat and suckling colostrum quickly not only aids thermoregulation and the acquisition of immunoglobulins and nutrients, but also aids gut closure. There are two major windows of opportunity for pathogens to enter the piglet's systemic circulation. The first is within the first 24 h of life and is influenced by delayed colostrum intake which can cause subsequent delay in gut closure. The second opportunity is between the time of declining antibody levels in the sow's milk and the transition from passive to active immunity in the piglet (Gaskins & Kelley 1995). Poor management practice with regard to hygiene and disease treatment will be major determinants of the risk of neonatal diseases and infections becoming fatal. Injuries may be acquired by the piglets as a result of over-lying or savaging by the mother and, if they are not immediately fatal, they may result in infection and a later death. It is also possible for piglets to sustain wounds from their siblings and/or abrasive floor surfaces during the competitive scrambling for the teat at milk let-down (Fraser 1975; Mouttotou & Green 1999; Drake et al 2008).

The influence of farrowing system

The farrowing system influences the prevalence of certain types of mortality. Riart et *al.* (2000) conducted post-mortem analysis in indoor and outdoor systems and found crushing to account for approximately 20% of total mortality in the indoor, crated system and 45% in the outdoor system. Starvation accounted for approximately 15% indoors and less than 10% outdoors. It is often reported that crushing is more prevalent in loose-housed or outdoor systems (alternative systems) where the sow is free to move about during farrowing and lactation, compared with the conventional system where the sow is confined in a crate (Marchant et *al.* 2000). However, this does not always equate to higher total mortality in these alternative systems and our recent

review comparing performance suggests that mortality rates can be relatively similar in both conventional and well designed alternative systems (Baxter et al. 2012a).

Higher levels of mis-mothering (e.g. savaging) are reported (Lawrence et al. 1994) when sows are confined in crates whereas this behaviour is rarely seen in alternative systems. Lawrence et al. (1994) found elevated cortisol levels in crated sows and hypothesised this was a consequence of the inability to perform natural behaviours and a likely factor contributing to increased savaging in restrictive environments (Lawrence et al. 1994; McLean et al. 1998; Jarvis et al. 1998; Jarvis et al. 2004).

The influence of piglet gender

There is evidence that female piglets have a greater chance of surviving from birth to weaning than males (Lay et al. 2002; Baxter et al. 2012b). Baxter et al. (2012b) demonstrated that, despite being born heavier than females, male piglets could not sustain this advantage and showed impaired thermoregulation compared to females, with significantly lower rectal temperatures at 24 h old. Moreover, piglets from male-biased litters showed reduced thermoregulatory abilities, were slower to suckle colostrum and were more likely to die from disease-related causes. These results suggest male-biased mortality, reflecting an intrinsic, size-related susceptibility to causal factors. This may be masked when competition for resources favours the larger, more dominant individuals but, where individual resources are sparse the effects may be more visible.

The influence of birth weight and litter size

Throughout the review the importance of birth weight and the disadvantages of large litter size have been alluded to, though they are very difficult to discuss separately, as they are not mutually exclusive. Roehe and Kalm (2000) reported 40% pre-weaning mortality in pigs with a birth weight of lower than 1 kg, 15% between 1-1.2 kg and only 7% when birth weight was above 1.6 kg. Within-litter birth weight variation is potentially more important than individual birth weight. High neonatal-weight variation generally results in lower survival and more variable weaning weights (Roehe 1999; Milligan *et al.* 2002; Quiniou *et al.* 2002). This is not an area new to the consideration of piglet survival (English & Smith 1975), yet is still one to effectively be addressed.

In the course of domestication, selective breeding has delivered more than 100% increase in litter size. However, the associated mortality levels are becoming unacceptably high with negative effects on other survival and production traits. Quiniou *et al.* (2002) have shown that increasing litter size from ≤ 11 to ≥ 16 resulted in a reduction of mean birth weight from 1.59 Kg to 1.26 Kg, corresponding to a mean decrease of 35 g for each extra piglet born. Recent reviews by Rutherford *et al.* (2013) and Baxter *et al.* (2013) have discussed the biological and managerial challenges of hyper-prolificacy, which will therefore not be in covered in this review. However, it is clear that improving piglet survival remains a significant challenge with increased selection pressure for prolificacy.

Solutions - How can we improve piglet survival?

A viable piglet is one that will adapt to the extra-uterine environment and survive the pre-weaning period. There are certain aspects of the piglet's behaviour and physiology which potentially aid this task. These include an optimum birth weight, favourable litter size, physiologically mature organ functions maintaining homeostasis and the ability to behaviourally adapt to obtain vital nutrients from the sow. Augmentation of existing environmental solutions and nutritional

programmes, as well as progression in genetic selection strategies which incorporate survivability within breeding goals, are possible ways of tackling piglet mortality.

Environmental and nutritional factors

Previous research to reduce piglet mortality was centred almost entirely around alterations to the farrowing environment. Supervision at the time of parturition (Holyoake *et al.* 1995; Andersen *et al.* 2009), increased control over the macro- and microclimate and nest area (Schmid 1994) and, of course, the introduction of the farrowing crate (Robertson *et al.* 1966) are some examples of tools put in place to improve survival. The various accommodation systems for farrowing and lactation and the welfare issues surrounding them have already been reviewed (Edwards & Fraser 1997; Barnett *et al.* 2001; Wechsler & Weber 2007; Baxter *et al.* 2012a). Adjustments to farrowing systems should consider solutions that optimise both sow and piglet welfare.

Nutritional interventions to improve embryo quality and subsequent birth weight and uniformity have included use of fermentable ingredients in sow diets prior to breeding (Van den Brand et al. 2009), and essential fatty acid supplementation in late gestation to increase piglet vitality (Rooke et al. 2001). Campos et al. (2012) offers a recent review on these offspring benefits, whilst Meunier-Salaün et al. (2001) and de Leeuw et al. (2008) discuss the influence of nutritional interventions on sow welfare.

To ensure that all piglets ingest vital colostrum soon after birth, targeted inputs by stockpeople can assist landmark behaviours (i.e. improve latency to reach the udder and suck colostrum). This is of particular importance when litter size exceeds functional teat number and when a greater number of low vitality, growth-retarded piglets require intervention (see Baxter *et al.* 2013 for managerial strategies). Improving maternal behaviour and farrowing progression by reducing stress in the periparturient sow can also indirectly assist the piglet. Farrowing duration, and risk of hypoxia, may be reduced by ensuring appropriate sow condition, minimising heat stress and providing enrichment to allow nest-building behaviour and reduce frustration (Thodberg *et al.* 1999; Jarvis *et al.* 2001, 2002; Damm *et al.* 2003, 2005). Providing substrate will not only facilitate improved maternal behaviour (Herskin *et al.* 1998) but will provide a microclimate for newborn piglets.

Selection for improved survival

Adjusting selection criteria to include neonatal survival, in addition to number born, is a more sustainable strategy and one that has achieved success in improving piglet survival rates (Roehe et al., 2009, 2010). The Danish pig industry, renowned for its success in increasing litter size, has recognized the accompanying significant increase in mortality which occurred (5% increase in total pre-weaning mortality). In 2004 it changed its selection criterion from total born to 'live piglets at day 5' (LP5) (Su et al. 2007) and, although mortality is still high, this has been stabilised with the net result of an increase of 2.3 pigs weaned/litter. This may reflect successful management of the surplus piglets, or indicate that selecting for survival may not result in as many compromised piglets (e.g. pathologically growth retarded) as selecting for number born. Reducing intra-litter variability, particularly with respect to birth weight, is an additional important breeding goal discussed by numerous authors (Rydhmer 2000; Knol et al. 2002a,b; Damgaard et al. 2003; Huby et al. 2003). Selecting for improved placental efficiency is another potential strategy to improve piglet outcomes (van Rens et al. 2005). Furthermore, breeding for improved maternal behaviour (Grandison 2005; Gade et al. 2007; Baxter et al. 2011) and investigating strategies for breeding a more robust piglet both have potential to reduce mortality.

Conclusions

Piglet mortality is multifaceted in nature and, as a result, finding solutions requires addressing all aspects of the problem. Little progress has been made over the last two decades in reducing piglet mortality. Nutritional interventions for the sow during gestation and lactation have scope to enhance piglet outcomes. Postnatal managerial interventions to assist vulnerable neonates must continue to be a focus for stockpeople, particularly as prolificacy increases. Coupling these environmental and nutritional interventions with a balanced selection programme offers the best chances of success in improving piglet survival.

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