Diabetes Mellitus and Chronic Kidney Disease in a Cat

Case Summary:

This report describes the nutritional management of an obese diabetic cat with chronic kidney disease. A nutrition plan was initially instituted to improve glycemic control and achieve weight loss. When the patient had lost weight and become non-insulin dependent, dietary intervention to address the chronic kidney disease was implemented for long-term feeding. The cat remained clinically stable in diabetic remission for 7 months while consuming an appropriate for diet for renal disease.

** This case report was originally submitted by an ACVN candidate but has been edited and reformatted to meet the Instructions for the Writing and Evaluation of ACVN Case Reports. The example case reports are intended to serve as an illustration of the report instructions, format and a clinical presentation that had been found acceptable in the past. Editorial liberties were taken to complete the information essential to the report. Example reports should not be taken as an ACVN's endorsement of any specific nutritional approach or rationale.*

A 12 year old spayed female indoor-outdoor domestic shorthair cat with diabetes mellitus (DM) and International Renal Interest Society (IRIS) stage 2 chronic kidney disease (CKD) was referred by an internist for a nutrition consultation due to poor blood glucose control over the previous 2 months. The Internist diagnosed the cat with DM 4 months previously, had prescribed glargine[a] and the dose was 7 units (U) SC q12h (hour) at the time of the nutrition consult. The cat's DM was uncontrolled based on high blood glucose spot checks and fructosamine concentrations (Table #1; day 1 is Candidate's first involvement). The patient was polyuric and polydipsic due to glucosuria (Table #2; day 1). The cat was receiving a histamine-2 receptor blocker for gastroprotection (famotidine[b] 5 mg PO q24h) although there had been no recent episodes of vomiting or diarrhea. Three months previously, the cat had been treated by the same Internist for presumed pyelonephritis secondary to an *Enterococcus faecalis* infection (Tables #1; day 1).

The Candidate obtained the dietary history and was informed that the cat had been eating free choice a dry feline renal diet (Diet A[c]) prescribed by the Internist. The dietary recommendation (Diet A) was appropriate for the CKD but the diet and feeding method were not appropriate for a diabetic cat.[1] No other food or treats were offered and although the daily caloric intake was unknown due to free choice feeding from a self-feeder, body weight (BW) had been stable for the past four months despite the unregulated diabetes. Due to the cat's disposition and indoor-outdoor lifestyle, the owner requested other options to decrease or eliminate the need for daily insulin injections.

The candidate examined the cat, recorded a BW of 6.2 kg with a body condition score (BSC) of 8/9, mild muscle wasting and mild periodontal disease.[2,3] The estimated ideal BW for this cat was 4.6 kg based on female cats having approximately 48% body fat at a BCS of 8/9 (Fig #1).[2] The physical examination was unremarkable except that the right kidney was small and irregular; left kidney was normal and systolic blood pressure was 130 mm Hg. Aside from glucosuria and azotemia, the cat's hemogram, serum biochemistry profile, urinalysis, and thyroid results were unremarkable (Tables #1, #2, #3; day 1). Based on the results of the historical information, physical examination, and review of laboratory data, this patient's problem list

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[a] Lantus®, 100 U/ml, Sanofi-aventis, Bridgewater, NJ
[b] Pepcid AC®, 10mg/tablet, Johnson and Johnson/Merck, Fort Washington, PA
[c] Hill's Prescription Diet® k/d Feline dry, Hill's Pet Nutrition, Topeka, KS (Diet A)

included obesity, unregulated diabetes mellitus, and IRIS stage 2, substage normotensive, non-proteinuric CKD.[4]

The candidate discussed the dietary strategies for each problem with the owner and Internist. Obesity, a risk factor for diabetes mellitus, is associated with insulin resistance, which contributes to beta cell exhaustion.[5,6,7] The mechanisms relating obesity to insulin resistance are complex but include impaired insulin secretion, down-regulation or impaired binding affinity of insulin receptors and post-receptor glucose transport defects.[8] Before clinical evidence of glucose intolerance, obese cats have decreased expression of insulin-sensitive glucose transporter GLUT4 in muscle and fat tissue, and as adiposity increases, concentrations of insulin sensitizing adiponectin decrease; hence in obese cats, insulin-independent glucose removal is inefficient.[9,10,11] Weight loss, however, in obese cats improves insulin sensitivity; therefore, a diet plan to achieve a 1-2% loss of body weight per week (optimally) was recommended.[6,8,12]

Historically diabetic cats have been managed with high crude fiber diets (>10-15% dry matter (DM) basis) because fiber was said to slow gastric emptying, delay glucose absorption, modulate gut hormone release and increase insulin sensitivity.[13,14] One study evaluating diets in naturally occurring diabetes showed improved blood glucose concentrations but no difference in insulin requirement in cats fed high (12.6% DM) compared to low (1.8% DM) dietary fiber.[15] However, in this study, the low fiber diet was achieved by adding more cornstarch, and therefore, the beneficial results attributed to the high fiber diet might have been due to a low starch concentration.

More recent studies have documented the benefit of feeding a diet with <40% carbohydrate DM and protein > 45% DM to diabetic cats.[16,17,18,19] Felids are obligate carnivores that evolved on an animal organ and muscle (meat) diet and maintain blood glucose concentrations eating a high protein/low carbohydrate diet relative to canids.[20] Cats have low concentrations of hepatic glucokinase (high K_m for glucose) and high concentrations of hepatic hexokinase (low K_m for glucose) relative to omnivores and maintain blood glucose primarily through hepatic gluconeogenesis from amino acids.[21,22] These metabolic features are exploited by the ‘low carbohydrate / high protein’ feeding strategy for diabetic cats. Studies have demonstrated improved glycemic control, lower insulin requirements, improved insulin sensitivity, and higher diabetic remission rates in cats consuming relatively low carbohydrate diets than previously recommended.[15-18] The optimal nutrient composition for diabetic cats is unknown; however, diets that contain at least 45% metabolizable energy (ME) from protein and less than 20% ME from carbohydrate have been recommended (Table #4).[23] Currently it is unknown whether the beneficial effect is due primarily to the protein or the carbohydrate concentration in these diets. Also relevant to this case, obese cats fed 36% protein ME diet had increased fat loss and decreased lean body mass loss when compared with cats fed 46% protein ME diet.[10,24] This patient's current diet (Diet A) contained 34% carbohydrate ME and 23% protein ME, i.e., more carbohydrate than recommended for a diabetic cat (Table #4). Therefore, a dietary change that increased protein and decreased carbohydrate intakes, relative to Diet A, fed in appropriate amounts to facilitate weight loss was recommended. This recommendation addressed obesity related insulin resistance and was expected to improve glycemic control and reduce insulin requirements.

The non-proteinuric, normotensive, IRIS stage 2 CKD of this patient also had to be considered in the nutritional plan. Prospective studies in cats with naturally occurring CKD have shown feeding a 'renal' diet was associated with decreased azotemia and improved life span.[25,26,27] Characteristics of therapeutic renal diets in general include avoiding excessive phosphorus, protein and sodium, and increased omega-3 polyunsaturated fatty acids, potassium, and B vitamins relative to a feline maintenance diet. Renal diets often include ingredients such as potassium citrate to minimize dietary acid load since CKD cats are at risk of metabolic acidosis.[28] Diet A had a nutrient profile appropriate for CKD (Table #4) with a protein concentration that met AAFCO minimum protein requirement and NRC recommended allowance (5 $g/BW_{kg}^{0.67}/d$) (Table #5). However, if Diet A were to be fed in restricted amounts (-25% kcal/d) sufficient for weight loss, protein intake would not have been sufficient (3.75 $g/BW_{kg}^{0.67}/d$) to prevent protein catabolism and skeletal muscle loss.[29,30]

The prospective clinical studies in feline CKD have not evaluated the influence of individual nutrients on outcome. An early study in cats with experimentally induced CKD demonstrated changes in renal histopathology with higher protein and energy intake; however, this study was confounded by hypokalemia, which adversely affects renal function.[31,32] A later study examining the influence of protein versus energy on renal function found no effect of various concentrations of dietary protein on renal histopathology.[33] Therefore, the degree of protein restriction necessary in non-proteinuric feline CKD is not clear and still debated. However, supplying sufficient non-protein energy to spare protein catabolism, selecting protein sources with high biological value, appropriate amino acid profiles, and addressing the effect of protein on acidosis were considered.[27]

Restricting dietary phosphorous intake to mitigate a calcium imbalance due to CKD was important to consider in this cat. As glomerular filtration decreases, renal phosphate excretion decreases resulting in phosphate retention and the relatively low serum calcium concentrations stimulate parathyroid hormone (PTH) secretion and, if allowed to progress, this compensatory response becomes counterproductive and detrimental to the patient. Reducing dietary phosphorus intake reduces renal mineralization, controls renal secondary hyperparathyroidism (calcium imbalance) in naturally occurring CKD; therefore maintaining serum phosphorus between 2.5-4.5 mg/dL has been recommended for IRIS stage 2 CKD cats.[24,34,35] This patient's serum phosphorus (Table #1; day 1) was 5.8 mg/dL while consuming a diet restricted in phosphorus (1.1 g/Mcal). A phosphate binder had been previously recommended but declined by the owner due to difficulty in administration and palatability concerns. In commercially available diets, usually concurrent dietary protein restriction is necessary to achieve the recommended restricted concentrations of dietary phosphorus, consequently, a phosphorus restricted diet would be lower in dietary protein than desired to manage this patient's DM while a higher protein diet would be associated with a higher dietary phosphorus intake than is recommended for IRIS stage 2 CKD.[26]

A nutritional plan to address both DM and CKD posed a challenge in this patient. The patient was normotensive and normokalemic; BUN and creatinine were above normal but historically had been stable and there was no evidence of metabolic acidosis; however, serum phosphorus was higher than desired. Most guidelines for patients with concurrent CKD and DM favor dietary management of CKD with dietary phosphorus and protein reduction, although others do not advocate lower dietary protein until the renal disease is more advanced.[6,20,36] The risk of causing clinical signs of uremia and promoting further progression of renal disease by feeding a high protein and phosphorous diet was discussed with the owners and Internist.

It was agreed to transition the cat to Diet B[d] with higher protein and lower carbohydrate concentrations relative to Diet A while decreasing exogenous insulin administration and monitoring blood glucose.[37] Current caloric intake was unknown and so an initial conservative

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[d] Purina Veterinary Diets® DM Dietetic Management® Feline canned, Nestlé Purina PetCare Company, St Louis, MO (Diet B)

calculation utilizing the NRC feline adult maintenance metabolizable energy (MER) equation for overweight cats [130 x (6.2 kg)$^{0.4}$ =270 Kcal/day] was used and a food dosage of 2/3 can (105 g) of Diet B fed twice daily providing 259 Kcal/day was recommended.[30] This plan was considered on a trial basis to assess glycemic control and to monitor body weight at a known caloric intake given individual energy requirements can differ widely (±50%) from predictive equations and the concurrent diabetic state compounded the challenge of estimating this cat's energy requirement.[38] Future adjustments in feeding amounts could be made based on changes in body weight to achieve the desired rate of weight loss. The Internist opted to discontinue the famotidine and would adjust the glargine dosage based on clinical signs and blood glucose monitoring. Although an initial PTH concentration was not determined, serial monitoring of renal parameters was recommended, and a dietary change would be instituted if there were indications of CKD progression, i.e., worsening azotemia. The owner was instructed to monitor appetite, activity concentration, water intake and urine frequency and volume if possible when indoors. Clinical signs of hypoglycemia were reviewed and at home urine glucose monitoring was recommended but the owner declined.

One week later the cat was admitted for a 7-day medical board under the supervision of the Internist. At admission, the owner reported the cat preferred Diet B, had abruptly stopped eating Diet A within days, and had been consuming the recommended amount of Diet B and receiving 7U glargine SC q12h with no change in clinical status. The owner stated subjectively that no change in water consumption or urination had been noticed. The cat weighed 6.1 kg (BCS 8/9), and blood work on admission showed BUN concentration was higher as anticipated with higher protein diet but creatinine concentration was unchanged (Table #1; day 7). The first evening of boarding, the blood glucose nadir was 52 mg/dL although the cat was eating and not showing clinical signs of hypoglycemia. The next morning, the cat was bright, alert, and responsive with no change on physical examination, and a pre-insulin blood glucose was 198 mg/dL. Insulin was reduced to 6U SC q12h based on the nadir the previous evening per published guidelines.[6] The second day of boarding, the glucose nadir was 48 mg/dL and the cat was quiet, but alert and eating. Based on a pre-insulin blood glucose concentration of 88 mg/dL, and the previous evening nadir, glargine was decreased by 50% to 3U SC q12h on the third day of boarding.[6] On the fourth day the cat again received 3U q12h of glargine based on no clinical signs of hypo- or hyperglycemia, pre-insulin and nadir glucose concentrations. On the fifth day of hospitalization, the insulin was reduced to 2U SC q12h due to a glucose nadir of 60 mg/dL. The cat was continued on 2U SC glargine q12h while eating 2/3 can (105 g) of Diet B twice daily for the remaining boarding period. The cat's insulin requirement during the medical boarding period most likely decreased due to the dietary change.

Before the cat was discharged from the 1-week of medical board, renal parameters (BUN, creatinine) were assessed as stable (Table #1; day 14). Serum phosphorus had not increased on the higher phosphorus diet, but remained above the desired goal for a cat in stage 2 CKD. The owner was pleased with the decreased insulin requirement and Diet B was continued with 2U SC glargine q12h and a recheck visit was scheduled in one week to reassess the daily food dosage for weight loss.

The cat was not seen again until three weeks later and without consulting the Candidate or Internist, the owner had decreased the food dosage to ½ can (80 g) of Diet B twice daily for a caloric intake of 194 Kcal/day and reported a relative decrease in water intake and urination. The patient weighed 5.6 kg (BCS 7/9) with mild muscle wasting along the topline i.e., the dorsal spinal epaxial musculature. The cat therefore had lost 0.6 kg or approximately 9.7% of initial body weight (6.2 kg) over the first five weeks averaging 1.9%/week, which is a safe rate of weight loss.[24] Laboratory renal parameters (BUN, creatinine, and phosphorous) had declined (Table #1; week 5) relative to day 1 but phosphorous was still higher than 4.5 mg/dL. Based on a pre-insulin value of 108 mg/dL, insulin was discontinued to test for diabetic remission. The Candidate spoke with the owner by phone one week later who reported the cat was doing 'very well' at home with no increase in water intake or urination.

Six weeks after transition to the Diet B, the cat no longer required insulin. As Diet B contained 2.3 g phosphorus/Mcal and fed long-term to a cat with CKD could be problematic, a follow-up nutrition appointment was made. The Candidate discussed feeding a lower phosphorus (Diet C[e]) with 11.9%ME carbohydrate and 23.5%ME protein albeit lower than typically recommended for diabetics (Table #4). Reduction in dietary phosphorus and restricted concentrations of high biological value protein would be more appropriate for long-term feeding if glycemic control was not jeopardized. Diet C contained more fat (64.6%ME) than the two previous diets and was higher than recommended for weight loss (Tables #4 and 5). This might have been a problem since one study documented an elongated glucose clearance and reduced

[e] Royal Canin Veterinary Diet® Feline Renal LP pouch, Royal Canin, St. Charles, MO (Diet C)

acute insulin response to glucose administration in cats fed a high fat diet.[39] The owner agreed to try the new diet and successfully transitioned the cat to 1 pouch (85 g) of Diet C twice daily over 7 days to provide 240 Kcal/day[f], and continued to monitor appetite, food, water intake and urinations. Additionally, monitoring of blood or urine glucose, body weight, and evidence of protein catabolism on this lower protein intake was recommended.[1]

The cat remained normoglycemic based on weekly random blood glucose concentrations with the local veterinarian for the next month and the cat continued to do 'very well' at home per phone conversations between the Candidate, local veterinarian and owner. The creatinine was stable, BUN was lower on Diet C and serum phosphorous was within the recommended range of 2.5-4.5 mg/dL (Table #1; week 9 and 11). By week 11, the cat weighed 5.4 kg (BCS 6/9; no changes in muscle mass) as assessed by local veterinarian, and although still overweight, had lost a total of 0.8 kg since the initial consultation (averaging 1.1% per week overall) and remained in diabetic remission. Further caloric restriction would reduce protein intake and would accelerate skeletal protein catabolism, and given the cat did not require insulin and was doing well clinically, no dietary changes were recommended at that time.

This case demonstrates the nutritional management of an obese cat with concurrent diabetes mellitus and chronic kidney disease. The patient went into diabetic remission shortly after transitioning to a relatively 'high protein/low carbohydrate' diet and decreased body fat by approximately 10% in a 3-month span. To then address the long-term dietary management of the CKD, a lower protein/low carbohydrate and phosphorus restricted diet formulated for feline renal disease was instituted successfully. The cat remained in diabetic remission such that at a routine annual examination (7 months after initial consultation), weighed 5.5 Kg (BCS 6/9) and was clinically stable with a satisfactory fructosamine concentration (Tables #1, #2, #3; month 7). The dietary recommendation was to continue feeding Diet C as prescribed with similar monitoring advised every 3 months.

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[f] 7% fewer calories than calculated MER = 259 Kcal/d [= 130 x $(5.6 \text{ kg})^{0.4}$] at new BW to simplify daily feeding amounts.

References:

[1] Sparkes AH, Cannon M, Church D, et al. ISFM Consensus Guidelines on the Practical Management of a Mellitus in Cats. J Feline Med Surg 2015;17(3):235-250.

[2] Laflamme DP. Development and validation of a body condition score system for cats: A clinical tool. Feline Practice - Nutrition. 1997;25(5-6):13-18.

[3] World Small Animal Veterinary Association. Global Nutrition Committee. Nutrition Toolkit [Internet]. Canada: WSAVA; 2014. Muscle Condition Score Available from: www.wsava.org/sites/default/files/Muscle%20condition%20score%20chart-Cats.pdf

[4] International Renal Interest Society. IRIS Staging of CKD (modified 2015). Available from: http://www.iris-kidney.com/pdf/003-5559.001-iris-website-staging-of-ckd-pdf_220116-final.pdf#page=7

[5] Scarlett JM, Donaghue S. Associations between body condition and disease in cats. J Am Vet Med Assoc 1998;212:1725-1731.

[6] Rand JS, Marshall RD. Diabetes mellitus in cats. Vet Clin Small Anim. 2005;35:211-224.

[7] Gerich JE. Is reduced first-phase insulin release the earliest detectable abnormality in individuals destined to develop type 2 diabetes? Diabetes. 2002;51(supp)117-121.

[8] Biourge V, Nelson RW, Feldman EC, et al. Effect of weight gain and subsequent weight loss on glucose tolerance and insulin response in healthy cats. J Vet Intern Med. 1997;11:86-91.

[9] Brennan CL, Hoenig M, Ferguson DC. GLUT4 but not GLUT1 expression decreases early in the development of feline obesity. Domest Anim Endocrinol. 2004;26(4):291-301.

[10] Hoenig M. The cat as a model for human nutrition and disease. Curr Opin Clin Nutr Metab Care. 2006;9:584-588.

[11] Hoenig M, Thomaseth K, Brandao J, et al. Assessment and mathematical modeling of glucose turnover and insulin sensitivity in lean and obese cats. Domest Anim Endocrinol. 2006;31:373-389.

[12] Hoenig M, Thomaseth K, Waldron M, et al. Insulin sensitivity, fat distribution, and adipokine response to different diets in lean and obese cats before and after weight loss. Am J Physiol Regul Integr Comp Physiol. 2007;292: R227-R234.

[13] Zicker SC, Ford RB, Nelson, RW, et al. Small Animal Clinical Nutrition. 4th ed. Topeka, KS: Mark Morris Institute; c2000. Chapter 24, Endocrine and Lipid Disorders; p. 854.

[14] Nelson RW. Diabetes mellitus. In: Ettinger SJ, Feledman EC, eds. Textbook of veterinary internal medicine. 6th ed. St. Louis: Elsevier Saunders, 2005:1756-1785.

[15] Nelson RW, Scott-Moncrieff JC, Feldman EC, et al. Effect of dietary insoluble fiber on control of glycemia in cats with naturally acquired diabetes mellitus. J Am Vet Med Assoc. 2000;216:1082-1088.

[16] Mazzaferro E, Greco DS, Turner AS, et al. Treatment of feline diabetes mellitus using an alpha-glucosidase inhibitor (acarbose) and a high protein, low carbohydrate diet. J Feline Med Surg. 2003;5:183-190.

[17] Frank G, Anderson W, Pazak H, et al. Use of a high protein diet in the management of feline diabetes mellitus. Vet Ther. 2001;2:238-246.

[18] Bennett N, Greco DS, Peterson ME, et al. Comparison of a low carbohydrate vs. high fiber canned diet for the treatment of diabetes mellitus in cats. J Feline Med Surg. 2006;8:73-84.

[19] Zicker SC, Ford RB, Kirk CA, et al. Small Animal Clinical Nutrition. 5th ed. Topeka, KS: Mark Morris Institute; c2010. Chapter 29, Endocrine Disorders; p.569.

[20] Kirk C, Debraekeleer J, Armstrong PJ. Small Animal Clinical Nutrition. 4th ed. Topeka, KS: Mark Morris Institute; c2000. Chapter 11, Normal cats; p291-347.

[21] National Research Council. Ad hoc Committee on Dog and Cat Nutrition. National Research Council Nutrient Requirements of Dogs and Cats. Washington DC: The National Academies Press; c2006. Chapter 4, Carbohydrate and Fiber; p.49-80.

[22] Morris JG. Idiosyncratic nutrient requirements of cats appear to be diet-induced evolutionary adaptations. Nutr Res Rev. 2002;15:153-168.

[23] Lutz, TA. Encyclopedia of feline clinical nutrition. Bogota: Aniwa SAS. C2008. Chapter 5, Feline diabetes mellitus: nutritional strategies.p.181-221.

[24] Laflamme DP, Hannah SS. Increased dietary protein promotes fat loss and reduces loss of lean body mass during weight loss in cats. Intern J Appl Res Vet Med. 2005;3(2):62-68.

[25] Barber PJ, Rawlings JM, Markwell PJ, et al. Effect of dietary phosphate restriction on renal secondary hyperparathyroidism in the cat. J Small Anim Pract. 1999; 40:62-70.

[26] Elliott J, Rawlings JM, Markwell PJ et al. Survival of cats with naturally occurring renal failure: effect of dietary management. J Small Anim Pract. 2000;41:235-242.

[27] Ross SJ, Osborne CA, Kirk CA, et al. Clinical evaluation of dietary modification for treatment of spontaneous chronic kidney disease in cats. J Am Vet Med Assoc. 2006;229:949-957.

[28] Burkholder WJ. Dietary considerations for dogs and cats with renal disease. J Am Vet Med Assoc. 2000;216:1730-1734.

[29] Association of American Feed Control Officials Incorporated. AAFCO Official Publication. c2009. Chapter 4, Model Regulations for Pet food and Specialty Pet food Under the Model Bill: p.150-151.

[30] National Research Council. Ad hoc Committee on Dog and Cat Nutrition. National Research Council Nutrient Requirements of Dogs and Cats. Washington DC: The National Academies Press; c2006. Table 15-12, Nutrient Requirements for Adult Cats for Maintenance; p.366-367.

[31] Adams LG, Polzin DJ, Osborne CA, et al. Effects of dietary protein and calorie restriction in clinically normal cats and cats with surgically induced chronic renal failure. Am J Vet Res. 1993;54:1653-1662.

[32] Dow SW, Fettman MJ, LeCouteur RA, et al. Potassium depletion in cats: renal and dietary influences. J Am Vet Med Assoc. 1987;191:1569-1575.

[33] Finco DR, Brown SA, Brown CA, et al. Protein and calorie effects on progression of induced chronic renal failure in cats. Am J Vet Res. 59:575-582.

[34] Ross LA, Finco DR, Crowell WA, et al. Effect of dietary phosphorus restriction on the kidneys of cats with reduced renal mass. Am J Vet Res. 1982;43: 1023-1026.

[35] Elliott J, Elliott, D. Encyclopedia of feline clinical nutrition. Bogota: Aniwa SAS. C2008. Chapter 7, Dietary therapy for feline chronic kidney disease. p. 250-281.

[36] Greco DS. Dietary treatment of diabetes mellitus in dogs and cats. In: Nestlé Purina Veterinary Symposium. Companion Animal Medicine. 2009. June 1-2. p. 3-10.

[37] Nestlé Purina. Veterinary Diets web site. Nestlé Purina Veterinary Diets® DM Dietetic Management® Feeding Guidelines [Internet]. Nestlé Purina PetCare Company. Cited 2009 May 16. Available from: http://www.purinavets.com/Product/DMDieteticManagementFelineFormula.aspx.

[38] Thatcher CD, Hand MS, Remillard RL. Small Animal Clinical Nutrition. 5th ed. Topeka, KS: Mark Morris Institute; c2010. Chapter 1, Small Animal Clinical Nutrition: An iterative Process; p. 17.

[39] Thiess S, Becskei C, Tomas, et al. Effects of high carbohydrate and high fat diet on plasma metabolite concentrations and on IV glucose tolerance in intact and neutered male cats. J Feline Med Surg. 2004;6:207-218.

Table #1: Serum Biochemistry Profiles*†

Parameter measured (units)	Normal Range	Day 1	Day 7	Day 14	Week 5	Week 9	Week 11	Month 7
Glucose (mg/dL)	64-170	**448**	**178**	**174**	108	137	161	152
BUN (mg/dL)	14-36	**44**	**50**	**37**	31	28	24	23
Creatinine (mg/dL)	0.6-2.4	**2.8**	**2.8**	2.3	2.4	2.3	2.2	2.3
Total protein (g/dL)	5.2-8.8	7.5				7.2	7.4	7.3
Albumin (g/dL)	2.5-3.9	3.3				3.3	3.2	3.0
Total bilirubin (mg/dL)	0.1-0.4	0.4						0.2
Alkaline Phosphatase (U/L)	6-102	46						36
Aspartate aminotransferase (U/L)	10-100	32						20
Cholesterol (mg/dL)	75-220	216						206
Calcium (mg/dL)	8.2-10.8	9.7				9.9	10.0	9.5
Phosphorus (mg/dL)	2.4-8.2	5.8		5.1	4.8	3.7	4.1	3.8
Sodium (mEq/L)	145-158	157				150	148	148
Potassium (mEq/L	3.4-5.6	5.6				4.2	4.4	4.9
Chloride (mEq/L)	104-128	118				114	115	113
Globulin (g/dL)	2.3-5.3	4.2						4.3
Fructosamine (umol/L)	‡	**691**						316
T4 (ug/dL)	0.8-4.0	2.1						1.2

* Bolded values are outside normal reference range
† Blank = test not done
‡ Fructosamine scale: <500 = good; 500-614 = fair; >614 = poor regulation

<u>Figure #1. Estimation of ideal body weight</u>

- Female cats with a BCS of 8/9 contained 48% body fat[2]
- BW = 6.2 x 0.48 = 2.98 kg of body fat and 6.2 kg - 2.98 fat = 3.22 kg lean body mass
- 3.22 kg lean body mass / 0.7 lean body mass at BCS 5/9 = 4.6 kg ideal body weight

Table #2: Urinalysis Result

Parameter	Reference Range	Day 1	Month 7
Source		Cysto	Cysto
Color	Yellow	Yellow	Yellow
Appearance	Clear	Clear	Clear
Specific gravity	1.015-1.060	1.016	1.018
PH	5.5-7.0	6.0	6.0
Protein	Neg	Neg	Neg
Glucose	Neg	3+	Neg
Ketone	Neg	Neg	Neg
Bilirubin	Neg	Neg	Neg
Blood	Neg	Neg	2+
Casts/LPF	0-3	None	None
WBC/HPF	0-3	0-3	0-3
Epithelial Cells/HPF	None-Few	Rare	Rare
RBC/HPF	0-3	0-3	0-3
Crystals/HPF	None-Few	None	None
Bacteria/HPF	None	None	None
Urine Culture	No growth	No growth	No growth

Table #3: Hemogram

Complete Blood Count (units)	Reference Range	Day 1	Month 7
HCT (%)	29-48	34.2	34.3
RBC ($x10^6$/uL)	5.92-9.93	7.52	8.26
Hemoglobin (g/dL)	9.3-15.9	11.5	12.1
MCV (fl)	37-61	45	42
MCH (pg)	11-21	15.3	14.6
MCHC (g/dL)	30-38	33.6	35.3
WBC (#/uL)	3500-16000	13900	15700
Band Neutrophils (#/uL)	0-150	0	0
Segmented Neutrophils (#/uL)	2500-8500	5977	7065
Lymphocytes (#/uL)	1200-8000	6533	7850
Monocytes (#/uL)	0-600	556	471
Eosinophils (#/uL)	0-1000	834	314
Basophils (#/uL)	0-150	0	0
Platelets ($x10^3$/uL)	200-500 (adequate)	Clump (adequate)	211

Table #4: Recommended Nutrient Profiles for Felines with Diabetes, Obesity or kidney Disease compared with commercial products fed in this case*

Nutrient	Diabetes	Weight Loss	Renal Disease	Diet A†	Diet B‡	Diet C$
% ME from:						
Protein	35-60	35-63	21-28	23.0	46.3	23.5
Fat	25-60	21-42		43.5	47.1	64.6
Carbohydrate	0-18			33.5	6.6	11.9
Phosphorous (g/Mcal)			0.8-1.2	1.1	2.6	0.9

* Adapted from: Nestle Purina PetCare Handbook of Canine and Feline Clinical Nutrition. Wilmington: Gloyd Group DE, c2010. pp 30, 36, 86. Blank = no recommendation

† Diet A: Hill's Prescription Diet® k/d Feline dry, Hill's Pet Nutrition, Topeka, KS 2010 product guide

‡ Diet B: Purina Veterinary Diets® DM Dietetic Management Feline canned, Nestlé Purina PetCare Company, St Louis, MO 2010 product guide

$ Diet C: Royal Canin Veterinary Diet® Feline Renal LP pouch, Royal Canin, St. Charles, MO 2010 product guide

Table #5: Comparison of Commercial Products to AAFCO and NRC Recommendations

Nutrient per Mcal	AAFCO Min[29]	NRC RA[30]	Diet A*	Diet B†	Diet C‡
Crude protein (g)	65	50	68	119.1	61.5
Total fat (g)	22.5	22.5	53.0	49.9	75.2
Carbohydrate (g)	NR$	NR	99.0	16.9	31.1
Crude Fiber (g)	NR	NR	4.0	7.6	1.8
Calcium (g)	1.5	0.7	1.7	2.4	2.1
Phosphorus (g)	1.25	0.64	1.09	2.30	1.01
Potassium (g)	1.5	1.3	1.8	1.7	2.0
Sodium (g)	0.50	0.17	0.56	0.80	0.80

* Diet A: Hill's Prescription Diet® k/d Feline dry, Hill's Pet Nutrition, Topeka, KS 2010 product guide

† Diet B: Purina Veterinary Diets® DM Dietetic Management Feline canned, Nestlé Purina PetCare Company, St Louis, MO 2010 product guide

‡ Diet C: Royal Canin Veterinary Diet® Feline Renal LP pouch, Royal Canin, St. Charles, MO 2010 product guide

$ No Requirement