Arginine Supplementation Increases Weight Gain, Depresses Antibody Production, and Alters Circulating Leukocyte Profiles in Preruminant Calves Without Affecting Plasma Growth Hormone Concentrations

J. M. Fligger*, C. A. Gibson*, L. M. Sordillo†, and C. R. Baumrucker*3

Departments of *Dairy and Animal Science and †Veterinary Science, The Pennsylvania State University, University Park 16802

ABSTRACT: The hypothesis that dietary L-arginine (L-Arg) supplementation would increase growth hormone (GH) secretion and antibody production in preruminant calves was tested. Sixteen newborn calves were randomly assigned to either Arg+ or Arg− treatment groups. Both groups were fed a single dose of Colostrx© within 6 h after birth followed by milk replacer twice daily until weaning. Beginning with the Colostrx feeding, calves in the Arg+ group were supplemented with L-arginine at 500 mg kg−1d−1, and the Arg− group received equivalent, but unsupplemented, diets. All calves were immunized against keyhole limpet hemocyanin (KLH) on d 4 and received a booster vaccination on d 14. The Arg+ treatment increased (P < .05) plasma L-Arg and urea concentrations an average of 2.8-fold and 26%, respectively, during the 4-wk supplementation period. Average daily gain (ADG) of Arg+ calves was increased (P < .10) during wk 1, 3, and 5 of life. The Arg+ treatment depressed (P < .05) total and KLH-specific IgG concentrations in plasma and caused a decrease (P < .01) in circulating leukocyte numbers. Differential counts revealed that the decrease in circulating leukocyte numbers was due to decreases in absolute numbers of lymphocytes, monocytes, and neutrophils. The Arg+ diet did not affect mean plasma GH concentrations during the first 3 wk of life, but GH mean concentrations were decreased (P < .01) during wk 4 due to depressed (P < .10) pulse amplitudes. The decrease in GH mean concentrations during wk 4 was paralleled by lower (P < .10) plasma IGF binding protein-3 concentrations. These data show that supplementary L-Arg does not increase plasma GH concentrations, but it increases ADG, depresses KLH antibody production, and alters circulating leukocyte populations in preruminant calves.

Key Words: Arginine, Immunity, Leukocytes, Somatotropin, Calves

Introduction

Recent advances in nutrition, immunology, and endocrinology indicate that specific dietary nutrients play important roles in regulating growth and immune function (Cunningham-Rundles, 1993). The amino acid L-arginine (L-Arg) is essential for optimal growth of young animals (Rose, 1949), and it promotes survival and nitrogen retention in animals suffering from sepsis (Madden et al., 1988), trauma (Kirk and Barbul, 1990), and severe burn (Saito et al., 1987). The beneficial effects of L-Arg on nitrogen retention and survival are thought to occur, at least in part, via enhanced immunity resulting from the ability of L-Arg to induce growth hormone (GH) secretion (Seifter et al., 1978; Kelley, 1990; Gala, 1991).

Finally, although the amino acid requirements of preruminant calves are nearly met by cows’ milk, the exceptions are L-cysteine and L-Arg (Williams and Hewitt, 1979). Even though the L-cysteine deficiency seems to be minor, milk may supply only approximately 60% of the L-Arg a calf needs for maximal growth (Williams and Hewitt, 1979). Thus, there is potential for depressed growth and immune function before weaning due to inadequate L-Arg in calf diets. Because L-Arg stimulates GH secretion in ruminants (Chew et al., 1984; Davenport et al.,...
1995), we hypothesized that supplementing prernumant calves with L-Arg would increase GH secretion, resulting in increased weight gain and antibody production in response to immunization.

**Materials and Methods**

**Animals and Diets**

Sixteen Holstein bull calves were obtained at birth from the Penn State Dairy Production Research Center. Calves were prevented from suckling their dams but were supplied with a single dose of Colostrx® (Protein Technology, Santa Rosa, CA) within 6 h after birth. Calves were subsequently fed milk replacer (22% crude protein from whey and skim milk sources, 20% fat, and medicated with oxytetracycline and neomycin; Land O’ Lakes, Fort Dodge, IA) twice daily at 10% of BW/d during wk 1 and 12% of BW/d from wk 2 until weaning. Beginning with the Colostrx feeding, calves in the Arg+ treatment group were supplemented with L-Arg base (Ajinomoto, Tokyo, Japan) at a rate of 500 mg·kg·BW·d⁻¹, and the Arg− group received unsupplemented diets. The L-Arg dose was based on reports that it stimulates GH secretion in a number of species (Sasaki et al., 1982; Chew et al., 1984; Barbul, 1986). Animals were given ad libitum access to water throughout the experiment, but alfalfa hay and starter grain (45% corn, 35% oats, 15% soybean meal, and 5% SoyPLUS®) were withheld until d 28 (±4 d) when weaning began. At weaning, L-Arg supplementation was stopped, and the milk replacer diet was gradually replaced with water over a 1-wk period.

All calves were immunized with 1 mg of keyhole limpet hemocyanin (KLH; Sigma Chemical, Saint Louis, MO) on d 4 and subsequently challenged with 500 µg on d 14. The KLH was dissolved in 500 µL of sterile water and subsequently mixed with an equal volume of incomplete Freund’s adjuvant (Sigma Chemical) prior to s.c. injection on contralateral surfaces of the neck. Keyhole limpet hemocyanin was selected as the antigen because it was previously used by Pollock et al. (1991), who demonstrated a humoral immune response in calves that was measurable by ELISA.

Blood was sampled repetitively at 48-h (±24 h) intervals throughout the 4-wk treatment period and intensively (8 h at 20-min intervals) on a single day when calves were 14 d (±4 d) and 28 d (±4 d) of age. Body weight data were collected at birth, 24 h after birth, and weekly through wk 8.

**Plasma L-Arginine Concentrations**

Plasma amino acid concentrations were determined by reversed phase HPLC of pooled samples that were derivatized online with o-phthalaldehyde (Pierce Chemical, Rockford, IL). For L-Arg quantification, the appropriate fluorescence peak was identified and integrated, and the area under the curve was compared with that obtained from a known quantity of L-Arg standard (Sigma Chemical).

**Plasma Urea Nitrogen Concentrations**

Plasma urea nitrogen concentrations were determined in weekly plasma pools using a diagnostic kit available from Sigma Chemical Company.

**Growth Hormone RIA**

Plasma GH concentrations were measured using the double antibody RIA of MacDonald and Deaver (1993). The assay of 50 to 200 µL of calf plasma resulted in a curve that paralleled the standard curve (CV of estimates corrected for dilution was 12.3%), and the recovery of 50 pg of GH standard from calf plasma was 119%.

Repetitive samples drawn at 48-h (±4-h) intervals were assayed, and the difference in mean GH concentrations between Arg+ and Arg− calves was calculated for wk 1, 2, 3, and 4. The treatment differences calculated for wk 2 and 4 were similar to those obtained after intensive sampling (20-min intervals) during wk 2 and 4, respectively. Thus, repetitive samples were used to assess plasma GH mean concentrations. Intensive samples were assayed, and the Pulsar algorithm (Merriam and Wachter, 1982) was used to calculate mean, baseline, and peak (pulse amplitude) GH concentrations from the diurnal GH secretory profile.

**Plasma Insulin-Like Growth Factor Binding Protein (IGFBP)-3 Concentrations**

Plasma IGFBP-3 concentrations were measured using the method of Skaar et al. (1994). Briefly, plasma was diluted 1:30 in 1× nonreducing buffer (Laemmli, 1970), and 3 µL was subjected to discontinuous SDS-PAGE. The proteins were transferred to nitrocellulose, and the blots were probed with 500,000 cpm of [¹²⁵I]rIGF-II. Radiolabeled bands were visualized using a Molecular Imager® (Bio-Rad Laboratories, Hercules, CA). The density of the 40- to 48-kDa band (previously immunoprecipitated in our laboratory and identified as IGFBP-3) was analyzed using Molecular Analyst® v1.40 software (Bio-Rad).

**Total and Differential Leukocyte Counts**

An aliquot of whole blood collected in EDTA was diluted 1:20 with .01 M HCl to lyse the erythrocytes. Total leukocyte numbers were subsequently determined using a hemocytometer.

Leukocytes in whole blood smears were microscopically differentiated as lymphocytes, monocytes, neutrophils, eosinophils, or basophils using Wright’s stain
(Sigma). The percentage of each cell type was multiplied by the total leukocyte count to obtain absolute cell numbers per unit of blood. Because greater than 90% of the cells were classified as neutrophils, lymphocytes, and monocytes, only the data concerning these cell types are presented.

Anti-KLH Titers

Anti-KLH titers were assayed using an antigen capture ELISA similar to that described by Korver et al. (1984) and Pollock et al. (1991). The KLH-specific antibodies in plasma samples were detected by incubation with a second antibody, rabbit anti-bovine IgG conjugated to horseradish peroxidase (The Binding Site, San Diego, CA), followed by addition of peroxidase substrate 3,3',5,5'-tetramethylbenzidine (Sigma). A plasma pool corrected for interassay variation. After 10 min, the pale blue reaction product was converted to a more stable yellow product by the addition of 1 M H2SO4 and read at 450 nm in an ELISA plate reader (Biotek Instruments, Winooski, VT).

Total IgG Concentrations

Total IgG concentrations were measured using an electrochemiluminescent (ECL) competitive binding assay (Deaver, 1995). Bovine IgG standards and plasma samples were diluted to a volume of 200 μL in ECL assay buffer (EAB; 1% gelatin, .9% NaCl, .01 M EDTA, .05% NaPO4, .01% thimerasol, and .01% heparin; pH 7.2) and incubated at ambient temperature overnight with 25 μL of a 1:10,000 dilution of rabbit anti-bovine IgG (Sigma) and a competing mass of ruthinium-labeled bovine IgG. Standards ranged from .39 to 50 ng/tube, and plasma samples were assayed at a final tube dilution of 1:25,000. Following incubation, 5 μg of sheep anti-rabbit Dynabeads® (Dynal A.S., Oslo, Norway) was added, and the tubes were agitated for 30 min at ambient temperature. Following agitation, 100 μL of EAB was added to each tube, and the percentage of ruthinium-labeled bovine IgG bound to the primary antibody was determined using an Origen® analyzer (Igen Inc., Gaithersburg, MD). Under these conditions, the curve generated by serial dilution of calf plasma paralleled the standard curve (CV of estimates corrected for dilution =3.2%).

Statistical Analysis

This experiment was designed as a split plot in time with treatment (Arg+ or Arg−) as the factor applied to each whole plot (calf). The number of weeks during which the treatments were applied constituted the levels of the subplot factor (age). With the exceptions of plasma L-Arg and urea concentrations, all data were analyzed using the Mixed procedure of SAS (1994) in conjunction with the repeated measures sub-statement [Animal (treatment × week)]. The model included terms for treatment, week, and the treatment × week interaction. Data are presented as least squares means ± SEM. The significance of treatment effects within week is indicated as follows: NSP < .10; *P < .01; **P < .05; ***P < .01. Because L-Arg was only administered during the preweaning period, weight gain data collected before and after weaning were analyzed separately.

Circulating L-Arg and urea concentrations were determined in Arg+ and Arg− pools (representing eight calves each) that were prepared separately for wk 1, 2, 3, and 4. Due to this pooling, it was not possible to analyze L-Arg and urea data using the statistical model described above. A t-test was used to establish whether treatment means differed (P < .05) over the entire 4-wk treatment period.

Results

Plasma Arginine and Urea Concentrations

Relative to the Arg− control calves, those that received the Arg+ treatment had greater (P < .01) circulating L-Arg concentrations during the 4-wk supplementation period (78 ± 11 vs 300 ± 23 μM). Average plasma urea concentrations were also increased (P < .05) from 12.9 ± 8 mg/dL in Arg− calves to 16.3 ± .5 mg/dL in Arg+ calves.

Weight Gain

Preweaning ADG of Arg+ calves (.18 ± .03 kg/d) was increased (P < .01) relative to that of control calves (.09 ± .03 kg/d). Postweaning ADG did not differ between the two groups. Analysis of the treatment effect by week revealed that the Arg+ diet increased (P < .10) ADG during wk 1, 3, and 5 (Figure 1).

Plasma GH and IGFBP-3 Concentrations

There was no effect of L-Arg supplementation on mean circulating GH concentrations measured at 48-h (± 24 h) intervals during the first 3 wk of life (Figure 2A). Analysis of intensive blood samples drawn during wk 2 revealed no effect of L-Arg on mean, baseline, or peak (amplitude) GH concentrations (Table 1). During wk 4, the Arg+ treatment decreased (P < .01) plasma GH mean concentrations in repetitive samples (Figure 2A) and in intensive samples (Table 1). The wk-4 decrease in mean GH concentrations was not associated with any change in baseline GH concentrations, but pulse amplitudes were depressed (P < .05) 67% (Table 1).

Plasma IGFBP-3 concentrations (Figure 2B) followed a pattern similar to that observed for GH mean concentrations (Figure 2A). The Arg+ treatment had no effect on mean plasma IGFBP-3 concentrations.
Figure 1. Dietary L-arginine supplementation increases average daily gain in calves immunized against keyhole limpet hemocyanin. Each point is the least squares mean ± SEM (n = 8). The significance of the treatment effect is indicated for each week: NS = P > .10; *P < .10; **P < .05.

Table 1. Growth hormone mean baseline and peak concentrations measured after intensive sampling during weeks 2 and 4.

<table>
<thead>
<tr>
<th>Variable and week</th>
<th>Arg−</th>
<th>Arg+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean, ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>7.0 ± 1.6</td>
<td>6.4 ± 1.7 NS</td>
</tr>
<tr>
<td>4</td>
<td>9.5 ± 1.6</td>
<td>6.0 ± 1.7†</td>
</tr>
<tr>
<td>Baseline, ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3.3 ± 0.6</td>
<td>3.8 ± 0.7 NS</td>
</tr>
<tr>
<td>4</td>
<td>3.0 ± 0.6</td>
<td>2.6 ± 0.7 NS</td>
</tr>
<tr>
<td>Peak, ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>8.9 ± 3.3</td>
<td>6.1 ± 3.7 NS</td>
</tr>
</tbody>
</table>
| 4                 | 16.8 ± 3.3 | 7.4 ± 3.7† | NS

*Data are least squares means ± SEM (n = 5). The significance of the treatment effect within week is indicated as follows: NS = P > .10; †P < .10; *P < .05.

Hematology

The Arg+ treatment decreased (P < .01) the total number of circulating leukocytes by 23% (Table 2). The suppression of leukocyte numbers resulted from a 26% decrease (P < .01) in circulating neutrophils, a 28% decrease (P < .01) in circulating lymphocytes, and a 41% decrease (P < .01) in circulating monocytes (Table 2).

Plasma Antibody Concentrations

Neither Arg+ nor Arg− calves produced a significant quantity of anti-KLH IgG after initial exposure to the antigen during wk 1 (Figure 3). After the secondary boost during wk 2, anti-KLH IgG concentrations increased in both groups but the Arg+ treatment depressed (P < .01) this response by 20%.

The Arg+ treatment did not affect total circulating IgG concentrations during wk 1, but it depressed (P < .10) circulating concentrations an average of 38% during wk 2, 3, and 4 (Figure 4).

Discussion

Dietary L-Arg supplementation increased plasma L-Arg and urea concentrations in preruminant calves but did not affect GH secretion during the first 3 wk of life, but it caused a 56% decrease (P < .10) during wk 4.

Plasma Antibody Concentrations

Neither Arg+ nor Arg− calves produced a significant quantity of anti-KLH IgG after initial exposure to the antigen during wk 1 (Figure 3). After the secondary boost during wk 2, anti-KLH IgG concentrations increased in both groups but the Arg+ treatment depressed (P < .01) this response by 20%.

The Arg+ treatment did not affect total circulating IgG concentrations during wk 1, but it depressed (P < .10) circulating concentrations an average of 38% during wk 2, 3, and 4 (Figure 4).

Discussion

Dietary L-Arg supplementation increased plasma L-Arg and urea concentrations in preruminant calves but did not affect GH secretion during the first 3 wk of life, but it caused a 56% decrease (P < .10) during wk 4.

Hematology

The Arg+ treatment decreased (P < .01) the total number of circulating leukocytes by 23% (Table 2). The suppression of leukocyte numbers resulted from a 26% decrease (P < .01) in circulating neutrophils, a 28% decrease (P < .01) in circulating lymphocytes, and a 41% decrease (P < .01) in circulating monocytes (Table 2).

Table 1. Growth hormone mean baseline and peak concentrations measured after intensive sampling during weeks 2 and 4.

<table>
<thead>
<tr>
<th>Variable and week</th>
<th>Arg−</th>
<th>Arg+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean, ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>7.0 ± 1.6</td>
<td>6.4 ± 1.7 NS</td>
</tr>
<tr>
<td>4</td>
<td>9.5 ± 1.6</td>
<td>6.0 ± 1.7†</td>
</tr>
<tr>
<td>Baseline, ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3.3 ± 0.6</td>
<td>3.8 ± 0.7 NS</td>
</tr>
<tr>
<td>4</td>
<td>3.0 ± 0.6</td>
<td>2.6 ± 0.7 NS</td>
</tr>
<tr>
<td>Peak, ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>8.9 ± 3.3</td>
<td>6.1 ± 3.7 NS</td>
</tr>
</tbody>
</table>
| 4                 | 16.8 ± 3.3 | 7.4 ± 3.7† | NS

*Data are least squares means ± SEM (n = 5). The significance of the treatment effect within week is indicated as follows: NS = P > .10; †P < .10; *P < .05.

Figure 2. Dietary L-arginine supplementation does not increase mean plasma growth hormone (GH) (A) and IGF binding protein (IGFBP)-3 (B) concentrations in calves immunized against keyhole limpet hemocyanin. Each bar is the least squares mean ± SEM (n = 8). The significance of the treatment effect is indicated for each week: NS = P > .10; †P < .10; **P < .01.
life. Surprisingly, L-Arg supplementation decreased mean circulating GH concentrations during wk 4; this decrease was due to a depression in GH pulse amplitudes. Further evidence to support the wk-4 depression in GH secretion is provided by the concomitant depression in plasma IGFBP-3 concentrations. Plasma IGFBP-3 is increased in cows treated with either GH or GH-releasing hormone (Vanderkooi et al., 1995), and IGFBP have been used as a non-pulsatile indicator of GH status in humans (Blum et al., 1990, 1993; Grinspoon et al., 1995). Our findings indicate that plasma IGFBP-3 may be a useful indicator of GH status in preruminant calves.

It is thought that L-Arg increases GH secretion by depressing somatostatinergic tone (Alba-Roth et al., 1988; Ghigo et al., 1990). Evidence to support this hypothesis lies in the fact that L-Arg has been shown to reinstate GH secretion in adult humans after secretion was suppressed by inducing somatostatin secretion with repeated growth hormone-releasing hormone (GHRH) injections (Ghigo et al., 1989). Our observation that L-Arg does not increase GH secretion in preruminant calves may indicate that GH secretion is not under strong somatostatinergic control at this age. There is evidence that GH secretion in children is not suppressed by repeated GHRH injection even though somatostatin secretion is increased (Ghigo et al., 1989; Bernasconi et al., 1992; Volta et al., 1995). This suggests that GH secretion in children is not under strong somatostatinergic control.

By what mechanisms might L-Arg supplementation enhance weight gain in calves without increasing GH secretion? One possible explanation involves the supplementary L-Arg fulfilling a deficiency in the calves' milk-based diet. The possibility that milk does not contain sufficient amounts of L-Arg to support maximal growth of calves was first set forth by Williams and Hewitt (1979), who calculated calf L-Arg requirements based on experimentally derived lysine requirements. Even though their evidence is indirect, our observations would support their calculations.

Another possible explanation for the positive effect of L-Arg on ADG, in the absence of any effect on GH secretion, involves the ontogeny of GH-dependent growth. Early hypophysectomy/pituitary extract replacement studies in rabbits (Vezinhet, 1968b) and lambs (Vezinhet, 1968a) indicated that postnatal growth was not pituitary-dependent during the first

---

Table 2. Arginine supplementation affects total and differential leukocyte numbers in immunized calves

<table>
<thead>
<tr>
<th>Treatment and week</th>
<th>Total</th>
<th>Neutrophils</th>
<th>Lymphocytes</th>
<th>Monocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arg−</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>14,978 ± 880</td>
<td>6,990 ± 794</td>
<td>5,927 ± 754</td>
<td>1,745 ± 259</td>
</tr>
<tr>
<td>2</td>
<td>14,496 ± 919</td>
<td>6,579 ± 740</td>
<td>5,974 ± 701</td>
<td>1,557 ± 247</td>
</tr>
<tr>
<td>3</td>
<td>11,931 ± 748</td>
<td>4,504 ± 632</td>
<td>6,344 ± 596</td>
<td>1,030 ± 227</td>
</tr>
<tr>
<td>4</td>
<td>11,091 ± 754</td>
<td>3,271 ± 518</td>
<td>6,665 ± 487</td>
<td>1,131 ± 201</td>
</tr>
<tr>
<td>Arg+</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>10,471 ± 824**</td>
<td>5,012 ± 740†</td>
<td>4,219 ± 701</td>
<td>954 ± 248**</td>
</tr>
<tr>
<td>2</td>
<td>11,578 ± 796*</td>
<td>5,192 ± 658NS</td>
<td>3,808 ± 622*</td>
<td>955 ± 230*</td>
</tr>
<tr>
<td>3</td>
<td>9,167 ± 800**</td>
<td>3,086 ± 550†</td>
<td>4,829 ± 525*</td>
<td>720 ± 213NS</td>
</tr>
<tr>
<td>4</td>
<td>9,018 ± 731*</td>
<td>2,594 ± 574NS</td>
<td>5,142 ± 541*</td>
<td>598 ± 214**</td>
</tr>
</tbody>
</table>

*Data are least squares means ± SEM (n = 8). The significance of the treatment effect is indicated as follows: NSP > .10; P < .10; *P < .05; **P < .01.
Figure 4. Dietary L-arginine supplementation depresses plasma total IgG concentrations in calves immunized against keyhole limpet hemocyanin. Each bar is the least squares mean ± SEM (n = 8). The significance of the treatment effect is indicated for each week: NS P > .10; * P < .10; ** P < .01.

weeks of life. More recently, Breier et al. (1988) suggested that energy supply and insulin may be more important regulators of neonatal calf growth than GH. Because L-Arg has been shown to stimulate insulin secretion in sheep (Sasaki et al., 1982; Sano et al., 1995), it is possible that the observed increase in ADG resulted from an insulin-mediated growth response.

Finally, increased water intake might have contributed to the weight gain advantage of Arg+ calves. Although water intake was not measured, Arg+ calves seemed to consume more water than Arg− calves. However, the Arg+ treatment increased ADG (prevented weight loss) during the first week of life, when it is unlikely that calves consumed an appreciable amount of water.

Many of the reported effects of L-Arg supplementation on immune function and wound healing are not realized when animals are hypophysectomized (Seifter, 1978). Based on this finding, Seifter (1978) suggested that the immunological effects of L-Arg supplementation might be attributed to pituitary GH secretagogue activities. Our findings indicate that L-Arg supplementation alters circulating leukocyte numbers and antibody production even in the absence of any change in plasma GH concentrations.

The observations that L-Arg supplementation depressed circulating lymphocyte numbers, anti-KLH titers, and total IgG concentrations in immunized calves indicate that Arg+ treatment may have inhibited humoral immune function. A metabolite of L-Arg, nitric oxide (NO), is thought to be an important antimicrobial agent synthesized by activated macrophages during an immune challenge (Granger et al., 1988, 1990). However, NO has also been shown to inhibit antibody synthesis by cultured mouse splenocytes (Takagi et al., 1994). Because L-Arg availability limits NO production (Granger et al., 1990; Norris et al., 1995; Wong and Billiar, 1995), it is possible that the Arg+ treatment allowed for overproduction of NO by macrophages presenting processed antigens to cells involved with antibody production.

Although humoral immune function seems to have been impaired by L-Arg supplementation, the antimicrobial activity of the cell-mediated branch of the immune system may have been enhanced as a result of increased NO production. Because no measure of cell-mediated immunity was made in this study, it is premature to state that L-Arg supplementation is detrimental to overall host defense.

Taken together, our data suggest that a cell-mediated immune function in preruminant calves without altering GH secretion. It is possible that some of the effects of L-Arg on immune function occur via regulation of NO production.

Implications

Calf morbidity and mortality are not only costly to farmers in terms of reduced growth rates and increased veterinary costs, but also because they undermine consumer confidence in the quality and safety of food animal products. Nutritional manipulation of immune function through dietary L-arginine supplementation may provide one means for improving animal health and productivity in a manner that is acceptable to consumers.

Literature Cited


