Monatshefte für Chemie 136, 1535–1544 (2005) DOI 10.1007/s00706-005-0327-6

Monatshefte für Chemie Chemical Monthly Printed in Austria

Effects of *L*-Carnitine Supplementation in Sows

Klaus Eder*

Institute of Nutritional Sciences, University of Halle, Halle/Saale, Germany

Received October 28, 2004; accepted December 22, 2004 Published online June 10, 2005 © Springer-Verlag 2005

Summary. In recent years *L*-carnitine has been used increasingly in animals. This review gives an overview of the effects of dietary *L*-carnitine supplementation during pregnancy and lactation on the reproductive performance of sows. In one investigation *L*-carnitine supplementation during pregnancy increased the number of piglets born to sows. Other studies showed heavier litters in sows supplemented with *L*-carnitine compared with control sows, and litters of *L*-carnitine supplemented sows gained more weight during the suckling period than litters of control sows. This effect might be due to more vigorous suckling by piglets of *L*-carnitine supplemented sows, causing the sows' milk production to rise. At negative energy balance during lactation *L*-carnitine supplemented sows are able to mobilize more energy from adipose tissue, which can be used for the production of surplus milk. In conclusion, recent studies clearly show that dietary *L*-carnitine supplementation increases the reproductive performance of sows. This finding suggests that the amount of *L*-carnitine synthesized endogenously does not cover the requirement for maximum sow performance during pregnancy and lactation.

Keywords. L-Carnitine; Sow; Reproduction; Milk production.

Introduction

L-Carnitine (*L*- β -hydroxy-4-(trimethylamino)butyric acid) is an important compound in mammals. Its primary function lies in the transport of long-chain fatty acids across the inner mitochondrial membrane to the place of their β -oxidative degradation, *e.g.*, energy production. In recent years, *L*-carnitine has been used increasingly as a supplement in livestock and sport animals. Feeding trials were designed to investigate whether dietary *L*-carnitine supplementation is capable of improving performance characteristics of livestock. Such studies have been conducted in sport horses [1–3], dairy cows or steers [4–6], laying hens and broilers [7–13], fattening pigs [14–16], suckling and weanling pigs [17–23], but also in boars [24]. In recent years, several studies have been performed which observed a

^{*} E-mail: eder@landw.uni-halle.de

series of beneficial effects of L-carnitine supplementation on the reproductive performance of sows [25–30]. This review gives an overview of the effects of L-carnitine in sows observed recently.

The Carnitine Status during Pregnancy and Lactation

In women, a strong decrease of plasma carnitine concentrations during pregnancy has been well established [31–33]. Concentrations of total *L*-carnitine at the 12^{th} week of gestation in pregnant women were only half of that of non-pregnant women [34]; during the further course of the pregnancy, plasma *L*-carnitine concentrations are further declining to values which are observed in patients with carnitine deficiency. The decline of total *L*-carnitine concentrations is mainly due to reduced concentrations of free *L*-carnitine while those of acylcarnitine are slightly increased compared to non-pregnant women. High ratios between acylcarnitine and free *L*-carnitine (>0.4) are designated as "carnitine insufficiency" [35]. In pregnant women, the concentration of acylated *L*-carnitine could be markedly decreased by supplementation of *L*-carnitine [36].

The behaviour of the plasma carnitine concentration in sows during the pregnancy seems to be different from that in women. In the study of *Musser et al.* [26], the concentration of free and total L-carnitine were measured in sows at days 10, 60, 90, and 110 of pregnancy. Plasma free and total L-carnitine concentrations at day 60 were lower than at days 10, 90, or 110. However, the concentrations at day 110 were even slightly higher than at day 10. This indicates that a strong decline of plasma L-carnitine during the late pregnancy as seen in women does not occur in sows. In our study, the concentrations of free and total L-carnitine in plasma of sows at day 95 of pregnancy were similar with those at day 21 of lactation (free *L*-carnitine: 6.1 vs. 6.9 μ mol/dm³ in pregnancy and lactation; total *L*-carnitine: 7.5 vs. 9.0 μ mol/dm³ in pregnancy and lactation). Supplementation of sows' diets with L-carnitine caused a moderate increase of plasma L-carnitine concentrations during pregnancy and lactation. In the study of Musser et al. [26] daily supplementation of 100 mg of L-carnitine increased the concentrations of free and total L-carnitine in plasma by approximately 10%. In our study, supplementation of 125 mg of *L*-carnitine per day increased the concentration of free *L*-carnitine in plasma from 6.1 to $8.4 \,\mu \text{mol/dm}^3$ and that of total *L*-carnitine from 7.5 to 11.1 μ mol/dm³ at day 95 of pregnancy. The ratio between acylated and free Lcarnitine, however, was not increased by L-carnitine supplementation of sows during pregnancy. L-Carnitine supplementation of sows during the lactation causes also an increase of L-carnitine concentrations in plasma and milk. In the study of Musser et al. [26], supplementation of the diets of lactating sows with 50 mg of Lcarnitine per kg of body weight increased the concentrations of free L-carnitine in plasma and milk by 22 and 5%, respectively, and that of total L-carnitine by 15 and 24%. In our study, a daily supplementation of 250 mg of L-carnitine per sow increased the concentration of free L-carnitine in the milk by 50% and that of total L-carnitine by 35% [28]. The L-carnitine concentration of the milk might be important for the development of the suckling piglets because they have a low capacity for an endogenous L-carnitine synthesis, particularly at the first days after birth [37-39]. Supplementation of newborn piglets with L-carnitine increases their activity of carnitine palmitoyl transferase-1 in the liver and their ability to oxidize fatty acids [19].

Impact of *L*-Carnitine Supplementation on Number and Weight of Piglets

The number of piglets born alive is one of the most important criteria of the reproductive performance of sows. Results concerning the impact of L-carnitine supplementation in sows during pregnancy on the number of piglets born are controversial (Table 1). In studies by Musser et al. [26] performed in the United States, sows supplemented with L-carnitine did not differ in the number of piglets born alive to unsupplemented controls. In our study, which was performed under commercial conditions in a sow unit with Leicoma sows, L-carnitine supplementation at a level similar with that used in the study of Musser et al. [26] increased the number of piglets born alive by 0.5 per litter [27]. In a more recent study performed in crossbred sow L-carnitine supplementation caused an even greater increase in the number of piglets born, both in the first and the second parity [28]. It should be noted, however, that this experiment was conducted with a small number of sows. Several studies concurred that L-carnitine supplementation of sows during pregnancy reduces the number of stillborn or non-viable piglets. In the study of Musser et al. [27] dietary L-carnitine supplementation reduced the number of stillborn piglets from 0.76 to 0.49 per litter (p < 0.05). In our study, the number of stillborn piglets remained unaffected by dietary L-carnitine but the number of nonviable piglets with a birth weight below 800 g was significantly lower in L-carnitine supplemented sows than in control sows [28, 29].

Birth weights of piglets are largely influenced by the intrauterine supply of nutrients to the fetuses. Our studies and those by *Musser et al.* [27–29] have shown that *L*-carnitine supplementation of sows during pregnancy increases birth weights of litters (Table 2). *Musser et al.* [27] found that *L*-carnitine supplemented pregnant sows have higher concentrations of insulin and IGF-1 in blood than control sows. This finding was confirmed by us (unpublished data) and *Woodworth* [30], who also found increased concentrations of insulin like growth factor-1 (IGF-1) in the

Reference	ference Supplementation level Total number Nu		Number	Number of piglets	
		01 50 83	Control sows	<i>L</i> -Carnitine supplemented sows	numbers
Musser et al. [26]	100 mg <i>L</i> -carnitine/sow/d	150	10.3	10.4	+0.1
Musser et al. [42]	$50 \mathrm{mg} L$ -carnitine/kg diet	232	11.5	11.1	-0.4
Ramanau et al. [27] Ramanau et al. [28]	125 mg <i>L</i> -carnitine/sow/d	175	10.6	11.1	+0.5
1 st birth	125 mg L-carnitine/sow/d	32	9.6	12.4	+2.8
2 nd birth	125 mg <i>L</i> -carnitine/sow/d	26	10.3	13.1	+2.8

 Table 1. Effects of L-carnitine supplementation of pregnant sows on the number of piglets born alive in various studies

Reference	Supplementation level ^a	Total number of sows	Control sows	<i>L</i> -Carnitine supplemented sows	Change
			Weights	of piglets (kg/piglet)	
Musser et al. [26]	P: 100 mg <i>L</i> -carnitine/sow/d L: 50 mg <i>L</i> -carnitine/kg diet	294	1.48	1.58	+0.10
Musser et al. [42]	P: 50 mg <i>L</i> -carnitine/kg diet L: 50 mg <i>L</i> -carnitine/kg diet	232	1.54	1.61	+0.07
Ramanau et al. [27]	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	175	1.38	1.48	+0.10
Ramanau et al. [28]					
1 st birth	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	32	1.54	1.39	-0.15
2 nd birth	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	26	1.70	1.53	-0.17
	21 200 mg 2 0amono/ 00 m/ a		Weights of litters (kg/litter)		
Musser et al. [26]	P: 100 mg <i>L</i> -carnitine/sow/d L: 50 mg <i>L</i> -carnitine/kg diet	294	14.6	15.6	+1.0
Musser et al. [42]	P: 50 mg <i>L</i> -carnitine/kg diet L: 50 mg <i>L</i> -carnitine/kg diet	232	17.9	18.2	+0.3
Ramanau et al. [27]	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	175	14.5	16.3	+1.8
Ramanau et al. [28]					
1 st birth	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	32	14.2	16.8	+2.6
2 nd birth	P: 125 mg <i>L</i> -carnitine/sow/d	26	17.3	19.6	+2.3

Table 2. Effects of *L*-carnitine supplementation of pregnant sows on birth weights of piglets and litters in various studies

^a P = pregnancy, L = lactation

L: 250 mg L-carnitine/sow/d

blood of *L*-carnitine supplemented pregnant sows. We also observed an increased concentration of growth hormone in the blood, which acts as a trigger for the release of IGF-1 [40]. IGF-1 is a key hormone for intrauterine fetal development. It promotes fetal secondary muscle fibre development [41]. *Musser et al.* [42] showed that muscle fibres in newborn piglets from *L*-carnitine supplemented sows are larger in diameter than those of piglets from control sows (p < 0.15). In the same study piglets of *L*-carnitine supplemented sows had more primary muscle fibres in the semitendinous muscle than piglets of control sows. These effects were presumably caused by a higher IGF-1 concentration in the blood. The mechanisms by which dietary *L*-carnitine increases the secretion of IGF-1 by sows are unknown and require further investigation. Improved intrauterine fetal nutrition due to dietary *L*-carnitine could explain the smaller number of stillborn or non-viable piglets born to sows supplemented with *L*-carnitine. *Woodworth et al.* [43] also reported an increased concentration of leptin in plasma of sows supplemented with *L*-carnitine, a hormone which plays a key role in reproduction. These

investigators suggested that *L*-carnitine influenced biochemical pathways involved in energy metabolism by an increased secretion of leptin. This suggestion is confirmed by a recent study which indicated that *L*-carnitine improves oral glucose tolerance in pregnant sows [30]. In our study, sows supplemented with *L*-carnitine had increased plasma concentrations of cortisol [40]. All these findings demonstrate that dietary *L*-carnitine alters the hormonal status of pregnant sows, resulting in improved fetal nutrition.

It has been shown that dietary *L*-carnitine supplemented to pregnant rats or women stimulates the development of the fetal lung [44–46]. A deficiency of the pulmonal surfactant factor which is composed mainly of phospholipids is the reason of the Respiratory Distress Syndrom (RDS) which is the most often cause of death of premature infants [47]. *L*-Carnitine supplementation of pregnant mothers has been demonstrated to increase the *L*-carnitine concentration of the fetal lung and the synthesis of dipalmitoyl phosphatidylcholine which is the most important component of the surfactant [48, 49]. It has not yet been investigated whether *L*-carnitine supplementation also stimulates the development of the fetal lung in sows. It cannot, however, be ruled out that such an effect could play a role for the lower rate of piglet losses after birth in sows supplemented with *L*-carnitine.

During the intrauterine phase, the supply of the fetus with amino acids, glucose, minerals, and fatty acids from the mother *via* the placenta is necessary for its development. The rate of fatty acid oxidation in the fetus is low [50]. However, immediately after birth the oxidation of fatty acids becomes extremely important because of the disruption of the supply with glucose and the rapid exhaustion of the glycogen storage [51]. Sufficient concentrations of *L*-carnitine in tissues are required for an efficient utilisation of fatty acids for the production of energy. It was shown that *L*-carnitine supplementation of mother's milk with *L*-carnitine reduced the rate of mortality in preterm babies from 22 to 6% [52]. We observed that *L*-carnitine concentrations in the whole body were 20% higher in newborn piglets of sows supplemented with *L*-carnitine than in newborn piglets of control sows. This could have contributed to the lower number of losses of piglets observed in sows supplemented with *L*-carnitine.

Development of Piglets during the Suckling Period

Several studies have shown that litters of sows supplemented with *L*-carnitine during pregnancy and lactation gain more weight during the suckling period than those of control sows (Table 3). Weight gain of litters during the suckling period is mainly dependent on the sow's milk production [53]. We therefore assumed that sows supplemented with *L*-carnitine produce more milk than unsupplemented control sows. Using the weigh-suckle-weigh method we were able to demonstrate that sows supplemented with *L*-carnitine produced more milk than control sows. The nutrient composition of the milk did not differ between control sows and sows supplemented with *L*-carnitine. The amount of nutrients and energy secreted with the milk, however, was higher in *L*-carnitine supplemented sows than in control sows [28]. This clearly indicates that increased litter weights of piglets during the

Reference	Supplementation level ^a	Litter weight gains (kg)		Change (kg)
		Control sows	L-Carnitine supplemented sows	(15)
Musser et al. [26]	P: 100 mg <i>L</i> -carnitine/sow/d L: 50 mg <i>L</i> -carnitine/kg diet	26.6	29.2	+2.6
Ramanau et al. [27]	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	51.2	57.9	+6.7
Ramanau et al. [28]	- , ,			
1 st birth	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	61.1	67.7	+6.6
2 nd birth	P: 125 mg <i>L</i> -carnitine/sow/d L: 250 mg <i>L</i> -carnitine/sow/d	91.4	99.2	+7.8

 Table 3. Effects of L-carnitine supplementation of pregnant sows on litter weight gains during the suckling period

^a P = pregnancy, L = lactation

suckling period are due to more energy and nutrients being transferred from the sow to the piglets with the milk.

Milk production of sows is influenced by several factors such as the age of the sows and their energy and nutrient supply. The suckling behaviour of the piglets is another important factor which has a bearing on milk production of sows. It is well established that the suckling interval and the vigour with which piglets stimulate the teats both affect milk yield. If piglets suckle more frequently they will obtain more milk, thus causing milk production to rise [54, 55]. Heavier piglets are able to massage the teats more vigorously and therefore obtain more milk at each suckling [56, 57]. We suspect that piglets of sows supplemented with *L*-carnitine can suckle for longer periods than piglets of control sows and therefore stimulate milk production by the sow. In order to investigate this, we recorded litters over a 24-h period by video. We found that piglets of *L*-carnitine treated sows have longer suckling times and shorter resting times (Table 4). We assume that the longer suckling time per day is the reason for higher milk production by sows and faster growth of piglets during the suckling period. This suggestion is confirmed by the

Table 4. Suckling activity of piglets of control sows and sows supplemented with *L*-carnitine at day 3 of lactation^a

	Control	+L-Carnitine
Number of sucklings per 24 h	43.0	44.3
Total suckling time $(h/24h)$	3.44	4.35 ^b
Average suckling time (min/suckling)	4.80	5.89 ^b
Average time between two sucklings (min)	28.3	25.3

^a *Birkenfeld et al.* (unpublished data), 10 litters in each group were observed; ^b significantly different (p < 0.05)

findings of *Musser et al.* [26, 58]. These investigators showed that *L*-carnitine supplementation of sows during pregnancy increased litter weaning weights. However, in sows which were not supplemented with *L*-carnitine during pregnancy, *L*-carnitine supplementation during lactation did not influence litter weaning weights. These studies showed that increased litter gains during the suckling period are due to the effects of *L*-carnitine during pregnancy, while *L*-carnitine during lactation has no effects in this respect if sows were not supplemented with *L*-carnitine during pregnancy. We also observed that sows supplemented with *L*-carnitine had larger and more active mammary glands at weaning than control sows [59]. The development of the mammary gland was probably stimulated by the suckling activity of the piglets. If piglets suckle more frequently or more vigorously they will obtain more milk, resulting not only in higher milk production but also in a higher rate of mammary growth [54].

The reason for the increased suckling time of piglets from *L*-carnitine supplemented sows is unclear. One possible explanation is that piglets of *L*-carnitine treated sows are already more vigorous at birth than piglets of control sows due to an increased nutrient supply during fetal development. Another possible reason why they are more vigorous than piglets of control sows is because they have higher tissue concentrations of *L*-carnitine at birth and moreover receive more *L*-carnitine with the maternal milk than control piglets. Newborn piglets have a very limited capacity for *L*-carnitine synthesis [37–39]. The milk is therefore an important source of *L*-carnitine for the piglets. Milk from *L*-carnitine supplemented sows contains higher concentrations of *L*-carnitine than milk from control sows [28, 58]. *L*-carnitine is required for both, the release of fatty acids from adipose tissue and the fatty acid utilization [60–62]. Therefore, higher tissue concentrations of *L*-carnitine supply through the milk might improve the release of energy from fatty acids which might contribute to the increased growth of suckling piglets of *L*-carnitine supplemented sows.

Mobilization of Energy from Adipose Tissue in Sows during Lactation

Mobilization of adipose tissue plays an important role in lactating sows because they are usually unable to cover their energy requirement for milk production from the diet alone. Previous studies in piglets and growing-finishing pigs have shown that dietary L-carnitine increases the utilization of fatty acids and spares protein [15, 19, 23]. This effect is caused by increased activity of carnitine palmitoyl transferase-1 (CPT-1) and increased utilization of amino acids for protein synthesis [16]. We assumed that sows supplemented with *L*-carnitine might be able to release more energy from adipose tissue during lactation, which can be used for producing surplus milk. To investigate this hypothesis we fed sows a diet low in energy and protein to stimulate mobilization of body pools. What we found was that sows supplemented with 125 mg of L-carnitine per day during the pregnancy and 250 mg of L-carnitine during the lactation were able to produce 18% more milk than control sows and their litters gained 20% more weight during the suckling period than those of control sows. Moreover, L-carnitine supplemented sows released 36% more body fat than control sows while the mobilization of body protein did not differ from untreated control sows [59]. These data suggest that in sows L-carnitine

stimulates the mobilization of energy from adipose tissue, which can be used for producing surplus milk. We also found increased concentrations of IGF-1 and adrenalin in plasma of *L*-carnitine supplemented sows, hormones which stimulate the lipolysis of adipose tissue [40]. The observation that mobilization of body protein was not increased although *L*-carnitine supplemented sows produced more milk, suggests that *L*-carnitine had a protein-sparing effect in sows. This is presumably due to increased secretion of IGF-1, which reduces protein catabolism.

Growth Performance of Piglets after Weaning

The observation by *Musser et al.* [42] that piglets of *L*-carnitine supplemented sows have more muscle fibres than those of control sows raised the question whether they also have a higher growth performance after weaning. To address this question we compared the growth performance of piglets of sows supplemented with 125 mg of L-carnitine per day during pregnancy and 250 mg of L-carnitine during lactation with that of piglets of control sows [63]. Feed intake, daily body weight gain, and gain: feed ratio of both groups of piglets did not differ. This means that Lcarnitine supplementation of sows does not influence the post-weaning growth performance of their offspring. However, supplementation of the piglets' diet with 50 mg of *L*-carnitine per kg of body weight increased their daily body weight gains. Piglets fed diets supplemented with L-carnitine also showed a tendency towards an increased gain:feed ratio (p = 0.06). This finding agrees with other studies in which L-carnitine supplementation increased the gain: feed ratio and protein accretion in piglets [19, 22, 23]. On the other hand, there are also studies in which L-carnitine supplementation did not alter the growth performance of piglets [21, 64]. These studies suggest that the effect of L-carnitine on piglet growth is influenced by other experimental factors such as the energy or lysine content of the diet [19, 23].

Conclusion

Studies reported in the literature clearly show that supplementation of diets with *L*-carnitine during pregnancy and lactation has beneficial effects in sows. This implies that the endogenous synthesis of *L*-carnitine by sows does not cover the amount required for maximum performance during pregnancy and lactation. Results of several studies indicate that *L*-carnitine influences the hormonal status of pregnant sows and thereby improves fetal nutrition. Molecular mechanisms by which *L*-carnitine influences the hormonal status of sows require further investigation.

References

- [1] Foster CV, Harris RC, Pouret EJ (1989) Vet Rec 25: 125
- [2] Rivero JL, Sporleder HP, Quiroz-Rothe E, Vervuert I, Coenen M, Harmeyer J (2002) Equine Vet J Suppl 34: 269
- [3] Zeyner A, Harmeyer J (1999) Arch Tierernährg 52: 115
- [4] LaCount DW, Drackley JK, Weigel DJ (1995) J Dairy Sci 78: 1824
- [5] Greenwood RH, Titgemeyer EC, Stokka GL, Drouillard JS, Loest CA (2001) J Anim Sci 79: 254

- [6] Citil M, Harmeyer J, Fürll M (2003) Berl Münch Tierärztl Wochenschr 116: 322
- [7] Leibetseder J (1995) Arch Tierernährg 48: 97
- [8] Harmeyer J, Schlumbohm C (1997) In: Schubert R, Flachowsky G, Bitsch R, Jahreis G (eds) Vitamine und Zusatzstoffe in der Ernährung von Mensch und Tier. Buch- und Kunstdruckerei Kessler, Weimar, pp 42
- [9] Richter VG, Schlumbohm C, Baumgartner M, Ochrimenko WI (1998) Arch Geflügelk 62: 1
- [10] Harmeyer J, Baumgartner M (1998) In: Jeroch H, Nonn H, Eder K (eds) 5. Tagung Schweineund Geflügelernährung. Wissenschaftlicher Fachverlag Dr. Fleck, Wittenberg, pp 195
- [11] Buyse J, Janssens GP, Decuypere E (2001) Br Poult Sci 42: 230
- [12] Celik L, Ozturkcan O (2003) Arch Tierernährg 57: 27
- [13] Rodehutscord M, Timmler R, Dieckmann A (2002) Arch Tierernährg 56: 431
- [14] Owen KQ, Ji H, Maxwell CV, Nelssen JL, Goodband RD, Tokach MD, Tremblay GC, Koo SI, Blum SA (1997) J Anim Sci 71 (Suppl 1): 62 (Abstr)
- [15] Owen KQ, Nelssen JL, Goodband RD, Tokach MD, Friesen KG (2001) J Anim Sci 79: 1509
- [16] Owen KQ, Ji H, Maxwell CV, Nelssen JL, Goodband RD, Tokach MD, Tremblay GC, Koo IS (2001) J Anim Sci 79: 3104
- [17] Wolfe RG, Maxwell CV, Nelson EC (1978) J Nutr 108: 1621
- [18] Kerner J, Froseth JA, Miller ER, Bieber LL (1984) J Nutr 114: 854
- [19] Heo K, Odle J, Han IK, Cho W, Seo S, Van Heugten E, Pilkington DH (2000) J Nutr 130: 1809
- [20] Heo KN, Lin X, Han IK, Odle J (2002) J Nutr **132**: 1989
- [21] Hoffman LA, Ivers DJ, Ellersieck MR, Veum TL (1993) J Anim Sci 71: 132
- [22] Rincker MJ, Carter SD, Real DDE, Nelssen JL, Tokach MD, Goodband RD, Dritz SS, Senne BW, Fent RW, Pettey LA, Owen KQ (2003) J Anim Sci 81: 2259
- [23] Owen KQ, Nelssen JL, Goodband RD, Weeden TL, Blum SA (1996) J Anim Sci 74: 1612
- [24] Kozink DM, Estienne MJ, Harper AF, Knight JW (2004) Theriogenology 61: 1247
- [25] Wittek T, Elze K, Scharfe S, Seim H (1999) Züchtungsk 71: 219
- [26] Musser RE, Goodband RD, Tokach MD, Owen KQ, Nelssen JL, Blum SA, Dritz SS, Civis CA (1999) J Anim Sci 77: 3289
- [27] Ramanau A, Kluge H, Spilke J, Eder K (2002) Arch Anim Nutr 56: 287
- [28] Ramanau A, Kluge H, Spilke J, Eder K (2004) J Nutr 134: 86
- [29] Eder K, Ramanau A, Kluge H (2001) J Anim Physiol a Anim Nutr 85: 73
- [30] Woodworth JC (2002) The Effects of Carnitine and/or Chromium on Blood Hormones and Metabolites of Gestating Swine. PhD Thesis, Kansas State University
- [31] Cederblad G, Fahraeus L, Lindgren K (1986) Am J Clin Nutr 44: 379
- [32] Genger H, Sevelda P, Vytiska-Binstorfer E, Salzer H, Legenstein E, Lohninger A (1988) Z Geburtsh u Perinat 192: 134
- [33] Cho SW, Park YO, Cha YS (2003) FASEB J 17: A733
- [34] Schoderbeck M, Auer B, Legenstein E, Genger H, Sevelda P, Salzer H, März R, Lohninger A (1995) J Perinat Med 23: 477
- [35] Böhles H, Evangeliov A, Bervoets K, Eckert I, Sewell AC (1994) Eur J Pediatr 153: S57
- [36] Lohninger A, Laschan C, Auer B, Linhart L, Salzer H (1996) Wien Klin Wochenschr 108: 33
- [37] Coffey TM, Shireman RB, Herman DL, Jons EE (1991) J Nutr 121: 1047
- [38] Baltzell JK, Bazer FW, Miguel SG, Borum PR (1987) J Nutr 117: 754
- [39] Borum PR (1983) Annu Rev Nutr 3: 233
- [40] Doberenz J, Ramanau A, Kluge H, Eder K (2004) In: Rodehutscord M (ed) 7. Tagung Schweineund Geflügelernährung. Wissenschaftlicher Fachverlag Dr. Fleck, Wittenberg, pp 93
- [41] Wigmore PMC, Stickland NC (1983) Growth 47: 67
- [42] Musser RE, Dritz SS, Goodband RD, Tokach MD, Davis DL, Nelssen JL, Owen KQ, Campbell RE, Hanni S, Bauman JS, Heintz M (1999) Swine Day 1999: 37

- [43] Woodworth JC, Minton JE, Tokach MD, Nelssen JL, Goodband RD, Koo SI, Owen KQ (2004) Domest Anim Endocrinol 26: 1
- [44] Lohninger A, Krieglsteiner P, Riedl W, Erhardt W, Blümel G (1978) Z Geburtsh u Perinat 182: 29
- [45] Salzer H, Husslein P, Lohninger A, Binstorfer E, Langer M, Schönbauer M, Wagner G, Simbruner G, Popow Ch (1983) Wien Klin Wochenschr 95: 724
- [46] Lohninger A, Böck P, Dadak C, Feiks A, Kaiser E (1990) J Clin Chem Clin Biochem 28: 313
- [47] Batenburg JJ (1992) Am J Physiol 262: L367
- [48] Lohninger A, Kriegelsteiner HP, Salzer H, Erhardt W, Eppl W, Kaier E (1985) In: Lohninger A, Kaiser E (eds) Carnitine – Its Role in Lung and Heart Disorders. Karger, Basel, pp 66
- [49] Salzer H, Lohninger A, Sevelda P, Legenstein E (1985) Gynäkologe 25: 72
- [50] Novak M, Monkus EF, Chung D, Buch M (1981) Pediatrica 67: 95
- [51] Warshaw JB, Curry E (1980) J Pediatr 97: 122
- [52] Strack E, Dieckhoff J, Theile L, Rotzsch W (1960) Z Kinderheilk 84: 458
- [53] Williams IH (1995) In: Hennessy DP, Cranwell PD (eds). Manipulating Pig Production V. Australian Pig Science Association, Werribee, Victoria, Australia, pp 71
- [54] Auldist DE, Morrish L, Wakeford C, King RH (1995) In: Hennessy DP, Cranwell PD (eds) Manipulating Pig Production V. Australian Pig Science Association, Werribee, Victoria, Australia, pp 137
- [55] Spinka M, Illmann G, Algers B, Stetkova Z (1997) J Anim Sci 75: 1223
- [56] Campbell RG, Dunkin AC (1982) Anim Prod 35: 185
- [57] Etienne M, Dourmad J-Y, Noblet J (1998) In: Verstegen MWA, Moughan PJ, Schrama JW (eds) The Lactating Sow. Wageningen Pers, Wageningen, The Netherlands, pp 285
- [58] Musser RE, Goodband RD, Tokach MD, Owen KQ, Nelssen JL, Blum SA, Campbell RG, Smits R, Dritz SS, Civis CA (1999) J Anim Sci 77: 3296
- [59] Eder K, Ramanau A, Felgner J, Kluge H (2004) In: Rodehutscord M (ed) 7. Tagung Schweineund Geflügelernährung. Wissenschaftlicher Fachverlag Dr. Fleck, Wittenberg, pp 90
- [60] Hahn P (1982) Annu Rev Nutr 2: 91
- [61] Novak M, Penn-Walker D, Hahn P, Monkus EF (1975) Biol Neonate 25: 84
- [62] Novak M, Penn-Walker D, Monkus EF (1975) Biol Neonate 25: 95
- [63] Birkenfeld C, Ramanau A, Kluge H, Spilke J, Eder K (2004) J Anim Physiol a Anim Nutr (in press)
- [64] Cho WT, Kim JH, Bae SH, Han IK, Han YK, Heo KN, Odle J (1999) Asian-Australian J Anim Sci 12: 799

1544