Scientific Works. Series C. Veterinary Medicine. Vol. LXIV (1), 2018
ISSN 2065-1295; ISSN 2343-9394 (CD-ROM); ISSN 2067-3663 (Online); ISSN-L 2065-1295

RISK FACTORS IN FELINE HYPERTHYROIDISM

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Abstract

The aim of this article is to synthesize and systematize the risk factors in the appearance of feline hyperthyroidism presented in the literature. It is based on data and statistics extracted from numerous articles made all around the world. Data were processed and systematized based on the following factors: age, race, gender, robe color and fur length, diet, living environment, litter usage, and interaction with various chemicals. Thus, it is noteworthy that hyperthyroidism is more common in senior cats, living indoor, consuming predominantly wet food, and having contact with PBDE and PCB type substances, as well as the fact that cats from a "color point" breed are less likely to develop hyperthyroidism. Many factors have been identified that may favor the disease, but no determinant.

Key words*: hyperthyroidism, cat, risk factors, etiology.*

INTRODUCTION

Hyperthyroidism affects up to 10% of geriatric cats and is associated with increased mortality and deleterious effects on several organ systems [(1)]. Hyperthyroidism was first reported in 1979 [(2)] and is recognized today as the most common endocrinopathy in cats. Although the pathologic changes associated with hyperthyroidism (adenomatous hyperplasia, adenoma of the thyroid gland) have been well characterized the pathogenesis of these changes remains unclear. It has been postulated that immunologic, infectious, nutritional (eg, iodine), environmental (eg, toxins or goitrogens), or genetic factors may interact to cause pathologic changes. However, a substantial difficulty with any of these factors is how they could cause disease to develop in cats on different continents within a relatively short period of time. It also has been suggested that the disease may not be new, but instead is simply being diagnosed more frequently as cats live longer and as owners seek more geriatric care. [(7)]

MATERIALS AND METHODS

In order to synthesize and systematize the risk factors in the appearance of feline hyperthyroidism presented in the literature numerous researches on the risk factors and the trigger factors of feline hyperthyroidism were studied. Until now, a number of risk factors have been studied and they are systematized below. Thus, the following aspects were investigated: age, gender, breed, robe color and fur length, diet, living environment, use of litter, interaction with various chemicals.

RESULTS AND DISCUSSIONS

From an age point of view, the disease affects especially cats aged between 4 and 16 years of age predominates cats with an average age of 10 years [(5)]. The prevalence of feline hyperthyroidism observed in some studies in USA has been reported to reach 10% of all cats older than 10 years, which is equivalent to a human age of over 60 years, and the prevalence appears to increase ever further with advancing age. Similar prevalence rates have been reported in other parts of the world over the last decade, ranging from 7.4% in London to 8.9% in Japan, 11.4% in Germany, and 20.1% in Warsaw [(3)].

So far no studies have demonstrated a clear involvement of the gender in feline hyperthyroidism development. [(6)]

Regarding breed, robe color and fur length, epidemiological studies have identified Siamese, Himalayan, and Burmese breeds to be at decreased risk of developing hyperthyroidism. The characteristic colorpoint coats of

these breeds result from temperature-sensitive mutations in the tyrosinase gene which limit conversion of the amino acid tyrosine to melanin pigment except at the cooler extremities. In addition to functioning as a precursor of melanin, tyrosine is an essential precursor of thyroid hormone. It has been hypothesized that the protective effect observed in colorpoint breeds may be related to the mutation in tyrosinase that leads to relatively greater tyrosine availability for thyroid hormone production. The authors commented that the study was underpowered. Further studies, to either support or refute their hypothesis, are lacking.

Overall, the results of studies do not provide consistent evidence in support of the proposed hypothesis for an association between coat color and hyperthyroid status. However, the studies were based on the assumptions that coat color is reflective of melanin concentration and that degree of pigmentation affects relative tyrosine availability for thyroid hormone production and was subject to certain limitations. Then genetics of coat color are complex, and cats could have been misclassified because of the use of owner-reported or receptionist-recorded information. Differences in the terminology used for coat colors by breeders and geneticists and lay terminology might also have compounded this effect. Studies results indicate that certain breeds have decreased risk of hyperthyroidism and that longhaired cats are at increased risk of hyperthyroidism. Further research is necessary to determine whether pigmentation plays a role in this breed protective effect or whether this association is as a result of alternative mechanisms. (1)

The diet based on wet food was associated with increased risk of hyperthyroidism. Dietary factors that represent a risk factor in this pathology are: high feed content in iodine, goitrogenic factors, soybeans, polyphenols and resorcinol. Compared to cats that did not eat canned food, cats that ate any canned food had an approximately 2-fold increase in disease risk. Although no obvious dose–response relationship existed, cats with diets reported by the owners as being 50–74% canned and 75–100% canned food had significantly increased risk. Little evidence was found of any relationship between dry or semimoist food and disease risk. Most cats almost exclusively ate commercial cat food. Of cats receiving 80% or more of their diet in the form of commercial cat food, the risk of hyperthyroidism decreased as the proportion of commercial cat food in the diet increased. (7)

Table 1. Coat color, hair length as risk factors for hyperthyroidism (1)

Risk Factor	Category	Euthyroid n (%)	Hyperthyroid n (%)
Color/pattern	Black (reference category)	567 (76)	176 (24)
	Brown and white	12 (100)	0 (0)
	Cream	10 (91)	1 (9)
	Colorpoint	27 (87)	4 (13)
	White	74 (82)	16 (18)
	Red	203 (78)	58 (22)
	Blue	59 (78)	17 (22)
	Blue and white	48 (77)	14 (23)
	Black and white	674 (76)	208 (24)
	Tabby	550 (75)	188 (25)
	Tabby and white	148 (74)	51 (26)
	Red and white	109 (74)	38 (26)
	Tortoiseshell	222 (73)	83 (27)
	Brown	18 (67)	9 (33)
	Tortoiseshell and white	48 (67)	24 (33)
White markings	No white (reference category)	1,656 (75)	539 (25)
	Some white	1,039 (76)	332 (24)
	All white	74 (82)	16 (18)
Dilute	Not dilute (reference category)	1,583 (76)	489 (24)
	Dilute	117 (79)	32 (21)
	Unknown	1,069 (74)	366 (26)
Base pigment	Black (reference category)	1,348 (76)	415 (24)
	Brown	30 (77)	9 (23)
	Red	322 (77)	97 (23)
	Unknown (tabby)	698 (74)	239 (26)
	Unknown (tortoiseshell)	**270 (72)**	**107 (28)**
	Unknown (colorpoint)	27 (87)	4 (13)
	Unknown (white)	74 (82)	16 (18)
Hair length	Short hair (reference category)	2,459 (76)	764 (23)
	Long hair	**310 (72)**	**123 (28)**

Many goitrogenic compounds can contribute to the development of adenomatous lesions in exposed cats. These may be of particular importance, because most are metabolised by glucuronidation, a metabolic pathway that is particularly slow in the cat. Most commercial cat foods contain relatively high levels of goitrogenic compounds (e.g. phthalates). (4)

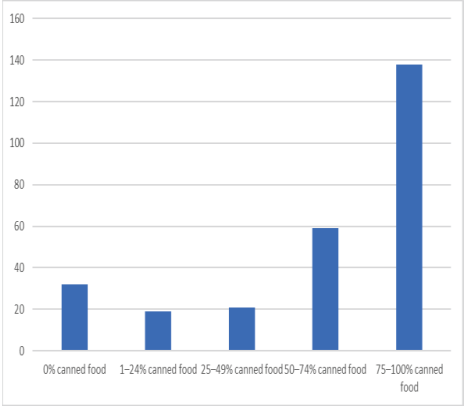

Figure 1. Relation between the percent of canned food and risk of hyperthyroidism in cats (7)

Even if some studies link the indoor living and the hyperthyroidism, in the absence of a clear explanation of the relation between litter use and hyperthyroidism, use of cat litter may simply be a marker for cats that primarily live indoors, receive better than average care, enjoy longer lives, and are more likely to reach the age at which cats develop the disease. [4]

Regarding the chemicals from the environment there where examined included those applied directly to cats to control ectoparasites, or to the environment as herbicides, insecticides, or fertilizers. Some flea-control products were associated with increased risk of developing hyperthyroidism. No obvious use–response relationship was discernable. For example, no difference in risk was found among low, moderate, and high use of flea collars. High use of flea spray and flea powder was associated with lowered risk whereas high use of flea shampoo was not associated with risk. Use of other flea products showed a monotonically increasing association with risk of hyperthyroidism, but these products were used in such a small number of cats that meaningful interpretation was not possible. Environmental use of insecticides, applied either by an exterminator or an owner, and environmental use of fertilizers and herbicides were not associated with increased risk of developing hyperthyroidism. [7]

Two classes of persistent organic pollutants, polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) are known to interfere with thyroid hormone signaling and regulation; thus, it is postulated that they contribute to the etiopathogenesis of feline hyperthyroidism and pose a risk to humans and other species. Studies demonstrate that elevated exposure to both PBDE and PCB congeners is associated with feline hyperthyroidism, supporting the hypothesis that these persistent organic pollutants may contribute to the etiopathogenesis of feline hyperthyroidism and suggests that they may have adverse impacts on thyroid health in humans and other animal species.[2]

CONCLUSIONS

Thyroid hormone function in the body is critical for proper execution of many developmental processes and the function of most organ systems throughout life across many species, thus there are many adverse implications of thyroid hormone disruption. [2]

Despite its increasing frequency, the origin and underlying pathogenesis of feline hyperthyroidism is not known, and therefore definitive recommendations in terms of prevention of the disease cannot as yet be made. [4]

Identification of a specific etiologic factor in feline hyperthyroidism will require randomized experimental trials. Such studies have limited feasibility because of the large number of cats necessary and the potential requirement for disease induction over many years. In the absence of such experiments, the need for observational studies continues. Future studies should determine detailed lifelong dietary history, micronutrient analysis of cat foods, and accurate measures of the frequency of pesticide applications on and around the cats studied. [7]

ACKNOWLEDGEMENTS

This research work was carried out with the support of Faculty of Veterinary Medicine Bucharest, Department of Clinical Sciences.

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