Revista de Educação Continuada em Medicina Veterinária e Zootecnia do CRMV-SP
Journal of Continuing Education in Veterinary Medicine and Animal Science of CRMV-SP
mv&z
ISSN 2596-1306 Versão on-line

FROM HUNTING TO THE BOWL: HOW NUTRITIONAL EVOLUTION INFLUENCES DIABETES IN CATS

Da caça à tigela: como a evolução nutricional e o manejo clínico influenciam a diabetes em gatos

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Cite as: CAMPOS, I. E. *et al*. From hunting to the bowl: how nutritional evolution influences diabetes in cats. **Journal of Continuing Education in Veterinary Medicine and Animal Science of CRMV-SP**, São Paulo, v. 24, esp.1, felinos, e38853, 2026. DOI: https://doi.org/10.36440/recmvz.v24.38853.

Como citar: CAMPOS, I. E. *et al*. Da caça à tigela: como a evolução nutricional e o manejo clínico influenciam a diabetes em gatos. **Revista de Educação Continuada em Medicina Veterinária e Zootecnia do CRMV-SP**, São Paulo, v. 24, esp.1, felinos, e38853, 2026. DOI: https://doi.org/10.36440/recmvz.v24.38853.

Article submitted to the similarity system
iThenticate

Abstract

Feline diabetes mellitus is a common endocrinopathy, with increasing prevalence linked to obesity. Environmental and behavioral factors such as sedentary lifestyle, indoor confinement, and energy-dense diets, combined with the species' anatomical and physiological traits, contribute to insulin resistance and hinder remission. This review examines how feline nutritional evolution and feeding behavior affect disease management, highlighting natural habits, unique carbohydrate metabolism, neutering effects, energy density, and food acceptance. It integrates pathophysiological mechanisms and evidence on diet types and specific nutrients, proposing multimodal strategies directed at remission. Understanding species-specific characteristics and applying them to nutritional and environmental management are essential to prevent treatment from being limited to glycemic control alone.

Keywords: Diabetes. Obesity. Cats. Nutrition. Behavior.

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Resumo

A diabetes mellitus é uma endocrinopatia frequente em felinos, com prevalência crescente associada ao aumento da obesidade. Fatores ambientais e comportamentais, como sedentarismo, confinamento e dietas altamente energéticas, aliados a particularidades anatômicas e fisiológicas da espécie, contribuem para resistência insulínica e dificultam a remissão. Esta revisão aborda como a evolução nutricional e o comportamento alimentar dos gatos influenciam o manejo da doença, destacando hábitos naturais, metabolismo peculiar de carboidratos, efeito da castração, densidade energética e aceitação alimentar. Integra mecanismos fisiopatológicos, evidências sobre tipos de dieta e nutrientes específicos, propondo estratégias multimodais voltadas à remissão. A compreensão das especificidades da espécie e sua aplicação prática no manejo nutricional e ambiental são fundamentais para evitar que o tratamento se restrinja ao controle glicêmico.

Palavras-chave: Diabetes. Obesidade. Felinos. Comportamento. Nutrição.

Introduction

Diabetes mellitus is one of the most prevalent endocrinopathies in felines, with incidence increasing proportionally to the rising prevalence of obesity in this species (Öhlund *et al*., 2015). Although its etiopathogenesis has not been fully elucidated, most cases are associated with excess body weight and physical inactivity, closely resembling the pathophysiological model of type 2 diabetes mellitus in humans, in which insulin resistance represents the central mechanism of disease development (Gilor *et al*., 2016; Sparkes *et al*., 2015).

Obesity and overweight constitute the main risk factors for the disease in cats. Genetic aspects, such as polymorphisms in the melanocortin 4 receptor gene, may predispose certain individuals; however, environmental and behavioral factors—including sedentary lifestyle, confinement, and feeding habits—also play a significant role in its development (Chandler *et al*., 2017).

Methodology

This narrative review was conducted between July and October 2025 with the objective of compiling recent evidence on the nutritional, metabolic, and therapeutic aspects of feline diabetes mellitus. Searches were performed in the PubMed, Scopus, and Web of Science databases using the descriptors *feline diabetes mellitus*, *feline obesity*, *SGLT2 inhibitors*, *GLP-1 agonists*, *microbiota*, and *nutrigenomics*.

Original articles, systematic reviews, and clinical guidelines published between 2021 and 2025 in English were included, with emphasis on pathophysiology, nutritional management, and emerging therapies in felines. Studies based exclusively on human or other animal models were considered only when clear translational applicability was evident. References were selected based on scientific relevance, timeliness, and methodological quality, prioritizing peer-reviewed and indexed studies.

Biology and Nutritional Evolution

The domestic cat (*Felis catus*) is an obligate carnivore, inheriting a dietary pattern based on the consumption of prey rich in protein, moderate in lipids, and low in carbohydrates. Growing kittens may require up to five times more protein than other species, and even in adulthood cats maintain a high protein requirement due to elevated nitrogen demands in metabolic pathways (Plantinga; Bosch; Hendriks, 2011).

From an anatomical and physiological perspective, the feline gastrointestinal tract exhibits features adapted to a carnivorous diet. The stomach has a smaller volumetric capacity compared with that of dogs, and the total digestive tract content is proportionally reduced, limiting the ingestion of large meal volumes and favoring fractional feeding throughout the day. The small intestine is relatively short, resulting in reduced transit time and absorption, while the large intestine has a small, minimally functional cecum with limited fermentative capacity compared with omnivorous species. Although the feline microbiota is capable of producing short-chain fatty acids, both the profile and magnitude differ from those observed in dogs, with a lower contribution of fermentative digestion (Verbrugghe; Hesta, 2017).

Behaviorally, cats retain patterns shaped by their evolution as solitary predators. Unlike dogs, which hunt in groups and share prey, felines hunt individually and consume small prey items of approximately 30 kcal each, in multiple daily feeding events, often ranging from 8 to 16 meals when free to choose. In natural environments, this behavior entails high energy expenditure related to hunting and movement, in contrast to the reality of confined and sedentary domestic cats. In domestic settings, however, feeding is often limited to a few meals per day or provided ad libitum, which deviates markedly from the natural feeding pattern (Delgado; Dantas, 2020).

The absence of feeding competition in nature also explains cats' low tolerance for the proximity of other individuals during meals. In multi-cat households, concentrated feeding resources may generate stress, anxiety, and conflicts (Crowell-Davis; Curtis; Knowles, 2004; Gourkow; Fraser, 2006). Therefore, strategies such as environmental enrichment are recommended to simulate hunting behavior, promote energy expenditure, assist in weight control, and improve overall welfare (Delgado; Dantas, 2020).

Another relevant trait is the expression of neophobia and neophilia—aversion or attraction to novel foods—modulated by prenatal and lactational feeding experiences, which influence dietary preferences in adulthood (Hepper *et al*., 2012). Palatability is determined by the interaction of aroma, texture, temperature, and chemical composition, and changes in these parameters may induce acceptance or rejection. Monotonous diets early in life tend to restrict dietary repertoire, whereas varied exposure promotes greater adaptability. From an evolutionary standpoint, neophobia may have functioned as protection against the ingestion of potentially toxic foods, while neophilia favored the exploration of new resources. This understanding is particularly relevant in diabetic cats, for whom acceptance of specific therapeutic diets is essential; food refusal may compromise glycemic control and preclude remission, making gradual dietary transitions and appropriate environmental management strongly recommended (Watson *et al*., 2023).

Metabolism and Dietary Implications

Carbohydrate metabolism in felines presents enzymatic particularities resulting from their evolution as obligate carnivores. Digestion begins in the intestine, as the species lacks salivary amylase; pancreatic amylase activity is relatively low, and the capacity to induce disaccharidase synthesis in response to high-carbohydrate diets is limited (Kienzle, 1993). Intestinal glucose absorption via the sodium–glucose cotransporter 1 (SGLT1) is slower than in dogs, possibly due to low expression of regulatory subunits (Mori *et al*., 2016; Batchelor *et al*., 2011).

Hepatic glycolysis in cats is characterized by the absence of glucokinase, a key enzyme for the rapid handling of large glycemic loads (Verbrugghe; Hesta, 2017), and glycogenesis is limited due to low glycogen synthase activity. Nevertheless, muscle glycogen stores are comparable to those of other species, owing to the high gluconeogenic capacity from amino acids (Hoenig *et al*., 2011).

Despite these physiological limitations, cats efficiently digest and metabolize carbohydrates present in commercial dry diets (Baldwin *et al*., 2010; Karr-Lilienthal *et al*., 2002). Coradini *et al*. (2011) observed that cats fed higher-carbohydrate diets gained less weight than those fed high-

protein diets, possibly due to greater caloric intake in the high-protein group. This finding demonstrates that carbohydrates alone cannot be held responsible for feline obesity; excessive intake of any macronutrient—carbohydrates, proteins, or lipids—may lead to weight gain. In this context, dietary energy density is a determining factor, with fat being the most energy-dense macronutrient and thus having the greatest potential to contribute to a positive energy balance.

Zhang *et al*. (2023) reported that nutrient digestibility and postprandial glycemic response vary according to the source of carbohydrates, highlighting that appropriately processed starches can be efficiently utilized by cats without compromising glycemic control. These results support the notion that ingredient quality and processing are more relevant determinants than the absolute amount of carbohydrates consumed.

Neutering is another relevant factor in energy metabolism, as it promotes increased food intake and, more importantly, a reduction in basal metabolic rate and spontaneous physical activity, thereby decreasing daily caloric requirements. An energy surplus of only 10 kcal/day may result in the accumulation of approximately 450 g of body fat per year, equivalent to about 10% of the ideal body weight in some individuals. These changes increase the risk of obesity and raise the likelihood of developing diabetes mellitus by two- to ninefold, in addition to reducing insulin sensitivity (Vendramini *et al*., 2020).

Pathophysiology and Nutrigenomics of Obesity and Diabetes

Obesity in felines is associated with metabolic alterations that favor insulin resistance and the development of diabetes mellitus. The particularities of feline carbohydrate metabolism were reinforced by Hoenig *et al*. (2011), who evaluated the impact of different macronutrients, as well as age and obesity, on postprandial glycemic response in cats. The authors demonstrated that obese animals exhibited greater glucose intolerance and reduced insulin sensitivity, especially within the 24 hours following a meal, compared with lean cats.

Excess adipose tissue promotes increased mobilization of free fatty acids and accumulation of lipid intermediates, such as diacylglycerols and ceramides, which interfere with insulin signaling pathways, characterizing the phenomenon of lipotoxicity (Brito-Casillas; Melián; Wägner, 2016).

Compensatory hyperinsulinemia stimulates the secretion of amylin by pancreatic beta cells. The accumulation of this protein may be associated with the formation of amyloid deposits in the pancreatic islets, a change frequently observed in diabetic cats (Yano; Hayden; Johnson, 1981). Although islet amyloidosis contributes to beta-cell dysfunction, it is not considered the sole etiopathogenic factor.

Adipocyte hypertrophy leads to tissue hypoxia, cellular apoptosis, and macrophage activation, resulting in a chronic low-grade inflammatory state. This process is mediated by the release of pro-inflammatory cytokines, such as tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6), which amplify insulin resistance. Conversely, the secretion of adiponectin—an insulin-sensitizing hormone produced by adipose tissue—is reduced in obese cats (Takashima *et al*., 2015).

Sustained insulin resistance, combined with persistent hyperglycemia, induces glucotoxicity, which impairs pancreatic beta-cell survival and function. The combination of lipotoxicity, glucotoxicity, and inflammation culminates in progressive beta-cell failure and the establishment of diabetes mellitus (Poitout; Robertson, 2008).

The molecular mechanisms linking obesity to reduced insulin sensitivity in cats are not yet fully understood. Among genes related to energy balance, the melanocortin 4 receptor (MC4R) plays a central role in the regulation of appetite and energy expenditure. A study conducted in Turkey identified six genetic variations in the MC4R gene in obese and non-obese cats, four of which were

novel, with a significant association observed between one of these variants and obesity. In addition, non-synonymous mutations—alterations that modify the amino acid sequence of the protein and may affect its function—were identified and correlated with feline body mass index (fBMI), suggesting that genetic variations in MC4R may influence susceptibility to obesity in cats (Mousa Basha; Akis, 2025).

In an investigation involving 54 cats (lean, overweight, and diabetic), gene and protein expression related to insulin and incretin signaling in the pancreas, liver, and skeletal muscle were analyzed. Diabetic cats exhibited hepatic and muscular fat accumulation, reduced expression of insulin, insulin receptors, and glucose transporters (GLUT), as well as alterations in key proteins of the insulin signaling pathway (IRS, Akt, PI3K). Treated diabetic cats showed partial improvement in incretin signaling (GLP-1 and GIP). These findings indicate that feline diabetes involves dysfunctions in insulin synthesis and signaling and ectopic lipid deposition, reflecting mechanisms similar to those observed in human type 2 diabetes (Patra *et al*., 2024).

The expression of genes related to obesity, glucose metabolism, and inflammation was investigated in 73 healthy, neutered domestic shorthair cats with varying body condition scores. A significant reduction in adiponectin expression was observed in the adipose tissue of obese cats, along with increased muscular expression of PPAR-γ2, a regulator of adipocyte differentiation and lipid metabolism. Increased expression of PAI-1, another protein associated with inflammation and insulin resistance, was also identified. No significant changes were detected in inflammatory cytokines. Further investigations are needed to evaluate the hypothesis that, as in humans, low-grade tissue inflammation plays an important role in feline obesity (Stenberg *et al*., 2023).

In addition, the interaction between the intestinal microbiota and lipid metabolism has been identified as a modulatory factor in insulin resistance. Changes in microbial composition may influence lipid absorption and storage, as well as regulate hepatic metabolic pathways associated with inflammation and glycemic homeostasis (Li *et al*., 2024). This bidirectional relationship suggests that modulation of the gut microbiota may represent a promising therapeutic target in the prevention and management of feline obesity and diabetes.

Therapeutic Management for Remission

Remission of feline diabetes mellitus is one of the primary therapeutic goals and is achieved more frequently when glycemic control is established early and maintained consistently (Roomp; Rand, 2009). Insulin therapy should be initiated as soon as possible, as the rapid reduction of glucotoxicity preserves residual pancreatic beta-cell function and favors disease reversal (Sparkes *et al*., 2015).

In recent years, insulinization protocols have become increasingly individualized, taking into account the cat's behavioral profile, feeding habits, and the owner's routine. This personalization is essential to ensure adherence and owner satisfaction, factors that directly influence therapeutic success and long-term treatment maintenance (Sparkes *et al*., 2015). Despite this individualization, management strategies must always be aligned with international consensus guidelines, such as those issued by the International Society of Feline Medicine and the American Animal Hospital Association, which provide evidence-based recommendations for dose adjustment, glycemic monitoring, and the definition of therapeutic targets.

The role of diet in glycemic control in insulin-treated diabetic cats was demonstrated by Hall *et al*. (2009), who evaluated the effects of different dietary formulations in animals receiving twice-daily glargine insulin. The authors observed that diet composition directly influences glycemic stability.

Feline obesity increases the risk of comorbidities such as hepatic lipidosis, diabetes mellitus, and urinary tract disease, and weight-control diets may assist in reducing body fat and improving metabolic status. In a study involving 24 obese adult cats, dietary restriction using either dry or wet diets resulted in significant reductions in fat mass, leptin, and insulin, along with increases in superoxide

dismutase and active ghrelin. These changes indicate improved insulin sensitivity, reduced oxidative stress, and more efficient regulation of appetite and energy metabolism, representing favorable effects on metabolic balance during weight loss. Modifications in the fecal microbiota were also observed, with greater bacterial diversity and changes in short-chain fatty acid profiles, indicating that diet type and formulation influence both metabolic and microbial responses to weight loss in obese cats (Opetz *et al*., 2024).

Accordingly, nutritional management should prioritize diets formulated to meet the physiological requirements of the species, with reduced energy density and meal fractionation. The goal is to promote gradual weight loss, between 0.5% and 1% of body weight per week, while preserving lean body mass (Zoran; Rand, 2013; Laflamme, 2020). Diets with low carbohydrate content (less than 15% of metabolizable energy) and high protein content (approximately 40% of metabolizable energy) reduce the need for exogenous insulin and are associated with higher remission rates (Bennett *et al*., 2006; Mazzaferro *et al*., 2003).

The inclusion of wet diets in the management of diabetic cats represents an effective approach to reducing caloric intake and assisting with weight control, a key factor in restoring insulin sensitivity and promoting disease remission. Experimental studies have shown that increasing the water content of feline diets reduces energy density and promotes lower spontaneous energy intake, resulting in weight loss or maintenance without adverse effects on tissue composition (Wei *et al*., 2011; Alexander; Colyer; Morris, 2014). Additionally, the addition of water to calorie-restricted diets further enhanced body mass reduction in cats, reinforcing the relevance of energy dilution as a practical nutritional management strategy (Cameron *et al*., 2011). Thus, the provision of wet diets, generally lower in caloric density and formulated to meet species-specific requirements, represents a valuable component of the multimodal treatment of feline diabetes mellitus.

More recently, sodium–glucose cotransporter 2 (SGLT-2) inhibitors have been investigated as an alternative or adjunct to insulin therapy, particularly in newly diagnosed and clinically stable cats. These drugs act by reducing renal glucose reabsorption, thereby promoting glycemic control independently of insulin secretion. Cook and Behrend (2025) and Romero-Vélez, Rejas, and Ruiz de Gopegui (2025) demonstrated that SGLT-2 inhibitors, such as bexagliflozin and velagliflozin, can be highly effective, with outcomes comparable to those achieved with insulin, providing a viable oral therapeutic option for the management of feline diabetes. However, their use requires rigorous monitoring and careful patient selection due to the risk of euglycemic diabetic ketoacidosis (eDKA), a potentially serious complication associated with this class of drugs.

Glucagon-like peptide-1 (GLP-1) receptor agonists and glucose-dependent insulinotropic polypeptide (GIP) agonists, as well as dipeptidyl peptidase-4 (DPP-4) inhibitors, are widely used in the treatment of type 2 diabetes mellitus in humans. These therapies are based on the action of incretin hormones, which stimulate glucose-dependent insulin secretion after nutrient intake and exhibit a more favorable safety profile than exogenous insulin (Haller; Lutz, 2024). Among drugs in this class, exenatide, a GLP-1 receptor agonist, has been investigated in felines for both its metabolic effects and its potential clinical application.

Haller and Lutz (2024) report that GLP-1 agonists represent one of the most promising fronts in modern feline endocrinology, as they combine hypoglycemic effects with potential weight loss and appetite reduction, in addition to improving pancreatic beta-cell function in experimental models.

The subcutaneous implant OKV-119, originally developed for human use, was experimentally adapted for prolonged exenatide release in obese, non-diabetic cats. The results demonstrated sustained drug release for more than 84 days, with reduced food intake and weight loss exceeding 5% in four of the five evaluated animals, suggesting potential for feline obesity control (Klotsman; Anderson; Gilor, 2024). Gilor *et al*. (2025) investigated 22 cats in recent diabetic remission and administered monthly long-acting exenatide (0.13 mg/kg) for up to two years. The authors found no increase in remission

duration; however, treatment contributed to the maintenance of glycemic control and stability of glycated hemoglobin, indicating metabolic benefits without prolonging diabetic remission.

The influence of lifestyle on the risk of developing diabetes mellitus in cats was highlighted by Slingerland *et al*. (2009), who investigated disease-associated factors in domestic felines. The authors observed that confinement to indoor environments and the resulting physical inactivity exert a greater impact on the development of type 2 diabetes than the proportion of dry food in the diet. Strategies that promote energy expenditure, such as environmental enrichment and encouragement of physical activity, as well as consideration of the animal's living environment in preventive and therapeutic management, should therefore be taken into account. Thus, in addition to nutritional interventions, lifestyle modification represents a fundamental component in reducing risk and controlling disease in predisposed cats.

Despite recent advances, the available body of evidence still presents important methodological limitations. Most studies are based on small sample sizes, short follow-up periods, and lack of standardization of the diets evaluated, which hinders direct comparisons. Furthermore, data on microbiota and nutrigenomics in cats remain scarce and are largely extrapolated from human or canine research. Consequently, new controlled, long-term studies are required to consolidate the role of emerging therapies, such as GLP-1 agonists and SGLT-2 inhibitors, and to fully elucidate the interactions among nutrition, microbiota, and feline metabolism.

Conclusion

Remission of feline diabetes mellitus requires a multimodal therapeutic approach that goes beyond simple glycemic control. Success depends on integrated interventions that consider species-specific biology, feeding behavior, and metabolic particularities. Factors such as confinement, sedentary lifestyle, hypercaloric diets, the effects of neutering, and deviations from the natural feeding pattern must be identified and corrected to optimize treatment response.

The combination of species-appropriate dietary strategies, adequate environmental management, and early, individualized insulinization protocols reduces insulin resistance, preserves pancreatic function, and promotes sustainable weight loss. This approach increases the likelihood of remission, minimizes disease-related complications, and contributes to greater longevity and quality of life for affected patients. &

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Received: September 8, 2025. Approved: October 31, 2025.

A publication of

CRMV SP
Conselho Regional de Medicina Veterinária
do Estado de São Paulo