The Giraffe Nutrition Workshop Proceedings

May 25 - 26, 2005

Lincoln Park Zoo

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In closing, we extend our personal thanks to everyone involved in making this workshop a success. Your hard work and dedication greatly enhanced our understanding of giraffe nutrition, giraffe health, and changes that can be made to our current practices to enhance the health of the captive population.

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Giraffe Nutrition Workshop Proceeding Contents

Please visit www.lpzoo.org to view the corresponding presentations.

The Giraffe Nutrition Workshop assembled a group of independent scientific experts in domestic ruminant nutrition and specialists in captive giraffe management to discuss and establish new feeding guidelines for zoo giraffe. The following document is a compilation of chapters on giraffe and ruminant nutrition, abstracts from the workshop, and a summary of the major scientific issues discussed at the Workshop. The opinions, finding, conclusions, and recommendations described in this publication are those of the authors and workshop participants and do not necessarily reflect the view of the agencies or organizations that provided support for this workshop. The sponsoring agencies and organizations are not responsible for the accuracy and validity of the information presented by individual authors.

Executive Summary

On May 25, 2005, 14 people interested in giraffe nutrition and health, convened at Lincoln Park Zoo in Chicago for a two-day workshop. The participants consisted of university and USDA ruminant nutrition researchers, zoo nutritionists, and veterinarians in addition to the giraffe nutrition and veterinary advisors and the PMP coordinator for giraffe.

Captive giraffe have a specific set of maladies that may be related to basic nutritional inadequacies. Peracute mortality (Fowler, 1978; Fowler and Boever 1986; Junge and Bradley, 1993), chronic wasting (Flach, 1997; Ball et al., 2002), energy malnutrition (Ball et al., 2002), mortality related to cold stress (Clauss et al., 1999), pancreatic disease (Lechowski et al., 1991), urolithiasis (Wolfe et al., 2000; Wolfe, 2003), neonatal health concerns (Miller et al., 1999), intestinal parasitism, and hoof disease may be influenced by traditional zoo diets.

The first day of the workshop was devoted to a series of presentations including:

Rumen Physiology of Digestion in Ruminants
- Guoyao Wu, Ph.D., Texas A&M University;

Physiology of Acidosis
- Monty Kerley, Ph.D., University of Missouri - Columbia;

Factors Affecting Rumen Microbial Ecology
- Jason Williams, Ph.D., Lincoln Park Zoo;

Clinical Problems of Captive Giraffe with a Possible Nutritional Basis
- Ray Ball, D.V.M., Busch Gardens;

The Giraffe Mortality Survey
- Judy St. Leger, D.V.M., D.A.C.V.P., SeaWorld San Diego;

Giraffe Nutrition: Facts and Unknowns
- Celeste Kearney, M.S., Saint Louis Zoo.

A complete list of all participants is included in this report.

With presentations concluded, the discussion section of the workshop began. Topics of discussion included: nutrient concentrations, zoo feeding practices, the need for a body condition scoring system, and research needed in giraffe nutrition. The scientific summaries of these discussions and recommendations for these topics are included with this report.

In summary, we worked together to gain a better understanding of ruminant nutrition and how it pertains to giraffe, to understand how the clinical problems in some captive giraffe may originate from traditional zoo diets, and how changing nutrient concentrations and feed types may alleviate serious health issues in the captive giraffe population. We have compiled a document that the participants feel reflects the best information presently available. As more giraffe nutrition research is completed and more information becomes available, the recommendations may be modified. By sharing the ideas and decisions that were developed from this meeting, we hope to improve the health and well-being of giraffe that are cared for by zoo professionals.

Scientific Summary

I. Review of Free-Ranging Giraffe Diets
C. C. Kearney, M.S.

Giraffe have been described as "browsers", "folivores`", and "concentrate selectors". Unlike grazing (grass-eating) herbivores, which consume relatively uniform feeds, wild giraffe choose their diet from an assortment of foods that are very different in nutrient content. Trees and shrubs generally comprise the bulk of the natural diet (in some cases > 93%), with limited vine and herb consumption.[4, 9] Grass intake is either non-existent [9] or negligible;[2] grass is apparently "eaten by accident when enmeshed with other food".[4]

The species of plants consumed by wild giraffe vary widely with season and geographic location. Giraffe in Tsavo National Park congregated along the river to feed on evergreen plants during the dry season, but scattered to feed on deciduous plants when increased rainfall promoted renewed plant growth.[9] Although Acacia *spp*. seem to be the most commonly reported dietary item,[1-3,5] giraffe in a given location may consume as many as 66 plant species over the course of a year.[9]

Although most reports suggest that the natural diet is primarily leaves and stems, these are not the only plant portions consumed by wild giraffe. Fruits, flowers, bark, thorns, and pods also are eaten, and in some cases, these plant parts are selected from plants whose leaves remain untouched. Plant species and portions selected vary with season.[1,3,9] The diet of giraffe in Niger was reportedly 86% leaves, 8.5% stems, and 5.5% flowers and fruits during the wet season. In contrast, during the dry season (December to April), the diet was composed of 45% leaves and stems, 44% fruits, and 11% flowers.[2] This seasonal shift reflected plant parts (foliage or fruits and flowers), which were at their highest growth productivity at the time.

With so many food items available to the free-ranging giraffe, intake does not always reflect the availability of plant species and parts in a habitat. When food is plentiful, giraffe may pass up plants that are abundant in the habitat to select less available foodstuffs; however, what drives selection is unknown. There seem to be few links between nutrient composition and animal preference.[3,11] Preference for both high- and low-protein foods has been noted, as has a preference for high-moisture leaves and young growing shoots.[1,11] Tannins, bitter tasting natural compounds in plant material, were low in leaves from the five most heavily consumed plant species in one study, but leaves with high tannin concentrations were not entirely avoided.[5]

Feeding is the most time-consuming activity of wild giraffe, taking up as much as 53% of daylight hours.[8] Giraffe feed at all hours of the day and night with morning and evening reported as peak feeding times. During observations in Nairobi, approximately 75% of visible giraffe were feeding in the morning, 50% at noon, and 90% in the evening.[3]

Giraffe use several browsing methods to obtain food. They may select specific parts of a plant by using the tongue or incisors to peel bark from stems, or to pull or cut chosen buds, twigs, leaves, or flowers. At other times, branches are rapidly stripped as a giraffe wraps its mouth around the branch and draws its head back, consuming all the material, including leaves, twigs, pods, fruits, thorns, and any attached insects. Rare odd food items reported include bird nests and ruminal contents of deceased antelope and giraffe are occasionally seen gnawing bones or licking salty soils.[3]

Despite the number of reports on feeding behavior, little is know about the actual nutrient intake of wild giraffe. Some of the plant species and parts eaten have been analyzed for nutrient content, but others have not. There is sufficient information available to indicate that the nutrient composition of the leaves, stems, flowers, fruits, bark, and pods consumed varies considerably, and therefore nutrients in one plant portion should not be taken as an indication of total nutrient intake. Ruminal contents of wild giraffe suggest that nutrient composition of the total diet varies with season, but which nutrients change and to what degree is still largely unknown.[6] At least one attempt to estimate average seasonal nutrient intake over the course of a year has been published.[10]

Because the water content of plants varies, all nutrient values in this report are on a dry matter (water removed) basis. Although water requirements have not been determined, giraffe seem to obtain much of their water from high moisture foods. The total daily dry matter intake by free-ranging giraffe has been estimated as 1.6% of body weight in males and 2.1% of body weight in females.[10]

Analyses of plants consumed by wild giraffe have focused on the major energy-producing nutrients (fats, proteins and carbohydrates). Vitamin and mineral concentrations in natural giraffe diets are, at this point, unknown. The reported concentrations of fat in various leaves consumed by giraffe are low, ranging from less than 1 to 3%,[1,4] and fat has been estimated to make up 3 to 5% of total intake.[10]

The protein content of plant portions eaten by wild giraffe varies considerably. Leaves from 10 of the species consumed by giraffe in Niger during the dry season ranged from 8.2 to 28.6% crude protein (CP).[1] Average CP content of plants eaten in the Narus Valley (Uganda) varied with season from 11.51 to 22.37% CP in leaves and 5.78 to 9.35% CP in stems.[4] Crude protein intake as a percentage of total diet during the wet and dry seasons in the Serengeti was estimated at 14 and 12% for males, and 19 and 15% for females, respectively.[10] It should be noted

that a portion of the protein in these plants is bound with tannins and lignin. This binding makes that protein unavailable for use by the giraffe and/ or its ruminal microbes; thus, digestible protein will be lower than the total CP values noted above.

Dietary carbohydrates can be separated into two major portions (fiber and non-fiber), which have very different fermentation and digestion characteristics. Reported concentrations of these nutrients in plants eaten by wild giraffe have ranged from 1.5 to 49% crude fiber (CF) and 6 to 72% non-fiber carbohydrates.[1,4] However, the analytical method used to obtain those values underestimates fiber and overestimates non-fiber carbohydrates to a degree that cannot be determined.[12] Using newer technology, acid detergent fiber (ADF) intake as a percentage of total diet during the wet and dry seasons in the Serengeti was estimated at 39 and 45% for males, and 26 and 36% for females, respectively.[10] Although ADF is an accurate analysis, it only represents two (cellulose and lignin) of the three (cellulose, lignin and hemicellulose) fractions that make up most of the structural dietary fiber in ruminant diets. Total fiber intake was therefore greater than the reported ADF value. The three major non-structural carbohydrates (sugar, starch and pectin) are more rapidly digested than fiber, but differ from each other in their digestion characteristics. Although initial investigations, along with basic plant physiology, suggest that wild giraffe consume mainly low-starch, high-pectin foods, further research would be necessary to verify this theory.[7]

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II. Ruminant Nutrition Review
M. L. Schlegel, Ph.D.

A. Anatomy

Ruminants and the closely related pseudoruminants are ungulates that are even-toed (Artiodactyla), chew their cud (ruminant) and have stomachs with three (pseudoruminants) or four (true ruminants) compartments.[1] The pseudoruminants include the Tragulidae (mouse deer or chevrotains) and the Camelidae (camels, llamas, alpaca, etc.) families. The true ruminants include three families; the Cervidae (deer), Giraffidae (giraffe and okapi), and Bovidae (ruminants with horns that are not shed).

The ruminant stomach is comprised of the reticulum, rumen, omasum, and abomasum. The first three compartments store and delay passage of ingested food. This allows time for remastication (cud-chewing or rumination) which decreases particle size and enhances fiber digestion (also called fermentation).[3] The reticulum is often called the 'honeycomb' due to its mucosal pattern. The rumen is the major site of fiber fermentation. The surface of the rumen is covered with papillae and micro papillae, which increase the absorptive surface area of the rumen. Papillae distribution, size, and number vary based on feeding habits and food availability.[3] The omasum controls the flow of digested or indigested food into the abomasums. Ruminants with limited fiber fermentation capacity have the ability to allow larger fibrous particles that can not be broken down sufficiently in the rumen to pass into the abomasum.[3] The fourth compartment (abomasum) is the true stomach and is lined with glandular mucosa which secretes hydrochloric acid and pepsin as in other mammals.[3,6] The ruminant animal is considered unique because of its four-compartment stomach, but specialization has occurred throughout the gastro-intestinal tract.[3] Additionally, ruminants have taken advantage of a wide variety of feedstuffs from high-fiber, low-protein grasses and tree parts, roots and tubers, to high-protein browse leaves.

B. Ruminal fermentation and digestion

A symbiotic relationship exists within the rumen providing an environment for bacteria, protozoa, and anaerobic fungi to live while these microorganisms provide the process through which fibrous feeds are digested and fermented, yielding protein, short-chain fatty acids, and vitamins utilized by the ruminant. Ruminal pH is normally between 6 and 7, and the rumen is buffered with the addition of saliva, balancing the acids produced during fermentation.[9]

The ruminants' protein requirement is met from two sources. The first is the dietary protein that bypasses ruminal degradation and reaches the abomasum and small intestine. The second source is from microbes that leave the rumen and are digested and absorbed in the abomasum and small intestine similar to dietary protein. Microbial protein can represent 40 to 60% of the non-ammonia nitrogen reaching the small intestine.[8] Ruminal microbes not only reproduce using dietary

protein (i.e., amino acids), but also ammonia-nitrogen. Ammonia-nitrogen is supplied through dietary non-protein nitrogen (e.g., urea) from the saliva and recycled from the animal's blood.[8] This continual recycling of nitrogen to the rumen allows ruminants to subsist on low-protein diets. In general terms, dietary nitrogen, in the form of protein, feeds the ruminal microbes and the microbes then become the protein source to the ruminant animal.

The majority of the diet consumed by ruminants is in the form of carbohydrates. These carbohydrates are polymers of glucose in the form of cellulose or starch.[2] Starch, made of α-1,4-linked glucose molecules, can be digested by the ruminant and other mammals because enzymes are produced that break the α-1,4 linkage. Cellulose, the main structural carbohydrate of plants, is also comprised of glucose molecules, but linked through ß-1,4 linkages. Enzymes to degrade ß-1,4 linkages are not made by mammals, but are synthesized by ruminal microorganisms. The ruminant and the microbes have a symbiotic relationship to efficiently utilize fibrous feeds.[10] The rate at which carbohydrates are fermented differs. Soluble carbohydrates (sugars and starches) are digested rapidly, followed by soluble fiber (pectins, hemicellulose), with the slowest fermenting fibers being cellulose.[7] The fermentation of forage diets, producing short-chain fatty acids (acetate, propionate, butyrate), provides 50 to 85% of the metabolizable energy used by the ruminant.[7]

In addition to the ruminal microbes providing energy in the form of short-chain fatty acids and nitrogen as microbial crude protein, the microbial population of the rumen also synthesizes the B vitamins and vitamin K required by the animal. The other fat soluble vitamins (A, D, and E) are required from the diet as occurs in non-ruminant mammals.[4] Vitamin D can also be synthesized in vivo.

C. Acidosis

Acidosis is a condition of high acidity of the blood or acidic conditions in the rumen.[5] The ingestion of a large quantity of readily fermentable carbohydrates provides the substrate for the rapid replication of microorganisms that synthesize lactic acid.[10] Sources of readily fermentable carbohydrates include tubers or roots, cereal grains, or immature, rapidly-growing forages.[5] Cereal grains are the most common source of readily fermentable carbohydrates that cause acidosis through the production of lactic acid.[10] Lactic acid is a stronger acid than the short-chain fatty acids. Due to the rapid production of lactic acid, ruminal pH can drop below 4, and if produced in large amounts, it is absorbed across the ruminal wall and into the blood resulting in systemic acidosis.[10] Acidosis can be prevented by limiting the quantity of readily fermentable carbohydrates in the diet, slowly adapting animals from a higher-fiber diet to a lower-fiber diet, monitoring daily changes in feed intake of diets high in readily fermentable carbohydrates, and providing fresh feed and water daily.[5]

D. Formulating ruminant diets

Ruminants are fed a wide variety of feed types with a range of moisture contents. For example, a pelleted diet or supplement contains 10% moisture (90% dry matter), whereas ear-leaf acacia leaves (*Acacia auriculiformis*) contain 69% moisture (31% dry matter). At first glance, one may consider the pellet a superior protein source if it contains 16% crude protein and the acacia leaves contain 7% on an as-fed basis (as it is consumed by the animal), but when expressed on a dry matter basis the pellets contain 17.8% crude protein and the acacia leaves contain 22.6%. When ruminant diets are formulated, nutritionists evaluate the nutrients on a dry matter basis, thereby, removing the variation of feed moisture content.

E. Summary

Ruminant animals have been uniquely adapted to take advantage of a compartmentalized stomach and a symbiotic relationship with microorganisms to efficiently utilize a high-fiber diet. This relationship in combination with the ability to recycle nitrogen makes them able to survive in many environmental niches from the artic to the desert. Although ruminants are adaptable to many different diets, sudden dietary shifts can produce digestive disorders and even death. Therefore, appropriate diets and enrichment items must be fed at all times.

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III. Dietary Physical Form Considerations
C. C. Kearney, M.S.

The physical form of a diet has a substantial effect on how foods are processed in the digestive tract. Physical form affects chewing,[12] saliva production,[2] development of ruminal papillae,[4] ruminal pH,[2] rate and extent of fermentation,[12] rate of passage,[1] and the proportion of unfermented nutrients that pass out of the rumen into the lower GI tract.[7] The concept of "physically effective fiber" takes into account both fiber content and physical characteristics of a feed.[12] Physical effectiveness is commonly measured by the ability of a feed to promote a chewing response in the animal. Chewing time is determined by the feed's physical size and the rate at which it is broken apart during mastication. Larger particle size feeds with higher fiber content, such as long-stemmed hay, promote more chewing from ingestion and rumination activities.

The rate of saliva flow increases during periods of eating and rumination.[3] Spending less time chewing can decrease the quantity of saliva produced over the course of a day, and the quantity of saliva produced per unit of feed consumed.[3,11] Saliva supplies approximately half the bicarbonate entering the rumen and, with a pH of 8.5,[5] saliva is a primary buffer against the acids produced during fermentation.[13] Saliva also is a major route of recycling minerals (especially phosphorus) out of the bloodstream and back into the digestive tract. Cattle consuming diets low in physically effective fiber chew less and have less salivary recycling, which contributes to excretion of greater amounts of minerals through the urine.

Ground ingredients used to create pelleted feeds are small in particle size. Direct animal observations indicate that pellets, which break apart easily, require little chewing during initial ingestion. After being swallowed, pellets dissolve into their ground form in the ruminal fluid. These ground particles are too small to stimulate normal regurgitation for cud chewing. As a result, pelleted feeds do little to promote chewing behavior.

Traditionally, the energy, vitamin, and mineral rich concentrates (e.g., pellets, sweet feeds) and physically effective fiber source (e.g., long-stemmed hay) have been offered to zoo ruminants as separate dietary components. This feeding practice may cause imbalanced nutrient intake, and can contribute to ruminal acidosis for two reasons. First, animals may consume less hay and more rapidly fermenting/high acid-producing concentrates than intended due to situations such as offering manufactured feeds ad libitum or in "limited" amounts that overestimate what giraffe are likely to consume, offering little to no hay/browse in proportion to manufactured feeds, and/or animal dominance influencing a large consumption of manufactured feed. This phenomenon of high concentrate/ low hay consumption has been documented in captive giraffe.[10] Second, animals often eat substantial quantities of concentrates at one time and hay hours later. When this happens, acid production from rapid fermentation of the sugars and starch in concentrates and

saliva production from chewing hay occur at separate times; saliva is not produced at the *time* it is needed to buffer the acids.

Total mixed rations (TMR) were developed for domestic cattle to encourage consumption of nutrient-rich dietary components and physically effective fiber at the same time and in correct proportions. A TMR blends all the dietary ingredients together to make a complete ration. High-fiber feeds that are large enough to require chewing (e.g., chopped hay, silage, chopped corncobs, etc.) are blended with rapidly fermentable carbohydrate sources (e.g., corn, oats, citrus pulp, etc.), protein, fat, vitamin, and minerals to make a nutritionally balanced complete feed. A moist binder such as molasses is generally included to decrease dust and to keep the loose individual ingredients from separating in the feed bunk. A TMR approach to feeding animals, such as the giraffe, could be a viable feeding management tool when animals are prone to selecting disproportionate amounts of either the nutrient-dense pellet or the hay component in their diet. If disproportionate selection of dietary components does not occur and the ration offered to the animal is similar to the diet selected by the animal, a TMR approach may not be necessary.

It also has been suggested that long-stemmed hays may be an inappropriate physically effective fiber source for captive giraffe.[6] Because of their physical shape, long-stemmed hays linger in the rumen.[1] Long hay strands form a mat that distends the rumen, causing a feeling of satiation and decreasing intake.[1] Domesticated grazers such as cattle are adapted to this, but wild giraffe have adapted to consuming smaller polygonal food particles (e.g., leaves, bark, etc.) that do not form a ruminal mat.[9] The unique digestive anatomy of the giraffe may be ill equipped to deal with long-stemmed hay and the associated ruminal mat formation (see abstract "Giraffe Nutrition: Facts and Unknowns"). If so, this may explain why giraffe fed hay-only diets had exceptionally low voluntary intake.[8] However, at this time, the optimal size, shape, and quantity of physically effective fiber for captive giraffe has not been determined. It is possible that animals consuming substantial quantities of long-stemmed hay and are offered their daily pellet ration over multiple, equal feedings may meet their physically effective fiber needs, whereas those not consuming sufficient browse or hay may benefit from a TMR. Given the importance of dietary physical form, further research should be conducted to define appropriate physically effective fiber for giraffe and other browsers.

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IV. Clinical Problems of Captive Giraffe with a Possible Nutritional Basis

R. L. Ball, D.V.M.

A. Introduction

The status of captive medicine and management of giraffe is underdeveloped compared with that of other terrestrial megavertebrates, the elephants and rhinoceros. Perhaps the fact that giraffe breed more readily in captivity than these species has discouraged critical evaluation of their overall health. Captive giraffe have a specific set of maladies that are likely related to nutritional inadequacies. Peracute mortality, chronic wasting, energy malnutrition, pica, mortality related to cold stress, pancreatic disease, intestinal parasitism, hoof disease, urolithiasis and neonatal health concerns may all result from problems associated with traditional diets. Pelleted feeds with high starch and protein content and low physically effective fiber coupled with low overall feed intake by captive giraffe may contribute substantially to these problems. Rumen pathology is the proposed basis for numerous secondary conditions.

B. Energy related health issues

Peracute mortality, chronic wasting, neonatal health concerns and cold stress are well documented both in literature and anecdotally and seem to be related to energy malnutrition.[1,4,7-9,13,16] As early as 1854 captive giraffe were noted to have nutritional problems that contributed to their death; this is likely the first recorded case of peracute mortality syndrome (PMS).[6] Peracute mortality syndrome has been seen worldwide in captive giraffe and has been defined as the sudden death of giraffe with a history of a stressor. Typical post-mortem findings include serous atrophy of fat and some degree of pancreatic degeneration.[8,9,13] Early investigations into PMS revealed several common denominators but no etiology. Suggestions were made that energy or, more likely, protein deficiency was at the core of the problem.[8,9] Even as recently as 2004, protein deficiency was hailed as the cause of this problem.[3]

Chronic hypoglycemia has been proposed as the proximate cause of PMS.[1] Pathology related to energy malnutrition is recorded in reports from the 1930's.[10] Energy-deficient giraffe also have been documented in European zoos due to a reported low feed intake.[5] Energy deficiency may be seen in wild giraffe after certain extremes in environmental conditions (Hofmeyer personal communication).[2]

Rumen morphology has been compared in captive and wild giraffe.[12] It was estimated that wild giraffe had nine times the absorptive capacity for short-chain fatty acids (SCFA, also referred to as volatile fatty acids (VFA)) than the captive giraffe examined. Short-chain fatty acids are essential for ruminants in that they provide up to 90% of the available energy and are directly absorbed through the

ruminal mucosa. Nutritional imbalances and pathology in the rumen can decrease the production and absorption of SCFA, resulting in energy deficient states.[17,18,20]

Energy deficiency may become particularly crucial during periods of increased energy demands such as pregnancy and lactation. Dystocia is uncommon and may be due to fetal maternal disparity; however, cases can be seen in dams of poor nutritional status that become exhausted and even show hypoglycemia during parturition (Murray, personal communication).[1] Maternal neglect and failure to produce adequate milk and/or colostrum are other common problems in giraffe and can be readily explained by a negative energy balance in a dam and/or an associated mastitis or irritation to the mammary gland by a calf attempting to nurse.

Nutritional imbalances of the dam alter milk production, which could be a contributing factor in the high (44%) captive first-year giraffe mortality rate (North American Regional Giraffe Studbook, NARGS, 1997). Retrospective assessments of mortality also have been performed on total life spans for animals at Busch Gardens Tampa Bay (BGT) from 1984 to 2002. The number of wild caught and captive born giraffe that died at BGT was compared and survivability curves were prepared. Wild born giraffe lived approximately five times longer than captive-born animals. This trend suggests a developmental problem associated with captivity.

Rumen development is most significant in the postnatal period, during the first three months of life. It is influenced heavily by diet and end products of microbial fermentation (SCFA).[19] A diet high in pelleted feeds and inadequate fiber led to abnormal papillae development, decreased absorptive surface area, and decreased mucosal SCFA transport capability in growing cattle.[18,20] In giraffe, the same dietary factors are likely to lead to a poorly developed rumen that differs significantly from one exposed to browse and high starch, low fiber feeds. Necropsy evaluations of giraffe at BGT revealed that wild-caught animals had better developed papillae and less pathology than captive-born animals. Hoffman and Matern (1988) compared wild and captive giraffe rumens and came to the same conclusion that captive giraffe have less-well-developed ruminal papillae.[12] Because similar management practices (i.e., diets) occur at many North American zoological facilities, there is a significant possibility that these problems are widespread.

C. Other nutritionally related health issues

Urolithiasis,[21] pancreatic disease,[15] dental disease (Montali personal communication), pica or oral stereotypes, gastrointestinal parasitism, and abnormal hoof growth/laminitis are all additional clinical problems noted in captive giraffe. These problems may be additional risk factors to chronic wasting and PMS, but likely have their etiology from the same nutritional causes. Consumption of feeds high in starch and low in fiber can be a cause of ruminal acidosis in domestic ruminants, affecting intake, feed digestibility, milk

production, hoof health, and overall animal health.[14,17] Identifying a common denominator for these problems is one of the cornerstones of medical diagnostic investigations. A central hypothesis is that dietary induced rumenitis and resulting changes in physiology are fundamental to the disease syndromes seen in captive giraffe. Figure 1 illustrates the proposed complex pathophysiology in captive giraffe associated with diets high in starch and low in fiber. There is limited information available about subacute ruminal acidosis leading to rumenitis and rumen mucosal degeneration in domestic ruminants and the condition is difficult to clinically diagnose.[11,17] It is possible that chronic and possibly subacute ruminal acidosis and rumenitis, resulting from feeds high in starch and low in fiber have a significant, but undetected occurrence in the captive giraffe population.

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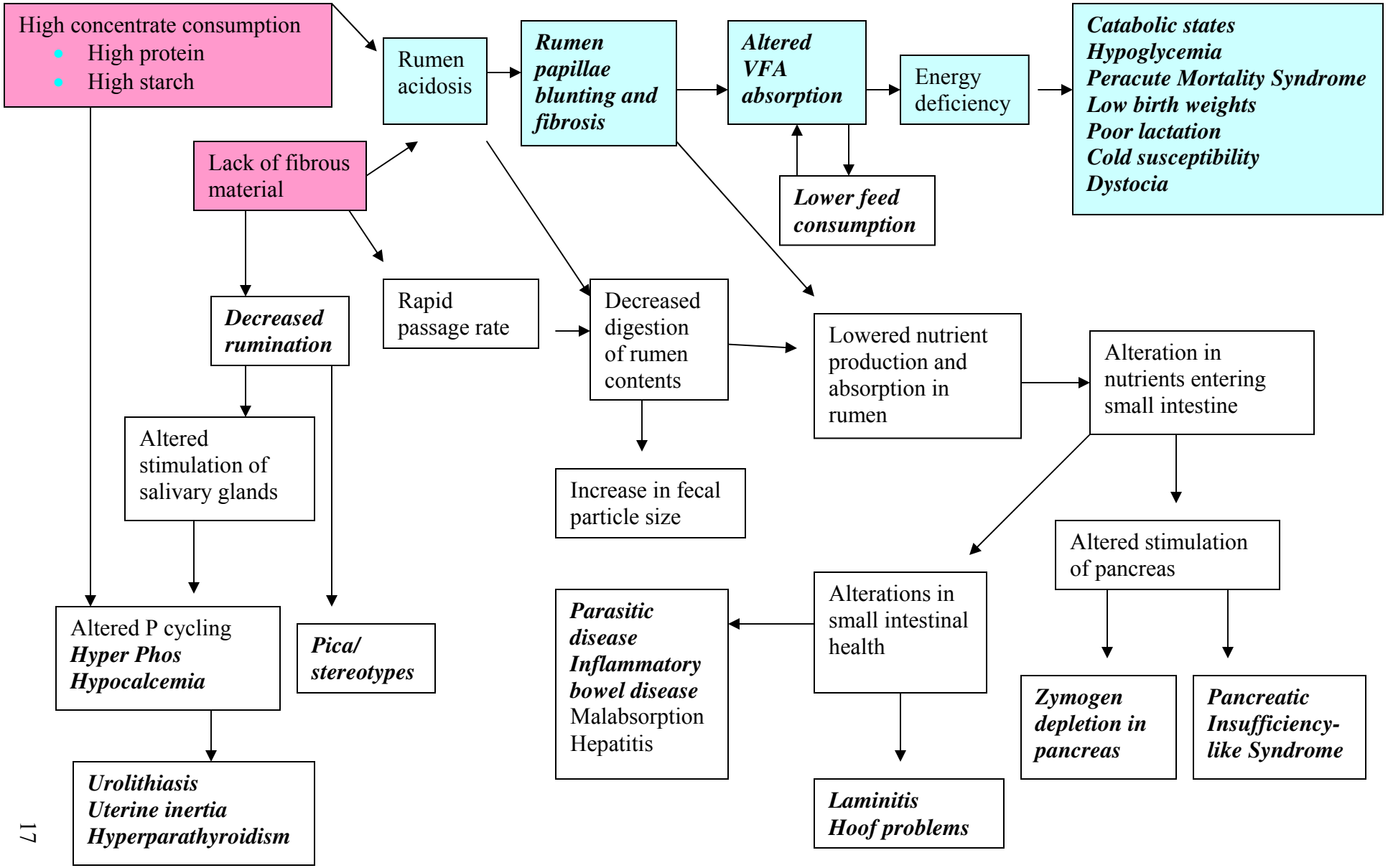
High concentrate consumption
High protein
High starch
Rumen acidosis
Rumen papillae blunting and fibrosis
Altered VFA absorption
Energy deficiency
Catabolic states
Hypoglycemia
Peracute Mortality Syndrome
Low birth weights
Poor lactation
Cold susceptibility
Dystocia
Lower feed consumption
Lack of fibrous material
Decreased rumination
Rapid passage rate
Decreased digestion of rumen contents
Lowered nutrient production and absorption in rumen
Alteration in nutrients entering small intestine
Altered stimulation of salivary glands
Increase in fecal particle size
Altered stimulation of pancreas
Altered P cycling
Hyper Phos
Hypocalcemia
Pica/ stereotypes
Alterations in small intestinal health
Parasitic disease
Inflammatory bowel disease
Malabsorption
Hepatitis
Zymogen depletion in pancreas
Pancreatic Insufficiency-like Syndrome
Urolithiasis
Uterine inertia
Hyperparathyroidism
Laminitis
Hoof problems

V. New Feeding Recommendations for Giraffe
D. A. Schmidt, Ph.D. and M. L. Schlegel, Ph.D.

Not all captive giraffe are afflicted with the clinical problems discussed in the previous chapter. However, the following recommended diet changes may help prevent the occurrence of the previously mentioned clinical issues.

A. Dietary changes

1) Protein - Recommended protein concentrations for giraffe have typically been 16 to 20% of the diet. Zoo diets for giraffe and other browsing animals are typically based on alfalfa hay and herbivore pellets. This diet easily meets and can even surpass the previously recommended requirements for crude protein. Alfalfa hay typically contains more protein than grass hays, depending on hay maturity and cutting stage. Herbivore pellets also tend to be alfalfa-based and contribute significantly to the total concentration of crude protein intake.

Protein for ruminants comes from two sources. One source is the diet. The second source of protein comes from microbial organisms that pass out of the animal's rumen and are digested in the small intestine. When too much of any nutrient (i.e., protein, starch, etc.) is fed, it tends to promote the growth of the microbial population that thrives on that item. When excess protein is fed, it supplies the bacterial populations with the nitrogen needed for bacterial reproduction. As bacterial concentrations increase, so will the production and concentration of short-chain fatty and lactic acids. When high concentrations of protein and carbohydrates occur in the diet, short-chain fatty acids are produced more rapidly. Animal absorption of these short-chain fatty acids and lactic acid may not keep up with their production, which allows the short-chain fatty acids and lactic acids to accumulate in the rumen, thereby decreasing ruminal pH and creating potentially unhealthy shifts in microbial populations.

A statement of agreement by the workshop committee regarding protein concentration in giraffe diets is: **"Given the nutrient requirements of domestic ruminants and diet studies of wild giraffe, there is no nutritional reason to expect that the total dietary crude protein requirement of a mature giraffe is more than 12% of the complete diet (dry matter basis; DMB) when dry matter intake is at least 1.2% of the animal's body weight.[1,2] Diets containing 10 to 14% crude protein (DMB) will likely provide the maintenance needs of adult giraffe."**

2) Starch and Simple Sugars - Too much starch or simple sugars, as is found in produce, can lead to ruminal imbalances as was discussed previously for protein. When high dietary concentrations of starch and simple sugars are fed to a ruminant animal there is an increased risk of the

animal developing ruminal and possibly systemic acidosis. Simple sugars and starch are rapidly fermented by ruminal microbes to lactic acid; in a healthy rumen with a low to moderate carbohydrate load, lactic acid concentrations are low. When large concentrations of simple sugars and starch are rapidly fermented, lactic acid concentrations increase significantly over a short period of time. Increases in lactic acid concentrations will dramatically lower the pH of the rumen, which results in microbial death, erosion of the rumen mucosal lining, decreased ruminal papillae size and number, liver abscesses, and, if systemic acidosis ensues, possibly death.

One of the most popular pellets used in giraffe diets is a commercially manufactured pellet known as ADF 16. This open formula pellet has an approximate starch content of 25-30% (DMB; Griffin, personal communication). The workshop committee agreed to the following statement: **"There is no indication that starch is a necessary dietary component and no more than 10% starch (DMB) should be included in the total diet of captive giraffe. Diets containing less than 5% starch are encouraged. High concentrations of simple sugars also can result in ruminal acidosis; therefore, care must be taken to also limit these in the diet."**

3) Fat - Fat has a variety of functions and can be used as a source of energy, as a carrier of fat-soluble vitamins, and is present in cellular membranes. Essential fatty acids are those that cannot be synthesized by the body and must be obtained through the diet. Linoleic and linolenic acids are believed to be essential fatty acids. Although their specific function is not fully understood, they are believed to be integral in cell membranes and are part of compounds called eicosanoids, which play an important role in hormone regulation and release. These fatty acids are typically in high concentrations in flaxseed and fish meal, but fish meal in a manufactured feed will have a fish odor and herbivores may not find the odor or taste appealing. Adding linoleic and linolenic acids to the diet may be useful, although additional research is still needed in this area, especially as it pertains to ruminants.

Total dietary fat concentrations should be in the range of 2 to 5% (DMB) for the complete diet.[2] Consideration should be given to evaluating the concentrations of linoleic and linolenic acids as part of the total fat concentration in manufactured feeds.

4) Fiber - Ruminants, unlike nonruminant (monogastric) animals, rely heavily on a symbiotic relationship with ruminal microbial organisms (i.e., bacteria, protozoa, and fungi) to supply a significant proportion of the animal's daily requirements for energy, protein, and even some vitamins. As discussed previously, microbial organisms themselves are digested by the animal as they escape the rumen and are used as a source of protein.

Microbial organisms also have the ability to synthesize the water-soluble B-complex vitamins as well as vitamin K, a fat soluble vitamin.

No mammals possess the enzymes necessary to digest plant fibers (i.e., cellulose, hemicellulose, and lignin). Ruminant animals have evolved to rely on a fibrous plant material diet by developing a symbiotic relationship with microbial organisms that possess the enzymes necessary for digesting plant fiber. The microbes break the particles into individual sugar units, use the sugars to meet their own energy needs, and excrete short-chain fatty acids (i.e., acetic, butyric, and propionic acids) as end-products (this entire microbial process is typically referred to as fermentation). The short-chain fatty acids are then absorbed by the animal and used as a source of energy. In essence, when feeding a ruminant, one is feeding the microbes that supply the animal with energy, protein, and some vitamins.

Fiber for ruminant animals is typically classified as neutral detergent fiber (NDF) and acid detergent fiber (ADF). Neutral detergent fiber consists of the hemicellulose, cellulose, and lignin plant fractions; all of these are considered structural fiber and they provide physical support for the plant. Acid detergent fiber concentrations measure the cellulose and lignin fractions.

The workshop committee agreed that maintenance diets for giraffe should contain a minimum of 25 to 30% ADF (DMB) for the complete diet.[1] Below this concentration problems with ruminal acidosis and general health of the giraffe are more likely to occur. This concept is not to be confused with the ADF concentration of the hay. The hay itself should have no more than 40% ADF to maintain digestibility.

For a more thorough explanation on ruminant nutrition, please see the "Ruminant Nutrition Review" section of these proceedings.

5) Vitamins - Vitamins are required by an animal to initiate and control many metabolic processes in the body. In ruminants, microbial organisms in the rumen and lower intestinal tract synthesize most of the water-soluble vitamins (i.e., B-complex, folate, biotin) and fat-soluble vitamin K to meet the animal's daily needs. Cobalt, a trace mineral, needs to be provided in adequate concentrations to ensure vitamin B_{12} synthesis. Based on work with dairy cows, biotin is considered to be potentially important for hoof strength.

In contrast to the B vitamins, fat-soluble vitamin A, D, and E requirements need to be met through the diet. Although vitamin D can be synthesized by the animal upon exposure to sunlight, nutritionists rely on the manufactured feed to provide these vitamins. The committee agreed that **because specific vitamin requirements for the giraffe are unknown and there is no**

reason to believe giraffe would differ dramatically from other ruminants in their vitamin requirements, recommendations are to feed vitamins A, D, and E at the concentrations recommended as part of the total diet for domestic ruminants[1,2]:

Vitamin	**A**	**3,900**	**IU/kg of diet (DMB)**
	D	**750**	**IU/kg of diet (DMB)**
	E	**60**	**IU/kg of diet (DMB)**

6) Minerals - Minerals are classified as macrominerals, needed in large concentrations in the diet, and trace minerals, needed in much smaller concentrations. The macrominerals are calcium, phosphorus, sodium, chlorine, potassium, magnesium, and sulfur. The microminerals required by animals for normal body processes include boron, cobalt, chromium, copper, iodine, manganese, molybdenum, fluorine, zinc, iron, silicon, and selenium. Forages can be variable in mineral concentrations (e.g., selenium) depending on where they are grown and should not be relied on to meet an animal's requirement. As with fat-soluble vitamins, nutritionists rely on a manufactured feed supplemented with appropriate concentrations of macro- and microminerals to meet the animals' needs. The following concentrations for the total diet (DMB) were agreed upon by the workshop committee:

Calcium[1,2] **0.65 to 1.0%; meets maintenance, growth, and lactation requirements**

Phosphorus[1,2] **0.35 to 0.5%; exceeding 0.5% P in the total diet may contribute to osteodystrophy and uroliths**

Magnesium[1,2] **0.3% minimum**

Copper[1,2] **10-15 ppm (mg/kg)**

Other mineral concentrations should follow those established for domestic ruminants.[1,2]

Mineral and salt blocks - A free-choice mineral block is not a sufficient source of mineral supplementation. However, giving giraffe free-choice access to salt is recommended. Salt intake may increase water intake and subsequently decrease potential issues with urolithiasis.

7) Feed intake - After initiating these changes, one would expect to observe an increase in food consumption compared with higher-starch, lower-fiber diets that are typically fed to captive giraffe. Based on captive studies, it is predicted that an adult giraffe will consume approximately 1.2% of its body weight in dry food each day, but this may vary slightly from animal to animal. Giraffe that are incurring greater energy demands, such as those

exposed to colder climates, that are pregnant, or lactating, should continue to receive the same nutrient concentrations, but may consume more feed than the predicted 1.2% of body weight.

8) Summary - The committee concluded that captive giraffe are currently being fed diets too high in starch, simple sugars, and protein. It is believed that the high concentrations of these nutrients may be leading to or exacerbating many of the health issues currently diagnosed in captive giraffe. By lowering the concentrations of simple sugars, starch, and protein and increasing the concentration of fiber in giraffe diets, zoos should be able to minimize the number of health problems associated with unhealthy rumens. By creating a healthy ruminal environment, issues with diarrhea also should be resolved. Feeding a lower protein diet also will help cut feeding costs because the quantity of higher priced, higher protein hays fed will be decreased.

B. Enrichment

Enrichment items are sometimes thought of as food offered in addition to an animal's "normal diet". Changing this concept is important. **ALL** food an animal is offered should be considered as part of the "total diet" and analyzed as such. **Enrichment items (not including browse) should be no more than 5% of the total diet on an as-fed (fresh or moisture included) basis.**

C. Browse

Browse is recommended for giraffe when available. Access to browse will not be a problem until it limits trace mineral and vitamin intake from the commercially manufactured feed. Only browse that has been approved for use with giraffe should be fed.

D. Public feeding programs

Most items (e.g., crackers, produce, etc.) selected for public feeding programs contain high concentrations of sugars and starch. Giraffe are typically fed during restricted time slots (large quantity fed in a short amount of time) or without restricting the quantity consumed by each animal. Dominant or more social animals will consume more public feeding items and are thereby at a greater risk for problems with lactic acidosis. The combination of high-starch/simple-sugar items, narrow public feeding time slots, and uncontrolled quantities fed to each individual can initiate and exacerbate issues with ruminal acidosis suspected to be a predominant problem with captive giraffe.

Public feeding programs for giraffe are becoming more popular among zoos. Finding items that are easy to handle, palatable to giraffe, and that will not create/extend problems with the rumen is a challenge. If browse is available, it may be a good choice. Developing an ideal food item that is easy for the public to feed requires further investigation. With patience, cooperation, and persistence

from giraffe-holding institutions, we should be able to develop a more nutritionally appropriate food item that can be used for public feeding programs.

E. Management recommendations

1) Feeding frequency - It is recommended that ruminant animals be offered at least two separate meals of the manufactured feed daily with hay available at all times. Increasing the number of meals offered will minimize the starch and sugar loads entering the rumen at one time and, in turn, lower the incidence of rapid microbial fermentation in the rumen. If an animal consumes an entire day's ration of pelleted feed at one time, the ruminal microbes that use (ferment) sugars, starch, and easily degraded protein have the potential to quickly ferment the nutrients, rapidly produce large concentrations of short-chain fatty acids, and multiply exponentially, all of which lead to problems with ruminal acidosis.

2) Feeder space - Offering adequate feeder space for giraffe that are housed together is another important consideration. Pelleted feeds are typically used to deliver the appropriate concentrations of vitamins, minerals, fats, and other nutrients required by the animal. If too few feeders exist, the dominant animal may consume a larger proportion of the pelleted feeds than is necessary to meet its nutritional needs, leaving smaller portions for the remaining animals. The dominant animal also will have a greater chance of incurring problems with ruminal acidosis when consuming large quantities of pelleted feed at one time due to rapid fermentation of starch, simple sugars, and highly degradable protein in pelleted feeds, as was discussed previously under "Feeding Frequency".

3) Weighing feed - Weighing and recording daily quantities of food offered is critical to monitoring giraffe intake. Anyone who has ever worked with hay knows bale density varies among fields, hay types, cuttings, and even between bales from the same field baled on the same day. Pelleted feeds also vary in density by type, lot, and manufacturer.

Quantifying food offered by weight rather than volume will enable a more precise understanding of the quantity of nutrients actually being fed. However, what an animal is fed is not always what an animal eats. For this reason it also is important to monitor and record amounts of food left over (in addition to food offered) for both hay and pelleted feeds. Knowing when an animal's intake changes can be the first sign of illness.

Based on captive studies, it is believed that an adult giraffe will consume approximately 1.2% of its body weight in dry food each day. Keeping track of giraffe body weights, food amounts offered, and food consumption will help to more quickly identify an animal that may be incurring health issues. The 1.2% of body weight value should be used as a guideline and will differ slightly between individuals.

4) Giraffe weights - Weighing giraffe on a regular basis is another tool that is used to monitor animal health. A change in an individual's food consumption is not always easily determined when giraffe are fed as a herd. These suspicions can be confirmed or dismissed if regular weights on animals are taken. Giraffe weights also will help identify how much food should be offered.

Visual assessments are not a reliable tool for determining fluctuations in weight and are subjective to the person doing the evaluation. However, the committee at this workshop discussed the potential of creating a body condition scoring chart to be used as an additional tool to monitor giraffe health. Additional information on this proposed project can be found under the "Research Needs" section of these proceedings.

1. National Research Council (NRC). 2000. Nutrient Requirements of Beef Cattle, 8th ed. National Academy Press, Washington, DC.

2. National Research Council (NRC). 2001. Nutrient Requirements of Dairy Cattle, 7th ed. National Academy Press, Washington, DC.

F. Diet and management recommendation summaries

Diet Nutrient	Concentration
Crude Protein	10 - 14% DMB
Starch	< 10% DMB, lower is better
Fat	2 - 5% DMB
ADF	25 - 30% DMB (minimum)
Vitamin A	3,900 IU/kg DMB
Vitamin D	750 IU/kg DMB
Vitamin E	60 IU/kg DMB
Calcium	0.65 - 1.0% DMB
Phosphorus	0.35 - 0.5% DMB (0.5% maximum)
Magnesium	0.3% DMB minimum
Copper	10 - 15 ppm DMB

- Other mineral concentrations to follow those established for domestic ruminants
- free choice access to salt block

Management

- Feed intake (DMB) should be approximately 1.2% of body weight.
- Enrichment should be less than 5% of total diet on as fed basis.
- Browse - access recommended, but not to the degree where it inhibits animal's intake of manufactured feed.
- Public feeding - Avoid items high in simple sugars and starch, such as crackers and produce.
- Feed at least two times each day (offering hay and pellets at each feeding), larger feeding frequencies is encouraged.
- Provide enough feeders for group housed animals to allow all animals access to manufactured feed and prohibit a dominant animal from consuming too much of the manufactured feed.
- Weigh and record all feed offered to the animals daily.
- Obtain animal body weights on a regular basis.

VI. Giraffe Diet Formulation Examples

The typical zoo giraffe diet of pelleted feed, with an ADF concentration of 16%, fed with alfalfa hay will not meet the proposed recommended feeding specifications. Table 1 lists average nutrient specifications for grass and legume hays, ADF-16 formula pellets, and a typical zoo giraffe diet consisting of 50% alfalfa and 50% ADF-16 pellets (DMB). The new recommendations are listed under Total Diet Target for comparison. The current zoo diet is too low in ADF and vitamin D and too high in protein, starch, calcium, and phosphorus with copper being slightly over the suggested concentration.

Table 1. Average nutrient composition (DMB) of hays, pellets, and a typical zoo diet compared to the new total diet recommendations.

	Hay		ADF-16		Total Diet
Nutrient	Alfalfa	Grass	Pellets	Zoo Diet[a]	Target
ADF (%)	31.2[b]	39.5[b]	17.2	24.2	> 25-30
Fat (%)	2.1[b]	2.6[b]	3.6	2.9	2-5
Crude protein (%)	20.2[b]	10.6[b]	19.0	19.6	10-14
Starch (%)	2.5[c]	1.2[c]	30.0[d]	16.3	< 10
Calcium (%)	1.5[b]	0.6[b]	0.9	1.2	0.65-1.0
Phosphorus (%)	0.3[b]	0.2[b]	0.8	0.6	0.35-0.5
Magnesium (%)	0.3[b]	0.2[b]	0.3	0.3	min 0.3
Copper (ppm)	9.0[b]	9.0[b]	22.2	15.6	10-15
Vitamin A (IU/kg feed)	[e]	[e]	7,220	3,610	3,900
Vitamin D (IU/kg feed)	[e]	[e]	1,330[f]	670	750
Vitamin E (IU/kg feed)	[e]	[e]	367	184	60

[a]Typical zoo diet: average alfalfa (listed) and ADF-16 pellets consumed in a 50:50 ratio.
[b] "hay, all samples", Nutrient Requirements of Dairy Cattle, 2001.
[c] "hay, all samples", Nutrient Requirements of Beef Cattle, 1996.
[d]Griffin, personal communication, 2005.
[e]Vitamin concentrations not given due to variability among hays; calculations are made with the assumption that no vitamins come from the hay.
[f]Vitamin D amount added.

National Research Council (NRC). 2000. Nutrient Requirements of Beef Cattle, 8th ed. National Academy Press, Washington, DC.

National Research Council (NRC). 2001. Nutrient Requirements of Dairy Cattle, 7th ed. National Academy Press, Washington, DC.

The following information consists of diet examples based on the new recommendations and uses the average hay values listed in Table 1 for formulation purposes. Manufactured feed nutrient specifications for each diet scenario are listed in Table 2. Please remember that the following are only examples and will vary by animal size, intake, hay quality, and available feeds. Please consult your veterinarian, nutritionist, consulting nutritionist, or feed manufacturer representative to assist in formulating appropriate diets for your giraffe using feeds available in your area.

Listed below are diet examples for a 1,000-kg animal that consumes approximately 12 kg dry feed/day based on a prediction of 1.2% of his body weight as intake. Table 2 describes the nutrient specifications needed for the manufactured feed to meet the new recommendations.

Diet A. A diet for this animal consisting of **25% alfalfa hay** (3 kg) and **75% manufactured feed** (9 kg). Please see Table 2 for manufactured feed nutrient specifications needed for this diet.

Diet B. A diet for this animal consisting of **50% alfalfa hay** (6 kg) and **50% manufactured feed** (6 kg). Please see Table 2 for manufactured feed nutrient specifications needed for this diet.

Diet C. A diet for this animal consisting of **35% alfalfa hay** (4.2 kg) and **65% manufactured feed** (7.8 kg). Please see Table 2 for manufactured feed nutrient specifications needed for this diet.

Diet D. A diet for this animal consisting of **25% mixed grass hay** (3 kg) and **75% manufactured feed** (9 kg). Please see Table 2 for manufactured feed nutrient specifications needed for this diet.

Diet E. A diet for this animal consisting of **25% mixed grass hay** (3 kg), **25% alfalfa hay** (3 kg) and **50% manufactured feed** (6 kg). Please see Table 2 for manufactured feed nutrient specifications needed for this diet.

Table 2. Target nutrient concentrations (DMB) for **manufactured feeds** based on diet examples described for a 1,000-kg animal consuming 1.2% (12 kg) of its body weight.

Nutrient	Example Diet Concentration Target	Manufactured Feed				
		A	B	C	D	E
ADF (%)	30	29.6	28.8	29.4	26.8	24.7
Fat (%)	5	6.0	7.9	6.6	5.8	7.7
Protein (%)	14	11.9	7.8	10.7	15.1	12.6
Starch (%)	5	5.8	7.5	6.3	6.3	8.1
Calcium (%)	0.8	0.57	0.50	0.50	0.87	0.55
Phosphorus (%)	0.4	0.43	0.50	0.45	0.47	0.55
Magnesium (%)	0.3	0.30	0.30	0.30	0.33	0.35
Copper (ppm)	15	17.0	21.0	18.2	17.0	21.0
Vitamin A (IU/kg)	3,900	5200	7800	6000	5200	7800
Vitamin D (IU/kg)	750	1000	1500	1154	1000	1500
Vitamin E (IU/kg)	60	80	120	92	80	120

These nutrient specifications for manufactured feeds were calculated using the average hay specifications listed in Table 1. Changes to the manufactured feed may be necessary if hay analyses differ from the averages listed.

VII. Research Needs

A. Body condition scoring system

Creating a standardized body condition scoring system for giraffe will help managers to more quickly and easily identify if and when animals may be developing health issues affecting feed intake. A standard method of cataloguing body condition is needed in giraffe to facilitate husbandry and to allow comparison of an institution's animals to an accepted standard. Towards this end, funding has been secured to provide a photographic record of all giraffe in North America. A panel will meet to discuss the classification, develop a standardized scale and classify each of the giraffe according to this standard. Anyone interested in serving on the panel please contact Joe Christman at Disney's Animal Kingdom.

Digital photos will be utilized. Each institution will be contacted and sent a digital camera with detailed instructions. Your support in this project is greatly needed and appreciated. Three photos of each animal are needed:

- Full front
- Left full side
- Full Rear

B. Diet form

Research is currently under way to determine whether the physical form of the diet affects giraffe intake, digestion, and possibly stereotypic behavior. A total mixed ration (TMR), which consists of "loose" feed items mixed with chopped hay, is being compared with pelleted feeds with normal, long-stemmed hays. Although a TMR helps decrease selectivity and encourages increased animal feeding time, it is not readily available at every feed manufacturer due to the need for special equipment.

C. Natural food item nutritional analyses

A more thorough investigation quantifying nutrients of diets consumed by free-ranging giraffe will help identify more appropriate feed items for the captive population. In addition to quantifying protein, fat, and fiber, it is important to measure starch and simple sugar concentrations. Identifying and quantifying the specific types of protein (e.g., amino acids), fat (e.g., fatty acids), fiber (e.g., hemicellulose, cellulose, lignin, pectin, gums, etc.), and sugars would further the understanding of giraffe nutrition.

D. Alternative public feeding items

Finding or developing food items higher in fiber and lower in starch and simple sugars that giraffe will readily accept is an important component to enhancing the rumen health of captive giraffe. The currently fed items (e.g., crackers, produce), which are readily accepted by giraffe, are much higher in starch and simple sugars than is considered appropriate for these ruminant animals. Some institutions have

used high-fiber primate biscuits, which are better than crackers or produce because they are nutritionally fortified, but they are still considered too high in starch for giraffe.

E. In vivo / in vitro digestibility studies

Conducting digestibility trials to compare both the current and recommended diets will help us better understand the giraffe's ability to digest and utilize dietary nutrients.

F. Blood urea nitrogen concentrations

Identifying normal blood urea nitrogen levels before and after making the proposed feeding changes and comparing those to free-ranging giraffe and domestic ruminant concentrations will help evaluate the proposed feeding changes. We hypothesize that giraffe on the current high-protein diets will have a much higher blood urea nitrogen concentration than giraffe on lower-protein diets.

G. Plasma ammonia concentrations

International Species Information System (ISIS)[1] data indicates an average value of 3.3 mM for plasma ammonia concentrations in giraffe, which is higher than is typically observed with domestic ruminants and may be reflective of the high-protein diets giraffe are receiving in captivity. It is hypothesized that feeding high-protein diets may be taxing on the kidneys, which work to rid the body of the excess nitrogen, a byproduct of protein digestion. Comparing plasma ammonia concentrations from giraffe on the current diet to concentrations from giraffe after the proposed diet recommendations have been made, those from free-ranging giraffe, and values from domestic ruminants will help us understand protein utilization by giraffe.

H. Nitrogen:creatine ratios

Quantifying the nitrogen:creatine ratios in urine from captive giraffe before and after dietary changes and comparing those to free-ranging giraffe (if urine is available) and domestic ruminant concentrations will help us validate that protein concentrations of captive diets is excessively high.

I. Serum for mineral and vitamin analyses

A current research effort is underway to establish "normal" concentrations of vitamins and minerals in free-ranging giraffe serum. These data will be used as a basis for evaluating the same nutrients in serum collected from the captive population. Dramatic differences will be an indication that additional changes with regard to minerals and vitamins need to be made to captive giraffe diets.

J. Urine mineral and pH analyses

Just as with serum, it is important to analyze urine mineral concentrations (especially calcium, phosphorous, and magnesium) and pH from giraffe on the current high-protein diets with those that have been transitioned to the proposed low-protein, low-starch/free-sugar, higher-fiber diets. Additional comparisons can be made with urine collected from free-ranging giraffe and domestic ruminants.

K. Serum and urine collection banking

Establishing a bank of frozen serum and urine from captive animals will easily provide samples for researchers interested in studying giraffe health and nutrition. Collecting additional serum and urine samples opportunistically when giraffe are undergoing other procedures, either in the wild or in captivity, would be the easiest way to begin a giraffe sample bank.

L. Fecal nitrogen concentrations

Nitrogen is typically measured in the laboratory as a predictive indicator of crude protein concentration. Measuring nitrogen concentrations in captive giraffe feces from giraffe on a current diet of alfalfa hay and pellets to giraffe that have been transitioned to the current diet recommendations, free-ranging giraffe fecal nitrogen concentrations, and values identified for domestic ruminants will help to further understand the giraffe's ability to digest, utilize, and clear the body of excess nitrogen. We hypothesize that nitrogen concentrations in feces of giraffe on current diets will be much higher than giraffe on the currently proposed diet and that of domestic ruminants.

M. Hoof slice at necropsy

At necropsy it is suggested that as part of a standard necropsy a sagital section of the foot be made with a band saw so that the first second and third phalanxes are included in the section. Then the distance between the proximal end of the pedal bone (P3) and the hoof wall and the tip or distal end of P3 and the hoof wall can be measured. They should be the same suggesting no separation of bone from hoof wall (i.e., the bone remains parallel to the hoof wall). But if the animal suffered bouts of laminitis, secondary to rumen acidosis in its life, the P3 tip tends to rotate downward and the distance between it and the hoof wall will be much greater than at the proximal end. This needs to do this with each claw. Lesions tend to be worse on front feet since they carry more weight, which causes more separation of hoof wall when the junction is weakened by laminitis. If feet are saved at the time of necropsy and shipped to Dr. Judy St. Leger, Sea World San Diego, she will perform this examination for the clinicians and send photos of the examined hoof. Please contact her prior to shipping the feet and see the full necropsy protocol at the end of this document.

N. Manufactured feed ingredients

Research investigating different pellets (based on different ingredient composition) while following the recommendations of this committee should be conducted. Assessment of the quality of these pellets should be performed based on the suggested parameters indicated above (e.g., serum mineral, serum vitamin, fecal nitrogen, among others). The use of chelated minerals or addition of buffers to the pellet should be investigated based on geographical/climatic needs (e.g., heat stress conditions).

1. International Species Information System (ISIS). 2002. *Giraffa camelopardalis*. Reference ranges for physiological values in captive wildlife (CD-ROM). www.isis.org.

VIII. The Giraffe Nutrition Workshop Participants

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IX. Abstracts

Rumen Physiology of Digestion in Ruminants
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A unique and important aspect of ruminant nutrition is the microbial digestion of complex carbohydrates, protein, and lipids as well as the microbial synthesis of organic substances (including B vitamins) in the rumen. This forestomach provides a continuous culture system for anaerobic bacteria, protozoa, and fungi. Importantly, rumen bacteria have different fermentative properties (e.g., cellulolytic, amylolytic, xylanolytic, pectinolytic, dextrinolytic, methanogenic, H_2-utilizer, soluble-sugar fermenter, proteolytic, and lipolytic). As a result, the products of microbial fermentation include CO_2, H_2, CH_4, formate, ethanol, lactate, succinate, short-chain fatty acids (SCFA; acetate, propionate and butyrate), ammonia, amino acids, and branched-chain fatty acids. The breakdown of dietary carbohydrates consists of two distinct stages: 1) the extracellular hydrolysis of complex ones (e.g., starch, fructosans, cellulose, hemicellulose, and pectins) to simple sugars; and 2) the intracellular degradation of simple sugars, with SCFA as the major products. The rate of SCFA production depends on the substrate: soluble carbohydrates > pectins > celluloses. In ruminants fed hay or other roughages, the rumen SCFA consist of 60-70% acetate, 15-20% propionate, and 10-15% butyrate. Bacteria are the principal micro-organisms responsible for protein degradation by proteases, peptidases, and deaminases, as well as urea hydrolysis by urease in the rumen. The resultant ammonia is incorporated into amino acids in the presence of various α-ketoacids via glutamine synthetase, glutamate dehydrogenase, and other enzymes. In the presence of carbohydrates, sulfur, and ammonia, rumen micro-organisms can synthesize all α-amino acids, which are subsequently utilized for microbial protein synthesis. About 90% of the total N in the rumen content exists in an insoluble form (e.g., undigestible dietary protein and microbial proteins), and the remaining N mainly as free amino acids and ammonia. Excess rumen ammonia is absorbed either directly through the rumen wall into blood or enters the small intestine. Bacteria and protozoa have very high activities of lipases, which attack triglycerides. The extensive hydrolysis of esterified dietary lipids yields long-chain fatty acids and glycerol. The latter is further fermented into SCFA. Because of the lack of O_2 in the rumen, fatty acid oxidation by rumen microbes is nearly absent. However, phospholipids are actively synthesized and unsaturated fatty acids are extensively hydrogenated by rumen bacteria and protozoa. This biohydrogenation process involves isomerization and results in the generation of conjugated fatty acids. Of note, high levels of long-chain fatty acids are toxic to cells, lower feed intake, decrease the number of protozoa, inhibit protein hydrolysis and amino acid catabolism, and reduce SCFA production in the rumen. Disorders in rumen digestion (particularly rapid production of SCFA, lactic acid, ammonia, and gasses) can result in ulceration of the forestomach, hypertonic rumen digesta, systemic acidosis, systemic dehydration, bloat (high intraruminal pressures that can cause cardiovascular collapse and death), neurological damage, and metabolic abnormalities. Therefore, understanding the rumen physiology of digestion is essential to optimizing feeding practice and health for ruminants.

Physiology of Ruminal Acidosis

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Ruminal acidosis is a disease characterized by low pH of the ruminal digesta. The lowered pH of the digesta is caused by excessive concentrations of lactic acid produced as an end-product of microbial fermentation in the rumen. The etiology of this disease is the overfeeding of starches to the rumen fauna unadapted to starch in the diet. The lack of adaptation can occur from including starch containing feeds (i.e. grain) abruptly into the diet or by offering excessive levels of starch containing feeds in the diet and/or fluctuating eating behavior. In captive ruminant herbivores, ruminal acidosis would be more likely to occur from the chronic overfeeding of starch in the diet given the diet type (high in plant structural carbohydrates and low to nonexistent in starch) that these animals select in the wild.

Ruminants are so named due to their compartmentalized stomach anatomy. In essence these animals are all similar in that they have a large compartment anterior to the secretory stomach that functions to symbiotically harbor microflora (bacteria, protozoa, and fungi) for the purpose of fermenting plant structural polysaccharides (cellulose, hemicellulose, and pectin) to short chain fatty acids that the animal can in turn absorb and metabolize for energetic reactions. The predominant fatty acids produced as fermentation end-products are acetic, propionic, and butyric acids. The addition of starch or free sugars (alpha glycosidic bonding) will be fermented to lactic acid. In adapted rumen ecosystems when the starch is not fed in an excessive mass, the lactic acid is further fermented primarily to acetic acid. However, when the rumen is not adapted to the production of lactic acid or when the mass of lactic acid production is great, significant concentrations of lactic acid can occur in the rumen. The pKa of lactic acid is 3.9, whereas the pKa of the short chain fatty acids is 4.8. Therefore, the increase concentration of lactic acid will result in substantially lowered ruminal digesta pH. The lowered pH of the rumen digesta will cause erosion of the rumen mucosa, which if severe enough can lead to microbial entry in the splanchnic blood flow and travel to the liver, causing liver abscess. Ruminal acidosis is characterized by a reduction in ruminal papillae size, and if severe enough the presence of liver abscesses.

The potential also exists for the level of rumen fermentable protein in the diet to enhance the potential susceptibility of a ruminant to ruminal acidosis. The microflora requires nitrogen for growth. The greater the supply of fermentable nitrogen in the diet, the greater supply of nitrogen to support growth of the bacteria, the greater the growth rates of the microflora and the more rapid the production of short chain fatty and lactic acids. Therefore, to prevent the potential for ruminal acidosis to occur care should be taken in diet formulation, primarily diets should be formulated not to contain excessive levels of ruminally fermented, or rumen degradable, protein.

Formulating diets for ruminant or nonruminant herbivores should be done to limit the amount of starch-containing feeds and the amount of rumen degradable protein. These two guidelines will do much to prevent high levels of lactic acid from accumulating in the rumen as well as help to prevent the rapid production of acids in the rumen. The result should be improved rumen health, and subsequently improved animal health.

Clinical Problems of Captive Giraffe with a Possible Nutritional Basis

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Captive giraffe have a specific set of maladies that are likely related to nutritional inadequacies. Peracute mortality, chronic wasting, energy malnutrition, pica, mortality related to cold stress, pancreatic disease, intestinal parasitism, hoof disease, urolithiasis and neonatal health concerns may all result from problems associated with traditional diets, especially high levels of concentrates with associated high starch content and low physically effect fiber, and low overall feed intake. Rumen pathology is the proposed basis for numerous secondary conditions.

Peracute mortality syndrome has been defined as the sudden death of giraffe with a history of a stressor. Typical post-mortem finding include serous atrophy of fat and some degree of pancreatic degeneration. Early investigations into peracute mortality syndrome revealed several common denominators, but no etiology. Suggestions were made that energy or more likely protein deficiency was at the core of the problem. Nutritional imbalances and pathology in the rumen can reduce the production and absorption of short chain fatty acids, resulting in energy deficient states.

While giraffe readily breed in captivity, a significant amount of reproductive and post-partum problems occur that appear to be related to energy malnutrition. Dystocia may be due to fetal maternal disparity but many cases can also be seen in dams of poor nutritional status that become exhausted and even hypoglycemic during parturition. Maternal neglect and failure to produce adequate milk and/or colostrum is another common problem in giraffe. Rumen development of the calf is most significant in the postnatal period, during the first 3 months of life. It is influenced heavily by diet and short chain fatty acid production. A diet of high concentrates and inadequate fiber leads to abnormal papillae development, decreased absorptive surface area, and decrease mucosal short chain fatty acid transport capability in growing cattle. Necropsy evaluations of giraffe at Busch Gardens Tampa reveal that wild caught animals have better developed papillae and less pathology than captive born animals.

Urolithiasis, pancreatic disease, dental disease, pica or oral stereotypes, gastrointestinal parasitism, and abnormal hoof growth/laminitis are all additional clinical problems noted in captive giraffe. These problems may be additional risk factors for chronic wasting and peracute mortality syndrome, but very likely have their etiology in the same nutritional causes as the previous health problems. Identifying a common denominator for these problems is one of the cornerstones of medical diagnostic investigations. A central hypothesis is that dietary induced rumenitis and resulting changes in physiology are central to the disease syndromes seen in captive giraffe. It is hypothesized that diets high in pelleted feeds and low in physically effective (chewable) fiber are initiating these problems.

Retrospective Evaluation of Captive Giraffe Mortality (1988-2005)

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SeaWorld San Diego
San Diego, California

Co-authors: Laurie Bingaman Lackey, Ray Ball, Erin Harper, and Pam Dennis

Giraffe are popular exhibit species in AZA facilities. Improved management requires direction based on an understanding of the population challenges and causes of mortality. Many of these causes appear to be related to dietary management and nutritional issues.

A survey was circulated throughout the facilities holding giraffe. Studbook information was utilized to create a perspective for the survey results. Forty-four facilities submitted reports on circumstances and assessment of mortality on 196 giraffe. Studbook data for this time period demonstrated 670 reported mortalities indicating that 29% of mortality data was captured in the survey. These results were examined by dividing reports into three groups of interest: neonates, sub-adults, and adults. Neonates were defined as animals less than 4 months of age; sub-adult animals as 4 months to 3 years of age; adults were greater than 3 years of age.

Significant causes of neonatal mortality included infections, often with maternal rejection and/or failure of passive transfer, non-specific failure to thrive, trauma or fracture. A significant number of animals in this group included stillborn animals with no specific diagnosis. Other noted conditions included anemia, and rumenitis. The role of proper maternal condition at the time of delivery may be significant to mortality in this group, affecting the rate of stillbirth, ability to produce milk and proper transfer of colostral antibodies.

Subadult mortalities were commonly associated with trauma or nutritional wasting. One specific limitation of investigations in both subadults and adults was a definite nutritional assessment performed at necropsy based on objective findings. Other causes of death included colitis, hemorrhagic enteritis, anemia, and nutritional myopathy.

Adult mortalities were most often associated with or due to musculoskeletal conditions such as arthritis and overgrown hooves. The next most significant cause of mortality was wasting and emaciation. Less frequent concerns include gastrointestinal diseases such as rumenitis, bloat, rumen acidosis, and colitis, dental issues, renal uroliths or chronic renal disease, pregnancy complications, myocardial necrosis, pneumonia and trauma.

These findings support the proposal that many causes of giraffe mortality may have a nutritional management component. Further definition of some of the concerns is progressing

Giraffe Nutrition: Facts and Unknowns

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Saint Louis Zoo
Saint Louis, Missouri

Because no true species-specific nutrient requirement data are available for giraffe and other captive browsing ruminants, domesticated ruminants such as cattle are commonly used as models for ration formulation. This appears to be a logical starting point, given the wealth of information available on cattle and sheep. However, differences between giraffe and domestic ruminant species, such as management objectives, natural feeding strategies, and digestive anatomy and physiology must be borne in mind.

The objective of feeding the majority of domestic cattle is to produce maximum amounts of meat or milk at minimum cost over a period of a few months (feedlot calves) to a few years (dairy cows). In these short-term production situations, little consideration needs to be given to the long-term health effects of rich, rapidly fermented rations. Because intensive feeding practices maximize short-term production of the overall herd, some individual animal mortality from digestive upsets is acceptable from an economic standpoint. In a zoo setting, such mortalities are not acceptable, and rapid growth and excessive milk production are unnecessary. Rather, the objective of feeding captive giraffe is to enhance longevity and maintain optimum health over many decades.

While many details of giraffe digestive physiology are still unknown, the known anatomical differences between cattle and giraffe, and grazing and browsing ruminants in general, suggest that they process foods differently. Rumen microbes produce large amounts of organic acids that can be absorbed through the rumen wall and used as energy by the ruminant animal. However, if too much strong acid and/ or total acid is present at any one time, a harmful reduction in pH (ruminal acidosis) occurs. Ruminants are equipped with mechanisms designed to prevent this: 1. production of saliva, which dilutes and buffers the rumen contents; 2. removal of acid from the rumen via absorption through the rumen wall; 3. removal of acid via flow to the next section of the digestive tract.

Browsing ruminants (BR) have larger salivary glands as a percentage of body weight than grazing ruminants (GR), suggesting a capability for greater saliva production, and therefore more rumen buffering. While rumen volume is similar in GR and BR of similar body weight, dense, even papillation and larger papillae size give wild BR much more absorptive surface area in the rumen. This suggests that BR may be able to absorb nutrients, including organic acids, more rapidly than GR. Other features imply that BR do not retain food in the rumen as long as GR do. In comparison with GR, BR have less rumen compartmentalization, a larger omasal orifice, and decreased omasal laminae. The stratification of ruminal contents and formation of a fibrous mat, natural phenomena in GR, slows rate of passage. This stratification does not appear to occur in wild BR, which consume polygonal leaf particles rather than long needle-shaped grasses. Measurement of passage rate in six captive giraffe suggested that food moves rapidly through the total

digestive tract, but results of nutrient digestibility studies in giraffe, while variable, fall within ranges reported for domestic cattle. Smaller foregut and larger hindgut fermentation chambers suggest that BR have a greater reliance on hindgut fermentation, and that perhaps nutrients reaching the lower digestive tract play a larger role in meeting nutritional needs. Together, these features raise the question of whether wild giraffe are adapted to more rapid rates of nutrient fermentation, digestion and absorption than GR.

The giraffe's unique digestive anatomy may reflect an adaptation to their natural feeding strategy. It is well know that wild giraffe consume different plants than cattle and other GR. While many terms, including "browser" and "folivore", are used to describe giraffe, greater understanding of nutritional needs may come from examining the physical and chemical properties of actual plant portions consumed. Although many researchers have documented feeding behavior of wild giraffe, most research has focused on animal/ environment interactions, competition among animal species, selectivity and other behavioral aspects. Few attempts have been made to quantify nutrient intake. While the natural diets of giraffe appear to differ in chemical composition and physical form from those of GR, much remains unknown.

Trees and shrubs appear to make up the bulk of the diet of wild giraffe, with Acacia *spp*. as the most commonly reported dietary item. Giraffe also consume vines and herbs, and may feed from as many as 66 plant species in a given habitat. Grass consumption has been reported as either non-existent or negligible. The plant fractions consumed are often leaves and stems, but also include fruits, flowers and bark. Both species and faction consumption vary widely with geographic location and season. There is scattered information on nutrient composition of some plant fractions consumed by wild giraffe, but without knowledge of the proportional contribution of these fractions to the total diet, these data must be viewed with caution. The proportion of leaves, bark, stems, fruit and flowers in a wild giraffe's diet, and the nutrient content of these plant portions, varies considerably with season and location. Leaves from a single plant species will not reflect the nutrient content of the total diet.

Based on analyses of some plant species consumed by wild giraffe, early researchers proposed that wild giraffe consumed a high-protein low-fiber diet. However, the nutrient analysis technique in use at the time is now known to underestimate fiber and overestimate soluble carbohydrates (sugar, starch, and pectin) to a degree that cannot be determined. A more recent attempt to quantify average daily nutrient intake using improved analytical methods suggests that wild giraffe consume more fiber and less protein than previously believed. Protein levels in individual diet components are highly variable, and giraffe have shown preferential selection of both high and low protein plants. Estimated total crude protein intake ranges from 12-19% of dietary dry matter, only slightly higher than levels fed to domestic ruminants. Tannins in many of the plants eaten by giraffe bind with dietary protein, reducing the availability of protein consumed. At this time, the amount of available protein consumed by wild giraffe is unknown.

The majority of captive giraffe are fed a basal diet of alfalfa hay and low fiber grain-based concentrates. Actual intake reported thus far (11 animals) shows an average

consumption of 25% hay and 75% concentrates. This type of intake pattern has caused overly acidic rumen conditions in domesticated ruminants. Commercial grain-based concentrates generally contain high levels of sugars, which are associated with rapid acid production, and/ or starch, which is associated with production of strong acids. Fiber, which ferments slowly and can serve to dilute the acid in rumen contents, is low in most concentrates offered to giraffe.

Captive giraffe also may not be as well equipped to deal with ruminally produced acids as their wild counterparts. Grains and pellets require little time to consume and little chewing. While wild giraffe appear to spend approximately half their time feeding, six captive giraffe fed a grain/ alfalfa hay diet spent only 12% of time feeding. Since oral activity such as chewing stimulates the flow of saliva, ruminal buffering by saliva may be greater in wild giraffe than in captive. The large papillae and dense, even papillation found in the rumen of wild giraffe was not noted in two captive specimens; those rumens appeared more similar to cattle than wild giraffe. Wild giraffe reportedly have nine times the absorptive surface area than was found in the two captive giraffe. Initial examination of rumens from additional captive giraffe has similarly shown sparse, blunted papillae, suggesting a possible decreased capacity for rapid nutrient absorption.

While a comprehensive picture of giraffe nutrition continues to emerge, much remains unknown. Based on current data, there appear to be subtle but significant nutritional differences between giraffe and domestic ruminants, and between captive and wild giraffe. Further research into nutrient intake in a wild setting, specific nutrient requirements, and characteristics of digestive anatomy and physiology unique to browsing ruminants will enhance captive health and nutritional status of not only giraffe, but other browsing species as well.

X. Giraffe Necropsy Protocol

The following worksheet is designed as a tool to standardize all necropsies of giraffe and to help investigate peracute mortality and related conditions in captive giraffe. The investigators will try to make themselves available for examinations if feasible and may be able to accommodate histopathology if the worksheet is completed and submitted. Please contact Ray L Ball, DVM at Busch Gardens, 813-987-5562, Fax 813-987-5548 or e-mail at dr.ray.ball@anheuser-busch.com if you anticipate the death of a giraffe.

Gross Examination Worksheet

Institution/Owner

Address

Country

Species Id# ISIS# Studbook#

Birth Date/Age / Sex Weight (Kg) Height at withers Total height (Actual☐/Estimate☐)

Death Date Death Location

Necropsy Date Necropsy Location PM Interval

Captive Born ☐Wild Caught☐

History (include clinical signs, circumstances of death, clinical lab work, diet & housing, Medarks records are welcomed an encouraged)

GROSS EXAMINATION

(If no abnormalities are noted mark as normal or not examined (NE); use additional sheets if needed)

Photographs if possible of dorsal sacrum withers, lateral hips, shoulders, neck, and anterior and lateral neck. Photos of abdominal cavity, subcutaneous space, mesentery, and perirenal fat are also requested.

Please collect samples of each system in parenthesis.

Blood: attempt to collect blood for serum if possible. If death was witnessed then collect heparinized plasma and keep on ice until spun and frozen back.

General Exam (Physical and nutritional condition, skin, body orifices, superficial lymph nodes)

See attached body-scoring system and score the carcass. Attached photos as requested above.

Musculoskeletal System (Bones, marrow, joints, muscles)

Body Cavities (Fat stores, pleura, thymus, lymph nodes)

Spleen

Respiratory System (Nasal passages, pharynx, larynx, trachea, bronchi, lungs, regional lymph nodes)

Cardiovascular System (heart, pericardial sac, great vessels, myocardium, valves, chambers). If blood is available freeze serum and plasma.

Digestive System (mouth, teeth, tongue, salivary glands, rumen, reticulum, abomasums,omasum,esophagus, small intestine, cecum, large intestine, rectum, liver and gallbladder, pancreas(large section), mesenteric lymph nodes)

Liver 200g frozen☐

Rumen contents in sterile/clean urine cup(2), parafilm sealed☐

(See collection protocols)

See diagram for collection of rumen sections. 10cmX10cm square piece is adequate for counting of papillae and histopathology.

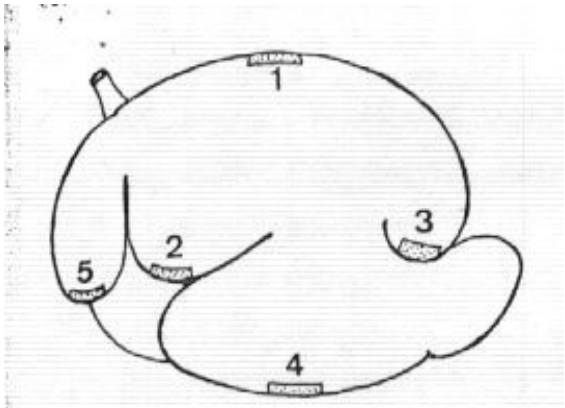

Fig. 1. Standardised sampling sites in the rumino-reticular test regions, left side. 1. rumen dorsal wall; 2. atrium ruminis; 3. saccus caecus caudodorsalis; 4. rumen ventral wall; 5. fundus reticuli (after Hofmann *et al.*, 1982).

Sampling sites from rumen.

Urinary System (kidneys, ureters, bladder, urethra). Freeze 20ml of urine for biochemical analysis.

Reproductive System (testes/ovaries, uterus & cervix, penis/vagina, urogenital canal, prostate, mammary gland, placenta)

Endocrine System (thyroids, parathyroids, adrenals, pituitary)

Central Nervous System (brain, meninges, spinal cord)

Sensory Organs (eyes, ears)

Additional Comments or Observations:

Prosector: Date:

Summarize Preliminary Diagnoses:

Laboratory Studies:

cytology☐

fluid analysis☐

urinalysis☐

serum chemistries☐

bacteriology☐

mycology☐

virology☐

parasitology☐

x-ray☐

photography☐

other☐

TISSUE CHECK LIST

Where possible freeze 3-5 cm blocks of tissue from lesions and major organs (e.g., lung, liver, kidney, spleen) in small plastic bags, preferably in liquid nitrogen to be kept ultrafozen at -70 degrees Celsius; freezing at conventional temperatures is acceptable if there is no access to an ultra-low freezer. Collect identical sets for histopathology in formalin.

Brain☐	Diaphragm ☐	Hemal node☐	Urethra ☐
Nerve (sciatic)☐	Liver ☐		Kidney ☐
Spinal cord☐	Mammary gland☐		Adrenal ☐
Eye☐	Spleen☐	Ovary/testis☐	Thymus ☐
Tongue☐	Pancreas☐	Epididymis☐	Rumen sections1-5 (see above diagram)
Esophagus☐	Stomach☐	Uterus/cervix ☐	Section 1☐
Trachea☐	Small intestine ☐	Vaginal/urogenital canal☐	Section 2☐
Thyroid☐	Large intestine☐	Penis☐	Section 3☐
Parathyroid☐	Cecum☐	Prostate☐	Section 4☐
Pituitary☐	Skin☐	Accessory sex organs ☐	Section 5☐
Heart/aorta☐	Bone with marrow☐	Ureter☐	
Muscle☐	Salivary gland ☐	Urinary bladder☐	
Lung☐	Lymph node☐		

Rumen Fluid collection:

The collection is the ventral sac of the rumen, but anywhere you can get fluid should be fine. Using a 14 - 16 gauge needle, 4- 6" long, pierce the abdomen and into the rumen. Use a sterile 10-20 ml syringe to aspirate the fluid. If the needle becomes clogged with ingesta, you can just force a small amount of air through the needle to clear it. Be careful not to create a negative pressure within the syringe, since that will let off CO2 and increase the pH. Aim for collecting 10-15 ml with a minimum of 5 ml. If possible, have a pH meter there and test pH immediately. (pH can change quickly after O2 exposure) Seal remaining fluid in airtight containers and freeze immediately. Ship frozen. Opening the rumen is another option but unless it is done shortly after death may alter the results. Please estimate the time from death to collection and the collection method used.