

CLINICAL ARTICLE

Prevalence of feline cataract: results of a cross-sectional study of 2000 normal animals, 50 cats with diabetes and one hundred cats following dehydrational crises

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Abstract

Objective In this study 2000 normal cats, 50 cats with diabetes and 100 cats with a history of dehydrational crises were examined ophthalmoscopically to determine presence of cataract.

Materials and methods The cats examined were predominantly from veterinary hospital populations but also from re-homing facilities and breeding catteries. Prevalence of cataract was determined for different age groups (year cohorts). The age at which prevalence of cataract was 50% (C_{50}) was determined indirectly from a fitted prevalence curve as previously described. C_{50} was determined for animals of different genders and different breeds as well as for those with diabetes and histories of dehydrational episodes related to chronic renal failure, chronic vomiting or chronic diarrhea.

Results The mean \pm standard deviation of C_{50} for all normal cats in the study was 12.7 ± 3.4 years. All cats over 17.5 years were affected by some degree of lens opacity. C_{50} for cats with diabetes was 5.6 ± 1.9 years (significantly different from normal cats at $P < 0.0001$). For cats with a history of dehydrational crises C_{50} was 9.9 ± 2.5 (difference from normal cats nearing statistical significance at $P = 0.06$).

Conclusion The study yields novel findings regarding the prevalence of age-related cataract in normal cats together with cats with diabetes and history of previous dehydrational episodes in which prevalence of cataract is increased.

Key Words: age-related, cataract, dehydration, diabetes, epidemiology, feline, lens

INTRODUCTION

We have previously reported the prevalence of cataract in 2000 dogs,¹ providing data for the prevalence of lens opacification in dogs at different ages. In this study we undertook the same size study for the cat but also sought to assess cataract prevalence in cats with diabetes and in those with a history of previous dehydrational episodes. Cats are generally accepted to have a lower prevalence of cataract than dogs.² While age-related cataract is reported in the dog, feline lens opacification is generally noted as either congenital or secondary to nutritional abnormalities, trauma or uveitis. Specifically, diabetic cataracts have been reported as rare in cats in contradistinction to the situation in dogs,³ apart from occasional cases in young kittens.⁴ However, as we noted in our previous canine study, such anecdotal statements have

yet to be supported or disproved by large-scale epidemiological studies. Here we seek to provide a cross-sectional survey to provide firm evidence of the prevalence and nature of age-related cataracts in cats.

Before we start, however, we might ask, from a comparative perspective, whether a large study to determine cataract prevalence in the cat is warranted. Globally it has been estimated from the most recently published studies based on surveys in 2002, that 22.8 million people are blinded by cataract in the developing world.⁵ Several large cross-sectional population-based studies show that around 75% of people over the age of 75 have sight-impairing lens opacification.⁶ Definitive documentation of the later onset of age-related cataract in the cat compared with the dog is, we suggest, the first step to defining what it is in the cat lens that delays the onset of age-related cataract, and renders these opacities less

sight-threatening than is the case in canine age-related cataract. Such future work could have significant implications for the prevention of human age-related cataract. This preliminary study to determine the prevalence of cataract in cats of different ages forms a basis for studies to identify what factors are associated with the slower development of cataract in cats compared with the rate of age-related lens opacification in dogs.

The human ophthalmic literature is well served with large cross-sectional population-based studies of ocular disease. The Beaver Dam study, for example, involves ocular examination of 4926 people from one geographic area in Wisconsin to determine prevalence⁷ and incidence⁸ of various ocular conditions including cataract, while in the Blue Mountains study over 3600 people from a region west of Sydney in Australia were subject to detailed ophthalmic examination with similar epidemiological end points.⁹ Performing a geographic population-based survey of the pet animal population is exceptionally difficult because census data for animals do not exist. It is possible, however, to examine cats presenting for nonocular conditions in first opinion and referral clinics as well as those normal animals housed in catteries and re-homing facilities, and thus to survey a relatively large number of animals from an ophthalmologically unremarkable population. Having undertaken such a cross-sectional study of normal cats what questions do we propose to ask of the data?

In his review of human age-related cataract, Taylor noted five 'D's as cataractogenic factors: daylight, diet, dehydration, diabetes and don't know!¹⁰ We cannot in this study population assess the influence of daylight and diet, and will seek to cover those areas in future reports of studies currently in progress in various species and population groups. However, the cat provides an excellent species for the determination of cataract prevalence in animals affected by both diabetes and dehydration: we postulate that cats with a history of diabetes or dehydrational episodes will have an increased prevalence of lens opacities. To determine this requires a normal population against which to compare the animals with diabetes and previous dehydration; the 2000 normal cats in this study provide just such a control group. In our canine study we specifically excluded animals with cataractogenic systemic conditions such as diabetes and hypocalcemia. In this study we sought to examine 2000 similarly normal cats but also to assess cataract prevalence in animals with diabetes and with previous dehydrational episodes.

Taylor's 'don't know' is most likely to be predominantly a genetic predisposition to increased age-related cataract.¹¹ In our canine study we showed a breed-related difference in prevalence of age-related cataract, with the C₅₀ value for different breeds being related to their longevity. Is the same association seen in the cat? Although Michel's previous work provided longevity data for different breeds of dog, there is no definitive data on the average lifespan of different cat breeds but for a paper on cemetery records for cats from the Far East in the mid 1980s,¹² data which are probably not of

great relevance to the cat population in this study. Anecdotal reports from cat breeders give some indication of the expected lifespan of specific breeds. <http://www.petplanet.co.uk/petplanet/breeds/catbreedprofile.asp>, for instance, states: 'The Persian Longhair may be expected to live for about 10–12 years while Siamese often live well into their late teens and it is not unheard of for them to live into their twenties'. Such anecdotal information can hardly, however, be considered scientifically reliable. We have thus, for these two breeds, undertaken to provide an age structure for pet populations seen in a group of first opinion veterinary clinics. While this cannot be considered as a definitive measure of longevity, it seeks to provide some confirmation of the statement on relative longevity obtained from the Internet.

This study only provides preliminary results for 2000 normal cats of different breeds and for 50 diabetic animals and one hundred cats with a history of previous dehydrational episodes. Yet we hope that these results for small numbers of cats will demonstrate the value of using a spontaneous animal model in the investigation of factors important in age-related cataractogenesis, and will form the basis for future research in companion animal age-related cataract.

MATERIALS AND METHODS

Animals

Cats from a number of populations were examined ophthalmoscopically as detailed below. The predominant group of normal animals was drawn from cats examined in eight first opinion clinics visited regularly by DLW (1045 cats). Three hundred and eighty-one normal cats were taken from the hospital population at the Queen's Veterinary School Hospital, Department of Clinical Veterinary Medicine, University of Cambridge, while 504 cats were examined at the Wood Green Animal Shelter, a large re-homing center where most cats were placed following geriatric owners entering residential care or where owners could not care for the animal after marital break-up. Forty Siamese cats and 30 Persian cats were examined in breeding catteries. The fact that a substantial number of the apparently normal animals examined were drawn from hospitalized populations is a potential failing in the study, as will be discussed further below, but no animals in this group were hospitalized for cataractogenic conditions and were otherwise ophthalmologically unremarkable.

The 50 diabetic animals and one hundred animals with a history of dehydration were drawn from the first opinion and referral hospital populations. Diagnostic criteria for diabetes included persistent blood glucose elevated above 10 μM ¹³ and a circulating fructosamine equaling or exceeding 400 μM .¹⁴ Eighteen animals had insulin resistant diabetes associated with acromegaly. Animals included in the dehydrated group had a history of chronic renal failure (85 cats), diarrhea (8 cats) or vomiting (17 cats) necessitating intravenous fluid therapy at least three times in the previous 12 months.

Ophthalmic examination

All animals were examined were screened using distant direct ophthalmoscopy at 0D and direct ophthalmoscopy at +10D. Pupils were dilated only by examining the animal in the dark, rather than by pharmacological intervention, in order to ensure that the investigation was entirely noninvasive and thus ethically valid, as owner consent was not available for a proportion of the animals examined. As discussed below this may be considered as a significant methodological failing of the study, yet pupil dilation sufficient to examine the majority of the lens was achieved merely by examination in the dark. The status of both lenses (either unaffected, with nuclear sclerosis or with frank cataract) was documented and if cataract was present the lens was examined by portable slit-lamp biomicroscopy (Kowa SL-14 slit lamp, Kowa, Tokyo, Japan). The position and extent of the lens opacity was documented graphically and scored using a scale from 0 (clear lens or nuclear sclerosis without opacity) to 10 (mature cataract). To avoid interobserver error only one ophthalmologist (DLW) examined all cats.

Statistics

The prevalence of cataract at different ages for the specific population sampled was derived from the cross-sectional data obtained in the study as described below. The proportion of animals affected in each year group was plotted to obtain a graph of prevalence proportion against age cohort. The prevalence proportion of an age cohort for an irreversible condition such as cataract is an estimate of the cumulative probability of onset up to and including that age. If it is assumed that the age at onset is normally distributed, by fitting a normal cumulative distribution curve to these data, using Microsoft Excel software, the mean and standard deviation of the underlying normal distribution of age of onset can be extracted. The age at which cataract prevalence was 50%, here termed C_{50} , given the similarity to LD_{50} in toxicological studies, is the mean from the cumulative probability curve. C_{50} for cataract for different subgroups may be compared by Student's *t*-test, using the means and standard deviations extracted from the fitted curve with the degrees of freedom based on the number of age cohorts used to determine the curve.

RESULTS

The median age of the 2000 cats examined was 8.3 years. The age structures of the four populations examined (referral hospital), first opinion clinic and nonhospital (re-homing center/breeding kennels) populations were not statistically different apart from a higher number of younger cats in the referral hospital population (Fig. 1). The overall cataract prevalence showed a C_{50} of 12.7 ± 3.4 years. The cataract prevalence by year group of these different populations was somewhat different, however, with C_{50} of 12.1 ± 2.7 , 13.5 ± 3.5 and 14.5 ± 3.6 in referral hospital, first opinion clinic and nonhospital populations, respectively (Fig. 2). However, the nonhospital population was difficult to model, as the

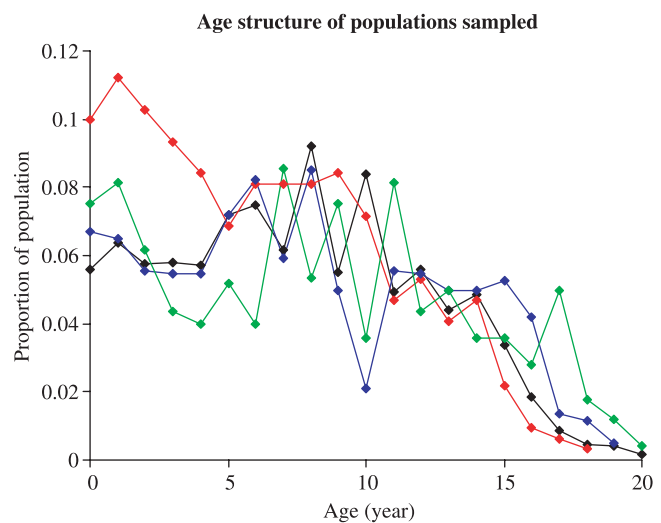


Figure 1. Age structure of the populations sampled. Black: All cats examined; red: veterinary hospital referral population; blue: first opinion clinic population; green: nonhospitalized population.

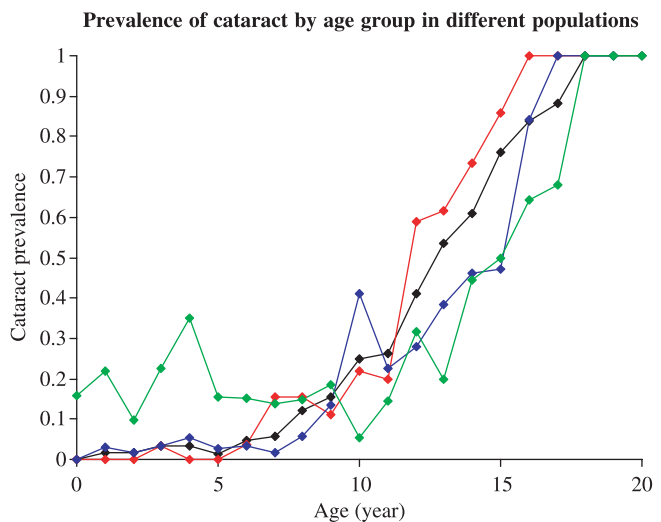


Figure 2. Cataract prevalence by year group in different populations examined. Black: all cats examined; red: veterinary hospital referral population; blue: first opinion clinic population; green: nonhospitalized population.

prevalence of cataract in the first year of life was greater than zero, the age structure of this population being significantly different from the others at $P = 0.02$.

There were only sufficient data for analysis of C_{50} values for male and female neutered animals rather than entire cats and for the Domestic Short-haired, Persian and Siamese breeds. The prevalence of age-related cataract in female and male neutered cats differed as is shown in Fig. 3, mirroring the difference in the age structure of the two gender populations (Fig. 4). C_{50} for the female neutered cats was 12.9 ± 3.2 years and for the male neutered cats was 11.7 ± 2.8 , these not being statistically significantly different. Cataract prevalence for Persian and Siamese cats is shown in Fig. 5. The age

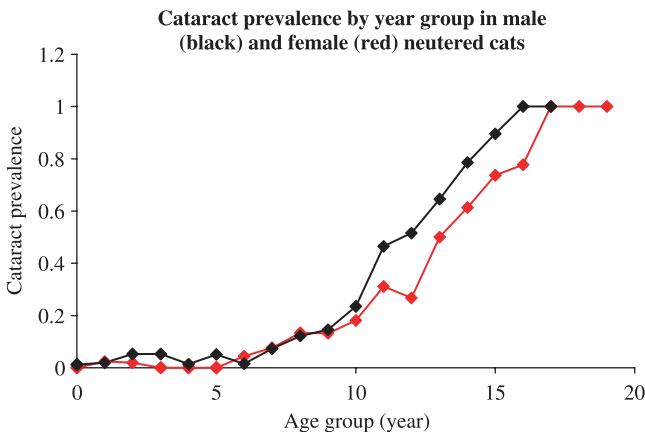


Figure 3. Prevalence of cataracts in cats examined by gender.

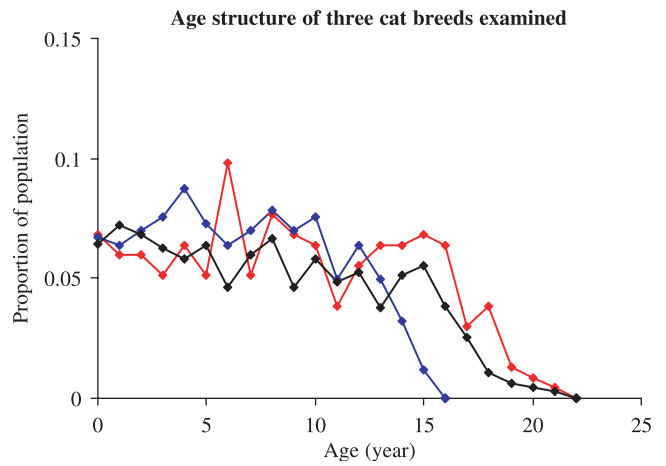


Figure 6. Age structure of three cat breeds examined in first opinion clinic population of 2388 cats. Black: Domestic Short-haired; red: Siamese; blue: Persian.

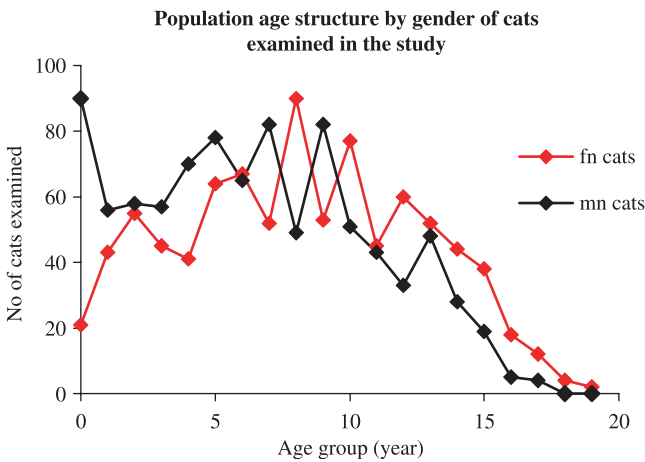


Figure 4. Age structure of cats examined by gender.

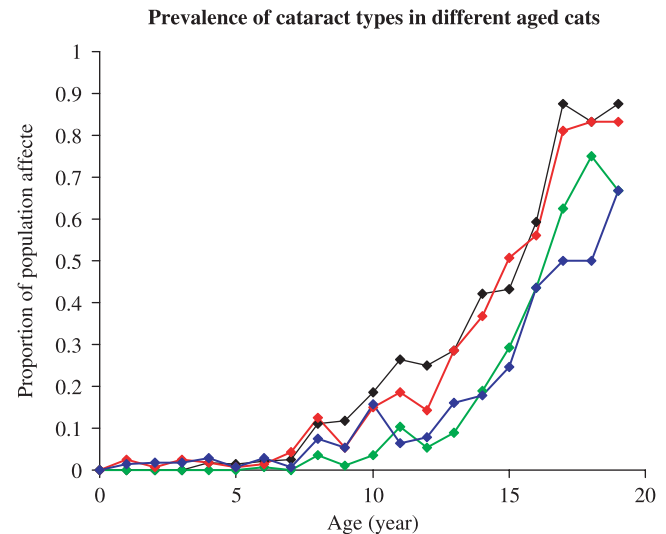


Figure 7. Prevalence curves for different cataract types. Black: nuclear sclerosis; red: posterior cortical cataract; green: nuclear cataract; blue: posterior polar subcapsular cataract.

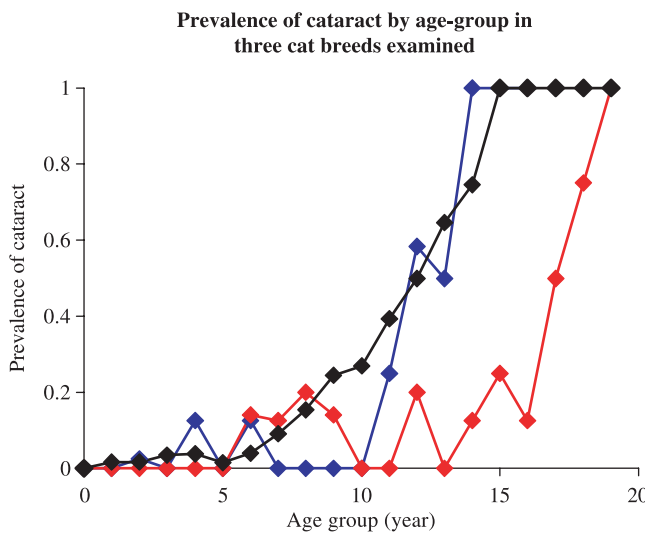


Figure 5. Cataract prevalence in three cat breeds examined. Black: Domestic Short-haired; red: Siamese; blue: Persian.

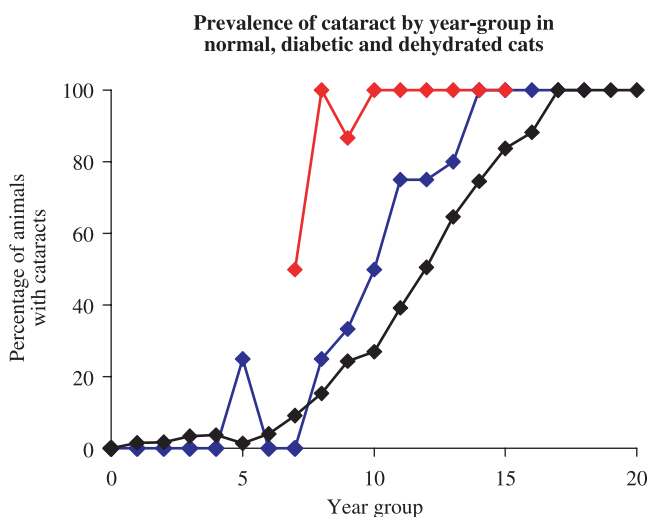
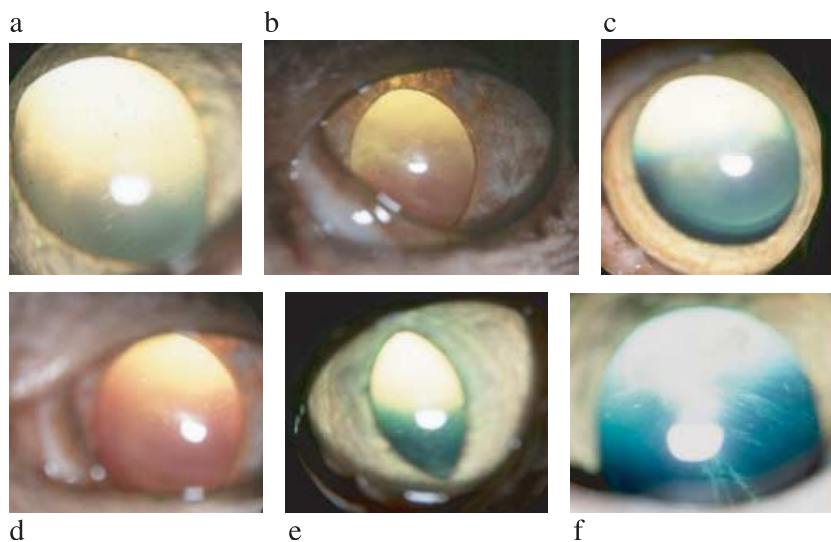
structures of populations of Persian, Siamese and Domestic Short-haired cat breeds in a first opinion clinic population of 2388 cats are shown in Fig. 6.

Prevalence curves of different cataract types are shown in Fig. 7. C_{50} for nuclear sclerosis is 14.6 ± 4.1 years, for nuclear opacities is 16.5 ± 3.2 years, for cortical cataract 14.9 ± 3.7 years and for posterior subcapsular cataract, 17.4 ± 4.6 years. These cataract groups were not easy to model, as the prevalence did not reach 100% at any age group.

The majority of cataracts were small linear opacities in the posterior cortex and, although older animals generally had more pronounced opacities, very few had mature lens opacities. A number of cats also exhibited congenital cataracts or opacities associated with trauma or intraocular inflammation.

Figure 8. Cataracts in normal-aged cats.

(a) 15-year-old, male neutered Domestic Short-haired cat with three linear posterior polar opacities. (b) 17-year-old, female spayed Burmese cat with nuclear and posterior cortical dot opacities. (c) 13-year-old, female spayed Domestic Short-haired cat with nuclear sclerosis and hazy nuclear opacities. (d) 17-year-old, male neutered Siamese cat with posterior cortical dot opacities. (e) 12.5-year-old male neutered Domestic Short-haired cat with posterior cortical short linear and dot opacities. (f) 20-year-old female spayed Domestic Short-haired cat with pronounced linear posterior cortical and capsular opacities.

**Figure 9.** Prevalence of cataracts in normal, diabetic and dehydrated cats. Normal: black; diabetic red; dehydrated: blue.

Six representative ophthalmic photographs of different types of cataract are shown in Fig. 8. Note that these photographs were taken focusing on the opacities, so where these were posterior cortical the iris may appear out of focus because it lies in a more anterior focal plane.

Prevalence of cataracts in the diabetic and dehydrated populations is shown in Fig. 9. C_{50} for cataract in diabetic cats was 5.6 ± 1.9 , differing from that of the normal population with a high statistical significance at $P < 0.001$, although the unusual distribution of these data rendered modeling of the C_{50} value difficult. Of cats with diabetes all but two animals had some degree of lens opacification. Twenty-two of these cats had linear posterior cortical opacities similar to those seen in the normal, aged cats. Twenty-six had more pronounced cortical cataracts or posterior subcapsular plaques. Photographs of cataracts in six representative diabetic cat eyes are shown in Fig. 10. C_{50} for cataract in

dehydrated cats was 9.9 ± 2.5 years, differing from that in the normal population with a P -value nearing significance ($P = 0.06$). All of the dehydrated cats had posterior cortical linear opacities similar to those seen in normal, aged cats, although on occasion these were multiple. Photographs of cataracts in six representative eyes from dehydrated cats are shown in Fig. 11.

DISCUSSION

The data accrued in this survey demonstrate that prevalence of cataract in the general feline population increases with age and that by the age of 17 years all cats were affected by some degree of lens opacity. This compares with the age of 13.5 years for all dogs to be affected, as shown in our previous study of 2000 dogs. While the dog has an average C_{50} of 9.4 ± 3.3 years, the cat has a significantly later C_{50} of 12.7 ± 3.5 ($P = 0.0005$). This difference is not particularly surprising, given that the lifespan of cats is significantly greater than that of dogs.^{15,16} Age-related cataract would seem to occur at a similar proportion of lifespan for these different species, although, clearly, considerable further research is required to substantiate such a hypothesis. This difference in age at cataract onset probably reflects the longevity of these two different species; age-related cataract would appear to occur at a specific fraction of the total lifespan of the individual. It is undisputed that, on average, cats live longer than dogs, so this difference in mean C_{50} is not unexpected. Such a correlation between the C_{50} value and lifespan was found in our previous study of 2000 dogs and does exist between the Persian and Siamese breeds in this study. The attempt at making this correlation is hampered by the lack of definitive longevity data for different cat breeds. Our evaluation of population ages of cats seen in several of the clinics visited during this survey aimed to estimate maximum lifespan in Domestic Short-haired, Persian and Siamese cats. The very rapid decline in older Persians may

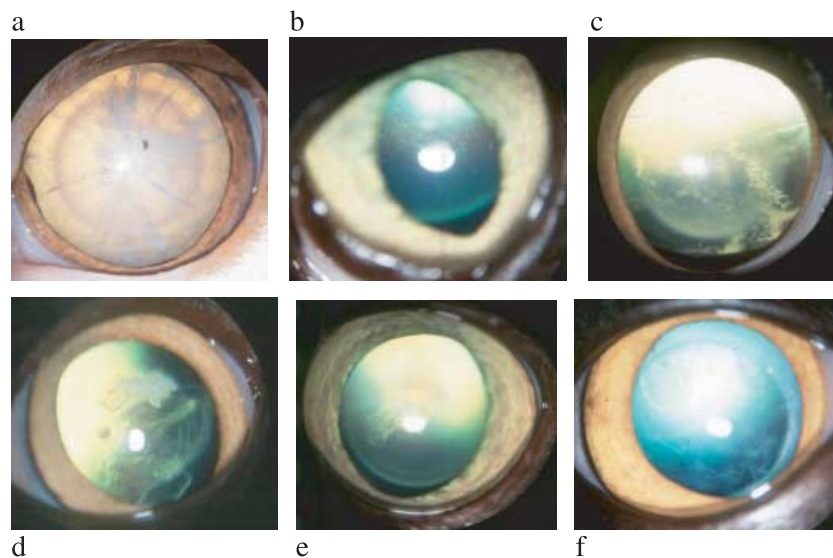


Figure 10. Cataracts in diabetic cats. (a) 18-year-old, male neutered Domestic Short-haired cat, diabetic for 3 years with pronounced nuclear and posterior cortical opacities after pharmacological mydriasis – dense focal paracentral opacity is corneal. (b) 15-year-old, female spayed Domestic Short-haired cat, diabetic for 2 years with nuclear sclerosis and posterior cortical opacities. (c) 12-year-old, female spayed Domestic Short-haired cat, diabetic and acromegalic for 6 months with pronounced anterior and posterior opacities and nuclear sclerosis. (d) 11-year-old, male neutered Domestic Short-haired cat, diabetic for 18 months with posterior subcapsular focal opacity, posterior cortical dot opacities, perinuclear plaque opacity and early nuclear sclerosis. (e) 13-year-old, female spayed Domestic Short-haired cat, diabetic for 2 years with posterior subcapsular plaque and nuclear sclerosis. (f) 14-year-old, female spayed Domestic Short-haired cat, diabetic for 6 months with early nuclear sclerosis and posterior cortical hazy opacities.

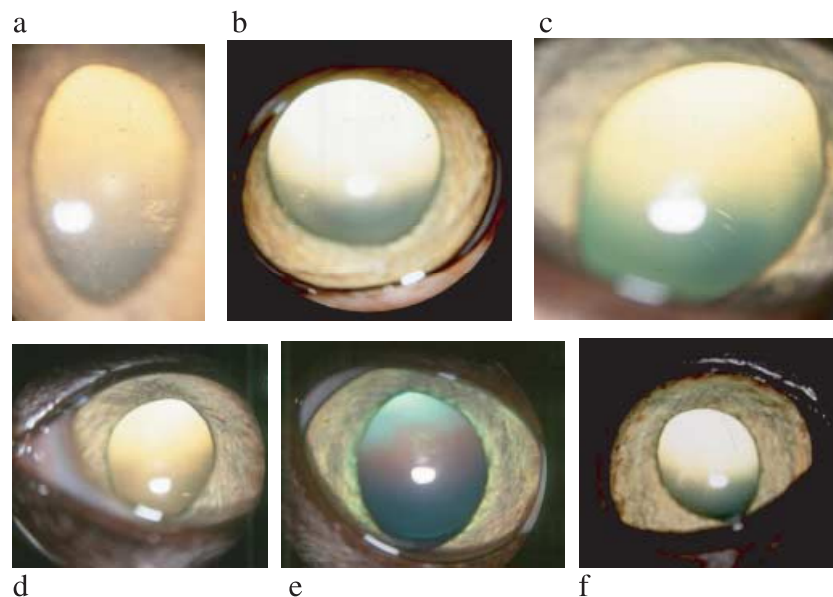


Figure 11. Cataracts in dehydrated cats. (a) 13-year-old, female spayed Domestic Short-haired cat with recurrent episodes of diarrhea for 2 years, requiring intravenous fluid therapy on four occasions with posterior cortical dot opacities. (b) 10-year-old, male neutered Domestic Long-haired cat with chronic diarrhea for 3 months requiring intravenous fluid therapy on three occasions with posterior cortical dot opacities. (c) 12-year-old, female spayed Domestic Short-haired cat with persistent episodes of vomiting for 2 years requiring intravenous fluid therapy on four occasions with two linear posterior cortical opacities. (d) 13-year-old, female spayed Domestic Short-haired cat with chronic renal failure requiring intravenous fluid therapy on three occasions with one linear posterior cortical opacity. (e) 14-year-old, male neutered Domestic Short-haired cat with chronic renal failure for 2 years requiring intravenous fluid therapy on three occasions with posterior cortical and subcapsular opacities. (f) 15-year-old, female spayed Domestic Short-haired cat with chronic renal failure for 2 years, requiring intravenous fluid therapy on three occasions with area of posterior cortical hazy opacity.

be associated with the high prevalence of polycystic kidney disease (PKCD) in this breed and highlights the difficulty in estimating true longevity: are cats dying of PCKD really dying of old age? Such questions must be postponed for a research paper on feline longevity and are peripheral to the current research. What is more relevant is that, while the cataract prevalence curve of Domestic Short-haired cats lies close to that of the Persian breed (Fig. 5), the population age structure shows the longevity of the Domestic Short-haired cat to be closer to the Siamese. The explanation to this may be that the Domestic Short-haired is less of a specific breed than a cross-breed. Resolution of this conundrum may have to wait until we have more concrete longevity data based on age at death in a substantially sized population, rather than the data on living aged cats as presented here.

Different types of cataract have different C_{50} values, possibly reflecting different etiopathologic pathways in the development of nuclear, cortical and subcapsular cataract, as first conjectured by Chylack in the mid-1980s.¹⁶ Photo-oxidation of crystalline proteins is widely held to be a key factor in age-related cataractogenesis, as discussed more fully in a companion paper in this issue of *Veterinary Ophthalmology*.¹⁷ The fact that nuclear proteins are present in the eye prior to birth leads one to expect that they would be most affected of all the lens tissue by photo-oxidative change. That said, the nuclear opacities in this study are less common and generally occur later (i.e. with a greater C_{50} value) than do posterior cortical opacities. Nuclear sclerosis is less commonly seen in the cat than in the dog and does not necessarily seem to be the precursor to nuclear opacification as it is in the canine lens. Further work on this will include a longitudinal study of the development of lens opacities in a smaller population of older cats. This is currently under development. The cortical opacities seen in aged cats are most commonly small linear opacities in the central and paracentral posterior cortex, probably representing opacification of individual lens fibers.

The data on lens opacities in normal cats are an essential backdrop to investigation of cataracts in animals with diabetes and previous dehydrational episodes. Both of these diseased populations are made up of older cats and, without the prevalence curve for normal animals, it would be impossible to assess whether a cataract in a specific individual was related to its disease or merely a function of its age. Figure 9 demonstrates that cataracts in both these diseased populations do occur at an earlier age than do lens opacities in comparatively aged normal cats.

Many of the cataracts in the diabetic cats were small linear opacities similar to those seen in the majority of normal, aged cats, but 56% of the diabetic cataracts were more pronounced posterior cortical opacities or posterior polar subcapsular plaques, as shown in Fig. 10. The data showing that almost all diabetic cats have lens opacities is contrary to findings reported to date in the literature. Salgado *et al.*, in the only large survey comparing diabetic cataracts in dogs and cats, state specifically that cataract is rare in diabetic cats,

in distinction to the case in dogs.³ We have shown here that lens opacities are common in diabetic cats, occurring in the vast majority of animals. How can this substantial difference be explained? We suggest that this disparity is related to the appropriation methods of the two studies. Salgado *et al.* analyzed diabetic cats and dogs using a retrospective survey of case records. Small cataracts may well have been missed using such a survey scheme; none of the diabetic cataracts here were mature blinding opacities and few of the diabetes-associated lens changes in our series (Fig. 10) would have been noted without a specific close ophthalmic examination. The fact that we have noted lens opacities in almost all of the diabetic cats does not mean that the lens changes in diabetic animals from these two species are to be seen as identical or equivalent. Cataracts in diabetic cats resemble cataracts in diabetic humans much more closely than do the mature cataracts characteristically seen in diabetic dogs.¹⁸ Mechanisms of diabetic cataractogenesis in the cat may involve processes other than the sorbitol generation classically considered responsible for the rapid development of the mature cataract characteristic of the diabetic dog.¹⁹ Other biochemical processes such as protein glycation and the formation of advanced glycation end products (AGEs) through the Maillard reaction are considered important in human diabetic cataractogenesis,^{20,21} and may also occur in the cat. Indeed, research from the Zurich group comparing the ratios of NADPH reductases and sorbitol dehydrogenase in the dog and cat lens suggested that sorbitol would accumulate at a faster rate in the cat than the dog lens; the difference in diabetic cataractogenesis between the two species cannot be explained by differences in enzyme ratio.²² Further work from the same group working under Professor Bernard Speiss demonstrated posterior cortical opacities in feline lenses cultured in high glucose concentrations though these occurred in young but not old cats.²³ In that study canine lenses developed equatorial vacuoles but not the characteristic mature cataract seen in 80% of dogs within 16 months of diagnosis in one recent study.²⁴ Clearly there is considerable work to be done on the pathogenesis of diabetic cataract in the dog and cat and the differences between the two species. Nonetheless, the findings in the present study are important in showing that almost all diabetic cats do have some signs of lens opacification, although substantially different from that seen in dogs.

Figure 9 also shows that cats with a history of previous dehydrational episodes requiring intravenous fluid therapy develop opacities in the lens at an earlier age than do normal cats. Again, as these cats are usually older animals, the baseline data on 2000 normal cats is needed, to compare against cataract prevalence in the dehydrated population. As noted above, dehydration is one of Taylor's five 'D's as factors predisposing to age-related cataractogenesis. Harding has particularly championed this as a major influence on the high prevalence of cataract in the tropical developing world,²⁵ suggesting that increased lens protein carbamylation may be responsible for the cataract formation with dehydration.²⁶

Other groups have not considered dehydration as important: those promoting daylight as the key factor in cataractogenesis in tropical areas may fail even to recognize it – Robman does not mention it in a recent survey of cataractogenesis from an Australian perspective, where clearly dehydration is of little importance.²⁷ Consider India, however, and the picture is very different. Zodpey *et al.*, working on a hospital-based population in Nagpur, Maharashtra State, show a relative risk of 3.1 of developing cataract in dehydrated individuals²⁸ while Minassian *et al.*, working on a similar central Indian population, estimated that dehydration from diarrhea or heatstroke might account for around a third of cataract cases in the affected area.²⁹ A second study on a different group, both geographically and from a socioeconomic perspective, strongly confirmed these findings with an estimated 38% of blinding cataract cases arising from repeated dehydrational crises.³⁰ It was in view of this research that we sought to investigate cataract prevalence in dehydrated cats.

We chose to take a historical perspective on the diagnosis of dehydration sufficiently severe and persistent or recurrent to cause lens changes similar to those seen in the Indian studies in humans. Thus, cats with three or more episodes of dehydration requiring intravenous fluid administration were included in the study. This necessarily resulted in a heterogeneous case population; cats with chronic renal failure, chronic diarrhea and chronic vomiting were all included and are not separated in the dehydrated group data. This could clearly be seen as a failing of the study; a wide variety of concurrent diseases was present in the population, some of which could have cataractogenic potential. Specifically, it may be that uremia is in itself cataractogenic,³¹ that hypocalcemia associated with renal failure can be responsible for cataract formation,³² or that oxidative stress, seen in chronic renal failure, is an important factor.^{33,34} Oxidative stress and reduced levels of intra-erythrocytic glutathione has been suggested as one of the causes of anemia associated with chronic renal failure,³⁵ but to our knowledge the association with cataracts has not previously been linked to intralenticular oxidative stress. The fact that we have seen similar cataracts in cats with chronic renal failure, chronic diarrhea and chronic vomiting, suggests that it is the common factor here, dehydration, which is the factor responsible for the cataract formation noted in these cats. The fact that the lens opacities are the same in form as those seen in older cats, suggests that dehydration is merely an acceleration of processes already giving rise to age-related cataract; however, the confirmation of such a hypothesis requires further clinical research. We aim to undertake a more comprehensive study of cataract formation in dehydrated cats, specifically using a case-control methodology rather than a cohort analysis as documented here. The difference in cataract prevalence between the dehydrated and normal groups narrows, but does not reach, statistical significance at $P = 0.06$. We hope that a case-control methodology with a larger population size will allow significance to be reached. Nevertheless, here we have

shown that increased cataract prevalence is seen in cats with a history of recurrent dehydration, a finding which is novel in a nonhuman population.

CONCLUSION

We have sought in this cross-sectional study to document cataract prevalence in a sizeable cat population, which, while having the disadvantage of being drawn mostly from a veterinary hospital setting, does provide concrete evidence with regard to the prevalence of cataract in the aging cat as well as showing increased numbers of cataracts in cats with diabetes and previous dehydrational episodes. It is hoped that this work will serve as a foundation for further in-depth studies of lens opacification in companion animal species.

REFERENCES

1. Williams DL, Heath MF, Wallis C. Prevalence of canine cataract: preliminary results of a cross-sectional study. *Veterinary Ophthalmology* 2004; **7**: 91–95.
2. Glaze MB, Gelatt KN. Feline Ophthalmology. In: *Veterinary Ophthalmology*, 3rd edn. (ed. Gelatt KN) Lippincott/Williams and Wilkins, Philadelphia, 1999; 1103–1033.
3. Salgado D, Reusch C, Speiss B. Diabetic cataracts: different incidence between dogs and cats. *Schweizer Archiv für Tierheilkunde* 2000; **142**: 349–353.
4. Thoresen SI, Bjerkas E, Alekandersen W *et al.* Diabetes mellitus and bilateral cataracts in a kitten. *Journal of Feline Medicine and Surgery* 2002; **4**: 115–122.
5. Resnikoff S, Pascolini D, Etya'ale D *et al.* Global data on visual impairment in the year 2002. *Bulletin of the World Health Organization* 2004; **82**: 844–851.
6. Klein BEK, Klein R, Lee KE. Incidence of age-related cataract. The Beaver Dam Eye Study. *Archives of Ophthalmology* 1998; **116**: 219–225.
7. Klein BE, Klein R, Lee KE. Incidence of age-related cataract over a 10-year interval: the Beaver Dam Eye Study. *Ophthalmology* 2002; **109**: 2052–2057.
8. Klein BE, Klein R, Linton KL. Prevalence of age-related lens opacities in a population. The Beaver Dam Eye Study. *Ophthalmology* 1992; **99**: 546–552.
9. Mitchell P, Cumming RG, Attebo K *et al.* Prevalence of cataract in Australia: the Blue Mountains eye study. *Ophthalmology* 1997; **104**: 581–588.
10. Taylor HR. Epidemiology of age-related cataract. *Eye* 1999; **13** (Pt 3b): 445–448.
11. Hejtmancik JF, Kantorow M. Molecular genetics of age-related cataract. *Experimental Eye Research* 2004; **79**: 3–9.
12. Hayashidani H, Omi Y, Ogawa M *et al.* Epidemiological studies on the expectation of life for cats computed from animal cemetery records. *Nippon Juigaku Zasshi* 1989; **51**: 905–908.
13. Plotnick AN, Greco DS. Diagnosis of diabetes mellitus in dogs and cats. Contrasts and comparisons. *Veterinary Clinics of North America: Small Animal Practice* 1995; **25**: 563–570.
14. Crenshaw KL, Peterson ME, Heeb LA *et al.* Serum fructosamine concentration as an index of glycemia in cats with diabetes mellitus and stress hyperglycemia. *Journal of Veterinary International Medicine* 1996; **10**: 360–364.
15. Hayashidani H, Omi Y, Ogawa M *et al.* Epidemiological studies on the expectation of life for dogs computed from animal cemetery records. *Nippon Juigaku Zasshi* 1988; **50**: 1003–1008.

16. Chylack LT Jr. Mechanisms of senile cataract formation. *Ophthalmology* 1984; **91**: 596–602.
17. Williams DL Oxidation, antioxidants and cataract formation: a literature review. *Veterinary Ophthalmology* 2006; **9**: 292–298.
18. Bron AJ, Brown NA, Harding JJ *et al.* The lens and cataract in diabetes. *International Ophthalmology Clinics* 1998; **38**: 37–67.
19. Basher AW, Roberts SM. Ocular manifestations of diabetes mellitus: diabetic cataracts in dogs. *Veterinary Clinics of North America: Small Animal Practice* 1995; **25**: 661–676.
20. Ahmed N. Advanced glycation endproducts – role in pathology of diabetic complications. *Diabetes Research in Clinical Practice* 2005; **67**: 3–21.
21. Stitt AW. The maillard reaction in eye diseases. *Annals of the New York Academy of Sciences* 2005; **1043**: 582–597.
22. Salgado D, Forrer RS, Spiess BM. Activities of NADPH-dependent reductases and sorbitol dehydrogenase in canine and feline lenses. *American Journal of Veterinary Research* 2000; **61**: 1322–1324.
23. Richter M, Guscetti F, Spiess B. Aldose reductase activity and glucose-related opacities in incubated lenses from dogs and cats. *American Journal of Veterinary Research* 2002; **63**: 1591–1597.
24. Beam S, Correa MT, Davidson MG. A retrospective cohort study on the development of cataracts in dogs with diabetes mellitus: 200 cases. *Veterinary Ophthalmology* 1999; **2**: 169–172.
25. Harding JJ, Rixon KC. Is diarrhoea a major cause of cataract in some tropical countries? *Metabolic and Pediatric Ophthalmology* 1981; **5**: 161–166.
26. Harding JJ, Rixon KC. Carbamylation of lens proteins: a possible factor in cataractogenesis in some tropical countries. *Experimental Eye Research* 1980; **31**: 567–571.
27. Robman L, Taylor H. External factors in the development of cataract. *Eye* 2005; **19**: 1074–1082.
28. Zodpey SP, Ughade SN, Khanolkar VA *et al.* Dehydrational crisis from severe diarrhoea and risk of age-related cataract. *Journal of the Indian Medical Association* 1999; **97**: 13–15.
29. Minassian DC, Mehra V, Jones BR. Dehydrational crises from severe diarrhoea or heatstroke and risk of cataract. *Lancet* 1984; **1**: 751–753.
30. Minassian DC, Mehra V, Verrey JD. Dehydrational crises: a major risk factor in blinding cataract. *British Journal of Ophthalmology* 1989; **73**: 100–105.
31. Beiran I, Scharf J, Tamir A *et al.* Influence of systemic diseases and environmental factors on age at appearance, location and type of acquired cataract. *Metabolic and Pediatric Ophthalmology* 1994; **17**: 34–37.
32. Chugh SK, Goel A. Bilateral cataracts as the presenting manifestation of chronic renal failure. *Journal of the Association of Physicians of India* 1992; **40**: 273–374.
33. Vrabec R, Vátavuk Z, Pavlovic D *et al.* Ocular findings in patients with chronic renal failure undergoing haemodialysis. *Collegium Antropologicum* 2005; **29** (Suppl. 1): 95–98.
34. Costagliola C, Iuliano G, Menzione M *et al.* Systemic human diseases as oxidative risk factors in cataractogenesis. II. Chronic renal failure. *Experimental Eye Research* 1990; **51**: 631–635.
35. Costagliola C, Romano L, Sorice P *et al.* Anemia and chronic renal failure: the possible role of the oxidative state of glutathione. *Nephron* 1989; **52**: 11–14.