Feline obesity and diabetes: a One Health perspective

Moira S. Lewitt

Address: School of Health and Life Sciences, University of the West of Scotland, PA1 2BE, UK.
MSL: 0000-0002-3859-1382.
*Correspondence: Moira S. Lewitt. Email: Moira.Lewitt@uws.ac.uk

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Abstract

There is increasing awareness of obesity and diabetes mellitus in cats that parallels the ‘epidemic’ of obesity and type 2 diabetes in humans. Likewise, understanding of the pathophysiology and management of feline obesity and diabetes has developed alongside and draws from human medicine. As in human medicine, overweight and obesity are associated with the development of diabetes; however, there are other genetic and environmental influences that contribute to this risk, some of which are shared. In cats, the body condition score appears to be a good diagnostic tool. It is reliable in the hands of owners and is likely to reflect visceral adiposity. However, the diagnostic label of feline obesity should be considered against the rate of success of any intervention, owner perspectives and impact on cat quality of life, as well as risk of future disease. The classification of human diabetes is evolving towards one that is more closely linked to disease outcome and therefore management. Consideration could be given to classifying the diabetes of hypersomatotropism and that associated with exogenous glucocorticoid administration in cats, separately in veterinary medicine. Rather regarding feline obesity and diabetes as models of human disease, a closer interdisciplinary ‘One Health’ approach between veterinary and human medicine is warranted to advance research and clinical care in this field.

Keywords: Feline, Diabetes, Obesity, Metabolic syndrome, One Health

Introduction

There is increasing awareness of obesity and diabetes mellitus in cats that parallels the ‘epidemic’ of obesity and type 2 diabetes in humans. Most knowledge of the pathophysiology of obesity derives from clinical and experimental studies in humans and rodent models which are used as the basis for understanding feline obesity and its management [1]. Conversely, feline obesity and diabetes are often regarded as models of human obesity and type 2 diabetes [2–4]. The aim of this review is to compare and contrast the definition, diagnosis, aetiology, pathogenesis and management of feline obesity and diabetes mellitus with the conditions in humans. The terms ‘feline’ and ‘obesity’ or ‘diabetes’ were used to search the PubMed and ScienceDirect databases. The title and abstracts of the items retrieved were searched manually for English language papers that focussed primarily on obesity or diabetes in cats, resulting in 702 relevant papers dating from 1946 being identified. The growth of the literature was found to fit an exponential curve ($r^2 = 0.996$), with a doubling time of 9.2 years (95% confidence interval 9.0–9.4 years). Two hundred were reviews or commentaries (28%), a proportion that has remained stable over time. Emerging themes guided the incorporation of relevant literature from human medicine.

Definitions and Diagnosis

Overweight and obesity

Weight can be used to track changes in body mass in individuals both human and feline. According to the World Health Organisation, overweight is ‘a condition characterised by excess weight relative to height’ [5] and recommends
use of the body mass index (BMI) to define overweight (25.00–29.99 kg/m²) and obesity (≥30.00 kg/m²). While these definitions are useful in adult populations, age, gender and pubertal status must also be taken into account in defining overweight and obesity in children. Furthermore, the BMI does not reflect adipose tissue distribution. Waist measurement is a significant predictor of health risk [6] and is a good marker of visceral adiposity [7]. Adult cats vary considerably in body mass, and the diagnosis of overweight and obesity in feline medicine rests to a large extent on observations of body shape. Using line drawings [8], owners able to identify a body condition score (BCS) and make judgements that closely agree with experienced veterinarians [9]. However, using a nine-point visual scale, where 6–9 is overweight or obese, owners perceive a BCS of 7 as indicating ideal body weight [10, 11]. Thus, while scoring might be considered reliable, owner education is needed if these are to have an influence on feline care. It has been suggested that a feline BMI score that utilizes the length from the top of the patella to the end of the calcaneous might be more sensitive and useful than the BCS [12]. However, as for waist measurement in human, feline BCS which assesses fat covering the ribs, abdomen and lumbar area might be a better marker of visceral adiposity than the feline BMI score.

Metabolic syndrome

The metabolic syndrome has emerged as a useful clinical concept in humans. The risk of cardiovascular disease is increased when abdominal adiposity is associated with evidence of glucose intolerance, dyslipidaemia or hypertension [7]. In cats, atherosclerosis does not present as a clinical problem. Nevertheless, since an elevated BCS is associated with insulin resistance [13], it has been suggested that an additional classification of feline metabolic syndrome, using BCS associated with increased blood glucose, dyslipidaemia and low adiponectin [14]. However, whether such a classification has practical clinical value is yet to be determined.

Diabetes mellitus

There is no agreed definition of diabetes mellitus in cats and this is considered a limitation in systematic reviews of this area [15]. In human health, there is a global consensus on the diagnostic criteria for diabetes [16], and there is also consensus on the definition of cure [17]. In feline clinical practice, the presence of signs (polyuria, polydipsia, polyphagia and weight loss), along with persistent hyperglycaemia or glycosuria, is used to make a diagnosis of diabetes mellitus, and treatment is commenced on that basis [18]. It would be useful to have a marker of prediabetes to use, for example, in cats with a high BCS score. In humans, the 75 g oral glucose tolerance test is used as a standard to screen for impaired glucose tolerance. It is preferred to intravenous glucose tolerance testing where there is no incretin response. In cats, administration of oral glucose requires careful handling techniques to minimize struggling and stress hyperglycaemia [19] and therefore intravenous glucose is used. Selection of a glucose concentration based on body weight, however, may lead to an overestimation in obese cats, and thereby an overdiagnosis of impaired glucose tolerance [20]. The lack of glucokinase in cats may account for a prolonged elevation of blood glucose after a meal [21] and needs to be considered in tests of glucose clearance.

Glucose is ubiquitously present in the urine of euglycaemic cats [22] and therefore cannot be relied on in the diagnosis or monitoring of feline diabetes. In the absence of stress, fasting blood glucose values do not exceed 7 mmol/l in cats and it has been proposed that ‘persistent values above this are diagnostic, if supported by documentation of elevated fructosamine or glycosylated haemoglobin, regardless of clinical signs’ [23]. Cut-off points of fasting blood glucose for the diagnosis of prediabetes in otherwise healthy older cats (>8 years) have been proposed [24]. In clinically healthy cats, fructosamine concentrations are positively associated with body weight and are higher in male cats than in female cats matched for age, weight and BCS [25]. While the use of fructosamine as a screening test for prediabetes is yet to be evaluated, it is well established as a measure of glycaemic control in cats with a diagnosis of diabetes [26].

Risk Factors for Overweight and Obesity

Genetic predisposition

In humans, there is an interaction between genes and environment in determining predisposition to obesity across childhood and adolescence [27]. Research using cat colonies indicates that obesity is also determined early in life [28] and there is likely to be a genetic component in its development [29]. Healthy weight intact male cats in a population genetically predisposed to overweight [29] have similar metabolic responses to a high carbohydrate to genetically lean cats, but lower fasting insulin levels [30] might indicate relative insulin sensitivity. A population study of cats in metropolitan Sydney, Australia, identified that mixed breed and British shorthair were more likely to be overweight than other purebred cats [31].

Nutrition and physical activity

It is well known that cats are obligate carnivores, which means evolutionary pressures have resulted in requirements for nutrients, including amino acids and vitamins, that
are met by consuming prey [32]. There are adaptations in feline carbohydrate metabolism which have been interpreted as indicating that high carbohydrate diets are a risk factor for the development of feline obesity. Consistent with this, feline overweight and obesity is associated with dry food consumption [11], as early as 1 year of age [33]. Commercial dry foods contain more carbohydrate than wet diets [34]. However, high wet food consumption at a young age and owners giving cat ‘treats’ are also associated with overweight [10] and it is likely that consumption of excess calories through overfeeding is the key risk factor [32]. Compared with veterinary physicians, owners are less likely to perceive that their cat is obese [10] and owner characteristics, such as age and income, are potential risk factors for feline obesity [35]. There is an interaction between food consumption and energy expenditure in determining predisposition to weight gain. Evidence shows that lean cats are more active than overweight cats, have a greater anticipatory physical activity prior to feeding and greater social interaction with humans [36]. Another study has shown a stronger diurnal activity pattern of cats when water is added to a dry food diet, compared with a wet canned diet of similar moisture content, the mechanism for which is unexplained [37].

Endocrine disruptors and hormonal influences

Perfluoroalkyl substances (PFAS) have endocrine disrupting effects and are associated with increased risk of childhood obesity [38]. In adult humans, higher plasma PFAS concentrations are associated with greater weight regain after a weight loss programme, particularly in women [39]. Since indoor cats have elevated PFAS levels that are similar to those in human sera [40], this environmental influence on obesity predisposition may be shared with humans and is worthy of further collaborative research.

In many countries, surgical neutering is a common procedure and considered a likely factor influencing obesity in cats [41]. Neutering in urban free-roaming cats is associated with an increased prevalence of feline obesity [42]. After neutering, male cats still have decreased insulin tolerance compared with female cats, and lower adiponectin levels [43].

Adipose tissue is an endocrine organ. Feline obesity is associated with increased adipocyte size and altered adipokine gene expression, with similar patterns to human obesity, with higher leptin and lower adiponectin levels in the circulation, and an increase in expression pro-inflammatory cytokines and chemokines [4, 44, 45]. In healthy cats, nearly 40% of the variance in final body weight after 8 weeks of ad libitum feeding is explained by initial body weight, energy requirements for maintenance and fasting leptin [46]. Adiponectin has been used in a classification of feline metabolic syndrome [14]. The role of other adipokines prediction and pathogenesis of overweight and obesity requires further study.

Is Obesity a Disease?

If feline obesity is a disease, it is one of the most prevalent. Prevalence of overweight and obesity in cats of 35% [47] and 39% [48] have been reported using a five-point BCS and as high as 45% [11] and 65% [49] using a nine-point scale. Despite suggestions that feline overweight and obesity might be increasing in prevalence in parallel with human obesity, a cross-sectional study in New Zealand found no evidence for an increase between 1993 and 2007 [50].

In humans, while obesity is increasing in prevalence, the level of risk appears to be decreasing. The lowest all-cause mortality in Denmark has increased from a BMI of 24 kg/m² in 1976–1978 to 27 kg/m² three decades later [51]. In a US study, while the risk of diabetes among obese individuals is the same, cardiovascular risk has declined over this time period [52]. Human obesity is classified as a nutritional disorder in the WHO International Classification of Disease [5]. However, there are calls to improve that definition based on aetiology, degree of adiposity and health risk [53]. The American Medical Association has classified obesity as a disease; however, this has generated debate around diagnostic criteria, classification and risk [54]. Recent studies in humans suggest that some individuals with obesity are in fact ‘metabolically healthy’. However, the risk of mortality and morbidity remains greater than in metabolically healthy normal weight individuals, and this difference is largely explained by cardiorespiratory fitness [55].

In cats, obesity is associated with reduced insulin sensitivity of glucose and fat metabolism [13] and a low or absent first phase insulin response following intravenous glucose [56, 57]. There are clear links between obesity and the development of diabetes and hepatic lipidosis in cats [58]. However, not all obese cats develop disease and it is not clear that all cats with obesity would have improved quality of life with weight loss. Useful predictive markers of disease in obese cats are yet to be identified. As in humans, prevention of obesity development and relapse is of key and importance in reducing prevalence of this condition [59].

Management of Obesity

It has been emphasized that, rather than focussing on the achievement of an ‘ideal weight’, the goal of weight loss should be established and a robust management approach designed in collaboration with the owner [60]. Restricted feeding of a moderate protein high-fibre diet is regarded as safe and effective for weight loss in cats [61]. Restricted high-protein diets might be beneficial for minimizing decreases in energy expenditure during weight loss [62]. In clinical practice the effectiveness is modest, with an average weight loss of 0.8% starting body weight per week and a 41% non-completion rate during a 3-month

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programme [63]. Regain in weight is common, particularly in younger cats [64]. Strategies that increase physical activity, such as increased feeding frequency [65, 66], running wheels [67] and social interaction with humans [36] may help in the prevention and management of overweight and obesity. It seems that, similar to humans, there is a stigma attached to a diagnosis of obesity in pets. Veterinarians are observed to use humour to cope with the discomfort surrounding the topic when talking to owners and the discussion is often avoided, suggesting the need for training towards history-taking and information-giving around nutritional intake [68] including communication skills [69].

**Feline Diabetes Mellitus**

There is a prevalence of feline diabetes mellitus of 0.58% in veterinary primary care clinics in England today [70]. In North America, the prevalence of feline diabetes presenting to a veterinary teaching hospital has increased, which may reflect changes in the willingness of owners to present or for veterinary physicians to refer, as well as increasing prevalence of risk factors [71]. Studies indicate an increased incidence in some cat breeds. An increased risk of diabetes has been observed in Burmese cats in England [70], Australia [72] and Sweden [73], which interestingly has not been reported in the USA, where breeding programmes have been kept separate [74]. An increased prevalence has been documented in Norwegian Forest cats [70, 73], Tonkinese [70] and in Russian Blue and Abyssinian [73], compared with other breeds. There is a higher prevalence of obesity in cats with diabetes [75]. As in humans, a polymorphism in the melanocortin 4 receptor gene is associated with diabetes in overweight cats [76].

In normal weight cats, there is an association between dry food intake and an increased risk of diabetes [77]. Since dry food is widely used by pet owners, it is important that this observation is further investigated.

In addition to the genetic predisposition and obesity, some of the factors associated with feline diabetes are also associated with type 2 diabetes risk in humans. Physical inactivity (and indoor confinement) [78] and advancing age [70] are associated with increased risk of feline diabetes. Male cats have lower total adiponectin and decreased insulin tolerance compared with female cats [43] and the risk of diabetes is higher [70–73]. In cats, it has long been recognized that exogenous glucocorticoid administration is associated with diabetes onset [79] and under these conditions, the pancreatic islets may be protected by insulin [80]. There is a greater chance of remission if cats have a history of corticosteroid treatment in the 6 months prior to diabetes diagnosis [21]. In cats with endogenous hyperadrenocorticism, it is reported that the incidence of diabetes is 80% [81].

Growth hormone has important lipolytic effects, and in humans with growth hormone excess (acromegaly), total body fat is reduced [82]. However, growth hormone has anti-insulin activities and acromegaly is associated with a high prevalence of glucose intolerance and diabetes, the severity of which is predicted by age, BMI and IGF-I concentrations [83]. Growth hormone-secreting pituitary tumours are well described in association with diabetes in cats [84], although cases of feline acromegaly without diabetes have also been reported [85]. Report of a prevalence of 25% of hypersomatotropism in feline diabetes in primary care settings in the UK [86] has led to a conclusion that screening for the disease should be undertaken in all cats presenting with diabetes [23]. However, many of these cases lack any other clinical features of acromegaly. Furthermore, while treatment that is directed at the tumour is likely to improve glucose tolerance, it is costly and invasive. In a series of 12 cases managed surgically, seven of the ten surviving cats achieved insulin independence [87]. A wide reference interval of 90–1207 ng/ml in a population of healthy cats [88] indicates that the current use of IGF-I concentration of >1000 ng/ml in screening might be set too high. Furthermore, IGF-I concentrations have been observed to increase in cats with diabetes in the weeks following the commencement of insulin [89]. Since abnormalities seen in pituitary imaging may be incidental, it would be prudent to continue to exercise caution in the diagnosis of feline acromegaly until further research is undertaken.

Veterinary medicine has adopted the nomenclature of human medicine, and currently uses the terms type 1 (T1DM) and type 2 diabetes (T2DM). Although there are reports of lymphocytic infiltration of the endocrine pancreas, T1DM is likely to be rare in cats [18]. Extensive pancreatic inflammation and destruction can, theoretically, cause insulin-deficient diabetes in any species. However, the converse may also be true. T2DM is associated with an increased risk of pancreatitis in humans [90]. Experimental hyperglycaemia in cats is associated with increased pancreatic neutrophils [91] and elevated serum pancreatic lipase activity and ultrasound findings consistent with pancreatitis have been documented in cats with diabetes, in the absence of clinical signs [92].

Historically, most feline diabetes was classified as insulin-dependent diabetes or type 1 [93], however it now clear that it resembles human T2DM, with a combination of insulin resistance, amyloid deposition in pancreatic islets and abnormal β-cell function [18, 94]. However, recently in human medicine it has been recognized that diabetes mellitus is a more heterogeneous disorder and a new substratification, based on disease progression and risk of complications, has been proposed [95]. The value of such an approach is to help in targeting management approaches. It is emphasized that protocols for diagnosis and treatment of diabetes are not appropriate for animals [96]. Thus there are limitations in using human aetiology-based classification systems for companion animals, and development of alternative approaches may be of more practical use for prognosis and management [23].

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Management of Diabetes

In diabetic cats, circulating insulin concentrations are reduced [97, 98]. Very early experimental work demonstrated that pancreatic islet lesions could be induced by administration of glucose [99] and glucotoxicity is now regarded as the key factor for declining β-cell function [18]. Recently, the mechanisms underlying the effect on β-cell function are the subject of intensive study using rodent models [100]. In humans, intensive insulin therapy in newly diagnosed patients with T2DM is associated with long-term restoration of insulin sensitivity and partial restoration of β-cell function [101]. Despite this observation, early intensive insulin regimens remain an unconventional approach to management in human medicine. In veterinary care, on the other hand, it is widely recognized that intensive management with insulin, along with a low carbohydrate diet and home blood glucose monitoring, which aims for near-euglycaemia, is associated with diabetes remission [102]. This success contrasts to earlier reports of high morbidity and mortality associated with the disease [103]. However, overall, the published evidence is moderate in quality at best [15] and, apart from a history of steroid administration [104] and the IGF-I response to insulin [89] being associated with an increased rate of remission, no single factor predicts successful independence from insulin therapy [15]. Intensive treatment regimens for feline diabetes impact on quality of life, with the risk of hypoglycaemia perceived by owners as one of the biggest concerns [105], despite the fact that it is uncommon in glargine-treated cats [106] and symptomatic hypoglycaemia is usually explained by vomiting after a meal or accidental double-dosing of insulin [107]. This highlights the importance of good communication between veterinary physicians and owners, in a collaborative approach that allows individualized care [108]. This requires that owners be provided with evidence and choice, as in the human domain, where the International Diabetes Federation recommends that diabetes self-management education is critical in laying the foundation for effective care in T2DM [109]. It has been reported that, amongst owners willing to take on the task of home blood glucose monitoring, there is a positive impact on quality of life for pet and owner, even in the absence of an impact on glycaemic control [110]. Continuous glucose monitoring systems have been tested in cats and would provide an alternative that reduces the number of venepunctures and therefore potential stress to the animal [111].

While insulin remains the mainstay of treatment of feline diabetes, a response to the oral hypoglycaemic agent glipizide is observed in approximately 50% of animals where there is residual β-cell function [112, 113]. Pioglitazone improves insulin sensitivity and improves lipid metabolism in obese cats [114] and therefore shows promise. Use of the α-glucosidase inhibitor, acarbose, along with a low carbohydrate diet, is associated with reduced insulin requirements, particularly in obese cats [115]. Alternative approaches are emerging that parallel the developments in human medicine. Glucagon-like peptide-1 (GLP-1) is an incretin, secreted in response to food intake and increasing insulin secretion and sensitivity. It is lower in obese, compared with lean cats, in the fasting state and following oral glucose [116] and increased in cats with diabetes compared with lean and obese animals [117]. GLP-1 receptor agonists reduce appetite and body weight, and increase insulin and decreases glucagon concentrations in healthy cats [118] and, when added to the insulin regimen in diabetic cats, are associated with weight loss and decreased insulin requirements [119]. A better understanding of the normal feline incretin response to dietary macronutrients [120] will contribute to understanding the role these agonists might play. The sodium-glucose cotransporter 2 inhibitor, velagliflozin, increases urinary glucose excretion in obese cats [121], and may be useful in the future management of hyperglycaemia.

One Health Approach to Obesity and Diabetes

One Health has been a long history of rediscovery [122], and has expanded even further since the American Veterinary Medical Association and American Medical Association resolved to increase collaboration between their communities [123]. Although there is no standard definition, the concept of how to improve animal and human health through interdisciplinary approaches is a useful one. The One Health concept thus implies that feline conditions should not be seen as simply models of human disease. In the context of obesity and diabetes, it is clear that a sharing of knowledge and clinical practice is helpful to feline and human medicine. There are shared lifestyle and environmental factors underpinning obesity that can be addressed concurrently and in interdisciplinary ways [124–126]. In diabetes care, human medicine could learn from the emphasis on intensive management while veterinary medicine could learn from the shift to precision medicine based on better disease classification.

Human–animal interaction is important in the management of obesity in pets [36, 127] and there is evidence that managing diabetes in pets pivots on their recognition as ‘sentient selves’ [128]. It is imperative to involve the owner closely in environment and behaviour modification [129] and in the complexities of diabetes management [108]. While there is evidence of a closer relationship between overweight cats and their owners, playing with the cat is more important to the owners of normal weight cats [130]. It has been suggested that styles of pet ownership might mirror parenting styles and provide comparative insights into the links to childhood obesity [131]. Behavioural addiction treatment methods are recommended for both overweight children and overweight pets, and studies of outcomes of these approaches are likely to be of value to both disciplines [132].
Summary and Recommendations

Our understanding of the pathophysiology and management of feline obesity and diabetes has developed in parallel to human medicine. The definition of human overweight and obesity, and the classification of human diabetes is being challenged. In the light of this, it is timely to reconsider feline obesity and diabetes. In particular, a reclassification of diabetes that is management-focussed would seem appropriate. The diabetes of hypersomatropism and that associated with exogenous glucocorticoid administration, for example, might be classified separately. The BCS appears to be a good diagnostic tool. It is reliable in the hands of owners and is likely to reflect visceral adiposity. Therefore, given the limitations in the use of BMI that have been identified in human medicine, efforts to develop a similar index for cats would seem unnecessary. As in human medicine, overweight and obesity are associated with the development with diabetes; however, there are other genetic and environmental influences that contribute to this risk, some of which are shared. Rather regarding feline obesity and diabetes as models of human disease, a closer interdisciplinary ‘One Health’ approach between veterinary and human medicine is warranted to advance research and clinical care in this field.

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