EFFECTS OF NEGATIVE DCAD (DIETARY CATION-ANION DIFFERENCE) AND VITAMIN D IN SOW TRANSITION DIETS TO INCREASE PIGLET WEANING NUMBERS, IMPROVE PIGLET WEANING WEIGHT, AND MINIMISE SOW CONDITION LOSS DURING LACTATION

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Executive Summary

There is increasing evidence that the period between late gestation and lactation, known as the transition period, is critically important in sow farrowing performance and in establishing a successful lactation (Theil 2015). However, feeding practices during this period are not optimised for the transition sow, but rather, focus on the nutritional needs for lactation. In the dairy industry, most cows are fed a transition diet with a negative dietary anion cation difference (DCAD) which increases the amount of calcium (Ca) available at parturition. Additionally, the vitamin D metabolite 25-OH-D_3 (calcidiol) enhances Ca absorption by the small intestine (Lean *et al.*, 2014), which is important for uterine contractions and milk production. Therefore, this project aimed to determine if a specific transition diet that featured a negative DCAD, calcidiol supplementation and increased fibre content would alter acid-base status, reduce the risk of piglets being born dead, improve sow body condition and increase piglet weaning number.

This study used 413 purebred Large White and Landrace primiparous and multiparous sows (parity 1 to 8; Myora Genetics) allocated to receive either a control diet (dry sow ration until entry to farrowing house then a lactating sow ration until weaning, *n* = 85), or one of four transition diets fed from day 103 of gestation until day three post-farrow 1) Negative DCAD, *n* = 84, 2) Negative DCAD + calcidiol, *n* = 84, 3) Positive DCAD, *n* = 81, or 4) Positive DCAD + calcidiol, *n* = 79.

Major outcomes of the project

- A significant reduction in stillbirths was observed in the negative DCAD + calcidiol and positive DCAD treatment groups compared to control sows (lactating sow ration).
- There was a significant reduction in mortality (↓ 4%) to day 120 of piglets offered the negative DCAD + calcidiol diet compared to piglets offered the control or positive DCAD + calcidiol diets.
- There was an interaction between treatment and parity with more than 1 additional piglet born in the subsequent litter for positive DCAD primiparous compared to control primiparous or multiparous sows.
- Urinary pH responses of sows to diets formulated to provide a positive DCAD diet indicated that there was acidification occurring in both positive and negative DCAD treatments. It is possible that the rapidly available starch in barley and wheat that comprised approximately 50% of the diets generated enough volatile fatty acids to reduce urinary pH.
- There were minimal differences in sow body condition during the experiment. However, negative DCAD + calcidiol-fed sows lost significantly less backfat during lactation than control-, negative DCAD- and positive DCAD + calcidiol-fed sows.
- Milk fat and protein, piglet weight and the number of piglets weaned was statistically similar for all five treatment groups. The lack of a significant effect on number of piglets weaned despite reductions in stillbirths and piglet mortalities was likely influenced by fostering.
- There were statistical differences reflected in blood gas, mineral and metabolite concentrations that are consistent with feeding of a negative DCAD diet, providing more evidence that negative DCAD diets may influence energy metabolism.

Relevance of the project's outcomes to the Australian pork industry

- A separate transition diet for sows that incorporates increased fibre content and an ability to induce metabolic acidification is recommended for Australian pork producers.

- Further research is required to define the optimal period of transition feeding, investigate the effects in gilts, determine the optimal urine pH to target for outcomes, and characterise the effects of carbohydrate fractions in the diet on urinary pH and metabolic acidification.

Contents

1. Introduction

The productivity of a breeding herd reflects the number of piglets weaned per sow per year and is a function of the number of piglets born alive, piglet survival to weaning and farrowing frequency. Therefore, maximising the number of healthy, robust piglets weaned per sow is a key objective for most producers. According to past Pork CRC Benchmarking figures, Australian producers are achieving on average 10.2 pigs weaned/litter (23.5 pigs weaned/sow/year). However, increasing this to 12 piglets weaned/litter (28 pigs weaned/sow/year), will substantially reduce cost of production (COP). For the Australian pig industry to remain globally competitive, strategies to reduce COP must be investigated; Australia currently has one of the highest COP in the world. Increasing the volume of pig meat produced can be achieved by increasing litter sizes; however, it's a well understood problem that large litter sizes exacerbate the issue of high piglet mortality with approximately 15-20% of piglets dying either during the farrowing process or in early lactation (Farmer and Edwards, 2021). Reducing this piglet loss prior to weaning is key to increasing weaned litter size.

Sows experience profound physiological and environmental changes in the transition period from gestation, through parturition and into lactation. The transition period is relatively short for sows and has been defined as the last 10 days of gestation to the first 10 days of lactation (Theil, 2015). During this transition period, sows change from an anabolic to a catabolic state, experience dietary changes, move from group housing to individual housing, undergo parturition, produce and secrete colostrum, and initiate and maintain milk production. Feeding practices may not adequately address these changes and there is evidence that the transition period is a strong determinant of a successful lactation (Theil, 2015). There are beneficial effects of specific transition diets, in particular high levels of fibre, on the incidence of stillborn piglets and piglet mortality (Hansen et al., 2012; Feyera et al., 2017).

In the dairy industry, it is common for producers to feed their cows a specific diet during this phase of transition. One key aspect of this diet is a negative dietary anion cation difference (DCAD). The DCAD is an index of the relative balance among the principal cations (potassium, K; and sodium, Na) and the principal anions (chloride, Cl; and sulphur, S) in the diet. A negative DCAD diet is commonly achieved by including an acidogenic feed protein meal in the ration. Negative DCAD diets are predominantly fed to reduce the incidence of milk fever (hypocalcaemia); however, sub-clinical milk fever is also recognised as a "gateway disease" for a number of other physiological conditions (Houe et al., 2001). Thus, negative DCAD transition diets can reduce the incidences of mastitis, metritis, dystocia, and retained placenta.

There are also recognised benefits for subsequent reproduction including reduced calving to conception rates and less services required per conception (Borsberry and Dobson, 1989). Furthermore, Degaris et al. (2008) found that the longer dairy cows are exposed to a negative DCAD transition diet, the higher the fat and protein yields of milk in lactation. Exposure to a negative DCAD pre-calving diet that incorporated Bio-Chlor™, a commercial anionic protein meal, improved milk yield by 7.4 L/day (DeGroot et al., 2010). Therefore, there is potential to improve milk quality and quantity, increase the growth rate and weaning weight of piglets which reduces the age at which a pig reaches slaughter weight (Wolter and Ellis, 2001).

In addition to the reduction in disorders associated with subclinical hypocalcaemia, there is increasing evidence in mice, human and cattle studies to suggest evidence of metabolic effects, orchestrated by osteocalcin produced by mature osteoblasts, which influence regulation of energy metabolism (Lean et al., 2014). It is hypothesised that the skeleton

plays an essential role in energy metabolism reflecting a crucial need to integrate the homeorhetic changes that are required to upregulate metabolism in response to the demands of lactation (Lee et al., 2007; Lean et al., 2014). Homeorhetic changes are defined as the 'coordinated changes in metabolism of body tissues necessary to support a physiological state' (Bauman and Currie, 1980). There is now evidence to support the hypothesis that the skeleton plays an important role in homeorhetic adaptation to lactation and that this relationship may be influenced by nutrition during this transition period. Understanding this mechanism in pigs may allow us to tailor a nutritional program that allows the sow to prepare and cope with the rapid and substantial increase in metabolism experienced during lactation.

While negative DCAD transition diets increase the available calcium (Ca) to the sow at parturition, inclusion of vitamin D in the diet is also important to ensure Ca uptake. This mechanism is well established in monogastric species whereby the vitamin D metabolite 25-OH-D_3 (calcidiol) enhances Ca absorption by the small intestine (Lean et al., 2014). While vitamin D_3 (cholecalciferol) can be included in the diet, cholecalciferol must then be converted in the liver to calcidiol. Calcidiol is the circulating form of vitamin D, which is then converted in the kidneys into the bioactive form calcitriol (1,25(OH_2)D_3). When there is an increased demand for Ca such as in lactation or growth, the ionised concentration of Ca (Ca^{2+}) decreases in blood. Release of parathyroid hormone (PTH) is then stimulated which targets both the bone and kidney to lift the supply of Ca^{2+} to the extracellular fluid. PTH induces increased secretion of calcidiol from the kidney which then increases active Ca transport across the small intestine (Lean et al., 2014). Most Australian sows are housed indoors, raising the potential for vitamin D insufficiency, notwithstanding cholecalciferol supplementation. Several studies have shown the benefits of including calcidiol in sow rations on the calcidiol status of the sow. Supplementation of calcidiol, a form of vitamin D in sow diets increases calcidiol concentrations in plasma (Lauridsen et al., 2010; Weber et al., 2014; Meuter et al., 2016; Sorensen and Nielsen, 2016), colostrum (Weber et al., 2014) and milk (Weber et al., 2014; Meuter et al., 2016). Inclusion of calcidiol in sow diets has also been associated with fewer farrowing complications (Meuter et al., 2016), increased piglet birth weight (Weber et al., 2014; Sorensen and Nielsen, 2016), reduced incidence of fever (Meuter et al., 2016), and, decreased stillborns when supplemented at levels of 1,400 IU or higher (Lauridsen et al., 2010).

In pigs, continual selection for highly productive breeding sows must be matched with appropriate nutrition to realise their genetic potential. Clinical cases of parturient hypocalcaemia in sows are not well documented; however, in recent years it has been hypothesised that increased productivity, including increased milk production, has led to an increase in unexplained sow mortality during the prepartum and early postpartum periods (Darriet et al., 2017). It was hypothesised that this is due to hypocalcaemic disorders. Three studies have shown that a negative DCAD diet in late gestation and early lactation increased Ca mobilisation from the skeleton (Darriet et al., 2017), tended to reduce stillborn piglets in an Australian study (Henman et al., 1999 (*unpublished data*); 2023), increased subsequent litter size (Roux et al., 2008) (Henman et al., 1999 (*unpublished data*); 2023), and increased lactation feed intake (Henman et al., 1999, *unpublished data*). While the Australian results were very positive, there were marked differences in response between gilts and sows, a pattern reflected in differences in response for cows and first calving heifers (Lean et al., 2014). When supplemented with Bio-Chlor 2 weeks prior to farrowing, gilts had a much greater reduction in stillbirth percentage when compared to the more moderate reduction observed in parity 3 sows (Henman et al., 1999 (*unpublished data*); 2023). In contrast, a recent Australian study which included 0.285% magnesium sulphate (an anionic salt) in a lactating sow ration fed during the transition period found no reduction in the incidence of stillborn piglets (Plush et al., 2018). Information is lacking, however, on the optimal DCAD value of the diet as well as the best time to transition to a positive DCAD post-parturition

and the optimal approach for gilts, as opposed to sows. Further, there is limited literature on the effect of implementing a negative DCAD transition feeding strategy on all aspects of production including farrowing performance, lactation performance, sow feed intake, pre- and post-weaning piglet growth and subsequent sow fertility.

This project addressed three research questions. Firstly, will feeding a negative DCAD transition diet from late in gestation to early lactation improve production outcomes? Secondly, is there evidence that skeleton regulates energy metabolism in the pig as it does in other species as indicated by changes in blood metabolites? And thirdly, is there a positive interaction of both DCAD and the inclusion of calcidiol in a transition diet?

2. Methodology

This study was approved by the Department of Primary Industries and Regions South Australia Animal Ethics Committee (#10/19) and was conducted in accordance with the 'Australian code for the care and use of animals for scientific purposes 8th edition' (National Health and Medical Research Council: Canberra, 2013). All animal work was conducted at Myora Farm's Breeder and Grower Facility, Glenburnie, South Australia.

Experimental design and diets

The aim of this experiment was to evaluate the performance, health and reproduction of commercial sows fed different diets over the transition period (approximately 14 days pre-farrowing until three days post-farrowing). Specifically, this study aimed to investigate the effect of transition rations with either a positive or negative DCAD diet alone or in combination with calcidiol 50 µg/kg (Rovimix HyD®; DSM Nutritional Products, Basel, Switzerland) and compare these to sows fed using the standard commercial practice of a dry sow ration fed until entry to the farrowing house, and then a lactating sow ration fed until weaning. All diets contained cholecalciferol (1000 IU/kg). Transition rations also had a higher fibre content than the dry sow and lactating sow rations.

This study was conducted from March 2020 to September 2020 over 10 weekly farrowing batches. A total of 413 purebred Landrace or Large White sows (Myora Genetics) were enrolled in the experiment and selected sows were either primiparous (*n* = 124) or multiparous/ (*n* = 289; average parity 2.58 ± 1.50; parity range 1-8). Sows were randomly allocated in blocks using the *ralloc* function in Stata version 14.1 (StataCorp LP) to one of five treatment groups based on their breed and their status of being primiparous or multiparous. Treatment groups were (1) Control, no transition diet - dry sow ration until farrowing house entry and lactating sow ration from farrowing house entry until weaning (*n* = 85); (2) Negative DCAD and cholecalciferol (*n* = 84); (3) Negative DCAD and calcidiol (*n* = 84); (4) Positive DCAD and cholecalciferol (*n* = 81); and (5) Positive DCAD and calcidiol (*n* = 79). Diet rations are outlined in Table 1, and nutrient specifications of each diet are shown in Table 2.

Table 1. Composition of dry sow, negative DCAD, and positive DCAD transition diets (+/- calcidiol) fed during the trial (kg per t).

Feed ingredient, kg	Dry sow	Lactating sow	Negative DCAD	Negative DCAD + calcidiol	Positive DCAD	Positive DCAD + calcidiol
Barley	548	187	255	255	255	255
Wheat	200	400	248	246	248	246
Lupins	75	150	120	120	120	120
Full fat soya	0	25	30	30	30	30
Canola meal expeller	35	20	30	30	30	30
Meat and bone meal	20	35	12	12	12	12
Soybean extract	20	30	0	0	0	0
Fishmeal	35	50	40	40	40	40
Lucerne chaff	0	0	70	70	70	70
Oat hulls	40	0	0	0	0	0
Bran	0	28	80	80	80	80
Sugar beet pulp pellets	0	0	35	35	35	35
Flax seed oil	5	25	34	34	34	34
Lysine HCl	1.2	1.8	1.1	1.1	1.1	1.1
MHA methionine	1.2	1.9	0.9	0.9	0.9	0.9
Threonine	0.9	1.5	0.9	0.9	0.9	0.9
Tryptophan	0.2	0.7	0.4	0.4	0.4	0.4
Valine	0.0	1.8	1.0	1.0	1.0	1.0
L-Arginine	0.5	0.0	0.5	0.5	0.5	0.5
Di-Calcium Phosphate	3.0	4.5	0.0	0.0	0.0	0.0
Salt	3.0	0.9	4.1	4.1	4.1	4.1
Sodium bicarbonate	6.0	10.5	0.0	0.0	4.0	4.0
Potassium Carbonate	1.0	3.0	0.0	0.0	0.0	0.0
BioChlor[1]	0.0	0.0	2.0	2.0	0.0	0.0
Magnesium sulphate	0.0	0.0	2.0	2.0	0.0	0.0
Rovimix HyD[2]	0.0	0.0	0.0	1.5	0.0	1.5
Yang[3]	0.0	0.0	0.4	0.4	0.4	0.4
Hilyses prebiotic[4]	0.0	8.3	10.0	10.0	10.0	10.0
Levucell SB10ME[5]	0.0	0.0	0.1	0.1	0.1	0.1
Betaine 96[6]	0.0	2.0	2.0	2.0	2.0	2.0
Opticell[7]	0.0	8.0	15.0	15.0	15.0	15.0
Mastersorb Gold MycoBind[8]	1.0	1.0	1.5	1.5	1.5	1.5
Activo[9]	0.10	0.15	0.25	0.25	0.25	0.25
Vitamin mineral premix[10, 11]	3.5	3.5	3.5	3.5	3.5	3.5
Choline Chloride 60%	0.5	0.5	0.5	0.5	0.5	0.5

[1] BioChlor (an acidogenic protein meal; Arm and Hammer Animal Nutrition, Princeton, NJ).
[2] Rovimix HyD (Division of Animal Nutrition and Health, DSM Nutritional Products LLC, Parsippany, NJ); [3] Yang (inactivated yeast fractions; Lallemand Animal Nutrition, Canada); [4] HiLysis (hydrolysed yeast; YorkAg Products Inc, York, PA).
[5] Levucell (active yeast; Lallemand Animal Nutrition, Canada).
[6] VistaBet (ABVista, Wiltshire, UK).
[7] OptiCell (fiber source; Agromed Austria GmbH, Austria).
[8] Mastersorb Gold MycoBind (mycotoxin binder; EW Nutrition, Adel, IA).
[9] Activo Xtract 6930 (phytochemicals; EW Nutrition, Adel, IA).
[10] Dry Sow Premix (Vitamin A 15.0000 MIU, Vitamin D 31.0000 MIU, Vitamin E 250.0000 G, Vitamin K3 4.0000 G, Vitamin B1 3.0000 G, Vitamin B2 10.0000 G, Vitamin B6 5.0000 G, Vitamin B12 0.0500 G, Biotin 0.8000 G, Pantothenic Acid 40.0000 G, Folic Acid -, Niacin 50.0000 G, Vitamin C 300.0000 G, Organic Copper 10.0000 G, Cobalt 0.3000 G, Iodine 1.2000 G, Organic Iron 60.0000 G, Organic Manganese 20.0000 G, Organic Selenium 0.3000 G, Organic Zinc 60.0000 G, Chromium 0.2000 G) [11] Transition and lactation premix. (Vitamin D3 1.0000 MIU, Vitamin E 250.0000 G, Vitamin K3 4.0000 G, Vitamin B1 3.0000 G, Vitamin B2 10.0000 G, Vitamin B6 5.0000 G, Vitamin B1 2 0.0500 G, Biotin 0.8000 G, Pantothenic Acid 40.0000 G, Folic Acid 6.0000 G, Niacin 50.0000 G, Vitamin C 300.0000 G, Organic Copper 10.0000 G, Cobalt 0.3000 G, Iodine 1.2000 G, Organic Iron 60.0000 G, Organic Manganese 20.0000 G, Organic Selenium 0.3000 G, Organic Zinc 60.0000 G, Chromium 0.2000 G)

Table 2. Nutrient analyses (calculated) of diets fed during the experiment.

Nutrient	Dry sow	Lactating sow	Negative DCAD (-/+ calcidiol)	Positive DCAD (-/+ calcidiol)
Dry matter, %	90.2	90.6	90.5	90.5
Digestible energy, MJ/kg	13.8	14.5	13.8	13.8
Crude protein, %	14.8	19.5	17.5	17.4
Crude fiber, %	5.37	4.94	7.36	7.35
NDF, %	15.2	13.0	18.3	18.2
ADF, %	6.52	6.24	9.11	9.10
Lysine (total), %	0.67	0.95	0.90	0.89
Digestible Lysine:energy, g/MJ	0.51	0.69	0.59	0.59
Calcium, %	0.81	1.15	0.83	0.83
Phosphorous, %	0.68	0.64	0.60	0.60
Magnesium, g/kg	1.44	1.57	1.92	1.69
Potassium, %	0.62	0.79	0.67	0.67
Sodium, %	0.35	0.41	0.34	0.34
Chloride, %	0.40	0.29	0.50	0.48
Salt, %	0.66	0.49	0.80	0.79
Sulphur, %	0.18	0.20	0.22	0.19
DCAD, mEq/kg	83.4	178	-2.1	68.4
Dietary electrolyte balance	198	301	136	186

Animal housing and management

Sows were previously mated by artificial insemination with either single sire Landrace or Large White fresh extended semen. Thus, sows produced either purebred or crossbred litters. Sows were pregnancy scanned at approximately 28 days post-mating via trans-abdominal ultrasonography (IMAGO.S, ECM International Inc, France). During gestation, sows were housed in groups of up to 35 animals on straw and sawdust bedding. Sows were fed in open full body stalls twice daily at approximately 0800 h and 1500 h via an automatic trickle feed system. On day 104 of gestation, sows commenced their experimental rations in the dry sow shed. At feeding time, all sows were locked in the individual feeding stalls, and transition sows (treatments 2 to 5) were hand-fed while control sows were fed via the trickle feed system. After approximately one hour, any feed residuals were recorded and removed from the feed bowl, and sows were released from the feeding stalls.

On day 109 of gestation, sows were moved from the dry sow group housing to individual farrowing crates where they remained until weaning. Sows had *ad libitum* access to fresh drinking water. Farrowing rooms were temperature controlled and each farrowing crate had a heat mat provided for an additional heat source for the piglets. Target temperatures in the house varied between 17 and 21C with floor heating also adjusted according to the physiological state of the sows. Sows were fed three times daily at approximately 0730 h, 1230 h and 1530 h. Prior to farrowing, sows received either 4 kg/day of the transition diets (treatments 2 to 5; hand-fed), or 3.5 kg/day of the lactating sow ration (control sows; automatic feeding system). Post-farrowing, feed bowls were checked every morning, and depending on the amount of feed left in the bowl, the amount of feed was either increased, decreased or remained the same. The amount of feed given each day was recorded. Sow feed intake in lactation increased from 3.5 kg on day one post-farrowing to a maximum of 13 kg by weaning.

Litters were processed within 24 h of farrowing. All piglets received a litter specific ear-notch number, teeth were clipped, and a 1 mL IM iron injection was given (Feron 200+B12, 200 mg/mL iron dextran and 40 ug/mL cyanocobalamin; Bayer Healthcare, Pymble, Australia). Where possible, cross-fostering occurred within treatment. Cross-fostering occurred if litter size exceeded the number of available functional teats and piglets that were failing to thrive were removed as required. Additional piglets from outside the experiment were fostered onto sows in the experiment only if necessary and if teat capacity

allowed. These piglets (*n* = 1,194) were excluded from individual piglet weight analysis; however, they were included in the total litter weights for each sow.

At 4 days of age, all piglets were tail-docked and received a 2 mL oral drench of coccidiocide (Baycox®, Bayer, Pymble NSW Australia) and a 2 mL injection of *Mycoplasma hyopneumoniae* vaccination (RespiSure ONE, Pfizer Animal Health, West Ride, NSW Australia). At 21 days of age, all piglets received a second injection of RespiSure and 1 mL IM CircoFLEX (porcine Circovirus associated disease vaccine, Boehringer Ingleheim Vetmedica, Berkshire, UK). Piglets were weaned at 27.5 ± 0.2 days of age and transported via trailer to the grower facility. Sows were moved to individual weaning pens on concrete slatted floors. Twice-daily oestrus checks were performed by walking a mature boar in front of each pen and performing a back pressure test to check for standing oestrus. Any sows that exhibited a standing reflex were inseminated twice 12 h apart.

Sow body composition

Upon entry to the farrowing house, on day 21 of lactation, and at weaning, sows were weighed and backfat at the P2 position was measured using an ultrasound machine and sector probe (IMAGO.S, ECM International Inc, France). Additionally, subset sows were weighed and P2 backfat was recorded on day one post-parturition.

Urine pH

To determine whether metabolic acidosis was being achieved in response to the different DCAD diets prior to farrowing, sow urine pH was measured. Urine was collected at 0700 daily from pregnant sows in the farrowing house, approximately 30 min before the morning feed. All sows were encouraged to stand, and approximately 20 mL of urine was collected mid-stream from any sows that subsequently urinated. Once a sow had two daily urine samples collected prior to farrowing, no further attempts were made to collect a daily sample. Urine sample date was recorded, and samples were immediately tested for pH using a handheld pH meter (HORIBA LAQUAtwin B-712, HORIBA Ltd., Kyoto, Japan). From 10 days post-farrowing, daily urine samples were collected from as many sows as possible using the same method as described above. A maximum of two daily samples were collected per sow.

Blood sample collection and blood gas analysis

Blood samples were collected from subset sows at entry to the farrowing house, and on days 1 and 21 post-partum. Sows were restrained by a snout snare and blood samples were collected via jugular venipuncture using an 18 g 1.5” vacutainer needle and 9 mL Lithium Heparin vacutainer tubes (BD Vacutainer, BD, Belliver Industrial Estate, Plymouth, UK). For day 1 post-parturition samples only, 5-10 µL of blood was immediately analysed for blood chemistry, metabolites and gases using an EPOC blood analysis system (Siemens Healthineers®; Ottawa, Canada). Blood samples were then placed on ice, transported to the laboratory, and processed within 2 h of collection. Blood samples were centrifuged at 1512 g for 20 minutes at room temperature and plasma was stored in triplicate at -20°C until analysis.

Blood sample analysis

Aliquots of frozen plasma were transported to The University of Sydney Veterinary Pathology Diagnostic Services (Camden, NSW, Australia) for 3-calcidiolroxybutyrate (BHB), Ca, cholesterol, magnesium (Mg), non-esterified free fatty acids (NEFA) and phosphorus (P) analysis; and The University of Adelaide Research Assay Facility (Adelaide, SA, Australia) for leptin and insulin analysis. A final aliquot of plasma was analysed for osteocalcin

concentration at South Australian Research and Development Institute's Turretfield Research Centre (Rosedale, SA, Australia). Plasma Ca, cholesterol and P were measured using Thermo Fisher Scientific Oy kits 981367/981772, 981813/981812 and 981890/981891, respectively, according to manufacturer's protocols on a Konelab 20XTi analyzer (Thermo Fisher Scientific, Oy). Plasma BHB, Mg and NEFA were measured using Randox Laboratories kits RB 1007, MG 3880 and FA 115 respectively according to manufacturer's protocols.

Plasma leptin and insulin were measured in duplicate using the radioimmunoassay (RIA) kits Multi-Species Leptin RIA Kit (cat. no. XL-85K, Merck Millipore, Darmstadt, Germany) and Porcine Insulin RIA Kit (cat. no. PI-12K, Merck Millipore, Darmstadt, Germany), respectively. The leptin and insulin assays were performed according to the manufacturer's instructions, including standard and sample tubes. Plasma osteocalcin was measured in duplicate using a commercially available enzyme-linked immunosorbent assay (ELISA) (N-MID® Osteocalcin Enzyme-Linked Immunosorbent Assay, IDS Immunodiagnostic Systems GmbH, Frankfurt am Main, Germany).

Farrowing characteristics and piglet weights

The number of piglets born alive, dead, and mummified was recorded for each sow. All sows that farrowed during the day were supervised by piggery attendants with sows checked every 30-60 minutes. An internal examination was performed if a piglet was stillborn, or, if an inter-piglet interval exceeded 45 minutes. The number of internal examinations and the number of piglets pulled alive and dead was recorded.

At the time of litter processing (day 0-1 post-parturition), piglet sex was recorded, and all piglets had an individual radio frequency identification (RFID) ear tag inserted. Piglets were individually weighed at litter processing, and at three, 21 days of age and at weaning. Any fostered piglets (including those with no RFID tags) were also weighed to calculate total litter weights for each sow. At approximately 115 days of age, all purebred progeny were weighed and backfat at the P2 position was measured using a Renco Lean Meater® (Renco Corp, Minneapolis, MN).

Colostrum and milk collection and analysis

For sows that farrowed during the day, a colostrum sample (5-10 mL) was collected across all teats after the birth of the first piglet. Colostrum was immediately analysed for total solid content (%) using a digital handheld refractometer (Starr Instruments: Model DBR-1). Colostrum was then frozen at -20°C until immunoglobulin G (IgG) analysis. The IgG concentration was determined by a previously validated radial-immuno diffusion assay developed by the University of Adelaide's Veterinary Diagnostic Laboratory (Roseworthy Campus, Roseworthy). Methods were utilised by a previous method described by Brougham et al. (2020) where 150 µL swine antigen, and 0.5, 0.25, 0.125 and 0.063 mg/ml of purified swine IgG was used in place of the ovine antigen and purified ovine IgG respectively. The colostrum samples were diluted with phosphate buffered saline to a 1:160 dilution prior to IgG analysis.

Two days post-weaning, milk samples (50 mL) were collected from subset sows only. Sows were walked to individual stalls, and milk was collected naturally and pooled from all functional teats. Following sample collection, sows were moved back to their weaning pen. Milk samples were placed into vials containing a milk preservative and shipped to a commercial milk analysis laboratory (Dairy Express Herd Recording Service, University of New England, Armidale, NSW, Australia) to determine fat, protein, and lactose percentage, and urea and somatic cell content.

Faecal consistency score

To determine the degree of sow constipation, a daily visual faecal consistency score was recorded for all un-farrowed sows in the farrowing house. Faecal scores were recorded each morning prior to faeces being scraped from behind the sows. A scoring system of 0 to 5 was used as described by Oliviero et al. (2009) with the following definitions: 0 (absence of faeces), 1 (dry and pellet shaped), 2 (between dry and normal), 3 (normal and soft, but firm and well formed), 4 (between normal and wet, still formed but not firm), and 5 (very wet faeces, unformed and liquid).

Sow and piglet health and mortalities

Sows and piglets were monitored daily for any signs of ill-health by trained piggery attendants. Any incidence of sow mastitis, udder oedema, udder engorgement, vaginal discharge or retained piglets/placenta was recorded as was any medication administered. Any sow or piglet deaths were recorded including date and cause of death, and piglets were euthanised if any deformities or health issues negatively impacted quality of life. Common causes of piglet mortality were stillborn, overlay, illthrift, diarrhoea, deformity and low birthweight. Deaths with a low prevalence were incorporated into the category "other" to enable analysis.

Sow survival and general censoring

Sows were terminated from their treatment on the date they were mated post-weaning, culled, died or reached day 30 post-weaning, whichever occurred first. Sows that died or were culled were terminated from the weaning data on the date they were removed from the herd. These sows were censored from the survival and reproduction data at that point.

Subsequent reproduction

Weaning to oestrus length (days) was recorded and subsequent reproduction was recorded. Measures included conception rate, pregnancy rate, litter size, and number of piglets born alive and dead.

Sample size estimations

The unit of interest used for sample size determination in this study was the piglet and the key outcome of interest was the stillborn piglet. We estimated an increase in the number of piglets born alive by 0.2 per litter (Effect Size 0.25) with the number of piglets per litter per treatment group being estimated from farm data at 12. The SD was 1.40 and mean number of stillborn was 1.07 based on 5,327 previous farrowings. This provided a power of 0.81 with an α of 0.05. The estimates were made using *rdpower* (Stata Corp Tx) with 70 clusters (sows) at level 2, 12 piglets per litter and an intraclass correlation of 0.2. The total piglet number estimated was 1680.

Statistical analysis

All statistical analysis was conducted using Stata Version 17 (StataCorp LLC, https://www.stata.com). Initial evaluation included tabulation of data by categorical outcomes and visual and statistical of appraisal of continuous variables for normality of distribution and the need to transform data to achieve a normal distribution. The unit of interest was the sow for most measures. For all piglet individual weight, weight gain and survival, piglet was the unit of interest. Data was analysed in two ways, firstly all five treatments were compared (five-way analysis) and secondly, only the four transition diets

were compared to look at the effect of DCAD and vitamin D source and their interactions (factorial analysis).

Due to the extensive and differing nature of the observed outcomes and differences in the unit of interest, several different statistical approaches were used. For continuous data for sows a mixed-models analysis (*mixed*) was conducted with the random effect of sow within block. For continuous data for piglets a mixed-models analysis was conducted with the random effect of piglet with sow within block. For count data, such as still birth, following initial exploration of the data, a Poisson regression indicated that the data were over-dispersed and use of a negative binomial model (*xtnbreg*) for data analysis was indicated (Rodney et al., 2016). Lactational incidence data including disease were either evaluated using a mixed-effects multi-level model using the *melogit* function provided an evaluation of the odds of disease. The models included sow within block as a random effect for sow data or piglet within sow within block. Survival data such as time to removal were evaluated using survival analysis methods including Cox's proportional hazards model (*stcox)* and results were evaluated based on Schoenfeld residual tests and Cox-Snell residuals. Visual appraisal of Schoenfeld and Cox-Snell residuals indicated an adequate fit with large values for time deviating from the ideal hazard function. Where the Cox model distribution was not suitable or tested against the Weibull parametric survival model using Akaike's information criterion and Bayesian information criterion and found to have a lesser fit and very similar estimates of effects, a Weibull model was used for these variables.

Post hoc analyses included analysis of the significance of main effects and interactions that were tested with *compare*, marginal means were calculated with *margins* and pairwise comparisons were made with *pwcompare*.

3. Outcomes

The Five-way analysis

Sow body composition

Sow liveweight, backfat at the P2 position, and changes in liveweight and backfat are presented in Table 3. At entry to the farrowing house (FH), positive DCAD sows were heavier than control, and both negative DCAD treatments ($P < 0.05$); however, by day 21 of lactation, this difference was no longer evident. Furthermore, sow liveweight changes over the course of the experiment did not differ among treatments. At farrowing house entry, primiparous sows were lighter than multiparous sows ($P < 0.05$), but by day 21 of lactation there was no difference in liveweight between primiparous and multiparous sows.

Backfat did not differ among treatments at farrowing house entry nor at days 1, 21 of lactation or at weaning. However, backfat change from farrowing house entry to weaning was reduced in negative DCAD + calcidiol-fed sows compared to control-, negative DCAD- and positive DCAD + calcidiol-fed sows ($P < 0.05$). Primiparous sows by day 21 had significantly higher backfat ($P < 0.05$) than multiparous sows. By weaning, this difference was no longer evident.

There was a significant treatment by parity interaction for backfat change from farrowing house entry to day 21 of lactation. Within primiparous sows only, positive DCAD + calcidiol-fed sows lost more backfat than both control- and negative DCAD + calcidiol-fed sows (-7.81 ± 0.92, -5.17 ± 0.84 and -4.97 ± 0.84 mm respectively; $P < 0.05$). For multiparous sows, control sows lost more back fat than negative DCAD + calcidiol-, positive DCAD- and positive DCAD + calcidiol-fed sows (-6.69 ± 0.53, -5.18 ± 0.52, -4.95 ± 0.58 and -5.09 ± 0.60 mm respectively; $P < 0.05$; Table 3).

Table 3. Sow liveweight, backfat at the P2 position, and liveweight and backfat change over the experimental period for diets: control, negative DCAD, negative DCAD + calcidiol, positive DCAD and positive DCAD + calcidiol. Values for treatments are marginal means and standard error.

	Treatment (T)					Parity (P)		*P*-value		
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	T	P	T x P
Liveweight, kg										
FH entry[1]	337.8 ± 3.2[a]	337.3 ± 3.3[a]	335.8 ± 3.2[a]	349.5 ± 3.3[b]	342.2± 3.3[ab]	310.5 ± 2.8	353.8 ± 1.9[a]	0.046	<0.001	0.965
Day 21[2]	300.0 ± 1.5	302.2 ± 1.5	300.6 ± 1.5	298.2 ± 1.6	301.3 ± 1.6	298.9 ± 1.4	301.2 ± 0.9	0.343	0.170	0.117
Weaning[2]	293.3 ± 1.9	295.4 ± 1.9	291.4 ± 1.9	289.1 ± 1.9	293.1 ± 2.0	291.2 ± 1.8	292.9 ± 1.2	0.137	0.409	0.524
Weight change, kg										
Day 1 to 21[3]	-23.1 ± 3.0	-20.1 ± 3.2	-20.7 ± 2.7	-20.4 ± 2.8	-19.4 ± 27	-22.6 ± 2.7	-19.7 ± 1.7	0.970	0.408	0.983
FH entry to day 21[2]	-39.8 ± 1.5	-37.6 ± 1.5	-39.2 ± 1.5	-41.7 ± 1.6	-38.5 ± 1.6	-41.0 ± 1.4	-38.6 ± 0.9	0.343	0.170	0.117
FH entry to weaning[2]	-46.8 ± 1.8	-44.6 ± 1.9	-48.6 ± 1.8	-50.2 ± 1.8	-46.9 ± 1.9	-48.7 ± 1.8	-46.9 ± 1.1	0.181	0.391	0.436
Backfat, mm										
FH entry	28.9 ± 0.6	28.6 ± 0.6	27.5 ± 0.6	28.5 ± 0.6	28.2 ± 0.7	27.8 ± 0.5	28.6 ± 0.3	0.698	0.217	0.992
Day 1	28.1 ± 0.6	27.3 ± 0.6	27.6 ± 0.6	27.2 ± 0.6	27.6 ± 0.6	28.5 ± 0.6	27.2 ± 0.4	0.964	0.069	0.863
Day 21[3]	22.1 ± 0.6	21.7 ± 0.7	23.0 ± 0.6	22.6 ± 0.6	22.7 ± 0.7	23.8 ± 0.6	21.8 ± 0.4[a]	0.840	0.008	0.912
Weaning[4]	22.1 ± 0.6	20.7 ± 0.6	21.6 ± 0.6	21.7 ± 0.6	21.6 ± 0.6	22.2 ± 0.5	21.3 ± 0.3	0.374	0.179	0.857
Backfat change, mm										
FH entry to day 21[5]	-6.2 ± 0.4	-6.3 ± 0.5	-5.1 ± 0.4	-5.5 ± 0.5	-5.9 ± 0.5	-6.3 ± 0.4	-5.6 ± 0.5	0.244	0.159	0.022
FH entry to weaning	-6.9 ± 0.5[a]	-7.2 ± 0.5[a]	-5.4 ± 0.5[b]	-6.0 ± 0.5[ab]	-6.4 ± 0.5[a]	-6.9 ± 0.5	-6.2 ± 0.3	0.047	0.180	0.213

FH = farrow house.[ab] Different superscripts within a row and within treatment or parity indicate pairwise comparisons with $P < 0.05$.
[1] Covariable was days on diet (P <0.02) and breed was not significant ($P > 0.10$).
[2] Covariables were breed ($P < 0.001$) and farrow house entry weight ($P < 0.05$).
[3] Covariable was significant for breed ($P < 0.001$). [4] Covariables was significant for farrow house entry weight ($P < 0.05$).

Feed intake

Feed intake from day of parturition until day four of lactation was higher in control sows (17.1 ± 0.1 kg) compared to negative DCAD- (16.3 ± 0.1 kg), negative DCAD + calcidiol- (16.2 ± 0.1 kg), positive DCAD- (16.2 ± 0.1 kg) and positive DCAD + calcidiol-fed sows (16.1 ± 0.1 kg; $P < 0.05$). However, there was no treatment effect ($P > 0.05$) on average daily feed intake from day of parturition until day 21 of lactation (overall mean 6.4 ± 0.1 kg) or total feed intake from parturition until day 21 of lactation (overall mean 135.0 ± 1.7 kg).

Urine pH and faecal consistency

Differences were observed in urine pH among treatments before farrowing. Control sows had higher urine pH values than all four DCAD treatments (Figure 1; $P < 0.01$), while both negative DCAD treatments had a lower urine pH than the two positive DCAD treatments (Figure 3; $P < 0.01$). Post-farrowing, all five treatments were similar in urine pH value (Figure 3; $P > 0.05$). There was no effect of parity on urine pH at any timepoint ($P > 0.05$).

Prior to farrowing, both control sows and negative DCAD-fed sows had slightly lower faecal consistency scores than negative DCAD + calcidiol, positive DCAD- and positive DCAD + calcidiol-fed sows (2.2 ± 0.1, 2.2 ± 0.1, 2.4 ± 0.1, 2.7 ± 0.1 and 2.6 ± 0.1 respectively; $P < 0.01$), but all were close to a 'normal' consistency. Primiparous sows also had a higher faecal consistency score than multiparous sows (2.6 ± 0.1 and 2.3 ± 0.1 respectively; $P < 0.05$).

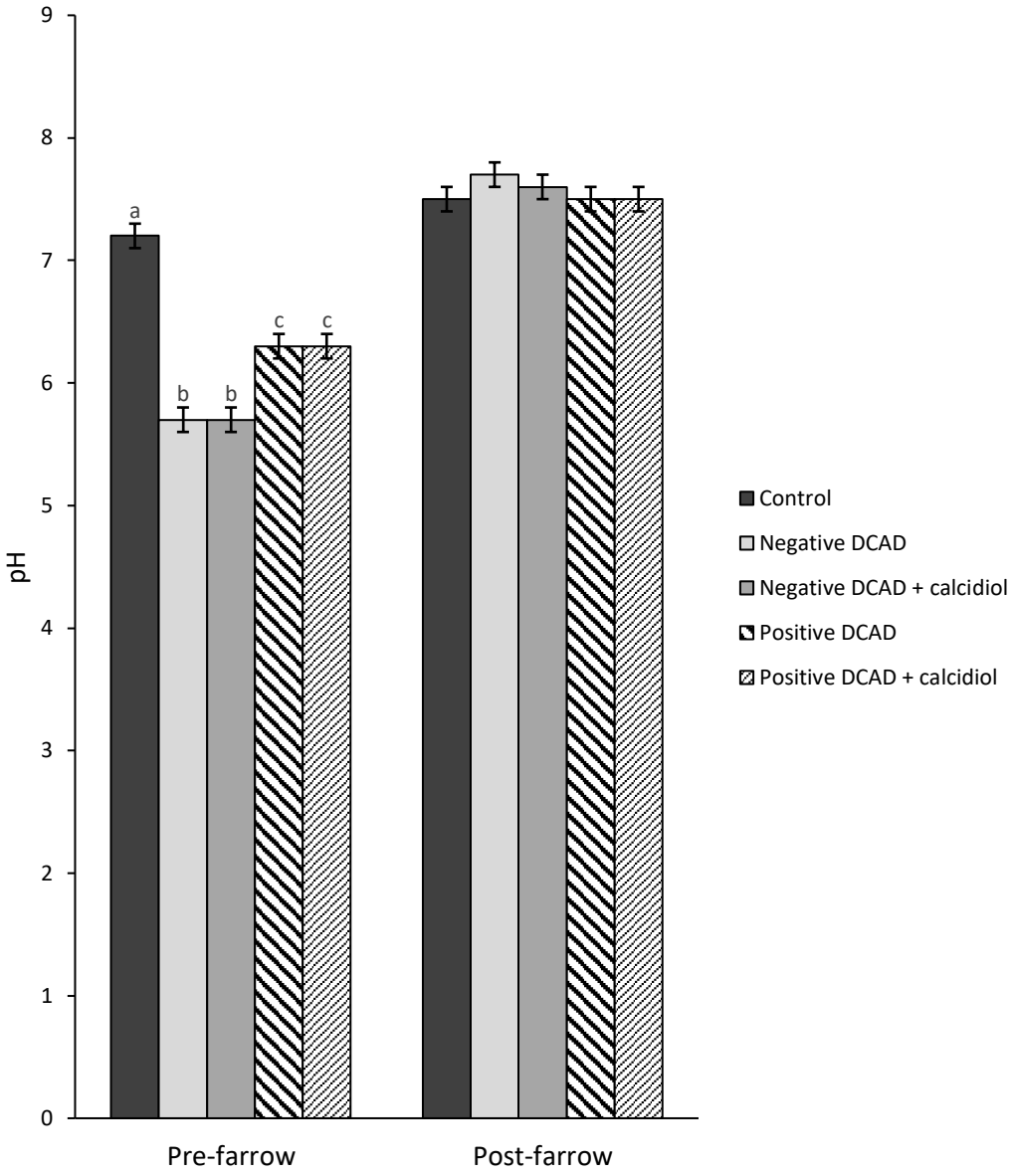

Figure 1. Sow urine pH prior to farrowing and post farrowing of control sows, and sows that received either a positive or negative DCAD diet, and with or without calcidiol. Different superscripts within a timepoint indicate $P < 0.01$.

Farrowing and litter characteristics

Total born piglets did not differ among all five treatments ($P > 0.05$); however, positive DCAD sows had more piglets born alive than control-fed sows ($P < 0.05$; Table 4). Litter birthweight, including both piglets born alive and stillborn, was unaffected by both treatment and parity.

The relative risk of a sow having a stillborn piglet was reduced for negative DCAD + calcidiol-fed sows (Table 5) compared to all other groups except negative DCAD and positive DCAD-fed sows. Multiparous sows had 1.47 ± 0.25 times greater risk ($P < 0.05$) of having a stillborn piglet compared to primiparous sows (Table 5). However, the negative DCAD + calcidiol-fed sows had an increased probability of having a mummified piglet within a litter compared to the positive DCAD-fed groups, but there was no effect of parity (Table 5; $P > 0.05$).

By day three post-partum, negative DCAD + calcidiol- and positive DCAD-fed sows had more piglets in each litter compared to control- and positive DCAD + calcidiol-fed sows. However, this was not accompanied by an increase in litter weight (Table 4). By day 21 post-partum, differences among treatments were no longer evident (Table 4).

At weaning, there was no effect of treatment or parity on the number of piglets weaned per sow, although there were significant ($P < 0.05$) treatment by parity interactions (Table 4). Within multiparous sows only, sows fed the negative DCAD + calcidiol diet weaned more piglets than negative DCAD-, positive DCAD-, and positive DCAD + calcidiol-fed sows (10.5 ± 0.2, 9.9 ± 0.2, 9.9 ± 0.2 and 9.8 ± 0.2 piglets respectively; $P < 0.05$). Positive DCAD-fed sows weaned more piglets than positive DCAD + calcidiol-fed sows ($P < 0.05$). Multiparous control sows weaned 10.10 ± 0.2 piglets.

Table 4. The number of total born piglets, piglets born alive, % stillborn, and litter weights and piglet numbers per sow throughout the experiment for diets: control, negative DCAD, negative DCAD + calcidiol, positive DCAD and positive DCAD + calcidiol. Values for treatments are marginal means and standard errors.

	Treatment (T)					Parity (P)		*P*-value		
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	T	P	T x P
Total born, *n*[1]	12.7 ± 0.4	13.5 ± 0.4	13.2 ± 0.4	13.9 ± 0.4	13.2 ± 0.4	12.8 ± 0.3	13.4 ± 0.2	0.169	0.186	0.346
Born alive, *n*[1]	11.5 ± 0.4[a]	12.1 ± 0.4[ab]	12.3 ± 0.4[ab]	12.9 ± 0.4[b]	11.9 ± 0.4[ab]	12.0 ± 0.3	12.2 ± 0.2	0.047	0.587	0.414
% Stillborn[2]	10.9 ± 1.2[a]	8.9 ± 1.2[ab]	6.2 ± 1.2[b]	7.1 ± 1.3[ab]	9.3 ± 1.2[a]	7.7 ± 1.1	8.8 ± 0.7	0.050	0.390	0.420
Litter birthweight, kg	21.5 ± 0.5	22.1 ± 0.5	22.0 ± 0.5	23.1 ± 0.5	21.9 ± 0.5	21.8 ± 0.5	22.3 ± 0.3	0.102	0.496	0.165
Day 3 post-farrow										
Litter size, *n*[3]	12.6 ± 0.1[a]	12.8 ± 0.1[ab]	13.0 ± 0.1[b]	13.0 ± 0.1[b]	12.6 ± 0.1[a]	12.8 ± 0.1	12.8 ± 0.1	0.014	0.967	0.703
Litter weight, kg	29.1 ± 0.5	28.4 ± 0.5	29.0 ± 0.5	28.5 ± 0.5	28.8 ± 0.5	28.8 ± 0.4	28.7 ± 0.3	0.870	0.786	0.480
Day 21 post-farrow										
Litter size, *n*[2]	10.1 ± 0.2	10.1 ± 0.2	10.3 ± 0.2	10.1 ± 0.2	10.0 ± 0.2	10.2 ± 0.1	10.1 ± 0.1	0.990	0.564	0.081
Litter weight, kg	87.1 ± 1.5	85.5 ± 1.6	87.0 ± 1.5	84.2 ± 1.6	86.7 ± 1.6	85.4 ± 1.5	86.4 ± 1.0	0.708	0.626	0.300
Number weaned[4]	10.0 ± 0.2	10.0 ± 0.2	10.3 ± 0.2	10.1 ± 0.2	10.0 ± 0.2	10.1 ± 0.1	10.1 ± 0.9	0.900	0.607	0.025

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$.
[1] Covariables were breed of sow and days on diet ($P < 0.05$).
[2] Covariable was farrow house entry weight ($P < 0.05$).
[3] Covariables were days on diet and farrow house entry weight ($P < 0.05$).
[4] Covariables were breed of sow and farrow house entry weight ($P < 0.05$).

Table 5. Number, relative risks (RR) and significance of piglets stillborn or mummified per sow for diet treatment groups: control, negative DCAD, negative DCAD + calcidiol, positive DCAD and positive DCAD + calcidiol.

	Treatment					Parity		Treatment RR[1] (*P*-value)				RR (*P*-value)
Disorder	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Parity[2]
Stillborn, *n per sow*	1.18 [b]	1.32[b]	0.81[a]	1.04[ab]	1.29[b]	0.68	1.32	1.02 (0.898)	0.68 (0.037)	0.76 (0.131)	1.07 (0.702)	1.47 (0.020)
Mummified, *n per sow*	0.45 [ab]	0.59 [ab]	0.75[b]	0.68 [a]	0.37[a]	0.45	0.62	1.21 (0.435)	1.51 (0.097)	0.89 (0.676)	0.74 (0.298)	1.00 (0.990)

[1]Reference group is Control.
[2]Reference group is primiparous.
[ab] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$.

Blood metabolites

At entry to the farrowing house, significant effects of treatment ($P < 0.05$) were found in some blood metabolite measures (Table 6). Control and negative DCAD-fed sows had lower cholesterol than negative DCAD + calcidiol- and positive DCAD + calcidiol-fed sows. Cholesterol was greater than all other groups for the positive DCAD + calcidiol-fed sows. Negative DCAD-fed sows had lower glucose and higher BHB concentrations than all other treatments, except for BHB in negative DCAD + calcidiol fed sows. Magnesium was higher in control sows compared to all other groups. Negative DCAD + calcidiol-fed sows had higher leptin concentrations than all other treatments. At farrowing house entry, treatment had no effect on Ca, P, insulin or osteocalcin ($P > 0.05$). Primiparous sows had higher phosphate, insulin and osteocalcin concentrations than multiparous sows at the time of farrowing house entry ($P < 0.05$).

Table 7 presents means and SEM for blood metabolite measures on day one post-parturition, including an evaluation of acid base status. Pairwise comparisons values and significance for the treatment by parity comparisons are in Supplementary Table 1.

Control-fed sows had higher blood pH, higher blood urea nitrogen and lower Cl- than other treatments ($P < 0.05$). Control-fed sows had higher oxygen partial pressure than other diet groups *(*$P < 0.01$). The base excess in the extracellular fluid compartment was lower for both negative DCAD-fed groups than for control sows and the positive DCAD + calcidiol-fed sows, but not significantly less than for the positive DCAD-fed sows ($P < 0.05$). Control- and positive DCAD + calcidiol-fed sows had greater base excess in blood than other sows ($P < 0.05$). Ionized calcium in blood was numerically lower in control sows ($P = 0.05$) than all other groups. Blood glucose concentrations were greater in negative DCAD + calcidiol-fed sows than all other groups except for the control-fed sows ($P < 0.05$).

The blood gas, mineral and metabolite concentrations are consistent with feeding negative or more negative DCAD diets and the tendency for an increase in osteocalcin (Table 7) is consistent with observations in dairy cows (Rodney et al., 2018). DeRouchey et al. (2003) lowered the dEB of lactating sow diets and found decreased base excess of blood and extracellular fluid, bicarbonate, partial pressure of carbon dioxide. Guo et al. (2019) found that a lower DCAD diet in late gestation and in-lactation reduced blood and urine pH; a finding consistent with this study. We found a tendency to increased blood Ca concentrations at farrowing for the transition fed sows (Table 7), which contrasts with Roux et al. (2008) but is consistent with studies in dairy cows (Rodney et al., 2018) and in lactating sows (Guo et al., 2019). DeRouchey et al. (2003) and Cheng et al. (2015) who fed diets with a lower dEB also found increased blood chlorine concentrations.

Primiparous sows had higher oxygen partial pressure and cholesterol than multiparous sows on day one after parturition ($P < 0.05$). Many significant treatment by parity interactions were present on the day after parturition and these highlight important differences in parity responses, especially in control-fed sows. The only significant pairwise difference for parity and treatment was within the control group for blood pH (primiparous 7.56 ± 0.03; multiparous 7.47 ± 0.02). The greatest pairwise difference for blood pH, carbon dioxide partial pressure, oxygen partial pressure, blood bicarbonate, base excess in blood, oxygen saturation %, sodium concentration, total carbon dioxide concentration, anion gap, hematocrit % and hemoglobin concentration was for the control group parity difference. These marked differences in metabolism within the control groups are attributable to parity effects and support observations in cattle that parity has a substantial influence on responses to diets in transition (Lean et al., 2014; 2019). The physiological basis for the difference in parity responses has not been established but may reflect effects of aging on

metabolic robustness and demands of previous gestation and lactation reflected in labile nutrient reserves.

Primiparous control sows had lower carbon dioxide partial pressure (39.33 ± 3.37) than all other treatment by parity groups, apart from primiparous negative DCAD-fed sows (46.19 ± 4.32), with all other groups having >50 mm of Hg for carbon dioxide partial pressure. Primiparous control (67.30 ± 4.24) and negative DCAD-fed primiparous sows (54.52 ± 5.38) had higher oxygen partial pressure all other treatment groups that did not exceed 40 mm Hg oxygen partial pressure. The blood bicarbonate concentration was least for primiparous controls (33.65 ± 1.12), and greatest for multiparous controls (38.25 ± 0.70) mmol/L, with other treatment by parity groups being intermediate to these concentrations. The base excess concentrations were similar for the treatment by parity groups, however, the multiparous controls had higher concentrations (14.63 ± 0.66 mmol/L) than all but the primiparous positive DCAD + calcidiol-fed sows (14.14 ± 0.86 mmol/L). The latter group had concentrations higher than the primiparous controls and the negative DCAD groups. The oxygen saturation percentage for the primiparous controls (90.54 ± 4.14%) were higher than all other comparisons except the negative DCAD group (78.97 ± 5.30%) and were markedly higher than the control multiparous sows that had similar concentrations (67.92 ± 2.57%) to all other treatment by parity groups. The blood sodium concentration was also least for the primiparous controls (143.84 ± 1.01 mmol/L) and differed from all other treatment by parity groups, except primiparous negative DCAD, negative DCAD + calcidiol and positive DCAD groups; which did not differ from any other comparison. Primiparous control sows (33.29 ± 1.22) had lower total carbon dioxide mmol/L than control primiparous sows (37.90 ± 0.76) and primiparous positive DCAD + calcidiol (36.61 ± 0.99). The positive four DCAD treatment by parity groups had similar total carbon dioxide concentrations in blood to the multiparous controls with the exception of the positive DCAD multiparous sows that were lower. The control and negative DCAD fed primiparous sows had the greatest anion gaps but did not differ to each other (control 16.30 ± 0.65, negative DCAD 15.33 ± 0.83 and negative DCAD + calcidiol 15.11 ± 0.52 mmol/L, respectively). The multiparous control sows had a lesser anion gap than the primiparous control, negative DCAD and negative DCAD + calcidiol sows (14.00 ± 4.04; $P < 0.05$) but were similar to all groups other than the multiparous positive DCAD sows that had a greater anion gap ($P < 0.05$). Control primiparous sows had markedly greater blood urea nitrogen concentrations (14.33 ± 1.22 mg/dL) than the control multiparous sows (9.45 ± 0.76) and all other groups. However, the multiparous control and negative DCAD (9.16 ± 0.75) sows had higher blood urea nitrogen concentrations than negative DCAD + calcidiol and positive DCAD + calcidiol-fed multiparous sows (6.76 ± 0.79 and 6.77 ± 0.79 mg/dL, respectively; $P < 0.05$), while other groups were similar in concentrations.

At 21 days post-parturition, there was no effect or treatment or parity on any measured blood parameter. Nor were there any significant interactions between treatment and parity (Table 8; $P > 0.05$).

Table 6. Blood metabolite measures of sows at entry to the farrowing house fed different diets. Data are presented as marginal means and standard errors with the effects of treatment, and parity and contrasts for treatment, parity, and their interaction.

	Treatment (T)					Parity (P)		*P* value		
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	T	P	T x P
Calcium, mmol/L	2.56 ± 0.03	2.49 ± 0.03	2.55 ± 0.03	2.57 ± 0.03	2.53 ± 0.03	2.55 ± 0.03	2.54 ± 0.02	0.308	0.670	0.985
Cholesterol, mmol/L[1]	1.82 ± 0.05[a]	1.79 ± 0.06[a]	1.98 ± 0.05[b]	1.92 ± 0.05[ab]	2.15 ± 0.05[c]	1.93 ± 0.04	1.94 ± 0.03	<0.001	0.794	0.761
Glucose, mmol/L	4.43 ± 0.09[a]	3.83 ± 0.11[b]	4.44 ± 0.09[a]	4.44 ± 0.09[a]	4.33 ± 0.10[a]	4.33 ± 0.08	4.31 ± 0.05	<0.001	0.937	0.053
Phosphate, mmol/L	1.90 ± 0.03	1.94 ± 0.03	1.92 ± 0.03	1.91 ± 0.03	1.89 ± 0.03	2.08 ± 0.02	1.83 ± 0.02 [a]	0.780	<0.001	0.151
BHB, mmol/L[2]	0.11 ± 0.02[a]	0.16 ± 0.02[b]	0.13 ± 0.02[ab]	0.11 ± 0.02[a]	0.12 ± 0.02[a]	0.13 ± 0.03	0.12 ± 0.02	0.030	0.564	0.513
Magnesium, mmol/L[1]	0.89 ± 0.02[a]	0.83 ± 0.02[b]	0.84 ± 0.02[b]	0.80 ± 0.02[b]	0.79 ± 0.02[b]	0.84 ± 0.01	0.82 ± 0.01	<0.001	0.403	0.558
NEFA, mmol/L	0.33 ± 0.08[a]	0.75 ± 0.08[b]	0.36 ± 0.07[a]	0.37 ± 0.07[a]	0.51 ± 0.07[a]	0.47 ± 0.06	0.45 ± 0.04	0.001	0.652	0.774
Insulin, uU/mL	1.82 ± 0.10	1.73 ± 0.12	1.96 ± 0.10	1.90 ± 1.10	1.94 ± 0.10	2.04 ± 0.10	1.79 ± 0.06[a]	0.722	0.049	0.893
Leptin, ng/mL	8.08 ± 0.83[a]	6.88 ± 0.91[a]	10.82 ± 0.77[b]	8.66 ± 0.82[a]	8.20 ± 0.84[a]	9.14 ± 1.09	8.29 ± 0.67	0.008	0.521	0.585
Osteocalcin, ng/mL	94.86 ± 8.20	96.94 ± 8.70	110.81 ± 7.14	94.00 ± 7.73	92.68 ± 7.59	112.2 ± 7.28	90.90 ± 4.54[a]	0.403	0.022	0.983

[abc] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$

[1] Covariable was breed of sow ($P < 0.05$).

[2] Covariable was days on diet ($P < 0.05$).

Table 7. Blood acid base, mineral and metabolite measures of sows on day one after parturition fed different diets. Data are presented as marginal means and standard errors with the effects of treatment, and parity and contrasts for treatment, parity, and their interaction.

	Treatment (T)					Parity (P)		*P* value		
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	T	P	T x P
pH	7.50 ± 0.01[a]	7.46 ± 0.02[b]	7.45 ± 0.01[b]	7.45 ± 0.01[b]	7.46 ± 0.01[b]	7.47 ± 0.01	7.46 ± 0.01	0.011	0.323	0.035
Carbon dioxide partial pressure, mmHg	48.52 ± 1.78	49.91 ± 1.99	51.08 ± 1.71	52.24 ± 1.68	52.05 ± 1.71	49.26 ± 1.43	51.62 ± 0.97	0.081	0.130	0.007
Oxygen partial pressure, mmHg	46.01 ± 2.27[a]	40.68 ± 2.51[ab]	36.66 ± 2.15[bc]	33.82 ± 2.12[c]	32.43 ± 2.16[c]	43.50 ± 1.96	34.75[a] ± 1.31	<0.001	<0.001	<0.001
Bicarbonate, mmol/L	36.73 ± 0.60[ab]	35.10 ± 0.66[a]	35.50 ± 0.57[ab]	36.13 ± 0.56[ab]	36.87 ± 0.56[b]	35.43 ± 0.48	35.42 ± 0.32	0.110	0.064	0.008
Base excess (extracellular fluid compartment), mmol/L	13.55 ± 0.57[a]	11.20 ± 0.63[b]	11.53 ± 0.54 [b]	12.16 ± 0.53[ab]	13.08 ± 0.53[a]	11.79 ± 0.45	12.58 ± 0.31	0.030	0.121	0.055
Oxygen saturation, %[1]	75.38 ± 2.21[a]	70.07 ± 2.44[ab]	68.97 ± 2.10[b]	64.74 ± 2.07[b]	64.25 ± 2.07[b]	72.83 ± 1.76	66.33[a] ± 1.19	<0.001	0.001	<0.001
Na+, mmol/L	146.10 ± 0.54	146.89 ± 0.59	146.42 ± 0.51	146.75 ± 0.50	147.14 ± 0.51	146.21 ± 0.45	146.89 ± 0.30	0.164	0.166	0.011
K+, mmol/L	4.34 ± 0.15	4.41 ± 0.16	4.34 ± 0.15	4.49 ± 0.15	4.47 ± 0.15	4.25 ± 0.22	4.49 ± 0.15	0.907	0.377	0.620
Ca++, mmol/L	1.22 ± 0.02[a]	1.28 ± 0.02[b]	1.24 ± 0.02[ab]	1.24 ± 0.02[a]	1.26 ± 0.02[ab]	1.25 ± 0.01	1.24 ± 0.01	0.052	0.626	0.122
Cl-, mmol/L	100.23 ± 0.55[a]	102.98 ± 0.61[b]	101.99 ± 0.52[b]	102.58 ± 0.53[b]	101.57 ± 0.52[ab]	101.82 ± 0.45	101.87 ± 0.30	0.013	0.850	0.412
Total carbon dioxide, mmol/L	36.44 ± 0.65	34.98 ± 0.72	35.22 ± 0.61	35.55 ± 0.61	36.28 ± 0.61	35.04 ± 0.52	36.19 ± 0.35	0.466	0.052	0.039
Anion gap, mmol/L	14.74 ± 0.35	14.28 ± 0.38	14.48 ± 0.38	14.24 ± 0.33	14.36 ± 0.33	14.76 ± 0.28	14.26 ± 0.19	0.222	0.112	0.002
Haematocrit, %	35.38 ± 0.81	33.27 ± 0.94	34.66 ± 0.81	34.17 ± 0.80	35.55 ± 0.81	35.69 ± 0.76	34.14 ± 0.48	0.241	0.097	0.336
Haemoglobin, g/L	120.13 ± 2.85	113.18 ± 3.16	118.09 ± 2.70	115.92 ± 2.67	120.81 ± 2.71	121.13 ± 2.41	116.11 ± 1.61	0.221	0.102	0.314
Base excess (blood), mmol/L	11.99 ± 0.49[a]	9.79 ± 0.54[b]	10.54 ± 0.46[bc]	10.54 ± 0.46[bc]	11.27 ± 0.46[a]	10.30 ± 0.40	10.94 ± 0.27	0.021	0.163	0.156
Lactate, mmol/[2]	3.28 ± 0.27	2.38 ± 0.30	3.04 ± 0.26	3.07 ± 0.25	3.17 ± 0.25	2.86 ± 0.22	3.09 ± 0.15	0.196	0.427	0.224
Blood urea nitrogen, mg/Dl[1]	11.06 ± 0.65[a]	8.60 ± 0.72[b]	7.09 ± 0.62[b]	7.46 ± 0.61[b]	6.86 ± 0.61[b]	8.76 ± 0.52	7.83 ± 0.35	<0.001	0.166	0.030
Creatinine, mg/dL	2.74 ± 0.09	2.63 ± 0.09	2.60 ± 0.08	2.78 ± 0.08	2.61 ± 0.08	2.60 ± 0.07	2.71 ± 0.05	0.467	0.145	0.125
Calcium, mmol/L	2.63 ± 0.04	2.65 ± 0.04	2.64 ± 0.03	2.59 ± 0.03	2.67 ± 0.03	2.65 ± 0.03	2.63 ± 0.02	0.803	0.592	0.781
Cholesterol, mmol/L[2]	1.47 ± 0.06[ab]	1.28 ± 0.06[c]	1.40 ± 0.05[abc]	1.33 ± 0.05[bc]	1.50± 0.05[a]	1.52 ± 0.05	1.34 ± 0.04[a]	0.113	0.003	0.519

Glucose, mmol/L[1]	5.49 ± 0.13[ab]	5.25 ± 0.15[a]	5.83 ± 0.13[b]	5.36 ± 0.13[a]	5.18 ± 0.12[a]	5.48 ± 0.11	5.40 ± 0.07	0.004	0.624	0.489
Phosphate, mmol/L	2.29 ± 0.08	2.25 ± 0.09	2.17 ± 0.07	2.29 ± 0.08	2.23 ± 0.07	2.36 ± 0.07	2.19 ± 0.04 [a]	0.797	0.023	0.894
BHB, mmol/L	0.09 ± 0.04	0.17 ± 0.04	0.11 ± 0.04	0.07 ± 0.04	0.08 ± 0.04	0.07 ± 0.04	0.12 ± 0.03	0.712	0.328	0.850
Magnesium, mmol/L[1]	0.77 ± 0.02	0.72 ± 0.02	0.72 ± 0.02	0.72 ± 0.02	0.68 ± 0.02	0.71 ± 0.02	0.73 ± 0.01	0.111	0.359	0.806
NEFA, mmol/L	0.29 ± 0.07	0.33 ± 0.08	0.38 ± 0.07	0.32 ± 0.07	0.35 ± 0.06	0.30 ± 0.06	0.35 ± 0.04	0.989	0.577	0.431
Insulin, uU/mL	3.13 ± 0.17	2.38 ± 0.20	3.15 ± 0.16	2.81 ± 0.17	2.79 ± 0.15	2.93 ± 0.16	2.84 ± 0.10	0.089	0.738	0.172
Leptin, ng/m[2]	7.67 ± 0.69	6.57 ± 0.82	7.47 ± 0.63	7.98 ± 0.69	8.68 ± 0.65	8.70 ± 0.71	7.23 ± 0.46	0.508	0.115	0.815
Osteocalcin, ng/mL	74.89 ± 5.61[a]	77.97 ± 6.33[ab]	79.20 ± 5.04[a]	66.06 ± 5.52[ab]	59.45 ± 5.28[b]	73.37 ± 5.20	70.24 ± 3.28	0.063	0.713	0.639

[1] Covariable was breed of sow ($P < 0.05$).
[2] Covariable was days on diet ($P < 0.05$).
[abc] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$

Table 8. Blood metabolite measures of sows on day 21 post-parturition fed different diets. Data are presented as marginal means and standard errors with the effects of treatment, and parity and contrasts for treatment, parity, and their interaction.

	Treatment (T)					Parity (P)		*P* value		
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	T	P	T x P
Calcium, mmol/L	2.64 ± 0.02	2.62 ± 0.03	2.64 ± 0.02	2.65 ± 0.02	2.66 ± 0.02	2.64 ± 0.02	2.64 ± 0.01	0.486	0.988	0.307
Cholesterol, mmol/L	2.16 ± 0.09	2.23 ± 0.11	2.17 ± 0.08	2.30 ± 0.09	2.36 ± 0.09	2.14 ± 0.09	2.29 ± 0.05	0.664	0.163	0.470
Glucose, mmol/L	5.57 ± 0.15	5.56 ± 0.18	5.53 ± 0.14	5.61 ± 0.16	5.48 ± 0.15	5.48 ± 0.14	5.58 ± 0.09	0.960	0.686	0.601
Phosphate, mmol/L	1.76 ± 0.04	1.68 ± 0.05	1.73 ± 0.04	1.69 ± 0.05	1.83 ± 0.05	1.68 ± 0.04	1.77 ± 0.03	0.169	0.124	0.632
BHB, mmol/L	0.03 ± 0.01	0.04 ± 0.01	0.05 ± 0.01	0.05 ± 0.01	0.05 ± 0.01	0.04 ± 0.01	0.05 ± 0.00	0.216	0.247	0.736
Magnesium, mmol/L	0.83 ± 0.02	0.86 ± 0.03	0.82 ± 0.02	0.84 ± 0.02	0.84 ± 0.02	0.82 ± 0.03	0.85 ± 0.02	0.835	0.435	0.983
NEFA, mmol/L	0.24 ± 0.04	0.12 ± 0.05	0.23 ± 0.04	0.15 ± 0.04	0.18 ± 0.04	0.22 ± 0.04	0.17 ± 0.03	0.145	0.377	0.091
Insulin, uU/mL	3.62 ± 0.19	3.48 ± 0.23	3.42 ± 0.19	3.72 ± 0.21	3.45 ± 0.20	3.51 ± 0.19	3.55 ± 0.12	0.734	0.996	0.210
Leptin, ng/mL	9.18 ± 0.82	10.00 ± 0.96	10.49 ± 0.78	9.94 ± 0.87	9.82 ± 0.87	9.45 ± 0.99	10.10 ± 0.60	0.885	0.596	0.813
Osteocalcin, ng/mL	116.54 ± 7.55	111.44 ± 9.05	114.94 ± 7.22	115.48 ± 8.34	105.40 ± 8.08	103.05 ± 7.66	117.86 ± 4.76	0.857	0.117	0.691

[abc] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$

Colostrum and milk composition

Colostrum IgG concentration (mg/mL) did not differ among all five treatments (control, 63.99 ± 4.14; negative DCAD, 70.82 ± 4.21; negative DCAD + calcidiol, 62.24 ± 4.39; positive DCAD, 65.91 ± 4.44; and positive DCAD + calcidiol, 72.94 ± 4.33; $P = 0.624$). Similarly, total solids content of colostrum was unaffected by treatment (overall mean, 26.1 ± 0.05 %; $P > 0.05$). Parity did not significantly affect colostrum IgG concentration or total solid %.

The composition of milk 2 days after weaning did not differ among treatments ($P > 0.05$). Across all five treatments, fat averaged 2.74 ± 0.33 %, protein averaged 8.18 ± 0.38 %, lactose averaged 4.61 ± 0.26 %, somatic cells averaged 6.84 ± 0.31 (cells ,000 per mL), and urea averaged 43.93 ± 7.04 mmol/L.

There was a significant treatment by parity interaction for milk somatic cells whereby in primiparous sows, positive DCAD-fed sows had a higher cell content than positive DCAD + calcidiol-fed sows (6.98 ± 0.49 vs 5.78 ± 0.48 cells respectively; $P < 0.05$). In multiparous sows, negative DCAD-fed sows had higher somatic cell content compared to control-, negative DCAD + calcidiol- and positive DCAD-fed sows (7.73 ± 0.38, 6.63 ± 0.34, 6.48 ± 0.36 and 6.55 ± 0.38 cells respectively; $P < 0.05$).

Sow health

The clinical health disorder with the highest incidence was vaginal discharge with an average of 14.5% of sows exhibiting a discharge during lactation. Mastitis had the second highest incidence with an average of 12.4% sows affected, followed by engorged udder with an average incidence of 11.5%. Neither treatment nor parity influenced the odds or probability per day of any of the health disorders occurring (Table 9).

Table 9. Percentage, odds ratio (OR) and significant risk of clinical health disorders for diets: control, negative DCAD, negative DCAD + calcidiol, positive DCAD and positive DCAD + calcidiol sows.

	Treatment (%)					Parity (%)		Treatment OR[1] (*P*-value)				OR (*P*-value)
Disorder	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Parity[2]
Mastitis	7.6	14.3	13.9	12.3	14.1	10.7	13.0	2.15 (0.169)	2.00 (0.210)	1.74 (0.330)	1.95 (0.239)	1.18 (0.718)
Oedema[3]	6.3	7.1	4.2	2.7	6.3	5.4	5.3	1.29 (0.705)	0.69 (0.531)	0.36 (0.231)	0.93 (0.914)	0.40 (0.174)
Engorged udder	17.7	8.6	8.3	13.7	9.4	11.6	11.8	0.43 (0.104)	0.42 (0.100)	0.74 (0.510)	0.49 (0.171)	1.05 (0.908)
Retained	7.6	4.3	1.4	0	4.7	3.6	3.7	0.47 (0.305)	0.15 (0.090)	–	0.55 (0.416)	1.07 (0.929)
Discharge[3]	13.9	15.7	13.9	15.1	14.1	10.7	16.3	1.23 (0.665)	1.05 (0.942)	0.94 (0.901)	0.92 (0.872)	0.72 (0.450)

[1]Reference group is Control
[2]Reference group is primiparous
[3] Covariable was farrow house entry weight ($P < 0.05$)

Piglet liveweight and average daily gain

At birth, piglet liveweight did not differ among treatments or parity group (Table 10). There was no effect of treatment on piglet liveweight at days three, 21 or 115 days of age (Table 10; $P > 0.05$). Parity affected piglet liveweight at 21 days of age whereby piglets born to multiparous sows were heavier than piglets born to primiparous sows (Table 10). Consequently, piglet average daily gain from birth to 21 days of age was greater in multiparous sows compared to piglets born to primiparous sows.

Table 10. Piglet liveweight and average daily gain (ADG) from birth to day 115 of age from sows fed different diets. Data are presented as marginal means and standard errors with the effects of treatment, and parity and contrasts for treatment, parity, and their interaction.

	Treatment (T)					Parity (P)		*P*-value		
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	T	P	T x P
Piglet weight, kg										
Birth[1]	1.68 ± 0.02	1.68 ± 0.02	1.66 ± 0.02	1.67 ± 0.02	1.65 ± 0.02	1.67 ± 0.02	1.66 ± 0.01	0.631	0.686	0.066
Day 3[1]	2.26 ± 0.03	2.23 ± 0.03	2.24 ± 0.03	2.23 ± 0.03	2.24 ± 0.03	2.24 ± 0.02	2.24 ± 0.02	0.752	0.795	0.117
Day 21[2]	8.53 ± 0.08	8.42 ± 0.09	8.40 ± 0.08	8.51 ± 0.08	8.53 ± 0.09	8.35 ± 0.07	8.54 ± 0.05[a]	0.659	0.017	0.264
Day 115[3]	84.35 ± 0.79	84.44 ± 0.71	83.17 ± 0.68	84.14 ± 0.71	83.53 ± 0.82	84.18 ± 0.71	83.83 ± 0.82	0.489	0.614	0.894
Day 115 P2 backfat	10.50 ± 0.13	10.75 ± 0.11	10.52 ± 0.11	10.73 ± 0.11	10.56 ± 0.13	10.61 ± 0.11	10.63 ± 0.06	0.382	0.890	0.786
Piglet ADG, g/day										
Birth to day 21	332.1 ± 3.62	316.3 ± 3.67	317.0 ± 3.52	319.5 ± 3.65	322.9 ± 3.99	314.0 ± 3.04	322.1 ± 2.09[a]	0.600	0.018	0.096
Birth to day 115	731 ± 0.63	731 ± 0.56	716 ± 0.54	724 ± 0.56	722 ± 0.65	726 ± 0.56	724 ± 0.31	0.194	0.763	0.712
Day 21 to day 115	832.5 ± 0.75	842.2 ± 0.67	819.6 ± 0.65	831.1 ± 0.67	825.4 ± 0.78	831.8 ± 0.67	829.6 ± 0.38	0.191	0.673	0.980

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$.

Piglet mortality

The most common causes of piglet mortality were stillborn, overlays, illthrift, and euthanasia due to deformity and low birthweight. The relative risk of a piglet being stillborn was reduced for negative DCAD + calcidiol-fed sows (Table 11) compared to control sows RR 0.56 ± 0.13; $P < 0.05$). The relative risk of a piglet dying as a result of low birthweight was higher in sows fed diets negative DCAD and positive DCAD compared to control piglets (Table 11). The mortality to day 120 was reduced by more than 4% for piglets from the negative DCAD +calcidiol-fed sows compared to the controls and positive DCAD + calcidiol-fed sows.

Table 11. Incidence, percentage of deaths (in brackets), relative risks (RR) and significant risk of piglet mortality causes for sows fed diets: control, negative DCAD, negative DCAD + calcidiol, positive DCAD and positive DCAD + calcidiol sows. Control sows are the reference group. All negative binomial models contained treatment, total born, days of transition and breed of piglet.

	Treatment (%)					Treatment RR[1] (*P*-value)			
Disorder (%)	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol
Stillborn	9.5[b] (46.3)	9.9[b] (50.0)	6.1[a] (38.3)	7.3[a b] (39.4)	9.8[b] (44.5)	0.96 (0.806)	0.63 (0.016)	0.71 (0.067)	1.01 (0.973)
Illthrift	1.2 (6.1)	1.4 (6.7)	2.0 (13.1)	1.5 (8.1)	1.2 (5.5)	1.06 (0.880)	1.54 (0.226)	1.11 (0.791)	0.98 (0.960)
Overlay[1]	5.4 (26.2)	4.3 (21.2)	4.6 (28.6)	4.7 (25.3)	6.3 (28.6)	0.79 (0.343)	0.90 (0.647)	0.85 (0.492)	1.22 (0.392)
Low birthweight[2]	0.19[a] (0.9)	1.3[b] (6.7)	0.46[ab] (2.9)	1.40[b] (7.1)	0.70[ab] (3.2)	5.12 (0.037)	2.10 (0.390)	5.30 (0.033)	3.50 (0.130)
Deformed	0.77 (3.7)	0.57 (2.9)	0.28 (1.7)	0.93 (5.1)	(0.70 (3.2)	0.78 (0.685)	0.34 (0.144)	1.24 (0.698)	0.89 (0.843)
Scours[3]	0.86 (4.2)	0.28 (1.4)	0.37 (2.3)	0.47 (2.5)	0.70 (3.2)	0.28 (0.066)	0.36 (0.095)	0.47 (0.193)	0.70 (0.503)
Mortality to D 120[4]	20.2[bc]	19.8[abc]	15.9[a]	18.2[ab]	21.8[c]	0.92 (0.486)	0.78 (0.029)	0.86 (0.167)	1.07 (0.519)

[1] Breed differences existed for overlay.
[2] Significant increased with increased litter size.
[3] Breed differences existed for scours and the covariate for days on transition was significant with increased days increasing risk.
[4] Breed differences existed for mortality to day 120.
[ab] Superscripts sharing a letter in the same row are not significantly different ($P < 0.05$).

Sow survival and subsequent reproduction

There was no difference among treatments for sows mated within 7 days of weaning, sow death rate or conception and pregnancy rates post weaning (Table 12). In the subsequent litter, treatment tended to increase the number of piglets born (Control, 13.3 ± 0.4; negative DCAD, 14.2 ± 0.5; negative DCAD + calcidiol, 13.6 ± 0.4; positive DCAD, 14.9 ± 0.4; and positive DCAD + calcidiol, 14.5 ± 0.5 piglets; $P = 0.067$). Consequently, there was a tendency for between 0.3 to 1.6 additional piglets to be born in the subsequent litter for the transition diets, all of which acidified metabolism compared to sows fed the lactating diet which did not acidify, nor provide calcidiol or additional fiber. The number of piglets born in the subsequent litter was similar ($P > 0.05$) between primiparous and multiparous sows (14.2 ± 0.2 and 12.89 ± 0.2 piglets, respectively). There was, however, a treatment x parity interaction (P = 0.017; Table 13). In primiparous sows only, positive DCAD-fed sows had more piglets born in the subsequent litter (15.6 ± 0.7) than both control (13.3 ± 0.7) and negative DCAD-fed (12.9 ± 0.7) sows (Table 13). The negative DCAD + calcidiol-fed primiparous sows also had more piglets born in the subsequent litter than negative DCAD-fed sows (15.1 ± 0.7 and 12.9 ± 0.7 piglets, respectively). In multiparous sows, controls produced less piglets in the subsequent litter compared to negative DCAD-fed sows (13.3 ± 0.5 and 14.8 ± 0.6 piglets, respectively; Table 13), and negative DCAD + calcidiol-fed sows produced less piglets in the subsequent litter than negative DCAD, positive DCAD and positive DCAD + calcidiol-fed sows (12.8 ± 0.5, 14.8 ± 0.6, 14.4 ± 0.5 and 14.4 ± 0.6 piglets, respectively; Table 13).

The number of piglets born alive in the subsequent litter did not differ between treatments (Table 13). However, there was a treatment x parity interaction ($P < 0.05$) whereby in primiparous sows, positive DCAD-fed sows had more piglets born alive in the subsequent litter than control- and negative DCAD-fed sows (14.5 ± 0.7, 12.4 ± 0.7 and 12.1 ± 0.7 piglets, respectively), and positive DCAD + calcidiol-fed sows had more piglets born alive than negative DCAD-fed sows (14.1 ± 0.7 and 12.1 ± 0.7 piglets, respectively). In multiparous sows, negative DCAD + calcidiol-fed sows had less piglets born alive than negative DCAD- and positive DCAD + calcidiol-fed sows (11.5 ± 0.5, 13.2 ± 0.5 and 13.0 ± 0.6 piglets, respectively).

These responses differ to Henman et al. (2023) who found similar numbers of piglets born to sows in the subsequent gestation, but a reduction in the number of stillborn piglets resulting in an average of an additional 0.5 piglets for the higher rate of acidogenic protein meal inclusion. The acidogenic protein meal was fed during lactation as well as the prior pre-farrowing interval in that study.

Table 12. Percentage, odds ratio (OR) and significant risk of sows being mated within 7 days of weaning, days to removal, the percent of sows that died during the experiment, and subsequent reproduction (pregnancy and conception rates, and percent of piglets stillborn or mummified in the subsequent litter) for sows fed diets: control, negative DCAD, negative DCAD + calcidiol, positive DCAD and positive DCAD + calcidiol.

	Treatment (%)					Parity (%)		Treatment OR[1] (*P*-value)				OR (*P*-value)
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Parity[2]
Mated within 7 days (%)	86.6	87.1	90.0	88.9	93.1	85.6	90.7	1.23 (0.709)	1.43 (0.507)	1.34 (0.590)	2.33 (0.185	2.33 (0.073)
Median days to mated or removed	5	5	5	5	5	5	5	1.12 (0.536)	1.07 (0.683)	1.05 (0.786)	1.16 (0.432)	1.02 (0.847)
Sow death (%)	1.2	4.8	2.4	2.5	2.5	2.4	2.8	0.92 (0.955)	2.03 (0.570)	1.04 (0.979)	1.01 (0.995)	1.25 (0.836)
% conception	97.0	93.5	98.6	98.4	94.8	99.0	95.4	0.41 (0.327)	2.28 (0.508)	2.05 (0.568)	0.84 (0.867)	.
% pregnant	97.0	91.9	97.1	96.8	93.1	99.0	93.5	0.31 (0.190)	1.08 (0.937)	1.11 (0.919)	0.43 (0.354)	1.00 (0.158)
Subsequent stillborn (mean SD)	1.17 (1.40)	1.28 (1.66)	1.35 (2.06)	1.63 (1.89)	1.29 (1.69)	1.06 (1.43)	1.49 (1.89)	1.04 (0.860)	1.03 (0.875)	1.14 (0.536)	0.87 (0.527)	1.20 (0.292)
Subsequent mummified (n)	0.61 (1.08)	0.51 (0.83)	0.60 (0.92)	0.56 (0.73)	0.44 (0.70)	0.48 (0.94)	0.56 (0.83)	0.74 (0.307)	0.92 (0.745)	0.69 (0.206)	0.65 (0.158)	1.12 (0.623)

[1]Reference group is Control
[2]Reference group is primiparous

Table 13. Subsequent total born and born alive for sows by parity fed treatment diets. Values for treatments are marginal means ± SE. Data are presented as marginal means and standard errors with the effects of treatment, and parity and contrasts for treatment, parity, and their interaction.

	Treatment (T)					Parity (P)		*P*-value		
	Control	-ve DCAD	-ve DCAD + Calcidiol	+ve DCAD	+ve DCAD + Calcidiol	Primiparous	Multiparous	T	P	T x P
Total Born Primiparous	13.31 ± 0.73^{abc}	12.86 ± 0.70^{ab}	15.06 ± 0.74^{cd}	15.62 ± 0.73^{d}	14.64 ± 0.74^{bcd}	14.30 ± 0.33	13.89 ± 0.24	0.067	0.419	0.017
Total Born Multiparous	13.31 ± 0.50^{ab}	14.82 ± 0.56^{cd}	12.80 ± 0.47^{a}	14.44 ± 0.54^{bcd}	14.36 ± 0.59^{bcd}					
Born alive Primiparous	12.35 ± 0.72^{abc}	12.06 ± 0.68^{ab}	13.47 ± 0.72^{bcd}	14.49 ± 0.72^{d}	14.10 ± 0.72^{cd}	13.27 ± 0.34	12.41 ± 0.24^{a}	0.115	0.036	0.049
Born alive Multiparous	12.16 ± 0.49^{ab}	13.20 ± 0.54^{bcd}	11.47 ± 0.46^{a}	12.50 ± 0.51^{abc}	12.99 ± 0.57 ^{bcd}					

^{ab} Superscripts sharing a letter for the same variable are not significantly different ($P < 0.05$).

The Factorial analysis

Sow body composition

Sow liveweight and backfat at the P2 position throughout the trial is presented in Table 14. At farrowing house entry, sows in the negative DCAD treatments weighed less than positive DCAD-fed sows ($P < 0.05$); however, this difference was not evident at day 21 post-partum nor at weaning. Primiparous sows weighed less than multiparous sow at the beginning of the trial ($P < 0.05$), yet at weaning there was no difference between parities. There was a significant interaction between DCAD and vitamin D at weaning whereby negative DCAD-fed sows were heavier than positive DCAD-fed sows in the absence of calcidiol. Furthermore, the negative DCAD-fed sows lost less weight from farrowing house entry to weaning than positive DCAD-fed sows.

Throughout the trial, P2 backfat was unaffected by either DCAD or vitamin D nor their interactions. However, post-farrowing, multiparous sows had consistently lower backfat than primiparous sows ($P < 0.05$).

Table 14. Sow liveweight, backfat at the P2 position, and liveweight and backfat change over the experimental period for sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). Data are presented as marginal means and standard errors.

	Negative DCAD		Positive DCAD		Parity		*P*-value				
		CA		CA	Primiparous	Multiparous	DCAD	Vitamin D	DCAD x Vitamin D	Parity	DCAD x Vitamin D x Parity
Liveweight, kg											
FH entry[1]	337.5 ± 3.2[a]	336.3 ± 3.1[a]	349.0 ± 3.3[b]	342.1 ± 3.3[a]	310.5 ± 2.9	354.7 ± 2.0[a]	0.010	0.223	0.349	0.000	0.807
Day 21[2]	303.0 ± 1.5	301.0 ± 1.5	298.7 ± 1.6	301.9 ± 1.6	298.7 ± 1.6	302.3 ± 1.0	0.147	0.398	0.155	0.073	0.713
Weaning[2]	296.2 ± 2.0[a]	292.2 ± 1.9[ab]	289.9 ± 1.9[b]	293.5 ± 2.0[ab]	290.8 ± 2.1	293.7 ± 1.3	0.169	0.974	0.036	0.234	0.749
Weight change, kg											
FH entry to day 1	-16.1 ± 2.6	-17.3 ± 2.1	-13.4 ± 2.2	-15.8 ± 2.1	-18.0 ± 2.3	-14.3 ± 1.5	0.896	0.359	0.921	0.318	0.191
Day 1 to 21	-20.0 ± 3.4	-21.3 ± 2.7	-21.4 ± 3.0	-19.9 ± 2.8	-21.2 ± 3.2	-20.4 ± 2.1	0.934	0.980	0.701	0.848	0.808
FH entry to day 21[3]	-37.5 ± 1.5	-39.4 ± 1.5	-42.2 ± 1.6	-38.6 ± 1.6	-40.5 ± 1.4	-38.9 ± 1.0	0.092	0.325	0.136	0.340	0.695
FH entry to weaning[4]	-44.4 ± 1.9[a]	-48.5 ± 1.8[ab]	-50.2 ± 1.8[b]	-47.7 ± 1.9[ab]	-49.7 ± 2.0	-46.9 ± 1.2	0.144	0.853	0.061	0.235	0.805
P2 backfat, mm											
FH entry	28.9 ± 0.6	27.6 ± 0.6	28.5 ± 0.6	28.2 ± 0.7	27.8 ± 0.6	28.5 ± 0.4	0.779	0.279	0.496	0.294	0.980
Day 1	27.4 ± 0.6	27.7 ± 0.6	27.2 ± 0.6	27.7 ± 0.6	28.9 ± 0.6	26.9 ± 0.4	0.799	0.647	0.685	0.012	0.568
Day 21	21.6 ± 0.7	23.0 ± 0.6	22.5 ± 0.7	22.6 ± 0.7	23.8 ± 0.7	21.9 ± 0.5	0.744	0.414	0.345	0.023	0.863
P2 backfat change, mm											
FH entry to day 1	-0.8 ± 0.4	-0.3 ± 0.4	-1.4 ± 0.4	-1.0 ± 0.4	-0.9 ± 0.4	-0.9 ± 0.3	0.208	0.268	0.862	0.976	0.418
Day 1 to 21	-4.9 ± 0.5	-4.4 ± 0.5	-4.3 ± 0.5	-5.1 ± 0.5	-5.3 ± 0.5	-4.4 ± 0.3	0.765	0.884	0.071	0.155	0.174
FH entry to day 21	-6.3 ± 0.5	-5.0 ± 0.4	-5.6 ± 0.5	-5.9 ± 0.5	-6.3 ± 0.5	-5.4 ± 0.3	0.452	0.240	0.055	0.111	0.325
FH entry to weaning	-4.9 ± 0.5	-4.4 ± 0.5	-4.3 ± 0.5	-5.1 ± 0.5	-5.3 ± 0.5	-4.4 ± 0.3	0.765	0.884	0.071	0.155	0.174
Day 1 to weaning	-6.1 ± 0.5	-5.7 ± 0.5	-6.1 ± 0.5	-6.2 ± 0.5	-6.5 ± 0.5	-5.8 ± 0.3	0.692	0.501	0.228	0.358	0.078

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$.

[1] Covariable was breed (Not significant $P > 0.05$).

[2] Covariables were breed ($P < 0.001$) and farrow house entry weight ($P < 0.001$).

[3] Covariable was breed ($P < 0.001$)

[4] Covariables were breed ($P < 0.001$), farrow house entry weight ($P < 0.007$), and sire of piglet breed ($P = 0.042$).

Sow feed intake

Sow feed intake throughout the experiment was not impacted by DCAD, Vitamin D, nor their interactions with each other. Average daily intake mean for all four transition treatments from day 1 to 21 post-farrowing was 6.4 ± 0.1 kg. However, multiparous sows ate 200 g more per day than their primiparous counterparts from day 1 to 21 post-farrowing (average intake 6.3 ± 0.1 kg and 6.5 ± 0.1 kg respectively; $P = 0.042$).

Urine pH and faecal consistency

Prior to farrowing, negative DCAD sows had a lower urine pH than positive DCAD sows (5.69 ± 0.07 and 6.29 ± 0.07 respectively; $P < 0.01$). Urine pH prior to farrowing was unaffected by vitamin D, parity or their interactions with DCAD ($P > 0.05$). Post-farrowing, urine pH was similar between all four transition treatments (overall mean 7.54 ± 0.12; $P > 0.05$).

Faecal consistency score was lower in positive DCAD-fed sows (2.26 ± 0.09) compared to negative DCAD-fed sows (2.59 ± 0.10; $P < 0.01$). In addition, there was a significant interaction between DCAD and vitamin D source whereby negative DCAD-fed sows had a lower faecal consistency score compared to the other three treatments (negative DCAD, 2.11 ± 0.09; negative DCAD + calcidiol, 2.40 ± 0.09; positive DCAD, 2.62 ± 0.10; and positive DCAD + calcidiol, 2.67 ± 0.10; $P < 0.05$). Parity also affected faecal consistency score, as multiparous sows had a lower score compared to primiparous sows (2.29 ± 0.06 and 2.67 ± 0.06 respectively; $P < 0.01$).

Farrowing and litter characteristics

The number of piglets born in a litter, piglets born alive and litter birthweight was not affected by DCAD, vitamin D, parity nor their interactions (Table 15). The percentage of stillborn piglets within a litter was not affected by DCAD, vitamin D or parity alone; however, a significant DCAD x vitamin D interaction was evident. Sows that received a negative DCAD transition diet in combination with calcidiol had a lower percentage of stillborn piglets than sows that received either a negative DCAD diet without calcidiol or a positive DCAD diet with calcidiol (Table 15). In addition, negative DCAD + calcidiol-fed sows had a 32% reduced risk of stillborn piglets compared to control sows ($P = 0.038$).

By day 3 post-farrow, litter size and total litter weight remained unaffected by DCAD, vitamin D and parity; however, there was a significant interaction between DCAD and vitamin D whereby negative DCAD with calcidiol- and positive DCAD without calcidiol-fed sows had 0.4 more piglets nursing than positive DCAD with calcidiol-fed sows ($P = 0.005$). By day 21 of lactation, there were no differences between DCAD, vitamin D, parity or their interactions on litter characteristics. Further, at weaning all sows weaned similar numbers of piglets (Table 16).

Individual piglet weight at birth, day 3, 7, 21 or 115 did not change in response to DCAD, vitamin D or their interaction (Table 16; $P > 0.05$). Furthermore, backfat at the P2 position was also unaffected by DCAD and vitamin D at day 115 of age (Table 16; $P > 0.05$). Parity had no impact on piglet weight at birth or at three days of age, although by 21 days of age, piglets born to multiparous sows were 290 g heavier than piglets born to primiparous sows (Table 16; $P < 0.05$). In addition, piglets born to multiparous sows grew 12 grams more per day from birth to day 21 (Table 16; $P = 0.001$). There were two significant interactions between DCAD, vitamin and parity for birthweight whereby piglets born to primiparous sows that received a positive DCAD in the absence of calcidiol were heavier than piglets born to primiparous sows that received a negative DCAD diet without calcidiol, and piglets born to

multiparous sows that received a positive DCAD without calcidiol diet (1.74 ± 0.04, 1.61 ± 0.04 and 1.63 ± 0.03 kg respectively; $P < 0.05$).

Piglet average daily weight gain from birth to day 21 and from birth to day 115, was not impacted by DCAD, calcidiol or their interaction; however, from day 21 to 115, piglets born to sows that received calcidiol grew slightly slower than piglets born to sows that did not receive calcidiol (Table 16; $P = 0.026$).

Table 15. The number of total born piglets, piglets born alive, and litter weights and piglet number per sow throughout the experiment for sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). Values for treatments are marginal means and standard errors.

	Negative DCAD		Positive DCAD		Parity		*P*-value				
		CA		CA	Primiparous	Multiparous	DCAD	Vitamin D	DCAD x Vitamin D	Parity	DCAD x Vitamin D x Parity
Total born, *n*	13.4 ± 0.4	13.2 ± 0.4	13.9 ± 0.4	13.2 ± 0.4	13.1 ± 0.4	13.5 ± 0.2	0.171	0.381	0.458	0.419	0.608
Born alive, *n*	12.0 ± 0.4	12.3 ± 0.3	12.9 ± 0.4	11.9 ± 0.4	12.2 ± 0.4	12.3 ± 0.2	0.180	0.338	0.072	0.893	0.717
% Stillborn	9.0 ± 1.1[a]	6.2 ± 1.0[b]	7.1 ± 1.1[ab]	9.2 ± 1.1[a]	6.6 ± 1.1	8.5 ± 0.7	0.663	0.674	0.043	0.178	0.863
Birth litter weight, kg	22.1 ± 0.5	22.1 ± 0.5	23.1 ± 0.5	21.9 ± 0.5	22.1 ± 0.5	22.3 ± 0.3	0.097	0.256	0.110	0.787	0.149
Day 3 post-farrow											
Litter size, *n*	12.8 ± 0.1[ab]	13.0 ± 0.1[a]	13.0 ± 0.1[a]	12.6 ± 0.1[b]	12.9 ± 0.1	12.8 ± 0.1	0.400	0.223	0.005	0.641	0.487
Litter weight, kg	28.4 ± 0.5	29.0 ± 0.5	28.4 ± 0.5	28.8 ± 0.5	28.9 ± 0.5	28.6 ± 0.3	0.864	0.720	0.751	0.672	0.704
Day 21 post-farrow											
Litter size, *n*	10.1 ± 0.2	10.3 ± 0.1	10.0 ± 0.2	10.0 ± 0.2	10.2 ± 0.2	10.0 ± 0.1	0.999	0.685	0.719	0.285	0.425
Litter weight, kg	85.3 ± 1.7	86.7 ± 1.6	83.6 ± 1.7	86.7 ± 1.7	84.7 ±1.8	86.0 ± 1.2	0.917	0.346	0.456	0.579	0.498
Number weaned	10.0 ± 0.2	10.3 ± 0.2	10.0 ± 0.2	10.0 ± 0.2	10.2 ± 0.2	10.0 ± 0.1	0.944	0.460	0.721	0.202	0.127

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons at $P < 0.05$.
[1] Covariable was breed of sow ($P < 0.05$).
[2] Covariable was farrow house entry weight ($P < 0.05$).
[3] Covariables were breed of sow ($P < 0.05$) and days on diet ($P < 0.001$).
[4] Covariables were breed of sow ($P < 0.05$) and farrow house entry weight ($P < 0.001$).

Table 16. Piglet weight, P2 back fat and average daily gain from sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). Values for treatments are marginal means and standard errors.

	Negative DCAD		Positive DCAD		Parity		*P*-value				
		CA		CA	Primiparous	Multiparous	DCAD	Vitamin D	DCAD x Vitamin D	Parity	DCAD x Vitamin D x Parity
Piglet weight, kg											
Birth[1]	1.67 ± 0.02	1.66 ± 0.02	1.67 ± 0.02	1.64 ± 0.02	1.67 ± 0.02	1.66 ± 0.01	0.731	0.303	0.269	0.778	0.047
Day 3[1]	2.23 ± 0.02	2.23 ± 0.03	2.22 ± 0.03	2.23 ± 0.03	2.23 ± 0.03	2.23 ± 0.02	0.333	0.641	0.872	0.901	0.336
Day 21[2]	8.41 ± 0.09	8.40 ± 0.08	8.49 ± 0.09	8.51 ± 0.10	8.25 ± 0.08	8.55 ± 0.06	0.343	0.794	0.976	0.001	0.443
Day 115[3]	84.7 ± 0.66	83.1 ± 0.63	83.7 ± 0.65	83.3 ± 0.76	83.6 ± 0.66	83.7 ± 0.39	0.743	0.207	0.208	0.873	0.354
Day 115 P2, mm[4]	10.8 ± 0.1	10.5 ± 0.1	10.7 ± 0.1	10.6 ± 0.1	10.6 ± 0.1	10.6 ± 0.11	0.588	0.086	0.403	0.829	0.211
Piglet ADG, g/day											
Birth to day 21[5]	316 ± 4	316 ± 4	319 ± 4	323 ± 4	309 ± 3	322 ± 2	0.352	0.973	0.656	0.001	0.704
Birth to day 115[6]	731 ± 6	717 ± 5	721 ± 6	719 ± 6	720 ± 6	723 ± 3	0.766	0.211	0.156	0.790	0.301
Day 21 to 115[6]	842 ± 7	818 ± 6	830 ± 7	822 ± 7	827 ± 7	827 ± 4	0.641	0.037	0.176	0.985	0.593

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons at $P < 0.05$.
[1] Covariables were breed of sow ($P < 0.001$) and litter size ($P < 0.001$).
[2] Covariable was litter size ($P < 0.001$).
[3] Covariables were breed of sow ($P < 0.001$), days of age at weighing ($P < 0.001$), and whether the piglet was fostered or not ($P < 0.001$).
[4] Covariables were breed of sow ($P < 0.001$) and whether the piglet was fostered or not ($P < 0.001$).
[5] Covariable was litter size ($P < 0.001$).
[6] Covariables were breed of sow ($P < 0.001$), days on transition diet ($P < 0.001$), and whether the piglet was fostered or not ($P < 0.001$).

Table 17. Mortality and neonatal disorders (%) of litters born to sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). The unit of interest is the sow litter. Estimated percentages for treated sows.

T	Treatment (%) Odds Ratio (SE)				(*P*-value)				
	Negative DCAD		Positive DCAD						
Mortality type		CA		CA	DCAD	Vitamin D	DCAD x Vitamin D	Parity	DCAD x Vitamin D x Parity
Stillborn	9.48 referent[a]	6.10 0.72 (0.19) [b]	7.21 0.75(0.19) [ab]	9.80 1.24 (0.30) [a]	0.671	0.483	0.020	<0.001	0.910
Illthrift	1.26 referent	2.00 1.36 (0.58)	1.41 0.95 (0.43)	1.10 0.80 (0.38)	0.361	0.839	0.447	0.309	0.468
Overlay[1]	4.22 referent	4.63 1.06 (0.29)	4.74 1.03 (0.28)	6.17 1.27 (0.34)	0.581	0.491	0.692	0.630	0.995
Low birthweight[2]	1.39 referent	0.47 0.32 (0.20)	0.96 0.70 (0.35)	0.71 0.49 (0.30)	0.981	0.091	0.339	NS	-
Scours	0.28 referent	0.37 1.29 (1.06)	0.48 1.66 (1.32)	0.71 2.48 (0.95)	0.270	0.533	-	NS	-
Other	0.90 referent	0.84 1.08 (0.56)	1.21 1.44 (0.72)	1.09 1.33 (0.67(	0.399	0.998	0.816	0.745	0.422

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$. Covariables tested were litter size, sex of piglet, breed of sow and sire.
[1] Covariable breed of sow ($P = 0.033$). [2] Interactions with parity could not be assessed and sex of piglet was a significant covariable ($P = 0.022$).

Sow blood parameters

Metabolic measures at farrowing house entry are in Table 18. At entry to the farrowing house and after approximately 5 days on diets, DCAD, vitamin D and the interaction between the two dietary components had no effect on circulating calcium, phosphate, insulin or osteocalcin. However, sows fed a negative DCAD transition diet had lower cholesterol and glucose, but higher BHB and magnesium compared to sows that received positive DCAD transition diets (Table 18). These results would be consistent with a lower intake of feed at this time. Sows fed calcidiol had higher circulating cholesterol, glucose and leptin, but lower NEFA than sows without calcidiol indicating less reliance on mobilized lipid and possibly a better energy balance. Sows fed negative DCAD had lower glucose and higher BHB and NEFA than sows that received that received all other transition diets indicating greater reliance on mobilized lipid. Sows fed the negative DCADCA ration had higher circulating leptin than the three other transition rations.

Parity had few effects on circulating blood measures at farrowing house entry except that primiparous sows had a higher P concentration than multiparous sows. There was one significant DCAD, vitamin D and parity interaction whereby primiparous sows fed a negative DCAD diet had higher BHB (0.29 ± 0.02 mmol/L) than all other groups (primiparous negative DCAD + calcidiol, 0.11 ± 0.03; primiparous positive DCAD, 0.09 ± 0.03, primiparous positive + calcidiol, 0.10 ± 0.02; multiparous negative DCAD, 0.15 ± 0.02; multiparous negative + calcidiol, 0.11 ± 0.03; multiparous DCAD, 0.10 ± 0.02; and multiparous positive + calcidiol, 0.12 ± 0.02 mmol/L).

The quite large effects of diet on metabolites after 5 days on diet and at entry to the farrowing house may reflect, in part, reduced DMI of acidogenic diets, as acidogenic diets can reduce DMI in cows (Zimpel et al., 2018). We did not evaluate DMI before farrowing crate entry, however, lower glucose and higher BHB both may reflect reduced DMI.

Table 19 presents sow blood parameters on day 1 post-parturition. The DCAD had a significant effect on several blood parameters with sows that received a negative DCAD transition diet having lower bicarbonate, base excess (extracellular fluid compartment and blood) and higher oxygen partial pressure and osteocalcin levels than sows that received positive DCAD rations ($P < 0.05$). All other blood parameters were similar between negative and positive DCAD rations.

Vitamin D type had minimal effects on most blood parameters at day 1 post-parturition. However, sows that received calcidiol in their transition ration had lower oxygen partial pressure and higher cholesterol than sows that received no calcidiol ($P < 0.05$).

There were minimal significant interactions between DCAD and vitamin D type on blood parameters on day 1 post-parturition, except for Ca ++ whereby sows that received a negative DCAD transition ration without calcidiol had higher circulating levels than sows that received a positive DCAD ration without calcidiol. In addition, negative DCAD plus calcidiol-fed sows had higher glucose levels than sows in the other three transition groups. Finally, sows that received a negative DCAD transition diet without calcidiol had lower insulin than all other groups, whilst sows in the negative DCAD + calcidiol treatment group had higher insulin than all other groups (Table 19; $P < 0.05$).

There were minimal DCAD X vitamin D X parity interactions, except for oxygen partial pressure whereby primiparous sows that received a negative DCAD diet had higher oxygen partial pressure (52.02 ± 5.24 mmHg) than all other groups (primiparous negative DCAD + calcidiol, 33.57 ± 3.10; primiparous positive DCAD, 33.92 ± 3.40, primiparous positive DCAD + calcidiol, 31.94 ± 3.20; multiparous negative DCAD, 34.12 ± 2.50; multiparous negative +

calcidiol, 38.89 ± 2.53; multiparous positive, 35.08 ± 2.70; and multiparous positive + calcidiol, 33.25 ± 2.44 mmHg; $P < 0.05$).

Sow blood parameters on day 21 post-parturition are presented in Table 20. There were no effects of DCAD on any blood parameter, although sows that that received calcidiol had higher P concentrations than sows than received no calcidiol in their transition ration ($P < 0.05$). All other blood parameters were similar between vitamin D types.

Multiparous sows had higher BHB on day 21 post-parturition than primiparous sows ($P < 0.05$). There were no interactions between DCAD, vitamin D or parity for any blood parameter on day 21 post-parturition.

Table 18. Blood metabolite measures of sow at entry to the farrowing house of sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). Values for treatments are marginal means and standard errors.

	Negative DCAD		Positive DCAD		Parity		*P*-value				
		CA		CA	Primiparous	Multiparous	DCAD	Vitamin D	DCAD x Vitamin D	Parity	DCAD x Vitamin D x Parity
Calcium, mmol/L	2.48 ± 0.04	2.55 ± 0.03	2.56 ± 0.03	2.53 ± 0.03	2.55 ± 0.04	2.53 ± 0.02	0.391	0.486	0.085	0.754	0.693
Cholesterol, mmol/L	1.77 ± 0.07	1.97 ± 0.05	1.91 ± 0.06	2.14 ± 0.05	1.99 ± 0.06	1.95 ± 0.04	0.013	0.001	0.960	0.605	0.430
Glucose, mmol/L	3.87 ± 0.12[a]	4.43 ± 0.09[b]	4.45 ± 0.10[b]	4.34 ± 0.09[b]	4.29 ± 0.10	4.30 ± 0.07	0.003	0.010	0.001	0.783	0.477
Phosphate, mmol/L	1.93 ± 0.03	1.94 ± 0.03	1.93 ± 0.03	1.90 ± 0.03	2.00 ± 0.03	1.88 ± 0.02	0.424	0.944	0.450	0.003	0.785
BHB, mmol/L	0.20 ± 0.02[a]	0.11 ± 0.02[b]	0.10 ± 0.02[b]	0.11 ± 0.02[b]	0.14 ± 0.02	0.12 ± 0.01	0.001	0.007	0.001	0.245	0.025
Magnesium, mmol/L	0.83 ± 0.02	0.83 ± 0.01	0.79 ± 0.01	0.78 ± 0.01	0.82 ± 0.02	0.80 ± 0.01	0.005	0.715	0.625	0.379	0.530
NEFA, mmol/L	0.76 ± 0.09[a]	0.35 ± 0.07[b]	0.34 ± 0.07[b]	0.51 ± 0.07[b]	0.57 ± 0.07	0.42 ± 0.05	0.070	0.039	<0.001	0.105	0.260
Insulin, uU/mL[1]	1.73 ± 0.13	1.96 ± 0.10	1.89 ± 0.11	1.94 ± 0.11	2.06 ± 0.12	1.80 ± 0.07	0.476	0.379	0.392	0.082	0.718
Leptin, ng/mL	6.46 ± 0.93[a]	10.77 ± 0.76[b]	8.68 ± 0.82[a]	7.73 ± 0.83[a]	8.52 ± 1.08	8.42 ± 0.65	0.425	0.017	<0.001	0.952	0.317
Osteocalcin, ng/mL	95.79 ± 9.20	111.26 ± 7.34	95.38 ± 7.99	92.71 ± 7.77	111.61 ± 8.35	92.29 ± 5.33	0.265	0.466	0.305	0.081	0.976

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$. A covariable tested was breed of sow. [1] (log transformed).

Table 19. Blood metabolite measures on day one post-parturition of sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). Values for treatments are marginal means and standard errors. Values for treatments are marginal means and standard errors.

	Negative DCAD		Positive DCAD		Parity		*P*-value				
		CA		CA	Primiparous	Multiparous	DCAD	Vitamin D	DCAD x Vitamin D	Parity	DCAD x Vitamin D x Parity
pH	7.45 ± 0.01	7.45 ± 0.01	7.45 ± 0.01	7.46 ± 0.01	7.45 ± 0.01	7.46 ± 0.01	0.734	0.782	0.448	0.611	0.173
Carbon dioxide partial pressure, mmHg	49.84 ± 1.92	51.02 ± 1.61	52.34 ± 1.60	52.08 ± 1.61	51.71 ± 1.46	51.22 ± 1.08	0.115	0.729	0.496	0.926	0.362
Oxygen partial pressure, mmHg	40.69 ± 2.23[a]	36.77 ± 1.87[ab]	34.34 ± 1.86[b]	32.74 ± 1.87[b]	38.70 ± 1.70[a]	34.43 ± 1.18[b]	0.003	0.032	0.170	0.018	0.009
Bicarbonate, mmol/L	35.07 ± 0.66[a]	35.61 ± 0.56[ab]	36.06 ± 0.55 [ab]	36.89 ± 0.56[b]	35.67 ± 0.51	36.09 ± 0.35	0.026	0.147	0.847	0.421	0.910
Base excess (efc), mmol/L	11.17 ± 0.63[a]	11.67 ± 0.53[ab]	12.07 ± 0.52[ab]	13.05 ± 0.53[b]	11.69 ± 0.48	12.22 ± 0.33	0.026	0.094	0.613	0.320	0.741
Oxygen saturation, %	70.05 ± 2.69	69.06 ± 2.25	65.28 ± 2.24	64.28 ± 2.25	69.30 ± 2.05	65.83 ± 1.42	0.034	0.402	0.690	0.123	0.204
Na+, mmol/L	146.5 ± 0.57	146.4 ± 0.47	146.8 ± 0.47	147.2 ± 0.47	147.8 ± 0.52	146.8 ± 0.35	0.229	0.578	0.389	0.808	0.611
K+, mmol/L[2]	4.45 ± 0.16	4.35 ± 0.15	4.50 ± 0.15	4.50 ± 0.15	4.23 ± 0.23	4.56 ± 0.16	0.465	0.597	0.612	0.244	0.990
Ca++, mmol/L	1.28 ± 0.01[a]	1.25 ± 0.01[ab]	1.24 ± 0.01[b]	1.26 ± 0.0[ab]	1.27 ± 0.01	1.25 ± 0.01	0.199	0.733	0.074	0.111	0.330
Cl-, mmol/L	103.0 ± 0.61	101.9 ± 0.52	102.6 ± 0.53	101.6 ± 0.52	102.4 ± 0.48	102.2 ± 0.46	0.916	0.090	0.851	0.846	0.561
Total carbon dioxide, mmol/L	34.95 ± 0.74	35.45 ± 0.62	35.86 ± 0.62	36.27 ± 0.62	35.23 ± 0.57	35.91 ± 0.39	0.127	0.419	0.781	0.285	0.521
Anion gap, mmol/L[2]	14.29 ± 0.35	14.53 ± 0.30	14.26 ± 0.31	14.36 ± 0.30	14.41 ± 0.28	14.34 ± 0.27	0.163	0.502	0.830	0.732	0.213
Haematocrit, %	33.24 ± 0.89[a]	34.68 ± 0.74[ab]	34.23 ± 0.74[ab]	35.65 ± 0.74[b]	35.12 ± 0.67	34.20 ± 0.47	0.113	0.080	0.860	0.322	0.604
Haemoglobin, g/L	113.1 ± 3.00[a]	118.1 ± 2.50[ab]	116.1 ± 2.49[ab]	121.2 ± 2.49[b]	119.3 ± 2.28	116.4 ± 1.59	0.122	0.066	0.868	0.347	0.593
Base excess (blood), mmol/L	9.76 ± 0.53[a]	10.14 ± 0.45[ab]	10.47 ± 0.44[ab]	11.26 ± 0.45[b]	10.08 ± 0.41	10.63 ± 0.28	0.044	0.102	0.556	0.249	0.566
Lactate, mmol/L[2]	2.34 ± 0.29	3.02 ± 0.24	3.11 ± 0.24	3.19 ± 0.24	2.65 ± 0.26	3.10 ± 0.16	0.089	0.209	0.203	0.120	0.671
Blood urea nitrogen, mg/Dl[3]	8.22 ± 0.68	7.06 ± 0.51	7.67 ± 0.54	6.69 ± 0.52	6.75 ± 0.55	7.67 ± 0.37	0.598	0.192	0.890	0.152	0.285
Creatinine, mg/dL	2.63 ± 0.10	2.59 ± 0.09	2.77 ± 0.09	2.61 ± 0.09	2.54 ± 0.08	2.71 ± 0.05	0.350	0.708	0.434	0.060	0.567
Calcium, mmol/L	2.65 ± 0.04	2.64 ± 0.03	2.59 ± 0.03	2.67 ± 0.03	2.67 ± 0.03	2.63 ± 0.02	0.846	0.420	0.389	0.552	0.351
Cholesterol, mmol/L	1.30 ± 0.06[a]	1.42 ± 0.05[ab]	1.32 ± 0.05[a]	1.49 ± 0.05[b]	1.48 ± 0.05[a]	1.35 ± 0.04 [b]	0.329	0.022	0.999	0.020	0.168
Glucose, mmol/L[4]	5.23 ± 0.17[a]	5.82 ± 0.13[b]	5.33 ± 0.13[a]	5.16 ± 0.12[a]	5.43 ± 0.11	5.34 ± 0.08	0.113	0.157	0.002	0.809	0.191
Phosphate, mmol/L[4]	2.26 ± 0.10	2.16 ± 0.07	2.29 ± 0.08	2.29 ± 0.07	2.36 ± 0.07[a]	2.16 ± 0.05 [b]	0.618	0.259	0.700	0.015	0.689
BHB, mmol/L	0.17 ± 0.05	0.11 ± 0.04	0.07 ± 0.04	0.09 ± 0.04	0.08 ± 0.04	0.12 ± 0.03	0.313	0.686	0.492	0.362	0.480
Magnesium, mmol/L[4]	0.73 ± 0.02	0.72 ± 0.02	0.72 ± 0.02	0.68 ± 0.02	0.70 ± 0.02	0.71 ± 0.01	0.321	0.144	0.561	0.762	0.435
NEFA, mmol/L	0.33 ± 0.08	0.38 ± 0.07	0.33 ± 0.07	0.35 ± 0.07	0.30 ± 0.06	0.37 ± 0.04	0.753	0.865	0.975	0.384	0.530
Insulin, µ IU/mL[1]	2.34 ± 0.19[a]	3.16 ± 0.15[b]	2.97 ± 0.16[b]	2.80 ± 0.16[ab]	2.82 ± 0.15	2.87 ± 0.11	0.256	0.053	0.006	0.810	0.655
Leptin, ng/mL[2]	6.31 ± 0.75[a]	7.59 ± 0.63[ab]	8.19 ± 0.64[ab]	8.62 ± 0.64[b]	7.72 ± 0.65	7.76 ± 0.45	0.066	0.173	0.476	0.953	0.782
Osteocalcin, ng/mL	78.5 ± 6.19[a]	78.4 ± 5.31[a]	64.9 ± 5.44[ab]	60.6 ± 5.57[b]	76.6 ± 4.96	67.4 ± 3.40	0.019	0.812	0.532	0.490	0.379

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons $P < 0.05$. [ab] Different superscripts within a row and within treatment indicate pairwise comparisons at $P < 0.05$.

[1] Log transformed.

[2] Covariable was days on transition diet ($P < 0.05$).

[3] Covariables were breed of sow and weight at farrowing house entry ($P < 0.05$). [4] Breed of sow ($P < 0.05$).

Table 20. Blood metabolite measures on day 21 post-parturition of sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). Values for treatments are marginal means and standard errors. Values for treatments are marginal means and standard errors.

	Negative DCAD		Positive DCAD		Parity		*P*-value				
		CA		CA	Primiparous	Multiparous	DCAD	Vitamin D	DCAD x Vitamin D	Parity	DCAD x Vitamin D x Parity
Calcium, mmol/L	2.60 ± 0.03	2.63 ± 0.02	2.64 ± 0.02	2.66 ± 0.02	2.62 ± 0.02	2.65 ± 0.01	0.069	0.267	0.954	0.348	0.372
Cholesterol, mmol/L	2.19 ± 0.11	2.15 ± 0.09	2.26 ± 0.09	2.37 ± 0.10	2.17 ± 0.08	2.28 ± 0.07	0.177	0.746	0.664	0.271	0.303
Glucose, mmol/L	5.63 ± 0.18	5.52 ± 0.16	5.44 ± 0.16	5.52 ± 0.17	5.59 ± 0.15	5.49 ± 0.10	0.912	0.479	0.938	0.832	0.192
Phosphate, mmol/L[2]	1.69 ± 0.04 [a]	1.72 ± 0.04[ab]	1.69 ± 0.04[a]	1.83 ± 0.04[b]	1.68 ± 0.04	1.76 ± 0.03	0.104	0.043	0.462	0.363	0.157
BHB, mmol/L	0.04 ± 0.01	0.05 ± 0.01	0.05 ± 0.01	0.05 ± 0.01	0.03 ± 0.01	0.05 ± 0.00	0.326	0.808	0.261	0.090	0.573
Magnesium, mmol/L	0.86 ± 0.02	0.83 ± 0.02	0.84 ± 0.02	0.84 ± 0.02	0.85 ± 0.02	0.84 ± 0.02	0.952	0.428	0.288	0.279	0.613
NEFA, mmol/L	0.13 ± 0.04	0.22 ± 0.03	0.16 ± 0.04	0.18 ± 0.04	0.17 ± 0.03	0.18 ± 0.02	0.661	0.193	0.477	0.702	0.868
Insulin, µIU/mL [1]	3.52 ± 0.21	3.45 ± 0.18	3.64 ± 0.19	3.50 ± 0.20	3.64 ± 0.18	3.47 ± 0.12	0.905	0.271	0.706	0.389	0.119
Leptin, ng/mL	10.07 ± 0.93	10.27 ± 0.82	10.35 ± 0.86	9.49 ± 0.91	9.28 ± 1.12	10.43 ± 0.72	0.706	0.954	0.767	0.390	0.242
Osteocalcin, ng/mL	111.9 ± 9.01	110.7 ± 7.68	110.9 ± 8.36	104.4 ± 8.78	109.3 ± 8.67	109.6 ± 5.71	0.926	0.970	0.594	0.950	0.494

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons at $P < 0.05$.

[1] Log transformed.

[2] Covariable was breed of sow ($P < 0.05$).

Colostrum and milk composition

The DCAD of the transition diet had no effect on any measured milk or colostrum parameter (Table 21; $P > 0.05$). Sows that received calcidiol in their transition ration had lower milk urea content than sows that received no calcidiol (Table 21; $P < 0.05$). There were no interactions between DCAD and vitamin D type for any milk or colostrum parameter measured in this study ($P > 0.05$). While parity had no effect on colostrum outcomes, in milk samples, primiparous sows had higher fat content than multiparous sows (Table 21; $P < 0.05$). No other differences were observed between the two parity groups.

There were several DCAD X Vitamin D X parity interactions. Primiparous sows that received a negative DCAD transition diet without calcidiol had higher milk fat (4.24 ± 0.71%) than primiparous sows which received a positive DCAD transition diet without calcidiol (2.72 ± 0.51%), multiparous sows that received a negative DCAD without calcidiol ration (2.38 ± 0.42%), and multiparous sows that received a positive DCAD both with calcidiol (1.90 ± 0.43%) and without calcidiol (2.20 ± 0.44%; $P < 0.05$).

Multiparous sows that received a negative transition ration without calcidiol had lower % total milk solids (13.50 ± 0.75%) than multiparous sows that received a negative DCAD ration with calcidiol (15.69 ± 0.77%), primiparous sows that received a negative DCAD ration without calcidiol (17.01 ± 1.38%), and primiparous sows that received a positive DCAD diet with calcidiol (17.56 ± 0.99%). In addition, primiparous sows that received a positive DCAD ration with calcidiol had higher milk % solids than both primiparous sows that received a negative DCAD ration with calcidiol (14.25 ± 1.03%) and multiparous sows that received a positive DCAD ration with calcidiol (14.67 ± 0.83%; $P < 0.05$).

Table 21. Colostrum and milk parameters of sows that received either a negative or positive DCAD transition diet and with or without calcidiol (CA). Values for treatments are marginal means and standard errors.

Item	Negative DCAD		Positive DCAD		Parity		*P*-value				
	No CA	CA	No CA	CA	Primiparous	Multiparous	DCAD	Calcidiol	DCAD × calcidiol	Parity	DCAD × calcidiol × parity
Colostrum solids, %	26.23± 0.58	25.71± 0.56	25.88± 0.60	25.65± 0.54	25.78 ± 0.52	25.88 ± 0.34	0.582	0.762	0.773	0.814	0.867
Colostrum IgG, mg/mL[1]	70.90± 4.36	62.84± 4.40	63.82± 4.59	73.35± 4.40	66.70 ± 4.74	68.37 ± 3.00	0.778	0.844	0.193	0.666	0.176
Milk fat, %[1]	2.71± 0.35	2.92± 0.30	2.52± 0.30	2.63± 0.32	3.16 ± 0.34	2.27 ± 0.22	0.494	0.713	0.521	0.067	0.003
Milk protein, %	7.67± 0.40	8.46± 0.41	8.15± 0.36	8.23± 0.40	8.44 ± 0.34	7.95 ± 0.24	0.742	0.338	0.670	0.241	0.189
Milk somatic cell[2]	7.12± 0.29	6.57± 0.27	6.73± 0.26	6.91± 0.29	6.51 ± 0.32	6.70 ± 0.21	0.817	0.404	0.177	0.653	0.031
Milk urea[3]	49.28± 6.97	39.11± 6.59	46.65± 6.09	41.39± 6.88	43.20 ± 5.70	46.14 ± 4.10	0.877	0.126	0.952	0.660	0.803
Milk lactose, %	4.99± 0.14	4.79± 0.14	4.74± 0.12	4.83± 0.14	4.50 ± 0.12	4.75 ± 0.08	0.462	0.450	0.221	0.085	0.511
Milk solids, %	14.83± 0.61	15.25± 0.59	15.48± 0.55	15.63± 0.62	15.67 ± 0.53	15.11 ± 0.37	0.340	0.870	0.508	0.680	0.005

[ab] Different superscripts within a row and within treatment indicate pairwise comparisons at $P < 0.05$.

[1] Covariable was farrow house entry weight ($P < 0.05$).

[2] Log transformed.

[3] Days on transition diet ($P < 0.05$).

Sow health and subsequent reproduction

Incidences of sow health issues in this experiment were not impacted by the DCAD of the transition diet, vitamin D type, parity, nor their interactions ($P > 0.05$). All sows had similar incidences of mastitis (13.7%), udder oedema (5.1%), engorged udder (10.0%), retained piglets and/or placenta (2.6%) and vaginal discharge (14.7%). Therefore, neither DCAD nor vitamin D type influenced the odds or probability per day of any of the health disorders occurring ($P > 0.05$).

The DCAD, vitamin D type and parity had no effect on conception rate or pregnancy rate after weaning ($P > 0.05$). In the subsequent litter, total born piglets did not differ between positive and negative DCAD (14.7 ± 0.3 and 13.8 ± 0.3 piglets respectively; $P = 0.057$) or supplementation with or without calcidiol (14.0 ± 0.3 and 14.5 ± 0.3 piglets respectively; $P = 0.597$). Parity did not impact subsequent total born; however, there were several significant DCAD x vitamin D x parity interactions ($P = 0.005$). Primiparous sows that received a negative DCAD diet without calcidiol and multiparous sows that received a negative DCAD diet with calcidiol had less piglets born in the subsequent litter compared to primiparous sows that received a negative DCAD ration with calcidiol, primiparous sows that received a positive DCAD diet without calcidiol, and multiparous sows that received a negative DCAD diet without calcidiol (12.7 ± 0.7, 12.9 ± 0.5, 14.9 ± 0.8, 15.5 ± 0.7 and 15.0 ± 0.6 piglets, respectively). Multiparous sows that received a negative DCAD diet with calcidiol also had less piglets born in the subsequent litter compared to multiparous sows that received a positive DCAD diet both with and without calcidiol (14.4 ± 0.16 and 14.5 ± 0.6 piglets).

Overall, sows that received a positive DCAD diet had more piglets born alive in the subsequent litter compared to negative DCAD-fed sows (13.3 ± 0.3 and 12.5 ± 0.3 piglets respectively; $P = 0.038$). Vitamin D type and parity did not affect subsequent born alive; however again, there were several significant DCAD x vitamin D x parity interactions ($P = 0.017$). In primiparous sows, those that received a negative DCAD diet without calcidiol (11.8 ± 0.7 piglets) had less piglets born alive than positive DCAD both with (13.7 ± 0.8 piglets) and without calcidiol (14.3 ± 0.7 piglets). In addition, multiparous sows that received a negative DCAD diet with calcidiol had less piglets born alive than positive DCAD + calcidiol primiparous sows, positive DCAD + calcidiol multiparous sows, and multiparous sows that received a negative DCAD diet without calcidiol (11.6 ± 0.5, 13.7 ± 0.8, 13.1 ± 0.6 and 13.4 ± 0.6 piglets respectively).

Application of Research

There are some notable and novel aspects of dietary formulation used in this study. The DCAD and calcidiol groups fed to sows were individually compared to an industry standard of feeding a lactating sow diet that differs in fibre and DCAD from the other diets. Therefore, while a causal relationship to differences in performance can be ascribed to diet, the source of the differences can only be speculated. Further, the urinary pH responses of the primiparous and multiparous sows to diets formulated to provide a positive DCAD indicated that there was acidification occurring in all these DCAD treatment groups, notwithstanding the formulation. It is possible that fermentation of the fibre fraction in barley and wheat, that comprised approximately 50% of the diets, generated enough volatile fatty acids to reduce urinary pH (Canh et al., 1997; Zhao et al., 2020). The control group fed the lactating diet was not acidified with a mean urinary pH 7.2 ± 0.1, whereas both the negative and positive DCAD groups were acidified with pH 5.7 and 6.3, respectively. The optimal range for urinary acidification has not been established for pigs before farrowing and it is possible that the optimum may be greater than 5.7.

Will feeding a negative DCAD transition diet from late in gestation to early lactation improve production outcomes

Feeding a negative DCAD transition diet from late in gestation to early lactation improved production outcomes. The most notable responses were the reduction in stillbirths for the negative DCAD + calcidiol-fed sows and positive DCAD-fed sows and the tendency for more than 0.9 additional piglets to be born in the subsequent litter for the negative DCAD- and both positive DCAD-fed groups of sows, compared to the controls. These results are similar to earlier studies evaluating responses to a negative DCAD diet in pigs (Heenan, 1999; 2023). The combination of negative DCAD + calcidiol in a diet led to a 32% reduction in stillbirth risk compared to control sows. It is worth noting that the negative DCAD groups had the highest incidence of mummified piglets in the first farrowing; however, this is unlikely to be due to the transition dietary treatments.

A feature of the four transition diets was also an increased fibre content achieved primarily by the addition of wheat bran and sugar-beet-pulp flakes. The reduction in stillbirths in all four transition diets compared to control-fed sows (both significant and numeric) is likely to have been due in part to the increased fibre content. There is considerable evidence that high fibre diets in the transition period reduce stillbirth rate of piglets due to a reduction in constipation and/or increased postprandial energy uptake due to increased volatile fatty acid production (Krogh et al., 2015; Feyera et al., 2017; Feyera et al., 2021). Separating the effect of fibre and DCAD value is difficult in this study as the lactating sow ration also had the highest DCAD value of all diets, therefore it cannot be concluded if the higher stillbirth rate is due to a lower fibre content or a positive DCAD value of 294 MEq/kg. However, the results when analyzed excluding the control group also demonstrated that the reduction in the numbers of stillborn piglets was greatest for the negative DCAD + calcidiol diet, suggesting that DCAD is important.

Overall, though, the significant reduction in mortality of 4% to day 120 of life for the negative DCAD + calcidiol-fed sows compared to controls or positive DCAD + calcidiol-fed sows, combined with the tendency to increased births in the subsequent litter, suggests considerable merit in further evaluation of transition diets in sows.

Despite randomization, there were differences in sow weight at entry to the farrowing house with the positive DCAD groups either weighing more or tending to weigh more than other treatment groups. These differences were accounted for by including farrowing house entry weight as a covariable in models and differences in body weight among treatments did not persist. While backfat remained similar between all five treatments at each timepoint during the experiment, negative DCAD + calcidiol-fed sows lost less backfat compared to control-, negative DCAD- and positive DCAD + calcidiol-fed sows.

Control sows ate slightly more in the first 4 days following farrowing. However, once all sows were on the lactating sow ration, there was no difference in feed intake between all five treatment groups. Interestingly, faecal consistency scores before parturition were lower in control- and negative DCAD-fed sows than other treatments indicating less constipation. This may be due to the higher volume of feed intake, and the inclusion of magnesium sulphate in the negative DCAD diets (Hou et al., 2014). While faecal consistency of the negative DCAD diet was similar to both positive DCAD groups, it was the lowest numerically of the three.

Overall, there was no effect of treatment on the number of piglets weaned in this experiment. Despite some differences at day three of lactation (higher litter size in negative DCAD + calcidiol- and positive DCAD-fed sows), these differences did not continue to day 21 or weaning. As mentioned earlier, there was a reduction in piglet mortality to day 120 of life for the negative DCAD + calcidiol group compared to controls or positive DCAD + calcidiol treatment, which would be expected to result in increased weaning numbers. It is possible that piglet fostering diluted this effect. Interestingly, when looking at multiparous sows only, negative DCAD + calcidiol-fed sows weaned more piglets than sows on the other three transition diets suggesting the effect may be more pronounced in older sows.

The lack of difference in weaning weight for the piglets is consistent with very similar bodyweights for the sows, although the positive DCAD-fed sows lost more body weight from farrowing house entry to weaning than the controls and negative DCAD-fed sows. Vitamin D source in the transition period had no impact on piglet weaning weights, which is in agreement with Weber et al. (2014) but contradicts Wang et al. (2020) who found calcidiol increased piglet growth rate and influenced genes which regulate milk fat synthesis. Differences between studies may be due to the period of feeding and time of sampling.

The lack of difference in piglet weights was consistent with a lack of difference in colostrum or milk fat or protein content. There was no evidence in this study of increased milk production similar to responses seen in cows (Lean et al., 2014; Lean et al., 2019; Santos et al., 2019). We found no differences in IgG content of colostrum between the five treatments. This is in agreement with Loisel et al. (2013) who showed no effect of a high fibre diet in late gestation on IgG content of colostrum.

Milk composition was also similar between the five treatments. This agrees with Krogh et al. (2015) who found no effect of fibre on fat or protein percentage of sow milk. However, it is important to note that we sampled milk after weaning when all sows had been on the lactating sow ration for in excess of 20 days compared to 36 hours post-partum in the Krogh et al. (2015) study. Furthermore, Guo et al. (2019) showed that lowering the DCAD of the diet during late gestation and lactation did not alter fat, lactose, protein and solids in sow milk at day 1 or 18 post-partum.

Interesting, primiparous sows had a higher fat content of milk than multiparous sows in this study, a finding that is supported by Pedersen et al. (2020). This did not translate to higher litter weights at day 21 of lactation.

Was there evidence that skeleton regulates energy metabolism in the pig as it does in other species as indicated by changes in blood metabolites?

There is ample evidence that the effects of dietary treatments were reflected in blood gas, mineral and metabolite concentrations and measures that are consistent with feeding a negative DCAD diet. These were most evident at day 1 after farrowing. Significant treatment differences consistent with feeding a negative DCAD diet were found at farrowing for blood pH, oxygen partial pressure base excess, oxygen saturation, chloride and base excess providing further insight to the metabolic changes caused by diet. Guo et al. (2019) also demonstrated a lower DCAD diet in late gestation and lactation leads to reduced blood and urine pH. Interestingly, blood Ca concentrations at farrowing did not differ; however, there was a tendency for ionized Ca concentrations to be higher in the four DCAD treatment groups. This finding contrasts with the marked increase in blood Ca concentrations in dairy cows exposed to negative DCAD diet (Rodney et al., 2018) and in lactating sows (Guo et al., 2019). Further, osteocalcin concentrations differ among groups at farrowing and the results for the diets excluding the lactating diet showed a significant increase with a negative DCAD. This finding supports associations between bone metabolism and energy and protein metabolism across a range of species (Lean et al., 2014). The current study identified trends and significant differences among treatment groups at farrowing in insulin and cholesterol and glucose, providing more evidence that negative DCAD diets may influence energy metabolism. None of these differences in metabolites were evident by day 21.

While disease did not differ among treatments, as the statistical power was too low to detect differences, there was approximately half the incidence of retained placenta in the DCAD treatments compared to the control.

Is there a positive interaction of both DCAD and the inclusion of calcidiol in a transition diet?

While there were few significant effects of calcidiol alone, apart from metabolic changes at entry to the farrowing house, there were many interactions with DCAD, calcidiol and parity in the analyses that excluded the lactating diet. At farrowing house entry, calcidiol had a positive effect on cholesterol, glucose and leptin and reduced concentrations of BHB and NEFA, all of which indicate improved energy metabolism. Notable interactions were those for stillbirths and litter size (Tables 14 and 16) in which the negative DCAD + calcidiol had the least stillborn piglets and as a percentage of litters.

Application of the research findings in the commercial world

The findings generated by this research have high applicability to commercial pork production both here in Australia and overseas. Much research has focused on optimising sow nutrition; however, the development of specifications for a specific sow transition ration is still lacking. Findings from this study are preliminary and require further refinement to define the optimal DCAD value, and relationship with vitamin D source. It is also important to understand how base feed ingredients can influence the acid-base status of the sow.

Many producers are becoming aware of the importance of feeding a specific diet during the transition period and have already implemented feeding systems to accommodate this. In this commercial piggery, the farrowing house had an automatic feeding system that could distribute two separate diets to sows in the farrowing house. However, handfeeding from a feed trolley could be practiced.

Reducing piglet mortalities is a high priority for the pork industry from both an economic and animal welfare perspective. Implementing a specific transition diet that incorporates high fibre and can induce a mildly acidic state is likely to be beneficial to help reduce piglet mortalities on commercial pig farms.

Opportunities uncovered by the research
This research has provided valuable information on the design of sow transition diets. Further studies should continue to look at feed ingredients, feeding duration, and the effect in gilts. We ideally wanted to collect data on farrowing duration and piglet birth intervals; however, this was difficult to accurately obtain on a commercial farm, therefore, it would be beneficial to look at the effect of negative DCAD diets on sow farrowing kinetics.

Commercialization/Adoption Strategies

- Potential benefits to cost of production: Reducing piglet mortalities and increasing weaning numbers will substantially reduce cost of production. The replacement of gestating sow and lactating sow rations with a transition ration from approximately 10 days pre-farrow to 4 days post-farrow is not likely to result in a significant increase in feed cost.
- Ease of adoption by producers: **High**; requires minimal additional infrastructure. Additional feed silos to accommodate extra diet would be required. Labour time not likely to increase, unless additional hand feeding is required.
- Impact of the research: **High**; research findings can be easily implemented on farms of all sizes. Most Australian sows are housed in farrowing crates/pens with individual feeding allowing for tight control over feeding times. While the optimal feeding duration is unknown, it is likely that transition feeding will need to commence in the dry sow accommodation. This may then require additional feed system infrastructure. Reducing stillbirth rate is a goal of many pig producers, and transition feeding may be helpful in achieving this goal.

4. Conclusion

Overall, all four transition diets resulted in the acidification of urine indicating a metabolic acidosis for both primiparous and multiparous sows which led to a reduction in stillbirth rate, increased piglet survival to day 120 of age, improved sow metabolism and a tendency to improve litter size in the subsequent litter. Further research is needed on the application of transition diets in sows to determine the optimal urine pH, DCAD value and possibly to characterise the effects of carbohydrate fractions in the diet on urinary pH and metabolic acidification.

Further studies understanding the relationship between DCAD value and vitamin D status would also be valuable. The analyses that included the control diet demonstrated the benefit of a transition diet that produced a metabolic acidosis and had higher fibre content. The analyses that excluded the lactating sow ration

indicated the benefits of a negative DCAD, but raise questions as to how acidified a diet should be to achieve optimal outcomes.

5. Limitations/Risks

This research has provided promising outcomes that further highlight the importance of a specific diet for the transition period in sows as opposed to feeding a lactating sow ration for the duration of the pre-farrowing period. The development of a diet to cater to the nutritional requirements of the sow from the period beginning in late gestation, through parturition and into early lactation should incorporate both high fibre content, the ability to induce a mild acidic state, and in the inclusion of calcidiol. While this study has shown benefits to the sow such as improved metabolic state, reduced risk of stillborn piglets and increased piglet survival to slaughter, there are several limitations to consider before implementation on farm:

Feeding systems – The provision of a specific transition diet to the breeding herd is likely to require additional infrastructure such as more silos, feed carts etc., which will have additional costs. Automatic feeding systems may need to be altered to incorporate an additional diet. Additionally, this trial fed the transition diets for approximately 10 days prior to farrowing, meaning diets commenced while sows were in the dry sow housing. This required hand feeding in the week prior to farrowing house entry.

Feed cost: The inclusion of an extra ration is likely to result in additional feed costs. In addition, the cost of the transition diets was slightly higher than both the lactating sow and gestating sow rations.

Carbohydrate fractions of diet: Characterising the effects of carbohydrate fractions in the diet on urinary pH and metabolic acidification is important. Understanding the acid-base balance of the herd in the transition period prior to incorporating a negative DCAD inclusion is worthwhile and is a simple as measuring urine pH of a subset of the herd. Further research to determine the optimal pH is required; however, this study achieved urine pH values of between 5.7 - 6.3 and benefits in reduction of stillbirths, survival and metabolism were observed.

Gilts: Many differences between primiparous and multiparous sow outcomes were found in this study. Understanding how an optimal transition diet for all parity sows is important as there are differences evident in metabolism. It is possible that gilts and sows may need to be fed differently during the transition period and this is likely to have logistical issues that would need to be overcome.

Genotype: This study was conducted on an Australian genotype which produces high litter birthweights (20-25 kg) and large 21-day litter weights (80 kg+), a factor of increased milk output of sows. Such high reproductive output exerts an enormous metabolic effort on the animal in which the mildly acidic state is expected to be beneficial. In addition, this genotype has a high feed intake in lactation, therefore repeating this study in a different genotype is recommended to determine if similar results will be observed.

Piglet fostering: As this study was conducted on a commercial operation, fostering of piglets was required to minimise piglet mortalities. While efforts were made to minimise the amount of fostering, and to foster within treatment groups, this was not always possible. As a result, total piglet movement equaled approximately 20%.

While effects on stillbirth rate would be a direct result of the diet fed prior to farrowing, any effects on piglet or litter growth may have been impacted by the movement of piglets both on and off a litter.

6. Recommendations

As a result of the outcomes in this study the following recommendations have been made:

- An optimal fibre content (type and amount) still needs to be defined, but this work suggests a need to further investigate this aspect of diet design.
- Feed a transition diet to achieve a mildly acidic metabolic state.
- Further research to is required to determine the following gaps in knowledge:
  - Determine the optimal period of transition feeding.
  - Understand how feed ingredients and diet composition influences metabolic acidification and urine pH irrespective of DCAD value.
  - Understand the differences between gilts and sows in the transition nutrition period.

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Supplementary Table 1. Blood acid base, mineral and metabolite measures of sows on day one after parturition fed different diets. Data are presented as marginal means and standard errors for the contrasts for the treatment by parity interaction. The model included treatment, parity, and the interaction of treatment by parity.

Treatment	Parity	pH	Carbon dioxide partial pressure, mmHg	Bicarbonate, mmol/L	Base excess (extracellular fluid compartment), mmol/L	Oxygen saturation, %	Na+, mmol/L	K+, mmol/L
Control	Primiparous	7.56 ± 0.03^{b}	67.30 ± 4.24^{b}	33.65 ± 1.12^{a}	11.36 ± 1.06^{a}	90.54 ± 4.14^{c}	143.84 ± 1.01^{a}	4.34 ± 0.28
Control	Multiparous	7.47 ± 0.02^{a}	35.54 ± 2.64^{a}	38.25 ± 0.70^{d}	14.63 ± 0.66^{c}	67.92 ± 2.57^{ab}	147.22 ± 0.63^{b}	4.33 ± 0.18
Negative DCAD	Primiparous	7.47 ± 0.03^{a}	54.52 ± 5.38^{b}	33.67 ± 1.44^{ab}	10.02 ± 1.36^{a}	78.97 ± 5.31^{bc}	145.73 ± 1.28^{ab}	4.25 ± 0.31
Negative DCAD	Multiparous	7.45 ± 0.02^{a}	34.02 ± 2.64^{a}	35.81 ± 0.69^{abc}	11.78 ± 0.65^{a}	65.69 ± 2.55^{a}	147.47 ± 0.62^{b}	4.49 ± 0.18
Negative DCAD + Calcidiol	Primiparous	7.45 ± 0.02^{a}	34.06 ± 3.37^{a}	35.13 ± 0.90^{ab}	11.16 ± 0.85^{a}	67.50 ± 3.30^{ab}	146.30 ± 0.80^{ab}	4.20 ± 0.25
Negative DCAD + Calcidiol	Multiparous	7.45 ± 0.02^{a}	37.85 ± 2.73^{a}	35.68 ± 0.72^{abc}	11.72 ± 0.69^{a}	69.69 ± 2.67^{ab}	146.48 ± 0.65^{b}	4.42 ± 0.18
Positive DCAD	Primiparous	7.43 ± 0.02^{a}	34.35 ± 3.75^{a}	36.26 ± 0.98^{abcd}	11.86 ± 0.93^{ab}	65.94 ± 3.61^{a}	146.36 ± 0.88^{ab}	4.22 ± 0.26
Positive DCAD	Multiparous	7.47 ± 0.02^{a}	33.55 ± 2.62^{a}	36.06 ± 0.70^{abc}	12.30 ± 0.66^{ab}	64.14 ± 2.57^{a}	146.95 ± 0.62^{b}	4.62 ± 0.18
Positive DCAD + Calcidiol	Primiparous	7.46 ± 0.02^{a}	31.86 ± 3.56^{a}	37.91 ± 0.91^{cd}	14.14 ± 0.86^{bc}	64.21 ± 3.36^{a}	148.50 ± 0.83^{b}	4.24 ± 0.26
Positive DCAD + Calcidiol	Multiparous	7.46 ± 0.02^{a}	32.71 ± 2.77^{a}	36.36 ± 0.73^{bcd}	12.51 ± 0.69^{ab}	64.26 ± 2.68^{a}	146.47 ± 0.66^{b}	4.58 ± 0.19

^{abcd} Different superscripts within a column indicate pairwise comparisons with $P < 0.05$.

Supplementary Table 1 (cont). Blood acid base, mineral and metabolite measures of sows on day one after parturition fed different diets. Data are presented as marginal means and standard errors for the contrasts for the treatment by parity interaction. The model included treatment, parity, and the interaction of treatment by parity.

Treatment	Parity	Ca++, mmol/L	Cl-, mmol/L	Total carbon dioxide, mmol/L	Anion gap	Haematocrit, %	Haemoglobin, g/L	Base excess (blood), mmol/L
Control	Primiparous	1.17 ± 0.03^{a}	99.75 ± 1.03^{a}	33.29 ± 1.22^{a}	16.30 ± 0.65^{d}	38.38 ± 1.59^{b}	129.85 ± 5.34^{c}	10.50 ± 0.92^{ab}
Control	Multiparous	1.24 ± 0.02^{abc}	100.46 ± 0.64^{ab}	37.99 ± 0.76^{c}	14.00 ± 0.40^{abc}	33.90 ± 0.99^{a}	115.35 ± 3.32^{ab}	12.72 ± 0.57^{c}
Negative DCAD	Primiparous	1.28 ± 0.04^{bc}	101.98 ± 1.32^{abcd}	33.53 ± 1.57^{ab}	15.33 ± 0.83^{bcd}	32.76 ± 2.03^{a}	110.98 ± 6.80^{ab}	8.90 ± 1.17^{a}
Negative DCAD	Multiparous	1.29 ± 0.02^{c}	103.46 ± 0.64^{d}	35.70 ± 0.75^{ab}	13.78 ± 0.40^{ab}	33.53 ± 0.99^{a}	114.27 ± 3.32^{ab}	10.23 ± 0.57^{a}
Negative DCAD + Calcidiol	Primiparous	1.27 ± 0.02^{bc}	101.54 ± 0.82^{abcd}	34.92 ± 0.98^{ab}	15.11 ± 0.52^{cd}	34.72 ± 1.27^{ab}	118.00 ± 4.24^{abc}	9.70 ± 0.73^{a}
Negative DCAD + Calcidiol	Multiparous	1.24 ± 0.02^{abc}	102.20 ± 0.67^{bcd}	35.48 ± 0.79^{ab}	14.19 ± 0.42^{abc}	34.63 ± 1.03^{a}	118.14 ± 3.44^{abc}	10.17 ± 0.59^{a}
Positive DCAD	Primiparous	1.27 ± 0.03^{bc}	103.58 ± 0.96^{cd}	36.36 ± 1.07^{abc}	13.18 ± 0.60^{a}	35.69 ± 1.40^{ab}	121.15 ± 4.70^{abc}	10.03 ± 0.81^{a}
Positive DCAD	Multiparous	1.22 ± 0.02^{ab}	102.11 ± 0.64^{abcd}	35.75 ± 0.76^{ab}	14.75 ± 0.40^{bc}	33.42 ± 0.99^{a}	113.34 ± 3.30^{a}	10.79 ± 0.57^{ab}
Positive DCAD + Calcidiol	Primiparous	1.28 ± 0.02^{bc}	102.16 ± 0.84^{abcd}	36.61 ± 0.99^{bc}	14.06 ± 0.53^{abc}	36.60 ± 1.32^{ab}	124.50 ± 4.44^{bc}	12.13 ± 0.75^{bc}
Positive DCAD + Calcidiol	Multiparous	1.24 ± 0.02^{abc}	101.29 ± 0.67^{abc}	36.12 ± 0.79^{abc}	14.50 ± 0.42^{abc}	35.03 ± 1.04^{ab}	118.99 ± 3.48^{abc}	10.84 ± 0.60^{ab}

^{abcd} Different superscripts within a column indicate pairwise comparisons with $P < 0.05$.

Supplementary Table 1 (cont). Blood acid base, mineral and metabolite measures of sows on day one after parturition fed different diets. Data are presented as marginal means and standard errors for the contrasts for the treatment by parity interaction. The model included treatment, parity, and the interaction of treatment by parity.

Treatment	Parity	Lactate, mmol/L	Blood urea nitrogen, mg/dL	Creatinine, mg/dL	Calcium, mmol/L	Cholesterol, mmol/L	Glucose, mmol/L	Phosphate, mmol/L
Control	Primiparous	3.93 ± 0.51^{b}	14.34 ± 1.22^{c}	2.82 ± 0.16^{bc}	2.63 ± 0.07	1.58 ± 0.10^{cd}	5.48 ± 0.25^{ab}	2.41 ± 0.15
Control	Multiparous	2.96 ± 0.32^{ab}	9.45 ± 0.76^{b}	2.71 ± 0.10^{abc}	2.63 ± 0.04	1.41 ± 0.07^{bcd}	5.49 ± 0.15^{ab}	2.23 ± 0.09
Negative DCAD	Primiparous	2.05 ± 0.65^{a}	7.45 ± 1.57^{ab}	2.30 ± 0.21^{a}	2.64 ± 0.09	1.35 ± 0.13^{abcd}	5.05 ± 0.32^{a}	2.48 ± 0.19
Negative DCAD	Multiparous	2.54 ± 0.31^{a}	9.16 ± 0.75^{b}	2.79 ± 0.10^{bc}	2.65 ± 0.04	1.25 ± 0.07^{ab}	5.35 ± 0.15^{a}	2.14 ± 0.09
Negative DCAD + Calcidiol	Primiparous	2.69 ± 0.40^{ab}	7.76 ± 0.97^{ab}	2.64 ± 0.13^{abc}	2.65 ± 0.05	1.50 ± 0.08^{cd}	5.88 ± 0.20^{b}	2.27 ± 0.12
Negative DCAD + Calcidiol	Multiparous	3.22 ± 0.33^{ab}	6.76 ± 0.79^{a}	2.58 ± 0.10^{ab}	2.64 ± 0.04	1.35 ± 0.07^{abc}	5.80 ± 0.16^{b}	2.13 ± 0.10
Positive DCAD	Primiparous	2.90 ± 0.44^{ab}	7.49 ± 1.06^{ab}	2.58 ± 0.14^{abc}	2.66 ± 0.06	1.56 ± 0.09^{cd}	5.68 ± 0.22^{ab}	2.38 ± 0.13
Positive DCAD	Multiparous	3.16 ± 0.32^{ab}	7.44 ± 0.76^{ab}	2.88 ± 0.10^{c}	2.56 ± 0.04	1.21 ± 0.07^{a}	5.21 ± 0.16^{a}	2.22 ± 0.10
Positive DCAD + Calcidiol	Primiparous	2.66 ± 0.41^{a}	7.05 ± 0.99^{ab}	2.61 ± 0.13^{abc}	2.67 ± 0.06	1.57 ± 0.09^{d}	5.24 ± 0.20^{a}	2.28 ± 0.12
Positive DCAD + Calcidiol	Multiparous	3.43 ± 0.33^{ab}	6.77 ± 0.79^{a}	2.61 ± 0.10^{abc}	2.67 ± 0.04	1.46 ± 0.07^{cd}	5.15 ± 0.16^{a}	2.21 ± 0.10

^{abcd} Different superscripts within a column indicate pairwise comparisons with $P < 0.05$.

Supplementary Table 1 (cont). Blood acid base, mineral and metabolite measures of sows on day one after parturition fed different diets. Data are presented as marginal means and standard errors for the contrasts for the treatment by parity interaction. The model included treatment, parity, and the interaction of treatment by parity.

Treatment	Parity	BHB, mmol/L	Magnesium, mmol/L	NEFA, mmol/L	Insulin, uU/mL	Leptin, ng/mL	Osteocalcin, ng/mL
Control	Primiparous	0.09 ± 0.07^{ab}	0.75 ± 0.04^{ab}	0.39 ± 0.13	2.77 ± 0.32^{abcd}	9.37 ± 1.30^{b}	83.74 ± 11.11^{ab}
Control	Multiparous	0.09 ± 0.05^{ab}	0.78 ± 0.02^{b}	0.24 ± 0.08	3.32 ± 0.20^{d}	6.75 ± 0.82^{ab}	70.46 ± 6.47^{ab}
Negative DCAD	Primiparous	0.09 ± 0.09^{ab}	0.71 ± 0.05^{ab}	0.38 ± 0.17	2.35 ± 0.43^{abc}	7.56 ± 1.71^{ab}	67.51 ± 13.59^{ab}
Negative DCAD	Multiparous	0.22 ± 0.05^{b}	0.73 ± 0.02^{ab}	0.30 ± 0.08	2.39 ± 0.21^{a}	6.05 ± 0.86^{a}	83.21 ± 6.57^{b}
Negative DCAD + Calcidiol	Primiparous	0.07 ± 0.06^{a}	0.69 ± 0.03^{a}	0.27 ± 0.10	3.11 ± 0.26^{bcd}	8.77 ± 1.04^{b}	82.07 ± 8.62^{b}
Negative DCAD + Calcidiol	Multiparous	0.13 ± 0.05^{ab}	0.74 ± 0.03^{ab}	0.44 ± 0.08	3.17 ± 0.21^{cd}	6.77 ± 0.84^{ab}	77.76 ± 6.56^{b}
Positive DCAD	Primiparous	0.07 ± 0.07^{ab}	0.71 ± 0.03^{ab}	0.26 ± 0.11	3.11 ± 0.28^{bcd}	8.44 ± 1.16^{ab}	70.43 ± 9.62^{ab}
Positive DCAD	Multiparous	0.07 ± 0.05^{a}	0.72 ± 0.03^{ab}	0.35 ± 0.08	2.65 ± 0.23^{abc}	7.73 ± 0.93^{ab}	63.88 ± 7.34^{ab}
Positive DCAD + Calcidiol	Primiparous	0.06 ± 0.06^{a}	0.69 ± 0.03^{a}	0.24 ± 0.10	3.15 ± 0.26^{bcd}	9.07 ± 1.13^{b}	62.32 ± 8.30^{ab}
Positive DCAD + Calcidiol	Multiparous	0.10 ± 0.05^{ab}	0.67 ± 0.03^{a}	0.40 ± 0.08	2.60 ± 0.20^{ab}	8.47 ± 0.83^{b}	58.01 ± 6.95^{a}

^{abcd} Different superscripts within a column indicate pairwise comparisons with $P < 0.05$.