Hypothermia in newly born piglets: Mechanisms of thermoregulation and pathophysiology of death

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Abstract Mortality in piglets during the perinatal period, especially the first days after birth, is frequently caused by non-infectious conditions, such as hypoglucemia or low birth weight, which can be associated with hypothermia experienced at birth. The thermal stability of newborn piglets is a fundamental aspect of neonatal care, so maintaining a constant, ideal temperature will substantially reduce newborn mortality. Species-specific characteristics, such as a limited capacity for thermoregulation, low energy reserves, a lack of brown adipose tissue (BAT) (-, and environmental conditions that are adverse for the piglet around the time of birth, including the absence of a microclimate, all of them contribute to difficulties in reaching thermal homeostasis in the first hours post-birth. Shivering thermogenesis and behavioral modifications to regulate body temperature through innate mechanisms allow animals to reduce their energy expenditures. Some body postures are effective in reducing contact with the floor and also nestling are useful to avoid heat loss, and also decreases heat dissipation. Achieving optimal development of thermoregulation is a challenge that newborns must confront to successfully adapt to extrauterine life. The objectives of this review, are to discuss the adverse factors that can lead to a death event due to hypothermia by analyzing the thermoregulation mechanisms at the central and cutaneous levels, also to analyze the harmful impacts that surviving neonate piglets confront in an unfavorable thermal environment, and to describe the pathophysiological mechanisms of death caused by hypothermia.

Keywords brown adipose tissue, cold stress, colostrum consumption, neonate, perinatal death, shivering thermogenesis

1. Introduction

Perinatal mortality continues to be one of the principal problems and concerns of the pork industry (Mota-Rojas 1996; Mota-Rojas and Ramírez-Neecochea; 1996; Mota-Rojas et al., 2002; Mota-Rojas et al., 2006; Houška et al 2010; Martínez-Burnes et al 2019), one closely-related to issues of animal welfare (Mota-Rojas et al 2011, 2012ab). Piglets can die due to a broad range of causes, but neonatal losses attributable to cold-induced stress are rarely registered as such, although the hypothermia that results from these events can lead to starvation, crushing, and/or disease (Curtis 1974; Kelley 1985; English 1993; Herpin and Le Dividich 1995; Mota-Rojas et al 2005abc; Jensen et al 2011). Hypothermia can be a significant cause of death in neonate piglets, and although this condition is not infectious, it is considered an important factor in death on swine farms, as it may go undetected due to several natural causes (Mount 1963; Mota-Rojas et al 2011, 2012ab). Because the newly born piglet has an immature thermoregulating center, homeostasis in its body temperature is affected within the first hours post-birth due, primarily, to the evaporation of placental fluids (Muns et al 2016; Mota-Rojas et al 2016). According to Nuntapaitoon and Tummaruk (2015), the newly born piglet is covered by approximately 23 g of amniotic fluid for each kilogram of live weight at birth, and about 50% of these fluids evaporate during the first 5-30 min after birth (Kammersgaard et al 2013; Muns et al 2016). For this reason, piglets experience an abrupt temperature decrease during the first hours post-birth with changes that begin from the moment of expulsion; that is, the transition from a thermoneutral, intrauterine environment to extrauterine life, an event that is accompanied by a severe reduction of the environmental temperature (ET) (approximately 15-20 °C in farrowing pens) at a very early stage of life (Herpin et al 2002; Vassdal et al 2011).

Besides, heat loss in newly born piglets is aggravated as they are born without brown adipose tissue (BAT) and with very little adipose tissue, both of which serve as insulators (Herpin et al 2002). This condition means that their only
resource for producing heat and raising their core body temperature (CBT) consists of mobilizing energy reserves present in the form of glycogen and fat and, as a last resort, catabolizing skeletal muscle. Of course, the environmental conditions of the installations and handling around the time of birth also drastically affect the thermoregulating capacity of newborn piglets, with consequences for their survival and growth (Muns 2013). In this regard, weaker piglets are usually unable to compete successfully for colostrum and milk, and they may become hypothermic (Swendsen et al 1986). Since hypothermia and the lack of nutrition weaken them even more, problems of orientation and locomotion often follow, increasing the risk of crushing (DeRoth and Downie 1976; Swendsen et al 1986). The decrease in ET that newborn piglets experience is likely the most immediate danger they confront upon leaving the intrauterine environment (Mota-Rojas et al 2008, 2011, 2016, 2018). It is important to keep in mind, as well, that piglets are born with a high surface/volume ratio due to their small size, and that they have little hair and very little adipose tissue for use as an energy source (also, no BAT is present in them). Another condition that exacerbate this situation is that the neonate’s skin is moist due to amniotic fluid (Herpin et al 2002). Hypothermia in piglets compromises various organs and systems that require additional study to fully understand the physiopathological mechanisms that lead to the death of newborn piglets by hypothermia. The objective of this review, is to discuss the adverse factors that can produce death by hypothermia in newly born piglets. To this end, we caused by non-infectious conditions, such as hypoglucleremia or low birth weight, which can be associated with hypothermia experienced at birth, analyzed thermoregulation’s mechanisms at the central and cutaneous levels, and the harmful effects that surviving newborn piglets face from an unfavorable thermal environment.

2. Thermal balance and adaptation to extrauterine life

The – newly born piglets adaptation to extrauterine life constitutes a considerable challenge for its survival and postnatal development (Baxter et al 2008) as almost immediately at birth, they experience temperatures markedly below their thermoneutral zone (Muns et al 2016). It is estimated that neonates can lose over 2 °C of body temperature (BT) between birth and their first ingestion of food (colostrum) (Tuchscherer et al 2000; Malmkvist et al 2006; Baxter et al 2008). During gestation, fetuses live at a uterine temperature that ranges from 38-40 °C. However, at birth neonates suffer a drastic environmental change as they are exposed to an ET around only 20-22 °C (the temperature that coincides with the sow’s thermoneutral zone) which makes them more vulnerable to stress-induced by cold (Berthon et al 1993; Tuchscherer et al 2000; Malmkvist et al 2006, 2009). Furthermore, a series of factors that include the lack of subcutaneous adipose tissue (-2%), low glycogen reserves (Herpin et al 2002; Le Dividich et al 2005), incomplete thermoregulation, reduced insulation (Muns et al 2016), heat loss by evaporation (due to moist skin), conduction (contact with colder surfaces), radiation (scarce hair), convection (airflow), and rapid heat dissipation due to their high surface/volume ratio attributable to their size (Theil et al 2014), resulting in many piglets suffering hypothermia in the first 24 hours post-birth (Mota-Rojas et al 2008, 2011; Baxter et al 2009; Shankar et al 2009; Pedersen et al 2011) (Figure 1). One technique to evaluate farm and companion animals’ surface thermal dynamics is the use of infrared thermography (IRT) (Mota-Rojas et al 2020ab; Casas-Alvarad et al 2020; Bertoni et al 2020ab). The thermal balance of newborn piglets is of primordial importance for neonatal care, so achieving and maintaining an ideal body temperature will significantly reduce perinatal mortality (Mota-Rojas et al 2008, 2011) (Figure 1). The use of IRT in both veterinary and human medicine to evaluate heat loss and gain in different corporal regions, and assess microcirculatory changes at the vascular level has proven to be effective (Kils et al 2017; Bruins et al 2018; Huggins et al 2018; Mota-Rojas et al 2020ac; Casas-Alvarad et al 2020; Bertoni et al 2020bc).

When the CBT of newborn piglets decreases to a level less than or equal to 35°C due to the exposure to a cold environment, the result is the condition called postnatal hypothermia (Haverkamp et al 2018; Muns et al 2016). Sosnowski et al (2015) pointed out that hypothermia can occur even when all thermoregulation mechanisms are totally functional, due to an organism’s prolonged exposure to cold that impedes it from taking conscious defensive measures. Lossec et al (1998), in turn, reported that hypothermia occurs naturally after birth in most mammalian neonates but that both the decrease in body temperature - and the time required for recovery vary widely among different organisms. The reduction in BT accompanied by a deficit in energy ingestion are factors that weaken newborn piglets even more and, consequently, increases the risk of neonatal mortality (Alonso-Spilsbury et al 2007; Mota-Rojas et al 2011; Mota-Rojas et al 2016, 2018). Piglet survival correlates with the degree and duration of the postnatal hypothermic condition (Tuchscherer et al 2000), so newborns must adapt quickly to extrauterine life through autonomous (e.g. thermogenesis) and behavioral mechanisms for heat conservation (Kammersgaard et al 2011).

When an organism experiences slight cooling, mechanisms designed to conserve heat begin to act. However, newborn hypothermia uses up glucose reserves (in the form of glycogen) and oxygen to produce heat (Le Dividich et al 2005), which constitutes an enormous energy cost for piglets. In contrast to other mammals, – newly born piglets, especially, have a limited thermoregulation capacity during the first hours of life. Thermal homeostasis is a biological priority for all endothermal species. In the case of piglets and up to 24 hours post-birth, a temperature range of 38-39°C indicates thermal homeostasis (Berthon et al 1993; Herpin et al 1994).

Thermoregulation in pigs, as in other mammals, is a process orchestrated by the central nervous system (CNS)
with the collaboration of the peripheral nervous system (PNS), through a series of autonomous and behavioral mechanisms that actively balance the production and dissipation of heat (Morrison 2016). However, the thermoregulatory response’s complexity depends on the anatomical, physiological, and behavioral characteristics of a given species (Angilletta et al 2019; Tan and Knight 2018). When the newborn piglet’s body temperature decreases, signals from the peripheral (cutaneous) and central (spinal cord, cerebral, visceral) thermoreceptors reach the preoptic area (POA) of the hypothalamus - through afferent pathways which process all the sensory information and activate thermoregulatory responses, also through afferent pathways (Figure 2). The POA is a thermosensitive area that regulates responses to different temperature changes and controls thermal sensitivity in the brain (Tan and Knight 2018).

At birth, the piglet passes from a dependent intrauterine environment to one that is independent. Hence, it must begin to regulate its body temperature to survive (Close 1992). To activate thermoregulating responses to the thermal stress caused by cold, the piglets’ cutaneous (type Aβ, Aδ, and C nerve endings) and visceral thermoreceptors perceive both absolute and relative temperature changes. These thermal stimuli cause the depolarization of thermoreceptors to generate transduction (Tan and Knight 2018). Cutaneous thermoreception is detected by the transient receptor potential (TRP) family of cation channels expressed in sensory neurons. In mammals, TRPM8 has been detected as the primary peripheral sensor for cold (Bautista et al 2007). Afferent signals ascend through ganglia and nerve nuclei along such pathways as the cortical spinothalamic tract - and the lateral parabrachial neuronal nucleus (LPB); they are then integrated at the level of the spinal cord (Plate I, dorsal rostrum, encephalic trunk, and hypothalamus (Nakamura and Morrison 2008; Pitoni et al 2011; Tan and Knight 2018). Thermal stimuli are also projected to the somatosensory cortex, which enables behavioral responses. Glutamatergic neurons are in charge of projecting the thermal information from the cutaneous, visceral, and spinal cord thermoreceptors from the LPB to the POA, where the

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information is processed (Tan and Knight 2018). The POA consists of various regions, including the medial preoptic area (MPO) and the medial preoptic nucleus (MnPO), which are considered the primary nervous structures responsible for temperature regulation (Morrison and Madden 2014; Tan and Knight 2018). Also, the POA connects to diverse brain nuclei, to maintain thermal homeostasis. Once the POA processes the thermal information, it enables the physiological responses to counteract the low temperature. This response occurs in the form of cutaneous vasoconstriction, piloerection, and/or thermogenesis (Romanovsky 2018) depending on a temperature threshold (Pitoni et al. 2011). The most important physiological responses to exposure to cold are the thermogenesis of BAT and the shivering of skeletal muscle to generate heat, accompanied by constriction of the blood vessels (vasoconstriction) to prevent heat loss. Though thermogenic BAT is considered highly necessary for thermoregulation after birth in mammal species (Mrowka and Reuter, 2016), it is not the case in pigs since there are reports that indicate that - newly born piglets lack this type of tissue (Trayhurn et al. 1989), and rely on shivering thermogenesis as the principal mechanism of thermoregulation (Berthon et al 1995). Reports on humans, however, suggest that shivering is less efficient than vasoconstriction as a defense against cold because much of the heat generated by peripheral muscles is released into the atmosphere instead of being retained in the core (Pitoni et al 2011). For this reason, skeletal muscle shivering generates heat, and peripheral vasoconstriction to prevent heat loss, together with piloerection that allows the formation of a layer of hot air around the body, constitute the mechanisms upon which the neonate piglet depends to reach thermal homeostasis (Figure 2).

3. Shivering thermogenesis

Sow’s fetuses experience constant temperature exchange through the placenta while in utero that maintains a thermostable environment. In stark contrast, the - newly born piglet is exposed to cold immediately at birth (Berthon et al 1994) and must depend on its immature thermoregulating mechanisms that were not required during intrauterine life. The essential components of thermogenesis are two fundamental mechanisms: shivering - and non-shivering thermogenesis. As mentioned above, newborn piglets have only a small amount of adipose tissue (1.5%) and, it seems, no BAT (Herpin et al 2002). However, this situation, changes quickly with age and development, to a point where fat content reaches perhaps 15% at weaning. Studies images A, B, and C, in spite of spot is located on red zones, however please note a marked temperature decrease in surface areas of the skin and such peripheral zones as the auricular pavilions, thoracic members and, above all, the rostrum,
particularly the snout (yellow zones). 3-month-old piglets have also detected small amounts of tissue similar to BAT (Dauncey et al 1981).

Determining the existence of a mechanism of non-shivering thermogenesis in piglets requires measuring, simultaneously, the magnitude of shivering and the level of heat production at temperatures that run from thermoneutrality to cold (Barré et al. 1985). However, the thermogenic response of newborn piglets does not include non-shivering thermogenesis. Shivering thermogenesis based on skeletal muscle, therefore, plays a major role in preserving homeothermy. Shivering thermogenesis—or simply “shivering”—refers to the production of heat through a repetitive process of muscular contraction that generates considerable amounts of heat, but rapidly consumes the newborn’s energy reserves (oxidized glycogen) (Theil et al. 2014; Berthon et al. 1994). Shivering is considered the first line of defense against acute exposure to cold in pigs (Bal et al. 2016; Berthon et al. 1995). During muscular contraction, heat is generated by hydrolysis of adenosine triphosphate (ATP) from three different ATPases: ATPase of myosin, which performs the contractile work, SERCA, and Na⁺/K⁺ ATPase (Rowland et al. 2014; Little and Seebacher 2013). Studies have demonstrated the contribution of skeletal muscle to the temperature increase in average up to 97% in response to cold, in five-day-old piglets (Lossec et al. 1998). Though shivering is the first response to acute exposure to cold, this reaction entails an extremely high energy cost and can even compromise muscular function (Periasamy et al. 2017). Due to the absence of BAT, the newly born piglet maintains its body temperature almost exclusively by shivering (Berthon et al. 1994). In this regard, free mitochondria have been detected in the skeletal muscle of two-month-old pigs adapted to cold (Herpin and Barre 1989). During muscular contraction due to cold-induced stress, a specific muscle-regulating mechanism has been identified that includes the activity of the carnitine palmitoyltransferase I enzyme (CPT I) in the interfibrillar mitochondria. Another example is the rhomboid muscle, where CPT I’s sensitivity to malonyl-CoA remains constant at ambient temperature, while the latter decreases under cold conditions. These changes could foster the oxidation of fatty acids in more oxidative muscles during shivering thermogenesis.

4. Participation of muscular glycogen

As the neonate pig has no BAT, its corporal reserves are essential for survival during the first hours of life, since it utilizes those glycogen and fat reserves as its primary energy substrates for producing heat in the first 12-24 hours post-birth (Berthon et al. 1994). At birth, glycogen reserves at birth range from 30-35 grams per kg of body weight, and are located almost entirely in the liver and muscles. Because the piglets’ subcutaneous fatty tissue is less than 2%, glycogen becomes the principal energy resource. It is consumed rapidly by the piglet’s metabolism to produce heat after birth by consuming segments of hepatic glycogen and 50% of the muscular tissue in just the first 12 hours of life (Charneca et al. 2010). The complications that arise from having energy values (glucose) below optimal levels, combined with diminished neonatal vitality, can lead to failure when the piglet seeks to begin sucking at its dam’s teat to ingest colostrum and obtain the nutrients it requires, as well as the benefit of adequate passive immunity. Also, piglets may be stressed due to hypothermia induced by hypoglycemia, which can cause them to fall into a coma quite quickly (Figure 3).

5. Birth weight and hypothermia

The piglet’s ability to overcome postnatal hypothermia by re-establishing its body temperature after the sudden decrease during the immediate postpartum period is directly related to two aspects: birth weight (Caldara et al. 2014), and its position in the corral during the first and second hours after birth. Kammersgaard et al. (2011) observed that the neonate’s low rectal temperature up to two hours after birth is related to birth weight of around 1.5 kg, and to piglets that remain on the floor for more time instead of approaching the dam’s udder during that time. Stanton et al. (1973) also observed that the performance of newborn piglets in a cold environment is closely-related to birth weight, noting that piglets weighing 1.02–1.15 kg have a higher surface-area/body volume ratio than larger neonates with weights of 1.27–1.38 kg., for this reason, the former are more prone to heat loss in cold environments.

6. Thermoregulating behavior

In addition to metabolic mechanisms, the capacity to conserve heat involves behavioral strategies. Behavioral adjustments in body posture provide efficient mechanisms for minimizing heat loss (Kammersgaard et al. 2011; Bertoni et al. 2020a, b; Guerrero-Lagarreta et al. 2020), principally because mammal species utilize specific postures to conserve energy and, in this way, limit heat dissipation. These postures reduce the surface-area/body volume ratio to minimize contact between the neonate’s surface and the air and, decrease the area susceptible to heat dissipation (Terrien et al. 2011). During the first day of the neonate piglet’s life, physical activity is responsible for 30% of the heat production they generate. The above underscores the importance of behavioral adaptations appropriate for minimizing heat loss, and necessary for increasing heat production in unfavorable thermal conditions. Some studies have shown that piglets have one thermal advantage in this regard: heat conduction through contact with the sow’s udder when they stay close to their dam for prolonged periods instead of remaining isolated. Moreover, when neonate piglets nestle or huddle with their littermates they also reduce the total surface area exposed to the low-temperature environment (Kammersgaard et al. 2011; Kammersgaard et al. 2013) and/or light air currents. This strategy is effective in reducing heat loss through what is called ‘social’ thermoregulation, since a litter of newborn piglets can increase a critically-low
temperature—below 25-30°C—though this is still below their thermoneutral zone (TNZ), which is around 34°C (Vasdal et al 2009; Close 1992). It is important to note that nestling or huddling behaviors lessen with age and as the piglets grew, their weight increases (Vasdal et al 2009; Boon 1981).

A second strategy that piglets employ to reduce heat loss consists of adjusting their position and posture. Conductive heat loss is diminished by adopting a sternal posture from a recumbent position (Vasdal et al 2009; Mount 1967), as this allows the neonate to reduce its contact surface with the floor. There are indications that during the first and second hours post-birth, the loss of body temperature correlates with the location of the neonate piglet in the farrowing pen (Kammersgaard et al 2011); for example, when the piglet is on the floor, far from the dam’s teat, its body temperature will decrease gradually (Kammersgaard et al 2011; Melišová et al 2014). In contrast, if it is close to its littermates, or the dam’s udder, its body temperature will begin to rise. The above emphasizes the vital importance of behavioral adaptations by neonates to reduce heat loss. Nestling or huddling in the warmest areas of the pen also allows heat transfer by conduction from warmer objects (Kammersgaard et al 2011; Kammersgaard et al 2013). These measures allow piglets to achieve homeothermy during the first hours of life (Vasdal et al 2009; Kammersgaard et al 2011) (Figure 4).

**Figure 3** Factors associated with hypothermia mortality in newborn piglets.

7. Colostrum consumption and neonatal metabolism

The behavioral patterns that the neonate piglet adopts to generate heat include ingesting colostrum, a substance that contains the energy necessary for regulating body temperature and growth (Herpin et al 2002; Le Dividich et al 2005). Colostrum is the first milk secreted by the mammary gland, which secretes it continuously from birth to 12-24 hours postpartum (Quesnel et al 2012). At that point, milk secretion becomes cyclical and piglets fighting to access the dam’s udder begins (Muns et al 2016; Auldist et al 2000). Colostrum is a rich source of digestible nutrients and several bioactive components, such as immunoglobulins (IgA, IgM and, particularly IgG), hydrolytic enzymes, hormones, and growth factors (Quesnel and Farmer 2019; Baxter et al 2011; Rooke and Bland 2002; Wu et al 2010). For this reason, colostrum, plays a key role in piglet’s thermoregulation, in the acquisition of passive immunity, and intestinal development and maturation (Quesnel et al 2012; Devillers et al 2007). Colostrum also supplies the newborn piglet with highly-metabolizable energy (Le Dividich and Herpin 1994) and a high content of fatty acids and lactose, which the piglet utilizes efficiently to cope with cold-induced stress by increasing its metabolic rate and maintaining homeothermic equilibrium during the first day of life (Le Dividich and Herpin, 1994). As a result, the rectal temperature of piglets at 24 hours of age correlates positively with colostrum ingestion (Muns et al 2016; Devillers et al 2011). According to Amdi et al (2017), piglets that consume colostrum within the first-hour post-birth increase their rectal temperature by 1°C compared to those that do not have access to colostrum (37.5 °C vs. 36.6 °C; P < 0.001). In other words, the piglet’s body temperature increases because colostrum provides large amounts of fat (30-40%) and can generate up to 60% of the energy that the neonate requires on its first day of life (Caldara et al 2014). Moreover, it activates the secretion of powerful lipases in the oral cavity (lingual lipase) (Theil et al 2014) that ensure the adequate degradation of the fats in the...
colostrum, making them easily digestible (Rooke and Bland 2002) and available to generate energy through glycogen oxidation (Herpin et al 2002; Theil et al 2014). In this sense, we consider that the degree at which the neonate increases its ingestion of colostrum, it increases heat production and so becomes capable of maintaining constant body temperature (Caldara et al 2014; Devillers et al 2011) therefore increasing its probability of survival.

8. Physiopathology of death by hypothermia in farm animals

Under normal conditions, the thermoreceptors in the skin and hypothalamus send nerve impulses to the preoptic region and the heat promoting center of the POA of the hypothalamus. The nerve impulses in the sympathetic nerve that reach the adrenal medulla stimulate the release of adrenaline and noradrenaline into the bloodstream. The nerve impulses from the heat promoting center, in turn, stimulate sympathetic nerves that cause constriction of the blood vessels in the skin to reduce the flow of warm blood, thus producing heat loss by organs internal to the skin (Casas-Alvarado et al 2020). The causes of heat loss in piglets’ range from cold-induced stress to an inadequate environment to metabolic diseases such as hypoglycemia, suprarenal insufficiency, hypothyroidism, or malnutrition (Mota-Rojas et al 2020a). Also, as relevant factors are the low-birth-weight and birth order, those piglets born at the end of parturition, in which the long time of the first contact with the udder of the dam and the intake of colostrum to generate heat is limited (Caldara et al 2014 Mota-Rojas et al 2008). Low-birth-weight piglets are weak, and it is challenging to stand and reach the dam’s heat protection, complicating the heat loss (Le Dividich and Herpin 1994; Kammersgaard et al 2011). These piglets suffer starvation, hypoglycemia, hypothermia, and eventually are crushed or die. Hypoglycemia, in turn, and the limited glycogen reserves of the newborn piglets leads to the use of hepatic or muscular glycogen conducting to metabolic acidosis. If piglets are born under perinatal asphyxia episodes, circulatory changes and hypoxia occur. Piglets are very susceptible to hypoxia, inducing nervous disorders that difficult orientation and piglets’ motion to reach the udder or the dam’s heat environment. Depending on the degree of the decrease in body temperature, hypothermia may be accompanied by the following effects: a sensation of cold, shivering, disorientation, vasconstriction, muscular rigidity, bradycardia, metabolic acidosis, hyperventilation, hypotension, loss of spontaneous movement (Mota-Rojas et al 2020c), and coma, possibly ending with death which, under these circumstances, is almost always caused by cardiac arrhythmias (Figure 4). Often, the piglets that die from hypothermia have a poor corporal condition, while animals that survive a long period of hypothermia develop very long hair. Another symptom that may appear is cyanosis in the animal’s extremities (Mota-Rojas et al 2016). These dead piglets are found to have empty stomachs and, therefore, low levels of blood glucose and hepatic glycogen that contribute to death by hypoglycemia (Mota-Rojas et al 2008; Mota-Rojas et al 2015; Mota-Rojas et al 2018).

Figure 4 Physical and postural aspects of hypothermic newborn piglets.
9. Final Considerations

The transition from intrauterine to extraterine life in newly born piglets is accompanied by a significant reduction of environmental temperature of as many as 15°C. One of the consequences of this situation is a drastic descent of the newborn’s body temperature that may fall to a critical minimum level. Heat loss effected by mechanisms like conduction, convection, evaporation, and radiation, added to certain innate characteristic of this species –scarce subcutaneous adipose tissue, lack of BAT, and limited glycogen reserves– are contributing factors that can result in many piglets suffering hypothermia during the first 24 hours postpartum. For this reason, regulating the body temperature of newborn piglets –a process governed by the CNS– depends primarily on producing heat through shivering thermogenesis, as the first line of defense, supported by thermoregulating behaviors. However, for neonates exposed to cold to achieve thermal homeostasis, they must spend their reserves of hepatic and muscular glycogen. Thus, it is important to ensure adequate ingestion of colostrum, which plays a vital role in providing the energy that piglets require to achieve thermoregulation. However, hypoglycemia, is not the only effect that is considered as a risk factor for hypothermia, as low birth weight also threatens the neonate piglets’ welfare. If hypothermia persists, piglets will suffer starvation leading to imminent death. In summary, the survival of neonate piglets results from the interaction between the dam, the piglet itself, and the environment.

Conflict of Interest

The authors declare that they have no conflict of interest.

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