

# Effect of fat supplementation on leptin, insulin-like growth factor I, growth hormone, and insulin in cattle

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## Abstract

We investigated the effect of fat supplementation on plasma levels of hormones related to metabolism, with special attention to leptin, in cows in early lactation and in feedlot steers. In experiment 1, 34 lactating cows received no fat or else 0.5 or 1.0 kg of partially hydrogenated oil per day in addition to their basal diet from day 20 before the expected calving date to day 70 postpartum. In experiment 2, part of the corn in the basal concentrate was replaced with 0.7 kg of the same oil such that the diets were isocaloric; 18 cows received the fat-substituted diet and 18 a control diet from day 20 before the expected calving date to day 75 postpartum. In experiment 3, calcium salts of fatty acids were added to the basal diet of 14 feedlot steers for 80 d; another 14 steers received a control diet. The basal plasma levels of leptin were higher in the cows than in the steers. Dietary fat supplementation did not affect the leptin levels in the lactating cows but lowered the levels in the feedlot steers despite greater energy intake and body fatness (body weight) in the steers receiving the supplement than in those receiving the control diet. The levels of insulin-like growth factor I and insulin were decreased with dietary fat supplementation in the lactating cows but were unaffected in the steers, suggesting that responses to fat ingestion depend on the physiological state of the animal, including age and sex. Finally, no effects of supplementary fat on the level of growth hormone were demonstrated in any of the models.

## Résumé

*L'effet de l'ajout de gras sur les niveaux plasmatiques d'hormones reliées au métabolisme, avec une attention particulière à la leptine, a été étudié chez des vaches en début de lactation et chez des bouvillons en parc d'engraissement. Dans l'expérience 1, 34 vaches en lactation n'ont reçu aucun gras ou bien 0,5 ou 1 kg/jour d'huile partiellement hydrogénée en supplément de leur ration de base commençant 20 jours avant la date prévue de vêlage et allant jusqu'à 70 jours post-partum. Dans l'expérience 2, une partie du maïs dans le concentré de base a été remplacé par 0,7 kg de la même huile de manière à ce que les diètes soient isocaloriques; 18 vaches ont reçu la diète avec le substitut de gras et 18 une diète témoin débutant 20 jours avant la date prévue de vêlage et allant jusqu'à 75 jours post-partum. Dans l'expérience 3, des sels de calcium d'acides gras ont été ajoutés à la diète de base de 14 bouvillons d'embouche pendant 80 jours; 14 autres bouvillons ont reçu une diète témoin. Les niveaux plasmatiques de base de leptine étaient plus élevés chez les vaches que chez les bouvillons. Un supplément de gras alimentaire n'a pas affecté les niveaux de leptine chez les vaches en lactation mais a diminué les niveaux chez les bouvillons d'embouche malgré un plus grand apport calorique et plus de gras corporel (poids corporel) chez les bouvillons recevant le supplément que chez ceux recevant la diète témoin. Les niveaux du facteur de croissance similaire à l'insuline de type I et l'insuline étaient diminués chez les vaches laitières recevant le supplément en gras mais non-modifiés chez les bouvillons, ce qui suggère que les réponses à l'ingestion de gras dépendent de l'état physiologique de l'animal, incluant son âge et le sexe. Finalement, l'ajout de gras n'a démontré aucun effet sur les niveaux d'hormone de croissance dans tous les modèles étudiés.*

(Traduit par Docteur Serge Messier)

## Introduction

Energy intake is directly related to production in beef- and milk-producing systems. Dairy cows in early lactation are usually in a negative energy balance. Supplementation of the diet with fat may be used to maintain a high dietary energy density while avoiding ruminal acidosis, which may result when excessive amounts of starch are provided. We recently reported improved production in grazing dairy cows in early lactation with the addition of hydrogenated

oil to the food concentrate (1) or with replacement of the starch by fat (2). Fat supplementation has also been used in growing and finishing beef cattle to increase production efficiency and improve meat quality (3,4).

Metabolic modifications due to fat intake have been reported to occur mainly at hepatic and adipose tissue levels (3,5). Alterations of blood concentrations of metabolic hormones have also been observed (4,6). Whereas most studies have reported that dietary fat supplementation lowered or did not change the blood concentration

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Received March 30, 2006. Accepted August 21, 2006.

of insulin-like growth factor I (IGF-I), contradictory results were obtained for serum growth hormone (GH) and insulin, their levels being as reported as augmented (5,7), unchanged (8), or, in the case of insulin, diminished (6).

Leptin is an adipose-derived hormone that regulates a wide variety of physiological processes. As a signal from energy stores to the hypothalamus, it is involved in food-intake regulation and energy homeostasis. Reciprocally, there is long-term regulation of leptinemia by nutritional status and short-term regulation by food intake (9). It has been shown that fat feeding increases the plasma leptin concentration in rats, depending on total energy intake and type of fat (10). In ruminants, no effect of fat ingestion on leptin levels could be consistently demonstrated (9), whereas in 1 study the leptin concentration was shown to increase in lambs given fat supplementation by means of rumen bypass (11).

In this study we examined the effect of fat supplementation on blood concentrations of leptin, IGF-I, GH, and insulin in adult lactating cows and young finishing feedlot steers.

## Materials and methods

The experiments were conducted at the National Institute of Agricultural Technology in Balcarce (37°45' south, 58°18' west), Argentina.

### Experimental designs

In experiment 1, 34 multiparous Holstein cows of mean body weight (BW) 570 kg (standard error 68 kg) were grouped according to the number of lactations and milk production registered during the first 70 d in milk (DIM) for the previous lactation and then randomly allocated to 1 of 3 dietary treatments: no supplemental fat (T0, 12 cows), 0.5 kg/d supplemental fat (T0.5, 12 cows), and 1 kg/d supplemental fat (T1, 13 cows), as previously described (1). The fat, partially hydrogenated oil (melting point 58°C to 60°C) containing C16:0 (30.3%), C18:0 (34.9%), C18:1 (21.8%), C18:2 (3.3%), and other fatty acids (9.7%), was added to a basal concentrate of ground corn (91.6%), fish meal (8%), and calcium chloride (0.4%). The concentrates were offered in 2 equal feedings during milking times (0600 and 1600 h) from day 20 before the expected calving date to day 70 postpartum. The cows grazed together on mixed pastures of alfalfa and orchardgrass in a daily-moving grazing system that provided each cow with a daily herbage allowance of 30 kg dry matter (DM). The daily total DM intake (DMI) per cow was 23.0, 19.8, and 19.6 kg and the estimated net energy of lactation (NE<sub>L</sub>) intake 36.7, 33.9, and 31.2 Mcal in the T0, T0.5, and T1 groups, respectively.

In experiment 2, 36 multiparous Holstein cows of mean BW 599 (standard error 49) kg were grouped according to the number of lactations and milk production registered during the first 70 DIM for the previous lactation and then randomly allocated to 1 of 2 dietary treatments, 18 cows per treatment. The control group was fed a concentrate containing corn grain (4.49 kg DM/d), fish meal (0.37 kg DM/d), and calcium chloride (0.02 kg DM/d), whereas the experimental group received a concentrate in which part of the corn grain was replaced by the same partially hydrogenated oil as in experiment 1 (corn grain, 2.87 kg DM/d; fat, 0.7 kg DM/d), as

previously described (2). The concentrates were isocaloric and were offered in 2 equal feedings during milking times (0600 and 1600 h) from day 20 before the expected calving date to day 75 postpartum. Again, the cows grazed together on mixed pastures of alfalfa and orchardgrass in a daily-moving grazing system. The daily total DMI per cow was 20.3 and 19.6 kg and the estimated NE<sub>L</sub> intake 30.1 and 30.4 Mcal in the control and experimental groups, respectively.

In experiment 3, 28 Aberdeen Angus and Aberdeen Angus–Hereford feedlot steers were weaned at 6 mo of age, at a mean BW of 124 (standard error 12.3) kg. After pairing by BW and strain, they were randomly assigned to 1 of 2 dietary treatments. The control diet was composed of corn silage (22.6%), corn grain (40.6%), sunflower meal (35.4%), and mineral salts (1.4%). The experimental diet consisted of the control diet plus commercial calcium salts of fatty acids (Full fat BP; Inagro, Buenos Aires, Argentina) containing 1.6% C14:0, 16% C16:0, 1.6% C16:1, 13.5% C18:0, 32% C18:1, 30% C18:2, 0.8% C18:3, 0.3% C20:0, and other minor fatty acids (4.2%). The lipid supplementation was adjusted every 15 d to 0.13% of the mean BW. Water was provided ad libitum. The animals were fed twice a day. The food offered and rejected was weighed daily to adjust the provision in order to have 10% food rejection.

### Sample collection and analysis

In experiments 1 and 2, after an adaptation period of 21 d (from 14 d before the expected calving date [−14] to 7 DIM), milk production was individually recorded daily. At 30 and 60 DIM for experiment 1 and at 15, 30, 45, 60, 75, and 105 DIM for experiment 2, blood samples were collected in heparinized tubes from the jugular vein immediately after the morning milking, and the body condition score (BCS) was recorded by 2 independent observers using a 5-point scale (1 = thin to 5 = fat). Plasma samples were stored at −20°C until assayed.

In experiment 3, the animals were weighed every 15 d for meal adjustment. The dorsal fat depth (DFD) was measured by ultrasonography with a 3.5-MHz linear transducer (Pie Medical Scanner 480; Pie Medical Equipment, Maastricht, the Netherlands) between ribs 12 and 13 on days 14 and 84 from the beginning of the experiment. On the same day, blood samples were collected in heparinized tubes from the jugular vein before the morning feeding. Plasma samples were stored at −20°C until assayed.

All plasma samples were assayed for leptin, IGF-I, GH, and insulin by radioimmunoassay (RIA). Leptin determinations were performed by a double antibody method with ovine-specific anti-serum and recombinant bovine leptin (DSLabs, Webster, Texas, USA) iodinated in our laboratory by the chloramine T method (12). The specificity of the ovine antibody for bovine leptin had been shown previously (13). The minimum detectable concentration of leptin was 0.4 ng/mL. For IGF-I RIA, acid-ethanol extraction was performed, as previously described (14); IGF-I antibody UB2-495 (National Institute of Diabetes and Digestive and Kidney Diseases [NIDDK], Rockville, Maryland, USA) was used, and the assay sensitivity was 60 pg per tube. The GH concentration was determined with use of an anti-ovine antibody (NIDDK) (15); the minimum detectable concentration was 0.8 ng/mL. The insulin concentration was measured as previously described (15) with use of anti-bovine insulin antibody (Sigma, St. Louis, Missouri, USA) and standard human

**Table 1. Results of experiment 1, in early-lactating cows, at 30 and 60 d in milk (DIM)**

Variable	DIM	Treatment; mean (and standard error)			Effect; P-value	
		T0 (n = 11)	T0.5 (n = 11)	T1 (n = 12)	Time	Diet
FCM (kg/d)	30	24.3 (0.9)	25.9 (1.4)	27.3 (1.7)	0.00093	< 0.05 T0≠T1
	60	22.8 (1.0)	23.7 (0.7)	25.5 (2.0)		
BCS <sup>a</sup>	30	2.35 (0.08)	2.33 (0.11)	2.17 (0.06)	0.0021	NS
	60	2.22 (0.11)	2.07 (0.10)	2.00 (0.08)		
Plasma level (ng/mL)						
	Leptin	30	2.91 (0.42)	2.40 (0.34)	3.08 (0.27)	0.00028
	60	4.98 (0.49)	4.31 (0.63)	4.24 (0.45)		
IGF-I	30	179.9 (20.6)	161.3 (24.2)	156.5 (23.1)	0.00077	NS
	60	235.5 (24.3)	216.4 (14.6)	187.8 (23.3)		
GH	30	2.21 (0.17)	2.59 (0.36)	2.36 (0.18)	NS	NS
	60	2.59 (0.10)	2.61 (0.20)	3.00 (0.26)		
Insulin	30	1.31 (0.32)	1.69 (0.28)	0.90 (0.22)	0.00017	< 0.05 T0≠T1≠T0.5
	60	0.62 (0.16)	0.44 (0.11)	0.10 (0.04)		

T0 — control diet (0 fat); T0.5 = control diet plus 0.5 kg fat daily; T1 = control diet plus 1 kg fat daily; FCM — fat-corrected milk yield; IGF — insulin-like growth factor; GH — growth hormone; NS — not significant.

<sup>a</sup> Body condition score on a 5-point scale (1 = thin to 5 = fat).

insulin provided by Laboratorios Beta (Buenos Aires, Argentina); the minimum detectable concentration was 0.05 ng/mL. The intra-assay and interassay coefficients of variation were always lower than 8% and 11%, respectively.

## Statistical analysis

A 2-factor analysis of variance for repeated measures was performed with Statistica software (StatSoft, Tulsa, Oklahoma, USA) to compare the effects of treatment and period on hormone levels. In all cases, if the *F*-ratio was found to be significant, individual means were compared by Tukey's honest significant difference test or Fisher's protected least significant difference test; if the *F*-ratio was not significant, groups of means were analyzed by the same tests. The Pearson correlation test, performed with InStat software (GraphPad Software, San Diego, California, USA), was used to evaluate for any correlation between leptin concentration and BCS in experiment 1.

## Results

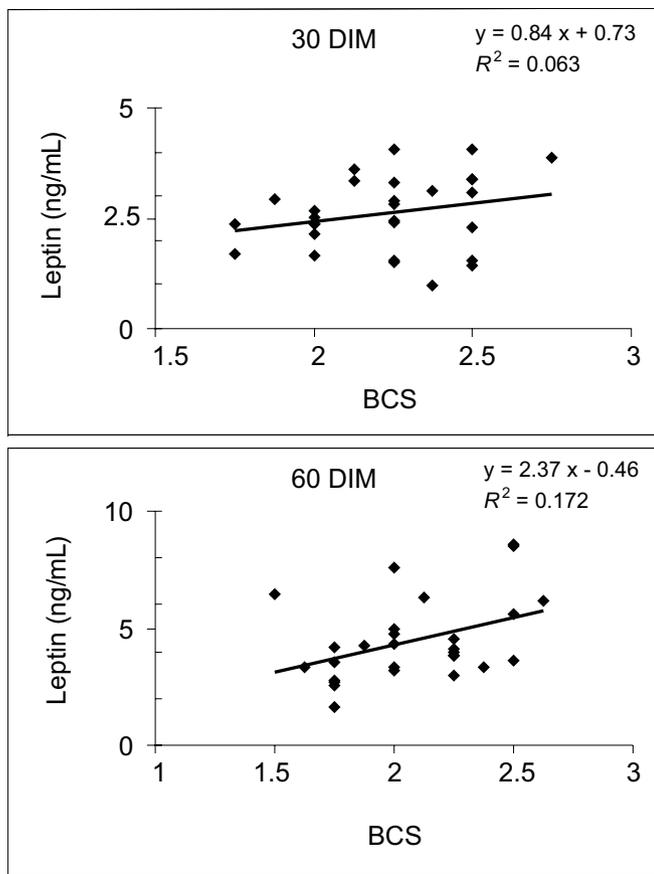
A mean of 19 (standard error 0.86) d and 15 (standard error 0.86) d before the calving date was the actual onset of experiments 1 and 2, respectively.

In experiment 1 (Table 1), the mean fat-corrected milk (FCM) yield was higher in the T1 group than in the T0 group at 30 and at 60 DIM ( $P < 0.05$ ), as previously reported (1). The mean BCS diminished between 30 and 60 DIM, but no significant differences were observed

between the dietary groups. The mean leptin and IGF-I plasma levels increased between 30 and 60 DIM ( $P < 0.01$ ), but no significant differences were observed between the treatment groups, although the mean IGF-I concentration tended to diminish with increasing fat. No differences were observed in the plasma GH value between times or between dietary groups. The mean plasma insulin level was lower at 60 DIM than at 30 DIM ( $P < 0.01$ ) and lower in the T1 group than in the T0.5 and T0 groups ( $P < 0.05$ ). Positive correlations ( $P < 0.05$ ) between leptin level and BCS were found at 30 and at 60 DIM (Figure 1).

In experiment 2 (Figure 2), in which corn grain was partially replaced by fat for half of the cows, the mean leptin level fluctuated erratically from 15 to 105 DIM; no interaction between time and treatment or significant differences in the effects of time or treatment were found. The mean plasma IGF-I level was lower in the experimental group than in the control group ( $P < 0.05$ ) regardless of lactation time; no effect of time was noticed for this hormone. The mean plasma GH level diminished gradually from 15 to 75 DIM, without differences between treatments. An interaction was found between treatment and time for the mean plasma insulin level, which increased between 15 and 45 DIM, decreased at 75 DIM, and then increased again; at 105 DIM, the mean insulin level was higher in the experimental group than in the control group ( $P < 0.05$ ).

In experiment 3, the individual daily DMI was not affected by diet in the feedlot steers, at a mean of 5.9 (standard error 0.3) and 5.5 (0.2) kg DM, in the experimental and control groups, respectively ( $P = 0.28$ ). However, the estimated metabolizable energy (ME) was



**Figure 1.** Correlation between the plasma leptin concentration and the body condition score (BCS) in the cows in experiment 1 (all dietary groups together) at 30 (upper panel,  $n = 33$ ) and 60 (lower panel,  $n = 31$ ) d in milk (DIM). The BCS was determined on a 5-point scale (1 = thin to 5 = fat).

higher ( $P < 0.05$ ) in those receiving the supplementary calcium salts of fatty acids than in those receiving the control diet, at a mean of 16.4 (0.6) versus 14.4 (0.7) Mcal. The mean BW and DFD increased with time in both groups and were higher in the fat-supplemented group at 84 d (Table II). The mean plasma leptin level was lower in the experimental group than in the control group and did not differ between times. The mean IGF-I and GH concentrations were not influenced by time or treatment. The mean insulin concentration increased with time but was not changed by fat supplementation.

## Discussion

The present discussion is focused on the effect of fat ingestion on hormones that are related to metabolic regulation and may be directly or indirectly involved in production. Special attention is paid to leptin, as it is a relatively novel hormone that has been related to body fatness, food intake, and energy balance (16). The fact that the addition of fat sources of energy to the diet enhanced the production of milk or meat is not discussed here, as it was the focus of other publications (1,2).

The first remarkable observation regarding leptin levels was that the basal concentrations of the hormone in this study were higher in cows than in steers, despite the cows' lactational hypoleptinemia (16) and the negative energy balance in early lactation, which likely

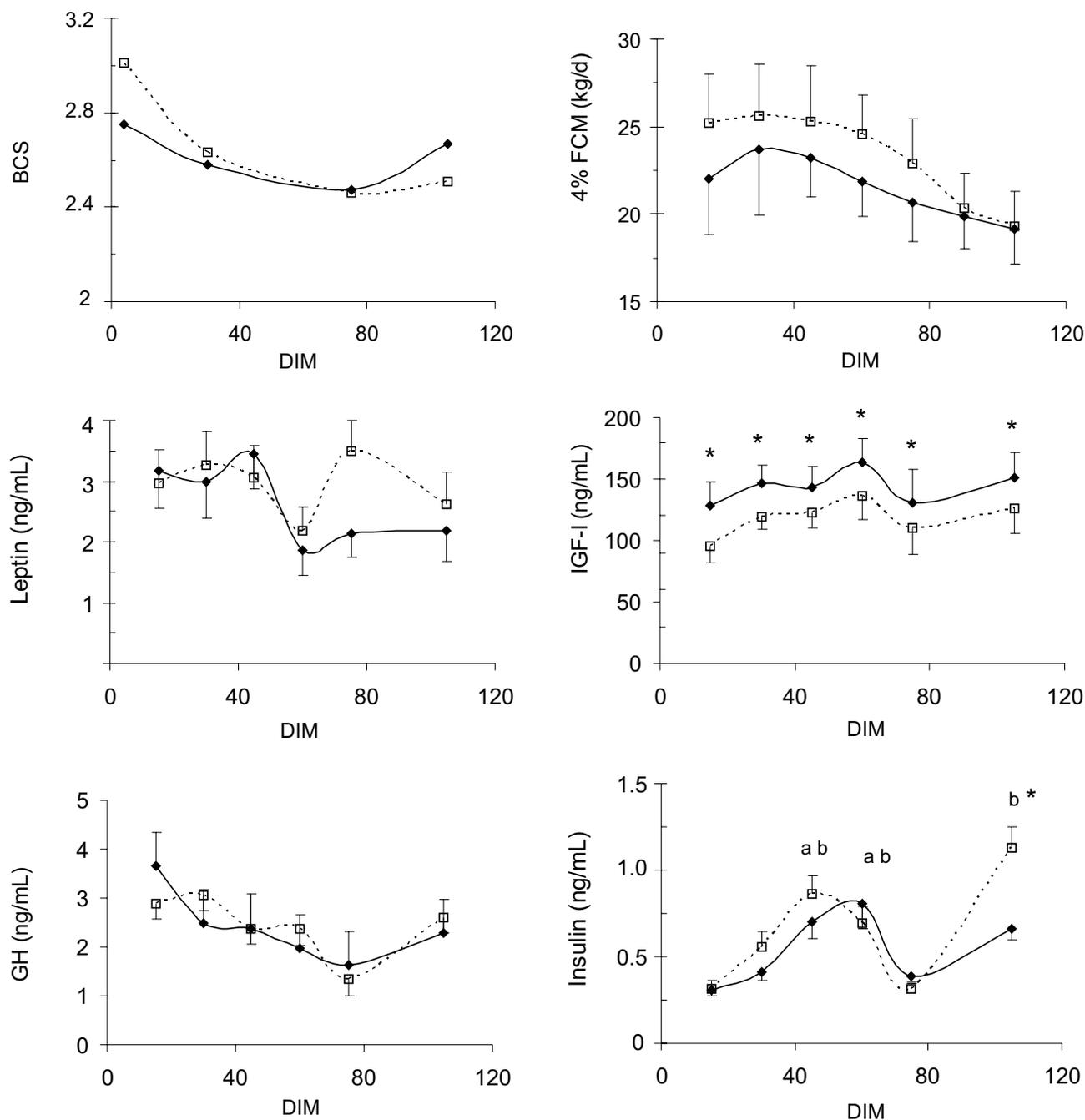
contributed to the depression of leptin levels (17). Sexual differences in the circulating levels of leptin have previously been reported in rodents, primates (including humans), and ovines and have been attributed to the stimulating effect of estrogens and the inhibiting effect of androgens on leptin production, as well as to the differential distribution of subcutaneous and omental fat in males and females (18,19). Whereas fat mass has been shown to be more important than age in determining blood leptin concentrations (20), differential effects of age and breed among the cows and steers in the present experiments cannot be ruled out. On the other hand, the higher basal levels of GH in the steers, in accordance with their growing phase, may have been involved in the lower leptin levels, as GH has been shown to inhibit leptin production in rodents (21). However, direct GH effects on leptinemia appear to be very low in cattle (16).

The mean plasma levels of leptin, as well as of insulin and IGF-I, were lower in the cows in experiment 2 compared with those in experiment 1, in accordance with the higher levels of nonesterified fatty acids (NEFA) and the lower estimated energy balance in the cows in experiment 2 (data not shown).

The dietary addition of, or energy replacement by, fat did not affect the circulating levels of leptin in the lactating cows. Similar results were observed in young heifers and lactating cows receiving a high-fat diet by means of sunflower seeds (4), calcium salts of palm oil or conjugated linoleic acids (22), corn oil or rumen-protected linoleic and linolenic acid (23), or linseed oil (24). Furthermore, when an aqueous emulsion of 18:2-rich oil was injected in the jugular vein of lactating cows, no effect was observed on leptin levels in early lactation, but an increase was reported in late lactation, suggesting different modulation of the hormone depending on the physiological status of the animal (25).

Surprisingly, the addition of calcium salts of fatty acids to the diet of growing steers produced a decrease in plasma leptin concentration, despite the increased DFD, suggesting a strong inhibitory effect of fat intake on leptin production that overrides the likely increase in secretion of the hormone due to both the larger fat stores and the higher net energy intake. Leptin is regulated by energy intake at 2 levels: a long-term effect that depends on adiposity and a short-term effect that is related to feeding level and diet composition (16). Therefore, plasma leptin concentrations are dependent on the amount of energy stored and strongly correlate with fat depots, as was demonstrated in humans (21), sheep (12), and cattle (26). This was also the case to some extent in the present study; however, the correlation was lower at 30 DIM than at 60 DIM, which could be related to a lower energy balance at 30 DIM. Similarly, the correlation between plasma leptin concentration and BCS was reported to be higher in dry late-pregnant cows than in early-lactation cows (26).

On the other hand, it has been shown that short-term fasting causes a rapid reduction in the plasma leptin concentration and that refeeding restores hormone levels in dairy and beef cattle (13,27). In the present experiments, no differences in total DMI were noticed between animals in the various groups. In both experiments with lactating cows, no differences in total  $NE_L$  intake were noticed between animals in the various dietary groups, but, in experiment 3, the total estimated ME intake was higher in the steers receiving the fat supplement than in the control steers. This difference in energy intake is consistent with higher body fatness, which would have been



**Figure 2.** Plasma hormone concentrations in early-lactating cows in experiment 2. The solid lines represent data for the cows that received the control diet; the dashed lines represent data for the cows whose corn grain was partly replaced by fat. Asterisks indicate a significant difference in means between treatment groups ( $P < 0.05$ ), whereas “a” and “b” indicate significant differences between days 0 and 75 in the control group and the experimental group, respectively ( $P < 0.05$ ). GH — growth hormone; 4% FCM — fat-corrected milk yield at 4% milk-fat content; IGF — insulin-like growth factor.

expected to increase, rather than decrease, leptin levels in the steers in the fat-supplemented group. In previous studies, leptin levels correlated with the fat content of the carcass but were unaffected by fat intake in growing dairy heifers receiving calcium salts of palm oil (22) and in beef feedlot heifers given supplemental corn or palm oil (23), although energy intake was not estimated. Nevertheless, a direct effect of fatty acids can be hypothesized, because free fatty acids have been shown to downregulate leptin production in rat adipocytes in primary culture (28). Another possibility is that a higher

intake of corn grain and corn silage in the control group stimulated glucogenic nutrient absorption, since the plasma glucose concentration increased with aging in this group, which could have played a role in the higher plasma leptin levels (16), in agreement with the fact that feeding corn silage increased leptinemia in growing cattle compared with pasture-fed controls that were managed to have the same body fatness (29).

Fat supplementation decreased (in experiment 2) or tended to decrease (in experiment 1) the plasma IGF-I concentration in the

**Table II. Results of experiment 3, in feedlot steers, at 14 and 84 d**

Variable	Day of treatment	Diet; mean (and standard error)		P-value		
		Control	Fat-supplemented	Time by diet interaction	Time effect	Diet effect
BW (kg)	14	173.8 (4.8)	170.7 (5.1)	0.0002		
	84	218.9 (4.6) <sup>a</sup>	233.6 (5.1) <sup>ab</sup>			
DFD (cm)	14	3.19 (0.20)	3.48 (0.17)	0.0047		
	84	3.70 (0.18) <sup>a</sup>	4.92 (0.21) <sup>ab</sup>			
Plasma level						
Glucose (mg/dL)	14	78.4 (3.2)	78.0 (1.6)	0.021		
	84	89.9 (2.4) <sup>a</sup>	81.2 (2.7)			
Leptin (ng/mL)	14	1.97 (0.22)	1.25 (0.22)	NS	NS	0.0051
	84	1.47 (0.20)	1.06 (0.17)			
IGF-I (ng/mL)	14	408 (24)	426 (40)	NS	NS	NS
	84	463 (25)	430 (19)			
GH (ng/mL)	14	7.45 (1.11)	8.48 (1.55)	NS	NS	NS
	84	10.26 (1.42)	10.43 (1.13)			
Insulin (ng/mL)	14	0.82 (0.08)	0.90 (0.10)	NS	0.0001	NS
	84	1.31 (0.08)	1.42 (0.07)			

BW — body weight; DFD — dorsal fat depth.

<sup>a</sup> Different from the previous time ( $P < 0.01$ ) with the same treatment.

<sup>b</sup> Different from the respective control ( $P < 0.01$ ) at the same time.

lactating cows but had no effect in the finishing feedlot steers (in experiment 3). The serum IGF-I level was also reported to be reduced in cows in a similar lactation period by the dietary administration of prilled saturated fatty acids (5) and in young heifers fed whole sunflower seeds as a fat source (4). In contrast, the IGF-I concentration was unchanged in lactating Jerseys given canola oil or oleamide supplements (30) or Holsteins infused postruminally with canola oil (31) and was increased during the estrous phase in lactating cows that received a diet rich in linoleic acid (32), suggesting that not only the fat source but also the physiological state of the animal and the endocrine milieu may account for hormone response.

Dietary fats are partitioned in the liver between various possible metabolic pathways, depending on fatty acid composition and hormone regulation. When NEFA are mobilized from adipose tissue, they are partitioned to accumulate as triglycerides in this organ, which results in poor liver function, as is often observed in cows at calving (33). Because the main part of serum IGF-I is liver-derived (34), we may speculate that a high-fat diet in early-lactating cows may result in impaired liver function and lower IGF-I secretion. However, no increase in liver fat accumulation was observed during fat supplementation in early-lactating cows (35), probably because

dietary fatty acids are mainly absorbed in esterified form. The lower plasma insulin levels in experiment 1, or insulin and GH resistance linked to lower energy balance in experiment 2, could have contributed to the lower plasma IGF-I levels in the fat-supplemented cows.

The plasma insulin concentration was lowered by the highest dose of fat in the lactating cows in experiment 1, whereas in experiment 2 the concentration was unchanged by diet during the trial but increased as a rebound after the end of fat supplementation, and in experiment 3 the plasma insulin concentration was not modified by fat treatment in the finishing steers. These results suggest that fat added to the diet could have an inhibitory effect on circulating levels of insulin in early to peak lactation in cows. Similar results were obtained in lactating cows given dietary supplements of calcium salts of long-chain fatty acids (6) or canola seeds (36) or infused postruminally with canola oil (31). However, no variations of insulin concentration by dietary fat were found in lactating Holsteins (32), lactating Jerseys (30), or beef heifers (8,37). On the contrary, in nonlactating adult beef cows given supplementary saturated, polyunsaturated, or highly polyunsaturated fats, the serum insulin concentration was markedly increased (7). After reviewing

insulin data from 17 fat-feeding studies in lactating cows, Staples et al (38) concluded that a lowered insulin concentration is the result of decreased energy status, in agreement with a review of 13 trials with 19 groups of fat-supplemented cows (3) that showed fat supplementation to increase the BW loss during early lactation and with the trend to lowered energy balance in experiment 1. Furthermore, high-fat diets sometimes decreased the molar proportion of ruminal propionate, which is a potent insulin secretagogue in ruminants (6), although the proportion was not changed in 48 groups of fat-supplemented cows (3).

Finally, we found that the plasma GH concentration was not modified by dietary fat supplementation in any of the present experiments, as has also been reported for prepubertal beef heifers (8) and primiparous postpartum beef cows (39) that received safflower seeds and for lactating dairy cows that received canola oil (36). However, in 3 other studies, the GH concentration was reported to increase in response to dietary fats in lactating dairy cows (5,31) and in undernourished beef cows (40). The different results were probably due to different fat source, as well as differences in the animals used and in their physiological state.

We conclude that dietary fat administration to cattle has diverse effects on metabolic hormones, which depend on the sex, energy intake and balance, and physiological state of the animal. Although fat supplementation does not affect circulating levels of leptin in lactating cows, it decreases the levels in feedlot steers despite increased energy intake and body fatness. On the other hand, circulating IGF-I levels are decreased by fat supplementation in lactating cows but not in feedlot steers, probably indicating a different liver susceptibility to fat intake or to a state of insulin or GH resistance, or both, in lactating cows with decreased energy balance. The GH levels, which were higher in steers than in cows, were not affected by fat administration.

## Acknowledgments

This research was supported in part by grants from the Agencia Nacional de Promoción Científica y Tecnológica (PICT 3632 and PICT 13666), Consejo Nacional de Investigaciones Científicas y Técnicas, and by Molinos Río de la Plata. We also thank NIDDK's National Hormone and Pituitary Program and Dr. Albert F. Parlow for the GH RIA kit and the IGF-I antiserum.

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