Assessment of the effects of feed restriction and amino acid supplementation on glucose tolerance in llamas

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Objective—To assess the effects of prolonged feed deprivation on glucose tolerance, insulin secretion, and lipid homeostasis in llamas.

Animals—9 adult female llamas.

Procedure—On each of 2 consecutive days, food was withheld from the llamas for 8 hours. Blood samples were collected before and 5, 15, 30, 45, 60, 120, and 240 minutes after IV injection of dextrose (0.5 g/kg) for determination of plasma insulin and serum glucose, triglyceride, and nonesterified fatty acid concentrations. Between experimental periods, the llamas received supplemental amino acids IV (185 mg/kg in solution). The llamas were then fed a limited diet (grass hay, 0.25% of body weight daily) for 23 days, after which the experimental procedures were repeated.

Results—Feed restriction decreased glucose tolerance and had slight effects on insulin secretion in llamas. Basal lipid fractions were higher after feed restriction, but dextrose administration resulted in similar reductions in serum lipid concentrations with and without feed restriction. Insulin secretion was decreased on the second day of each study period, which lessened reduction of serum lipid concentrations but did not affect glucose tolerance.

Conclusions and Clinical Relevance—Despite having a comparatively competent pancreatic response, feed-restricted llamas assimilated dextrose via an IV bolus more slowly than did llamas on full rations. However, repeated administration of dextrose reduced insulin secretion and could promote hyperglycemia and fat mobilization. These findings suggested that veterinarians should use alternative methods of supplying energy to camelids with long-term reduced feed intake or consider administering agents to improve the assimilation of glucose. (*Am J Vet Res* 2004;65:996–1001)

Camelids have poor glucose tolerance,[1-3] which is the result (at least in part) of a weak insulin response to hyperglycemia.[3-5] Under normal conditions, this is clinically unimportant, most likely because camelids are not naturally challenged with large quantities of glucose and the weak insulin response is sufficient to maintain glucose and lipid homeostasis. However, pathologic syndromes such as hyperosmolar disorder, hepatic lipidosis, ketonemia, and hyperlipemia develop when excessive glucose or lipid fractions accumulate in the blood.[6-9]

These disorders are detrimental to camelids for a variety of reasons. Hyperglycemia increases blood osmolarity, leading to fluid shifts from the intracellular space. As glucose is excreted in the urine, this fluid is lost and results in dehydration, metabolic acidosis, and electrolyte depletion. The pathologic effects of high lipid concentration in the blood are more complex and poorly understood. In camelids, hyperlipemia has not been linked to the development of atherosclerosis or pancreatitis as it has in other species, but does represent a derangement in body energy metabolism that may be linked to development of hepatic lipidosis in some animals.[7,8] High concentrations of ketone bodies and **nonesterified fatty acids (NEFAs)** have been associated with the development of anorexia and mental derangements in ruminants (perhaps as a result of their effects on blood pH or combined effects with hypoglycemia) and may also inhibit glucose metabolism.[10] The accumulation of energy substrates (carbohydrate or lipid) in the blood may represent failure of adequate tissue uptake and, hence, energy starvation of cells.

The causes of these syndromes are not fully understood. Risk factors for fat mobilization in ruminants are not always present in affected camelids; factors other than pregnancy, lactation, and dietary caloric insufficiency deserve investigation. Impaired insulin secretion or action could be contributory to the development of these disorders because insulin generally acts to decrease circulating glucose and lipid concentrations. Anorexia is another possible risk factor, especially for the development of disorders associated with fat mobilization. It is possible that anorexia promotes lipolysis through suppression of insulin production or activity, but this has not been investigated in camelids.

Partial parenteral nutrition has been used with some success to treat camelids with anorexia and fat-mobilization disorders.[9] The mixtures that are administered to these animals usually contain glucose and amino acids; lipid energy supplements are usually avoided because of high concentrations of lipid already in the animals' circulation. Except for some successes with partial parenteral nutrition in clinical cases, the efficacy of glucose and amino acid supplements in the

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treatment of anorexia and fat-mobilization disorders in camelids has not been evaluated.

The purpose of the study reported here was to assess the effects of prolonged feed deprivation on glucose tolerance, insulin secretion, and lipid homeostasis in llamas. Additionally, the effects on those variables of IV administration of amino acid supplements to feed-deprived llamas were examined.

Materials and Methods

Animals—Nine adult nonpregnant nonlactating female llamas from the university herd were catheterized[a] in the right jugular vein and acclimated to their stalls and handling areas in the university clinic for 96 hours. A physical examination and basic clinicopathologic tests were performed to ascertain health status. The 9 llamas were housed in groups of 3 to minimize separation stress. These llamas were also used in a study of hydrocortisone challenge conducted in sequence with the glucose trials.[11]

Investigational procedures—This study was conducted with approval of the Institutional Animal Care and Use Committee of Oregon State University. Feed was withheld from the llamas for 8 hours overnight. By use of established protocols,[1] simplified glucose tolerance tests were performed on 2 successive days; on each day, blood samples were obtained before (time 0) and 5, 15, 30, 45, 60, 120, and 240 minutes after IV injection of dextrose (D-glucose monohydrate; 0.5 g/kg). Blood samples were divided between tubes containing no anticoagulant and tubes containing lithium heparin. Heparinized blood samples were immediately placed on ice and centrifuged in a refrigerated centrifuge within 20 minutes of collection to obtain plasma. Plasma was frozen at –70°C for ≤ 90 days before analysis. This interval is within the established stability period for insulin.[12] Blood in tubes containing no anticoagulant was allowed to clot, and samples were centrifuged within 1 hour for immediate analysis of sera. Plasma insulin concentration was measured by use of radioimmunoassay.[b] Serum concentrations of glucose, triglycerides, and NEFAs were measured by use of an automated analyzer.[c] All assays have been validated previously for scientific trials[1,4] or diagnostic testing in our clinical laboratory.

Between the 2 days on which blood samples were obtained, llamas were fed grass hay ad libitum until midnight; at approximately 4 to 6 PM, llamas were administered a solution[d] of crystalline amino acids (185 mg of amino acids/kg) that had been further diluted with lactated Ringer's solution to a total volume of 12.5 mL/kg. Following sample collections on the second of the 2 days, llamas received restricted rations of feed for 23 days; on days 22 and 23 of the restricted feed period, samples were collected as before, including overnight feed removal and between-trial amino acid administration, with the only difference being that the amount of hay available between trials was the restricted quantity.

Feed restriction—Llamas were weighed daily and fed a restricted diet of hay; each llama received an amount of hay equivalent to 0.25% of its body weight each day. This protocol has been used to induce hepatic lipidosis in pregnant and lactating llamas.[13] Blood samples were obtained every 2 to 3 days during the 22-day period to monitor fat mobilization and health. At the conclusion of the period of feed restriction, llamas were fed a pelleted mineral supplement (250 g/llama) and grass hay ad libitum. A final health assessment was performed 5 days after conclusion of the last trial, and the llamas were returned to the herd the following day.

Statistical analyses—Mean ± SEM plasma concentration of insulin and serum concentrations of glucose, triglycerides, and NEFAs were each analyzed for changes over time, differences between values before and after feed restriction, and differences between values with or without amino acid supplementation by use of a 2-way ANOVA for repeated measures.[e] Differences between mean values were detected by use of a Tukey test.[14] Values of $P < 0.05$ were considered significant in these comparisons. For comparison with mean values, median values were calculated at all time points for NEFAs.

Results

All 9 llamas appeared to be clinically normal during the period of feed restriction and were deemed to be in good health at the end of the study. Mean weight loss during the period of feed restriction was 4.8 ± 2.9% of body weight.

Serum glucose concentration—Baseline and peak serum glucose concentrations were similar in llamas under all experimental conditions (**Figure 1**). Glucose clearance curves were statistically similar for camelids with or without amino acid supplementation before feed restriction. In these llamas, glucose concentrations decreased significantly from each time point to the next in both experiments, except between 30 and 45 minutes after administration of dextrose in the absence of amino acid supplementation ($P = 0.062$).

Glucose clearance curves were also statistically similar for camelids with or without amino acid supplementation after feed restriction. In these llamas, glucose concentrations decreased significantly from each time point to the next in both experiments, except between 30 and 60 minutes after administration of dextrose ($P = 0.078$ to 0.586). Serum glucose concentrations in llamas after feed restriction were significantly ($P < 0.007$) higher than values in the prerestriction trials at 120 and 240 minutes.

Plasma insulin concentration—In llamas without feed restriction or amino acid supplementation,

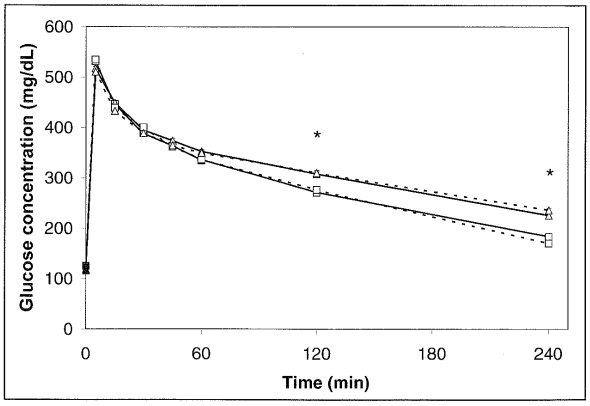

Figure 1—Mean ± SEM serum concentrations of glucose before and after IV administration of dextrose (0.5 g/kg) in 9 llamas that were (triangle) or were not (square) feed-restricted, with (dashed line) or without (solid line) previous administration of an amino acid solution. Glucose was administered immediately after obtaining the first blood sample (ie, at 0 minutes). Open symbols indicate values that are significantly different ($P < 0.05$) from baseline values for that treatment group. *Time point at which values for llamas that were feed-restricted (with or without amino acid supplementation) are significantly ($P < 0.05$) different from the values for llamas that were not feed-restricted (with or without amino acid supplementation).

plasma insulin concentrations increased by > 100% after dextrose administration, compared with baseline values (**Figure 2**). The response curve appeared biphasic, with the first peak (6.16 ± 2.16 μU/mL) at 30 minutes and the second peak (6.89 ± 2.49 μU/mL) at 60 minutes. At all time points, plasma insulin concentration was significantly higher than the baseline value (3.02 ± 1.01 μU/mL). On the following day, after amino acid supplementation, the insulin curve had a similar biphasic shape, but the peaks were lower and detected at 15 (5.29 ± 0.67 μU/mL) and 120 minutes (5.45 ± 1.13 μU/mL). At all subsequent time points, plasma insulin concentration was significantly higher than the baseline value (3.44 ± 0.33 μU/mL); however, the increase in plasma insulin concentration was < 100% of the baseline value (ie, the increase did not reach a value double that of baseline). From 30 minutes to the end of this experiment, plasma insulin concentrations were significantly lower than values obtained the previous day.

After feed restriction with no amino acid supplementation, the insulin curve had a biphasic shape with the first peak (6.12 ± 1.16 μU/mL) at 30 minutes and the highest value (7.65 ± 1.94 μU/mL) at the last time point. At all time points, plasma insulin concentrations were significantly higher than the baseline value (3.24 ± 0.32 μU/mL); plasma concentration of insulin increased to more than double the baseline value. After feed restriction with no amino acid supplementation, plasma insulin concentrations were significantly lower at 60 minutes and higher at 240 minutes after administration of dextrose than values obtained at those time points in llamas without feed restriction or amino acid supplementation. Plasma insulin concentrations in llamas after feed restriction with no amino acid supplementation were also higher than those in llamas on full rations with amino acid supplementation from 30 minutes until the end of the experiment.

Plasma insulin concentrations in llamas after feed restriction and amino acid supplementation were significantly lower than values in llamas after feed restriction without amino acid supplementation and llamas without feed restriction (with or without supplementation) at baseline (2.15 ± 0.16 μU/mL) and all time points. The insulin response in llamas after feed restriction and amino acid supplementation was similar to that detected under the other experimental conditions; however, plasma insulin concentrations were only significantly higher than baseline values at 15 ($P = 0.004$), 30 ($P = 0.011$), and 240 ($P < 0.001$) minutes after dextrose administration and did not increase by 100% of the baseline value.

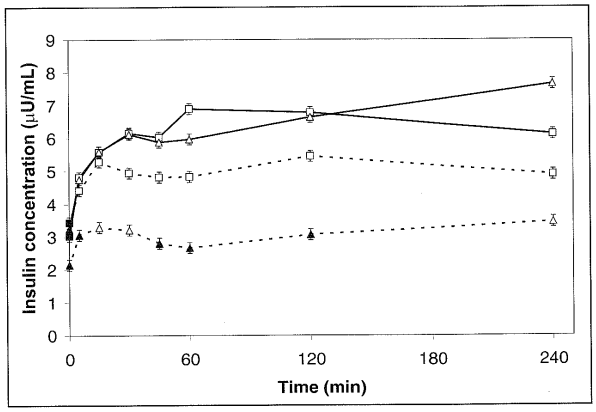

Figure 2—Mean ± SEM plasma concentrations of insulin before and after IV administration of dextrose (0.5 g/kg) in 9 llamas that were (triangle) or were not (square) feed-restricted, with (dashed line) or without (solid line) previous administration of an amino acid solution. Glucose was administered immediately after obtaining the first blood sample (ie, at 0 minutes). *See* Figure 1 for key.

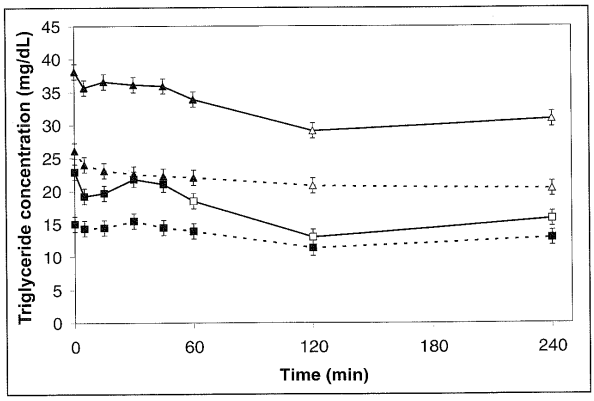

Figure 3—Mean ± SEM serum triglyceride concentration before and after IV administration of dextrose (0.5 g/kg) in 9 llamas that were (triangle) or were not (square) feed-restricted, with (dashed line) or without (solid line) previous administration of an amino acid solution. Glucose was administered immediately after obtaining the first blood sample (ie, at 0 minutes). *See* Figure 1 for key.

Serum triglyceride concentration—Serum triglyceride concentrations in llamas before feed restriction (without amino acid supplementation) decreased after administration of dextrose (**Figure 3**). The response curve appeared to be biphasic with the first trough value (19.2 ± 7.1 mg/dL) at 5 minutes and the second trough value (13.0 ± 9.3 mg/dL) at 120 minutes. At 60, 120, and 240 minutes after dextrose administration, serum triglyceride concentrations were significantly lower than the baseline value (22.9 ± 8.4 mg/dL) and intertrough highs (30 and 45 minutes). In llamas that

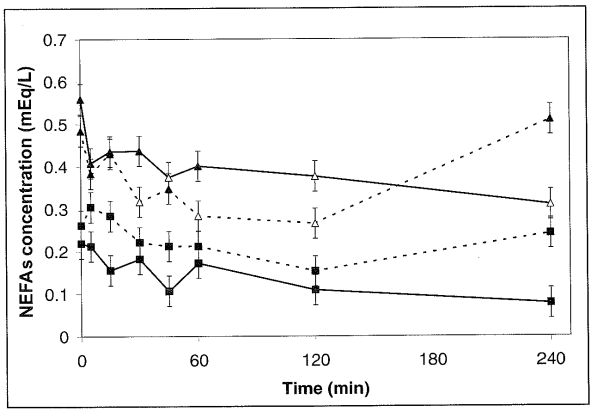

Figure 4—Mean ± SEM serum concentrations of nonesterified fatty acids (NEFAs) before and after IV administration of dextrose (0.5 g/kg) in 9 llamas that were (triangle) or were not (square) feed-restricted, with (dashed line) or without (solid line) previous administration of an amino acid solution. Glucose was administered immediately after obtaining the first blood sample (ie, at 0 minutes). *See* Figure 1 for key.

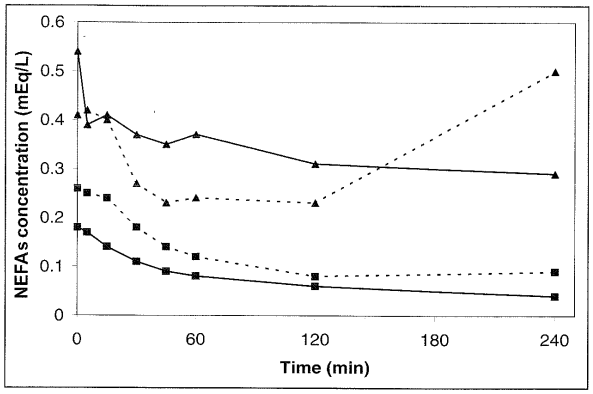

Figure 5—Median serum concentrations of NEFAs before and after IV administration of dextrose (0.5 g/kg) in 9 llamas that were (triangle) or were not (square) feed-restricted, with (dashed line) or without (solid line) previous administration of an amino acid solution. Glucose was administered immediately after obtaining the first blood sample (ie, at 0 minutes).

were not feed-restricted but received amino acid supplementation, changes in serum triglyceride concentrations after dextrose administration followed the same general pattern, except that none of the changes were significantly different from the baseline value. Compared with serum triglyceride concentrations obtained when the llamas were not feed-restricted and did not receive amino acid supplementation, values in llamas that were not feed-restricted and did receive the supplementation were significantly lower at the onset of the trial (15 ± 4.0 mg/dL) and between the trough values (30 to 45 minutes).

After feed restriction (with no amino acid supplementation), serum triglyceride concentrations also decreased in a biphasic manner after dextrose administration. Trough concentrations were also detected at 5 (35.7 ± 8.7 mg/dL) and 120 minutes (29.1 ± 6.0 mg/dL) after dextrose administration. At all time points including time 0 (baseline concentration, 38.1 ± 8.5 mg/dL), serum triglyceride concentrations were significantly higher than values obtained in llamas that were not feed-restricted (with or without amino acid supplementation). Serum triglyceride concentrations in feed-restricted llamas without supplementation at 120 and 240 minutes were significantly lower than the baseline value and intertrough highs (5 to 60 minutes).

In llamas after feed restriction with amino acid supplementation, serum triglyceride concentrations decreased with time in a simple curve; values at 120 and 240 minutes were significantly lower than the baseline value (26.1 ± 8.4 mg/dL). Serum triglyceride concentrations for feed-restricted llamas that received amino acid supplementation were significantly ($P < 0.005$) lower than values obtained for feed-restricted llamas that did not receive amino acid supplementation at all time points, significantly ($P < 0.002$) higher than values obtained for llamas that were not feed-restricted but did receive amino acid supplementation at all time points, and significantly ($P = 0.002$) higher than values for llamas that were not feed-restricted and did not receive amino acid supplementation at only 120 minutes after dextrose administration.

Serum NEFAs concentration—Serum concentration of NEFAs in llamas that were not feed-restricted and did not receive amino acid supplements decreased after administration of dextrose (**Figure 4**). These changes were not significant. The curve appeared biphasic, but examination of the median values revealed that sporadic spikes in serum concentration of NEFAs in individual llamas at single time points affected the shape of the curve (**Figure 5**). Such spikes were relatively rare in this group of llamas that were not feed-restricted and did not receive amino acid supplementation (4 events). In llamas that were not feed-restricted but received amino acid supplementation, serum concentrations of NEFAs decreased in a similar manner after administration of dextrose, especially in regard to median values. These changes were also not significant and not significantly different from values in llamas that were not feed-restricted and did not receive the supplement. Spike values identified among the median concentration values were rare (3 events).

In llamas that were feed-restricted but did not receive the amino acid supplement, serum concentrations of NEFAs decreased after administration of dextrose during the experimental period; compared with the baseline concentration (0.56 ± 0.13 mEq/L), values were significantly lower at 45 (0.37 ± 0.10 mEq/L; $P = 0.016$), 120 (0.38 ± 0.17 mEq/L; $P = 0.018$), and 240 minutes (0.31 ± 0.10 mEq/L; $P < 0.001$). Serum concentrations of NEFAs were significantly higher in llamas that were feed-restricted (without amino acid supplementation) than values for llamas that were not feed-restricted (without amino acid supplementation) at all time points and significantly higher than values for llamas that were not feed-restricted but did receive amino acid supplementation at all time points, except 5, 15, and 45 minutes after dextrose administration. Spike values identified among the median concentration values were more common (8 events). In feed-restricted llamas that received amino acid supplementation, serum concentrations of NEFAs decreased in a manner similar to that observed in the llamas under the other experimental conditions, except for the final time point. Compared with the baseline value (0.48 ± 0.20 mEq/L), the concentrations in feed-restricted llamas that received amino acid supplementation were significantly lower at 30 (0.32 ± 0.14 mEq/L; $P = 0.044$), 60 (0.28 ± 0.12 mEq/L; $P = 0.006$), and 120 minutes (0.27 ± 0.16 mEq/L; $P = 0.002$), but increased to a significantly high value (0.51 ± 0.20 mEq/L) at 240 minutes. Spike values identified among the median concentration values were rare (3 events), except for this last time point (5 events). The baseline serum concentration of NEFAs in feed-restricted llamas that received amino acid supplementation was significantly higher than values in llamas that were not feed-restricted (with or without amino acid supplementation). Serum concentrations of NEFAs in feed-restricted llamas that received amino acid supplementation were higher than values for the llamas that were not feed-restricted and did not receive the supplementation at 15 and 45 minutes after administration of dextrose. At 240 minutes after administration of dextrose, serum concentration of NEFAs in feed-restricted llamas that

received amino acid supplementation was significantly ($P < 0.04$) higher than those values obtained for llamas under all other experimental conditions.

Discussion

Our investigation has provided some interesting data regarding glucose clearance in llamas; some of these findings were expected and others were not. Of primary interest was the reduced glucose clearance associated with feed restriction. Healthy camelids are known to have poor glucose tolerance, compared with that of ruminants and other species,[1-3] and our data have suggested that sick camelids may be even more poorly glucose tolerant. Dehydration reduces glucose tolerance in camels, but whether this occurs in New World camelids has not been investigated.[15] These considerations influence clinical management of the different species. Negative energy balance in ruminants is commonly treated with IV administration of glucose, which is often effective because ruminants that are mobilizing excess fat retain normal glucose tolerance.[16] Our data suggested that IV administration of glucose (especially as a bolus) is of little value and potentially more likely to have adverse effects in camelids after a period of anorexia, compared with its effects in healthy camelids. Thus, the value of glucose in the treatment of anorexic, normoglycemic camelids is in doubt unless glucose assimilation could be improved by some method.

Feed restriction had small, though significant and potentially important, effects on insulin secretion. In llamas that were or were not feed-restricted (without amino acid supplementation), the first peak plasma concentration of insulin was similar in timing and magnitude and is likely representative of similar amounts of preformed insulin stored in islet cells. Feed-restricted llamas (without amino acid supplementation) had a delayed second peak in plasma insulin concentration, which may be the result of impairment of insulin synthesis. Indirect evidence of impaired insulin synthesis in the feed-restricted llamas included slow glucose clearance during the middle portion of the experimental period (ie, 30 to 60 minutes after dextrose administration) and resultant higher concentrations toward the end of experimental period, as well as slower significant reduction in serum triglyceride concentration, compared with llamas before feed restriction. In the llamas of this report, the difference in insulin production before and after feed restriction was small (< 1 μU/mL), compared with the insulin response to glucose tolerance testing detected in sheep,[3] ponies, or cattle[16] (40 μU/mL or more), but this difference must be considered in light of the normal low insulin production by camelids.

Assessment and comparison of the lipid response to hyperglycemia in llamas were difficult because llamas on full-feed rations had near-basal concentrations of circulating lipid. Thus, suppressed mobilization or enhanced clearance of lipid was difficult to detect. Suppression of mobilization of NEFAs by administration of exogenous glucose in camelids has been identified[1]; results of the study reported here confirmed a reduction in serum triglyceride concentration after IV administration of dextrose in llamas that were or were not feed-restricted. In support of the role of the insulin response in triglyceride clearance, the decreases in serum triglyceride concentration were biphasic in general, with the exception of the decrease observed in feed-restricted llamas that received amino acid supplementation (which also had the poorest insulin response).

Changes in the serum concentration of NEFAs monitored in the llamas during the 4 experimental periods were more complex; theoretically, the serum concentration of NEFAs could simultaneously decrease as a result of inhibited adipose breakdown and increase as a result of accelerated triglyceride clearance (intravascular lipolysis). The serum concentration of NEFAs decreased after IV administration of dextrose in a biphasic manner that mirrored the peaks in plasma insulin concentrations to some degree. In llamas that were not feed-restricted, there was little change in serum concentration of NEFAs, which is coincident with their smaller circulating lipid fractions. In llamas that were feed-restricted, serum concentration of NEFAs decreased more substantially but was also more variable from one time point to the next. In feed-restricted llamas, fat mobilization did not decrease to values that had been detected in llamas that were not feed-restricted, which suggested that glucose alone is not sufficient to abate fat mobilization in anorexic camelids or camelids from which food has been withheld. The variability in serum concentration of NEFAs between time points, especially in feed-restricted llamas, suggested that multiple blood samples may be needed to accurately assess adipose mobilization in camelids with inadequate dietary energy intake.

The differences in the measured variables observed between llamas that received or did not receive the amino acid supplement were difficult to attribute to that supplementation alone. In the study of this report, llamas were administered 185 mg of amino acids/kg; this dose is much lower than the dosage recommended by the manufacturer for nutritional maintenance in humans (1.0 to 1.5 g/kg/d)[d] but more similar to that used in our clinic for the overnight treatment of sick camelids (approx 28 mg/kg/h). Compared with findings in llamas that had not received amino acid supplementation, serum triglyceride concentrations in llamas that received the supplement were lower; this may have been the result of residual effects of the glucose tolerance test performed on the preceding day or better triglyceride clearance in amino acid-treated camelids. The lower plasma insulin concentrations detected in llamas that received amino acid supplementation, compared with values detected in llamas that did not receive the supplementation, were more difficult to explain. Suppression of pancreatic function after the administration of solutions of amino acids has not been reported, but such an effect would be a contraindication for such administrations in animals with excess fat mobilization. On the contrary, postprandial surges in plasma concentrations of amino acids are expected to stimulate insulin release. More likely, the difference in the insulin response curves was a consequence of performing glucose tolerance tests on suc-

cessive days in the same group of llamas. This would suggest that camelids are susceptible to a reduction of pancreatic function after chronic stimulation (pancreatic exhaustion) and that clinicians should avoid long-term administration of glucose; administer exogenous insulin at the same time as the glucose treatment; or at least carefully monitor blood concentrations of ketones, triglycerides, and NEFAs for evidence of excess mobilization as a result of possible iatrogenic hypoinsulinemia. The possible effects of pancreatic exhaustion were best illustrated by the changes in serum concentrations of NEFAs in llamas that received the amino acid supplementation, compared with those detected in llamas that did not receive the amino acid supplementation; without the supplement, llamas had a continual decrease in serum concentrations of NEFAs and their lowest values were at the end of the experimental period, whereas insulin-mediated suppression of fat mobilization in amino acid-treated llamas appeared to have ceased to be effective by the last time point. However, our study did not involve differentiation between the effects of administration of amino acid solutions and those associated with performance of multiple glucose tolerance tests, so these clinical recommendations remain speculative. Regardless, the lack of changes in glucose clearance detected in our study emphasizes the minor role of exogenous insulin in glucose clearance in camelids.

Administration of glucose to sick or anorexic camelids is common clinical practice. It has been reported[1,2] that treatment with a bolus of glucose stimulates an insulin response and reduces serum concentration of NEFAs; our data have suggested that there is also a slight reduction in serum triglyceride concentration associated with such treatment. In the study of this report, the reduction in blood concentrations of NEFAs and triglycerides may be attributable solely to the insulin response. It is not known whether any of those potential benefits would be elicited by slower rates of glucose infusion.

Detrimental effects associated with administration of glucose in camelids have been reported in neonates only.[6] However, because of the slow glucose clearance in camelids and the possible deleterious effects of hyperglycemia, hydration status and changes in blood osmolarity and electrolyte composition should be monitored in all camelids receiving glucose. On the basis of our data, these recommendations are especially applicable to camelids with chronically reduced feed intake, which are highly likely to receive glucose treatment. Also, veterinarians should be able to justify the administration of glucose (which may exercise its beneficial effects through the weak endogenous insulin response) to camelids rather than the direct administration of insulin or possibly administration of the 2 agents in combination.

[a]Long-term polyurethane catheter, MILA International Inc, Erlanger, Ky.
[b]Coat-A-Count insulin kit, Diagnostic Products Corp, Los Angeles, Calif.
[c]Hitachi 717 serum biochemical analyzer, Boehringer Mannheim Diagnostics Division of Boehringer Mannheim Corp, Indianapolis, Ind.
[d]Aminosyn 8.5%, Abbot Laboratories, North Chicago, Ill.
[e]SigmaStat, version 2.0, SPSS Inc, Chicago, Ill.

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