POSTNATAL STRATEGIES TO INCREASE MYOFIBRE PROLIFERATION

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Executive Summary

Lean meat deposition is positively correlated with the total number of myofibers in pigs. Challenging the classical theory that the myofiber number is fixed by late gestation, some studies have shown that the myofiber number increases with the proliferation of tertiary myofibers during the first four weeks after birth, implying a novel time window for interventions. N-carbamyl glutamate (NCG) and L-carnitine are the two additives that have been reported to stimulate endogenous growth hormone and thus may promote postnatal myofiber proliferation in young pigs. We hypothesised that oral drenching NCG or L-carnitine during the suckling phase can increase total myofibre number and thereby improve finisher phase growth performance.

The experiment followed a 2 x 3 factorial design based on birth weight (light vs. normal) and oral supplements during the suckling phase (placebo, NCG, and L-Carnitine). A total of 60 normal weight (1.3-1.7 kg range) and 60 light weight (0.8-1.2 kg) piglets were allocated to the three oral supplements. The piglets received the respective water-dissolved supplements via oral drench (placebo (2 mL water), 250 mg NCG or 400 mg) once daily from birth to 21 days of age. Piglets were transferred to the weaner facility at 21 days of age and then to the grower/finisher facility at 10 weeks of age. Pigs were housed individually from weaning and slaughtered at an average age of 150 days. Growth performance of the pigs was measured during the following age ranges: 0-3 weeks, 3-10 weeks, 10-16 weeks, and 16 weeks of age to sale. Blood samples were taken from 21-day-old piglets to measure amino acids and IGF-1, reflecting the anabolic effect of the proposed supplement. Carcass weight and backfat thickness were measured post-slaughter. Loin samples from focal carcasses were taken to quantify the total number of myofibers.

Results showed that the 21-day oral drenching of NCG and L-carnitine increased the concentration of certain amino acids in the plasma but did not affect the IGF-1 concentration. The total number of myofibers measured in the loin sample post-slaughter was not affected by the oral drench treatment. The lifetime growth performance and carcass traits were not affected by the oral drench treatment. Born-light pigs had a similar total myofiber number as the born-normal piglets, but the born-light pigs had a poorer average daily intake and growth rate.

In conclusion, oral administration of NCG or L-carnitine is not an effective strategy for improving postnatal myofiber proliferation and lifetime growth performance. Pigs born light are not disadvantaged in terms of total myofiber number. Other physiological factors that dictate the poor growth performance of the born-light piglets need to be investigated.

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1. Introduction

Lean meat deposition is positively related with the total myofibre number of pigs (Dwyer *et al*. 1994a; Rehfeldt *et al*. 2000). Myofibres can be classified as primary, secondary and tertiary myofibres based on the formation sequence during fetal development phase. In pigs, the fetal primary myofiber number reaches the maximum at about 60 days of gestation. The fetal secondary myofiber starts to form around the surface of primary myofiber at approximately 54 days of gestation and reaches the maximum number at 90 days of gestation (Swatland 1973; Wigmore and Stickland 1983). As the primary and secondary myofiber accounts for more than 95% of final total myofiber population (Wigmore and Stickland 1983), the classical theory is that that the myofiber number is fixed by late gestation in pigs. However, some recent studies showed that the myofibre number increases with the proliferations of tertiary myofibre during first four weeks after birth (Rehfeldt *et al*. 2008; Berard *et al*. 2011). Histologically the tertiary myofiber are smaller in diameter and randomly scattered among the secondary myofibres (Berard *et al*. 2011). The discovery of postnatal proliferation of tertiary myofibers in pigs implies a novel time window for applying interventions to boost postnatal myofiber proliferation.

Nutritional strategies during the suckling phase may be able to increase myofibre number postnatally. Oral supplementation of NCG (100 mg/kg body weight per day) has been reported to increase endogenous growth hormone secretion and protein synthesis in neonatal piglets (Frank *et al*. 2007). The effect of stimulating growth hormone is one of the mechanisms for myofibre proliferation (Oksbjerg *et al*. 2004). Oral supplementation of L-carnitine (400 mg/d) from day 7 to 28 increased muscle fibre number in the low-birth-weight piglets (Lösel *et al*. 2009). A more profound effect may occur if supplementation begins earlier (e.g., from birth). We hypothesised that oral drenching NCG (250 mg/d) or L-carnitine (400 mg/d) from day 1 to 21 of age in piglets would increase the total myofiber number, improve finisher phase growth performance, and reduce carcass backfat.

2. Methodology

All procedures that involved animals in the current study were in accordance with the Australian Code for the Care and Use of Animals for Scientific Purposes (8th edition, 2013), and the Animal Ethics Committee of Rivalea Australia Pty Ltd, Corowa, NSW, Australia, approved the research protocols (24-007).

Animals and Experimental Design

The experiment was conducted at a pig research facility at the Research and Innovation Unit, Rivalea Australia Pty Ltd, Corowa, NSW, Australia. The experiment used PrimeGro genetics pigs as the experimental animals (Large White × Landrace × Duroc; Primegro™ Genetics, Corowa, NSW, Australia).

The experiment followed a 2 x 3 factorial design based on birth weight (light vs normal) and oral supplements during the suckling phase (placebo, NCG, and L-carnitine). A total of 60 normal weight (1.3-1.7 kg range) and 60 light weight (0.8-1.2 kg) piglets were allocated to the three oral supplements. The piglets received

a respective 2-mL oral drench solution containing placebo (2 mL water), 250 mg NCG or 400 mg L-carnitine once daily from day 1 to 21 days of age. Oral drench ceased after 21 days of age when pigs were weaned. Creep feed was not provided in the farrowing house.

Growth performance measurements

Growth performance was measured during the suckling (0-3 weeks), weaner (4-10 weeks of age), grower (10-16 weeks of age), and finisher phases (16 weeks of age-slaughter). The key nutrients of diets supplied to each phase are presented in **Table 1**.

Piglets were weaned and moved to individual weaner facility at three weeks of age. Pigs were fed *ad libitum* after weaning throughout the experiment. Pigs were individually housed in an enclosed, climatically controlled building (28°C down to 22°C). Pens consisted of half slatted plastic floor and half solid plastic floor. Pens were divided by a metal fence, allowing pigs to have visual and nose-to-nose contact with other experimental pigs. There was one feeder and a nipple drinker in each pen. Feed delivery and refusals were recorded weekly to calculate average daily feed intake (ADFI).

Pigs were moved to a grower/finisher facility at 10 weeks of age. Pigs were individually housed (4.16 m^2 floor space per pig) in an enclosed, climatically controlled building (20°C). Pens consisted of half slatted plastic floor and half concrete floor. Pens were divided by a metal fence, allowing pigs to have visual and nose-to-nose contact with other experimental pigs. There was one feeder and a nipple drinker in each pen. All experimental pigs were treated with Draxxin (0.25 mL per kg live weight; Draxxin®, Zoetis, US) at entry and received Lincomycin in water for three days every two weeks from entry for preventing respiratory and enteric illness.

The body weight of live pigs was measured on the starting day and one day before slaughter. Feed delivery and refusal were recorded weekly to calculate ADFI. Pigs were slaughtered at an average of 150 days of age. Days from entry to reaching the slaughter weight were recorded, and average daily gain (ADG) was calculated. The feed conversion ratio (FCR) was calculated as the ratio between the actual feed consumption and body weight gain.

Carcass trait measurements

The commercial carcass traits, including hot standard carcass weight (HSCW), loin depth and backfat thickness (P2 site: last rib, 65 mm from the midline; Hennessey Chong's Probe method) were measured in the abattoir. The hot standard carcass weight was measured after trimming of visceral organs (Australian Trim 1 standard). The dressing percentage was calculated as the ratio of the hot standard carcass weight to the live weight.

Myofibre number counting

Measuring muscle fibre number in slaughtered finisher pigs is a more acceptable and humane method than measuring muscle fibre in euthanised piglets. This method has

been used in many previous studies, for example, Dwyer *et al*. (1994b). It is known that the myofibre number does not change from 7 to 20 weeks of age in wild or domestic pigs (Rehfeldt *et al*. 2000); therefore, any muscle fibre number change due to the treatment imposed during 0-21 days of age should be reflected in the slaughtered finisher pigs. The current experiment has adopted this refined method for measuring muscle fibre number due to the above consideration.

A 3 cm-thick loin sample was taken from focal carcasses between the 5th and 6th ribs for counting the total myofiber number. The loin area was measured using the loin photo taken against a grid board (**Figure 1**). A muscle cube (1×1×1 cm^3) was taken from the centre of the loin sample using a surgical blade. The muscle cube was slightly trimmed to ensure the myofibre longitudinal direction (i.e. visual check) is perpendicular to the base. The muscle cube was glued (Tissue Tek) to a 2 cm × 2 cm cork board, then snap-frozen in a liquid-nitrogen-cooled isopentane solution (Sigma Aldrich). The snap frozen samples were stored at -80ºC before being transported to The University of Melbourne for the myofiber histology analysis.

The longissimus samples were sectioned at a thickness of 5 µm using a cryostat (Leica Biosystems, Victoria, Australia). The sectioned samples were subjected to Hematoxylin and Eosin (H&E) staining using the automated H&E procedure using the Melbourne Histology platform. The stained slides were scanned at 40 × magnifier using a P480 slide scanner (3DHISTECH, Budapest, Hungary) in Phenomics Australia, The University of Melbourne. The image was viewed and analysed using the slide viewer (1.2 RX, 3DHISTECH). Six 0.5 × 0.5 mm areas were randomly selected on each slide to count the number of muscle fibres. The average count of muscle fibres in each area was calculated. The cross-sectional area (CSA) of the muscle fibre was expressed as the ratio of the area of measured muscle fibres to the average fibre count.

Plasma amino acid concentration and composition

Blood samples from 21-day-old piglets were taken for amino acids and IGF-1 measurement to reflect the anabolic effect of the proposed oral supplement. The blood sample was collected into a heparinised vacutainer (BD Vacutainers, BD Diagnostics, Oxford, UK). Blood samples were centrifuged at 1,600 × g for 10 min at 4°C (Heraeus Megafuge 16R, Thermo Fisher Scientific, North Ryde, NSW, Australia) for harvesting plasma. Plasma samples were stored in a freezer at -20 °C pending analysis. Plasma samples (n=6 born-light and n=6 born-normal pigs per oral drench treatment group) were assayed in single for quantifying 20 amino acid concentrations. Briefly, plasma samples were hydrolysed using 6 M HCl to generate Individual amino acids; then, the amino acid peaks were quantified by ultra-performance liquid chromatography. The composition of each amino acid was calculated using the plasma concentration of the amino acids divided by the plasma total amino acid concentration. The lab analysis was conducted at the Australian Proteome Analysis Facility (APAF) Centre of Macquarie University, Australia.

Plasma IGF-1 concentration was measured using Pig Insulin-like Growth Factor-1 ELISA kit (Cusabio: CSB-E06829p, Millennium Science, Vic). The samples were

processed in one batch with an intra-assay CV% < 8%. The lab analysis was conducted at The University of Western Australia, Australia.

Statistical analyses

The pig was the experimental unit. All data were analysed for the main effect of oral drench (control vs. NCG and L-carnitine), birth weight (light vs. normal), and their interactions using the UNIVARIATE procedure in SPSS (IBM SPSS Statistics for Windows, v27, Armonk, NY). Sex (female vs male) was used as a block factor.

3. Outcomes

Growth performance

Oral drench treatment did not affect ADG, ADFI or FCR in any growth phase (i.e. suckling (0-3 weeks), weaner (3-10 weeks of age), grower (10-16 weeks of age), and finisher phase (16 weeks of age-slaughter)) (**Table 2**). Born-light pigs had lower body weights (all $P < 0.01$) than the born-normal cohort at weaning, 10 weeks of age, 16 weeks of age, and slaughter. The ADFI and ADG of born-light pigs were both lower than those of born-normal pigs in the above phases (all $P < 0.05$), except for the grower phase (10-16 weeks of age). The FCR was similar between the two birth weight groups in all the above phases. The interaction between oral drench and birth weight group was not significant for ADFI or ADG variables.

However, this interactive effect was significant ($P < 0.01$) for FCR during the 16-week age to sale, such that the L-carnitine group had a higher FCR than the NCG group in the born-light cohort but a lower FCR than the NCG group in the born-normal cohort.

Carcass traits

The oral drench treatment did not affect carcass weight, backfat, loin depth, or dressing percentage (**Table 3**). The born-light pigs had lower ($P<0.05$) carcass weight than born-normal pigs. The carcass backfat, loin depth, or dress percentage was similar ($P > 0.05$) between born-light and born-normal pigs.

Myofibre number

The oral drench treatment did not significantly affect area, myofiber density, cross-sectional area of myofiber or the total myofiber number in the loin area (**Table 4**). Born-light pigs had a smaller loin area and greater myofiber density than the born-normal pigs (both $P < 0.05$). However, the cross-sectional area of myofibers and total myofiber number did not differ significantly between the born-light and born-normal pigs.

Plasma amino acid concentration

The NCG oral drench tended to increase ($P = 0.072$) plasma total amino acids and increased blood lysine concentration ($P < 0.05$), regardless of the birth weight group (**Table 5**). Both NCG and L-carnitine oral drenches increased (both $P < 0.05$) the plasma proline concentration. Born-light piglets tended to have higher ($P < 0.10$)

concentrations of glutamic acid and had a higher ($P < 0.05$) tyrosine concentration than the born-normal cohort. The interactive effect between oral drench and birth weight cohort was not significant.

Plasma IGF-1 concentration

Neither birth weight group nor oral drench treatment affected the plasma IGF-1 concentration measured at 21 days of age (both $P > 0.10$) (**Figure 4**).

Table 1 Dietary nutrient levels

Nutrients	4-6 weeks of age	6-9 weeks of age-	9-15 weeks of age	15 weeks of age to sale
Digestible energy, MJ/d	14.9	14.4	13.9	13.5
Crude protein, %	18.2	21.5	18.1	15.0
available Lysine, %	1.28	1.22	0.97	0.81
Calcium, %	1.4	1.3	0.71	0.61
available phosphorus, %	1.2	1.1	0.46	0.40

Table 2. Lifetime growth performance of pigs received oral drench during 0-21 days of age

Variables	Born-light			Born-normal			SEM	P-values		
	Control (n=13)	NCG (n=14)	L-carnitine (n=16)	Control (n=17)	NCG (n=14)	L-carnitine (n=16)		Drench	Birth Weight	Interaction
Body weight (birth), kg	1.20	1.22	1.27	1.62	1.58	1.64	0.033	0.95	<0.001	0.15
Body weight (weaning), kg	5.9	6.5	5.8	7.4	7.2	7.2	0.35	0.62	<0.001	0.48
Body weight (10 weeks), kg	30.9	32.8	31.5	35.4	35.6	35.4	1.12	0.60	<0.001	0.75
Body weight (16 weeks), kg	69.0	69.8	72.3	73.2	76.3	76.8	2.1	0.25	0.003	0.82
Body weight (sale), kg	108.5	113.0	104.8	114.0	114.5	118.9	2.55	0.78	0.004	0.11
Age at sale, days	150.5	150.2	150.4	150.2	149.5	150.5	0.75	0.71	0.60	0.88
ADG (birth to wean), kg	0.20	0.22	0.20	0.25	0.24	0.24	0.015	0.61	0.004	0.56
Weaning-10 weeks of age										
ADG, kg	0.60	0.64	0.62	0.69	0.69	0.69	0.023	0.60	<0.001	0.77
ADFI, kg	0.74	0.78	0.76	0.03	0.84	0.86	0.85?	0.62	<0.001	0.92
FCR	1.23	1.21	1.23	1.22	1.23	1.23	0.019	0.86	0.92	0.73
10-16 weeks of age										
ADG, kg	0.91	0.91	0.95	0.9	0.95	0.97	0.033	0.52	0.36	0.53
ADFI, kg	2.21	2.31	2.26	2.32	2.31	2.38	0.082	0.27	0.77	0.75
FCR	2.44	2.45	2.50	2.6	2.59	2.46	0.058	0.77	0.097	0.20
16 weeks of age to sale										
ADG, kg	1.07	1.10	0.97	1.15	1.10	1.18	0.048	0.75	0.015	0.074
ADFI, kg	3.21	3.40	3.25	3.54	3.63	3.45	0.118	0.40	0.019	0.88
FCR	3.03	3.12	3.38	3.13	3.36	2.96	0.112	0.35	0.78	0.008
Weaning-Sale										
ADG, kg	0.85	0.88	0.82	0.89	0.90	0.92	0.025	0.53	0.008	0.17
ADFI, kg	1.94	2.05	1.96	2.09	2.16	2.10	0.056	0.23	0.005	0.97
FCR	2.30	2.33	2.40	2.37	2.41	2.28	0.042	0.73	0.79	0.34

ADG: Average daily gain; ADFI: average daily feed intake; FCR: feed conversion ratio.

Table 3. Carcass traits of finisher pigs received oral drench during 0-21 days of age

Variables	Born-light			Born-normal			SEM	P-values		
	Control (n=11)	NCG (n=10)	L-carnitine (n=11)	Control (n=14)	NCG (n=11)	L-carnitine (n=13)		Drench	Birth Weight	Interaction
HSCW	83.5	84	79.5	87.6	84.5	89.4	2.27	0.84	0.018	0.16
Backfat	17.0	14.4	15.1	15.0	15.5	15.7	0.95	0.11	0.63	0.11
Loin Depth	52.2	55.5	54.6	56.5	53.5	55.1	2.06	0.96	0.56	0.298
Dress%	77.0	77.4	76.7	77.9	76.2	77.2	0.54	0.43	0.90	0.097

Covariates appearing in the model are evaluated at the following values: Age at Sale = 150.94.

Table 4. Myofibre number of finisher pigs received oral drench during 0-21 days of age

	Born-light			Born-normal			SEM	P-values		
Variables	Control (n=9)	NCG (n=8)	L-carnitine (n=10)	Control (n=9)	NCG (n=8)	L-carnitine (n=9)		Drench	Birth Weight	Interaction
Loin area, cm^2	57.2	59.3	56.4	64.9	61.5	60	2.8	0.54	0.053	0.57
Myofibre density, number per mm^2	242	276	263	235	241	228	18.8	0.58	0.090	0.70
Cross-section area of myofibre, um^2	4311	3857	3990	4340	4302	4668	329.5	0.70	0.16	0.59
Total myofibre number, thousands	1406	1608	1498	1521	1478	1373	149.2	0.55	0.60	0.54

Table 5. Plasma amino acid of piglet received oral drench during 0-21 days of age

Variables	Born-light			Born-normal			SEM	P-values		
	Control (n=6)	NCG (n=6)	L-carnitine (n=6)	Control (n=6)	NCG (n=6)	L-carnitine (n=6)		Drench	Birth Weight	Interaction
Total amino acids, μg/mL	565	625	630	516	660	579	42.8	0.072	0.54	0.52
Essential amino acids										
Lysine, μg/mL	33	44	46	35	52	37	4.6	0.014	0.99	0.20
Methionine, μg/mL	10	11	10	9	11	10	1.5	0.52	0.86	0.90
Threonine, μg/mL	18	18	18	12	16	17	4.2	0.77	0.37	0.77
Tryptophan, μg/mL	1.0	1.1	1.0	0.9	1.1	0.9	0.12	0.4	0.62	0.92
Histidine, μg/mL	19	19	19	17	21	20	2.4	0.84	0.92	0.64
Valine, μg/mL	30	29	30	29	35	29	3.1	0.6	0.64	0.44
Phenylalanine, μg/mL	14	16	15	13	14	14	1.4	0.78	0.21	0.93
Isoleucine, μg/mL	16	15	15	14	16	13	1.8	0.76	0.51	0.66
Leucine, μg/mL	25	26	26	23	26	23	2.1	0.62	0.32	0.71
Non-essential amino acids										
Alanine, μg/mL	56	71	66	55	74	62	6.8	0.064	0.96	0.89
Asparagine, μg/mL	11	11	11	8	11	10	1.2	0.31	0.58	0.53
Serine, μg/mL	21	25	27	22	29	26	2.5	0.086	0.48	0.67
Glycine, μg/mL	77	79	86	78	93	80	7.4	0.52	0.64	0.35
Aspartic acid, μg/mL	2.6	3.1	3.3	2.5	2.9	3	0.3	0.23	0.44	0.95
Glutamic acid, μg/mL	25	26	29	20	24	23	2.9	0.53	0.086	0.71
Conditional amino acids										
Arginine, μg/mL	25	26	25	25	29	32	4.2	0.66	0.32	0.67
Proline, μg/mL	59	75	82	56	83	73	7.2	0.009	0.77	0.48
Glutamine, μg/mL	93	96	91	74	95	83	11.1	0.53	0.32	0.72
Tyrosine, μg/mL	27	33	29	19	26	23	3.0	0.15	0.008	0.99

Cystine, µg/mL	2.1	1.9	1.4	3.5	1.6	1.9	0.66	0.18	0.35	0.50

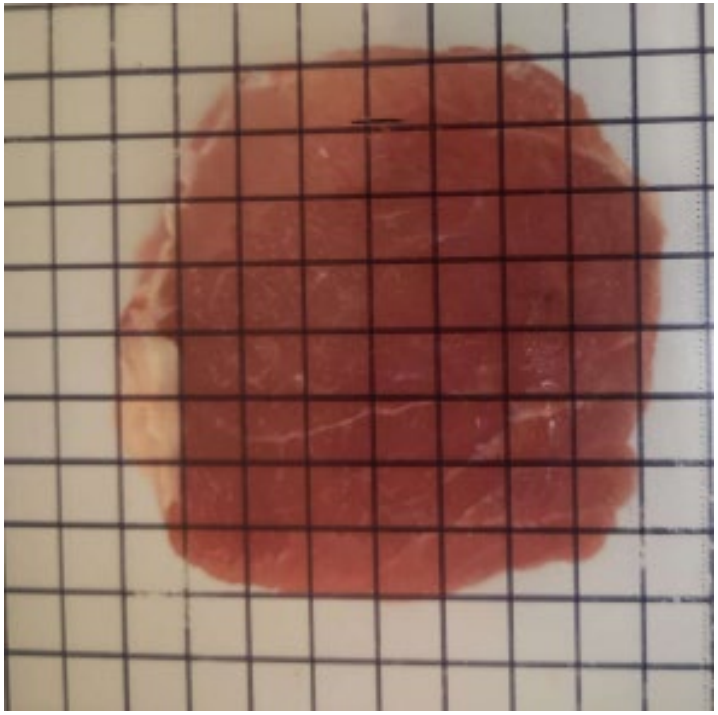

Figure 1. An example of a loin sample.

Figure 2. An example of a loin cube glued to a cork board before snap-freezing in isopentane.

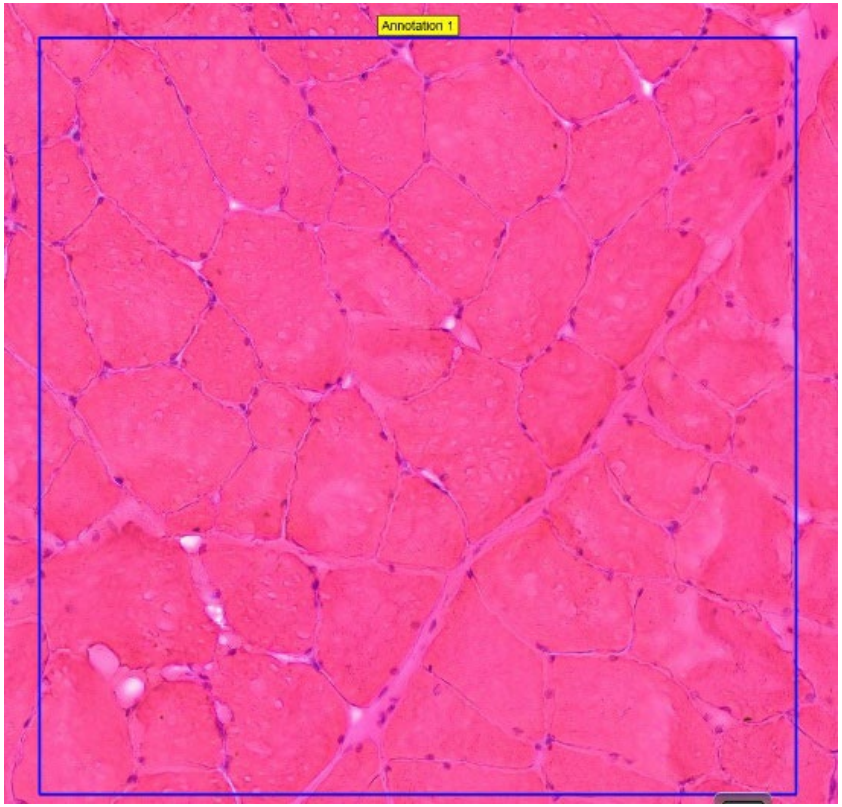

Figure 3. An example of a loin section photo for myofiber counting.

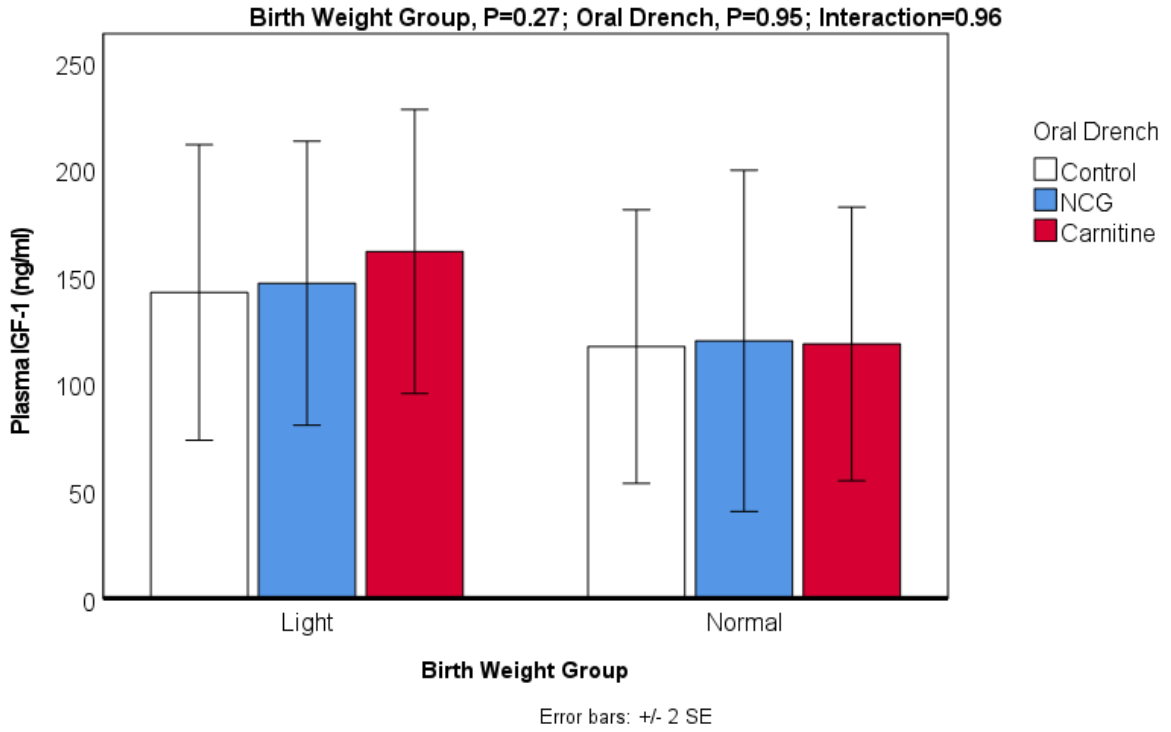

Figure 4. Plasma IGF-1 concentration of piglets that received oral drenches for 21 days.
The plasma IGF-1 concentration of piglets at 21 days of age was not affected (P>0.1) by birth weight group, oral drench, or their interactive effect. N=26, 22 and 27 for the oral drench of control (placebo), NCG and L-carnitine, respectively.

4. Application of Research

The main finding of the current experiment was that oral drenching with 250 mg/d NCG and 400 mg/d L-carnitine during the 0-21 d period managed to improve the availability of certain plasma amino acids but did not improve total myofiber number, lifetime growth performance, or carcass traits. Therefore, the main hypothesis was rejected.

The oral drench of NCG increased the total amino acid concentration by 18%, the lysine concentration by 40%, and the proline concentration by 38%, respectively. Similarly, Ye *et al*. (2017) and our previous study (Liu *et al*. 2022) in Australia reported increased circulating essential amino acid and proline concentrations of finisher pigs fed NCG. N-carbamyl glutamate is a precursor for the *de novo* synthesis of arginine (Palencia *et al*. 2018). For example, 0.1% NCG supplementation increased arginine concentration by 15% (Li *et al*. 2021) or 100% in finisher pigs (Ye *et al*. 2017). Plasma arginine concentration was not affected by NCG supplementation in piglets in the current study. However, proline, an intermediate precursor for arginine, was increased by the NCG oral drench. Interestingly, L-carnitine supplementation numerically increased plasma total amino acids concentration by 10% and significantly increased proline concentration by 32%. The improved availability of amino acids may not be a consequence of growth hormone stimulation, as the blood IGF-1 concentration was not affected by the NCG or L-carnitine oral drench. The fact that L-carnitine oral drench increased plasma amino acid concentrations may be related to its effects in upregulating skeletal insulin signalling and inhibiting muscle atrophy (Keller *et al*. 2011).

The total myofiber number was not affected by the NCG or L-carnitine oral drench. These two treatments did not affect the loin area or the myofiber density. Lösel *et al*. (2009) reported that L-carnitine oral supplementation between d7 and 27 of age increased the total myofiber number by 14% in the semitendinosus of the piglets born less than 1.1 kg but did not affect the myofiber number in the cohort born between 1.16 to 1.38 kg. However, a later study by Lösel and Rehfeldt (2013) showed that L-carnitine did not improve the total myofiber number in the born-light piglet selected between 0.8 and 1.26 kg. In our experiment, the born-light piglets were selected from a body weight range of 0.8 to 1.2 kg, which is slightly heavier than the weight range used in their first study. This weight difference may explain the disparity of the results. Between the born-light and normal pigs in our experiment, the total myofiber number did not differ, although born-light pigs tended to have smaller loin area but higher myofiber density. In the former study (Lösel *et al*. 2009), the born-light piglets (<1.1 kg) had 15% less total myofiber number than the born-normal cohort (1.16-1.38 kg). The magnitude of body weight difference may explain the difference in total myofiber numbers. The effect of NCG on the total number of myofibers has not been studied before.

Our results suggested that NCG oral drench can improve amino acid availability (i.e., higher total amino acid, lysine, and proline concentrations in the plasma) for suckling piglets, but this benefit is not effective in stimulating myofiber proliferation. Based on the IGF-1 results, the stimulation of the somatotropin axis by NCG oral drench was not successful in this study. Thus, the hypothesis that a stimulation of the somatotropin axis during neonatal phase can increase postnatal

myofiber proliferation has not been tested, highlighting a future direction to be investigated.

Insulin-like growth factor 1 is a downstream hormone of the somatotropin axis and is secreted mainly from the liver. Insulin-like growth factor 1 was chosen as a focal measurement to investigate if the NCG and L-carnitine can stimulate the somatotropin axis, because IGF-1 is more consistently secreted, and somatotropin secretion follows a pulsatile manner. The lack of effect of NCG and L-carnitine on IGF-1 concentration is consistent with our study on finisher pigs (Liu *et al*. 2022), whereas Frank et al. (2007) reported a postprandial increase in somatotropin hormone in neonatal pigs drenched with a similar dose of NCG. The disparity in results may indicate that the stimulation of the somatotropin axis by NCG may be only transient (e.g., postprandial). With regard to the effects of L-carnitine on the IGF-1 secretion, our data suggested that the L-carnitine oral drench did not improve IGF-1. L-carnitine feeding in other stage of pigs (e.g. lactating and gestating sows) showed inconsistent effects on circulating blood IGF-1 concentrations (Waylan *et al*. 2005; Birkenfeld *et al*. 2006; Brown *et al*. 2007).

Interestingly, the carcass backfat thickness were numerically lower in the L-carnitine and NCG drenched piglets within the born-light cohort although it was not statistically significant. However, this numerical difference in backfat did not exist in born-normal cohort, implying that the muscle deposition rate of born-light piglets may be benefited more from the improved postnatal nutrients availability. The supplementation of NCG in the finisher phase was reported to improve loin length (Zhu *et al*. 2020) and reduce carcass backfat (Liu *et al*. 2022).

5. Conclusion

Oral drenching daily with NCG and L-carnitine from 0 to 21 days of age increased the concentration of some plasma amino acids but did not affect growth performance or total myofiber number measured after slaughter. Born-light piglets had a poorer growth rate than the born-normal piglets throughout their life, although the total myofiber number was not different between the birth weight groups.

6. Limitations/Risks

None.

7. Recommendations

Oral administration of NCG or L-carnitine is not an effective strategy for improving postnatal myofiber proliferation. Pigs that are born light are not disadvantaged in terms of total myofiber number. Other physiological factors that dictate the inferior growth performance of the born-light piglets need to be investigated.

8. References

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