

# Leptin and lactogenesis in the periparturient dairy goat\*

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## ABSTRACT

We aimed to study the variation in milk leptin around the time of parturition in dairy goats, and whether milk leptin can be absorbed and play a role in systemic regulation in the neonate. The results indicate that colostrogenesis in goats does not involve accumulation of leptin in colostrum, but secretion of leptin into milk increases and peaks within the first 2 days of lactation. This is likely the result of local regulation in the mammary gland in relation to lactogenesis. Our results did not suggest that milk leptin may play a role in systemic regulation in the neonatal goat.

KEY WORDS: goat, milk, pre-partum secretion, insulin, cortisol, neonate, colostrogenesis

## INTRODUCTION

Leptin has primarily been studied in regulation of energy intake and expenditure. It acts as an endocrine signal from adipose tissue, the main site of leptin production, to the hypothalamus (Casanueva and Dieguez, 1999; Houseknecht and Spurlock, 2003). Detection of leptin in milk of various species including ruminants (McFadin et al., 2002; Smith and Sheffield, 2002) has led to the suggestion that leptin among other physiological functions may be involved in neonatal regulation of feed intake, thermoregulation, immune response, and development of intestinal function (Baratta, 2002). The objectives of this study

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were to investigate the variation in leptin levels in milk during colostrogenesis and lactogenesis, and to investigate whether milk leptin can be absorbed and possibly play a role in systemic regulation in the neonate.

## MATERIAL AND METHODS

All animal experimental procedures conformed to national Danish legislation. Four multiparous cross-bred Saanen and Black Danish Dairy goats, and their offspring ( $n=8$ ) were used. Does were previously prepared with exteriorised carotid arteries and with both superficial epigastric caudal veins (milk veins) exteriorized in skin covered loops. They were fed, and during lactation, milked twice daily at appr. 08.30 and 15.30 h. Does were fed hay *ad libitum* and a concentrate ration calculated to fulfil Danish requirements for energy and protein. Does were weighed weekly pre-partum, biweekly post-partum, and kids daily during the first week. Blood samples were taken by vacutainers from carotid artery and milk veins at regular intervals from week 5 pre-partum to 7 post-partum concentrated around parturition. Blood samples from kids were taken by venipuncture before and 6 h after first colostrum meal, day 3 and 7 post-partum. Pre-partum milk samples were obtained from only one gland app. 25, 12, 7, and 3 days pre-partum, and post-partum from both glands at each milking the first 10 days of lactation, and days 20 and 50 post-partum. All samples were stored at  $-20^{\circ}\text{C}$  until assayed. Plasma and milk concentrations of leptin were measured by RIA as described by Blache et al. (2000). Plasma insulin and cortisol were determined by ELISA (Sheep Insulin ELISA, EIA-2339, DRG Instruments GmbH, Germany) and RIA (Immulite<sup>®</sup> Cortisol, LKCO51, Diagnostic Products Corporation, LA, CA, USA), respectively. Data were analysed by CORR and MIXED procedure for repeated-measures data using SAS (SAS Institute Inc., Cary, NC, USA).

## RESULTS

Milk leptin increased after parturition and peaked within 2 days postpartum (Figure 1). Apart from that there were no differences between milk leptin levels in prepartum milk-like secretions, colostrum, and milk during established lactation. Plasma leptin levels were higher prepartum than postpartum ( $P<0.05$ ), decreased at parturition and during the first few days of lactation (Figure 2). Plasma leptin levels were positively correlated to systemic plasma insulin and serum cortisol ( $P<0.001$ ) throughout the study period, except that a cortisol peak was observed at the time of parturition (Figure 3). Slightly higher correlations were found to leptin levels in milk veins compared to arteries ( $r=0.64$  and  $0.52$  for insulin, respectively, and  $r=0.55$  and  $0.42$  for cortisol, respectively;

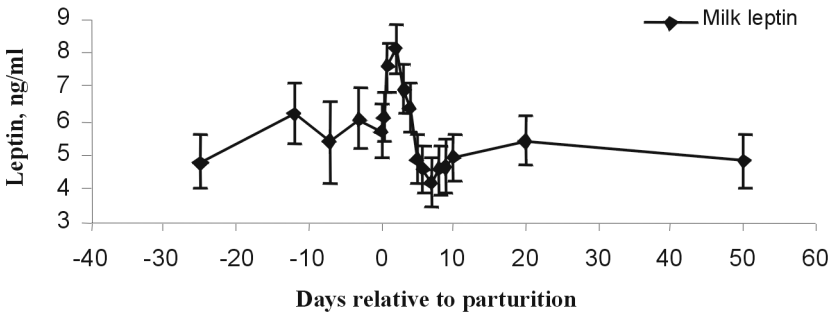


Figure 1. Milk leptin concentrations (LS means ± SE)

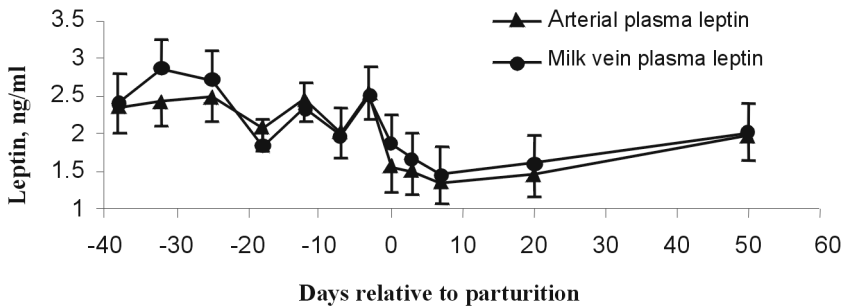


Figure 2. Leptin concentrations in plasma from carotid arteries and milk veins plasma respectively (LS means ± SE)

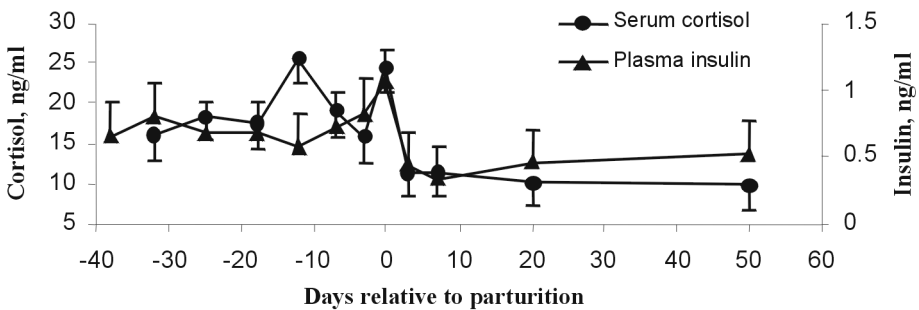


Figure 3. Serum cortisol and plasma insulin concentrations (LS means ± SE)

P<0.01). Arterial-venous differences for leptin across the mammary gland were at no time significantly different from 0. Intake of the first colostrum meal had no influence on plasma leptin levels in the kids. Plasma leptin levels in the kids were also independent of milk leptin levels as well as maternal plasma leptin levels.

## DISCUSSION

Milk leptin levels were not affected by circulating leptin levels in plasma. It has been reported that leptin levels are highest in colostrum, and that leptin may be accumulated in the mammary gland during colostrogenesis in late gestation (McFadin et al., 2002). Contrary to this we found that secretion of leptin in milk did not peak until 2 days postpartum, which indicates that secretion of leptin increases in connection with onset of lactation. It has been suggested that milk leptin may play a role in the neonate. However, in this study plasma leptin levels in kids were not affected by the first colostrum meal nor leptin levels in the milk.

## CONCLUSIONS

Colostrogenesis in goats does not seem to involve accumulation of leptin in colostrum. Rather increasing leptin levels in milk during the first 2 days of lactation suggests a local regulation of leptin secretion in milk in relation to onset of lactation. Our results do not support the hypothesis, that absorption of leptin from colostrum and milk might play a role in systemic regulation in the neonatal goat. It can, however, not be ruled out that leptin may play a local regulatory role in the gut or as a cytokine in stimulation of neonatal immune function.

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