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Abstract

An increased incidence of cataract and fatty liver in plains viscachas (Lagostomus maximus) was noted for many years at the Zurich Zoo. Based on elevated serum fructosamine and glucose, diabetes mellitus was diagnosed, and these parameters normalized when the diet of the animals was changed from a low-fiber to a high-fiber diet. Here, we evaluate 177 necropsy reports from before and after the diet change for the incidence of cataracts and fatty liver. Sixteen of 56 animals (29%) that were born before the diet change developed cataract. In contrast, only two of 121 animals (1.65%) that were born after the diet change developed cataracts. The prevalence of cataract and fatty liver in animals born after the diet change (1% and 0%, respectively) was significantly lower than in animals born before (9% and 6%, respectively) the diet change. The results suggest that the plains viscachas at Zurich Zoo were affected by an alimentary induced diabetes mellitus (type 2) before the diet was changed.
OCCURRENCE OF CATARACT AND FATTY LIVER IN CAPTIVE PLAINS VISCACHAS (LAGOSTOMUS MAXIMUS) IN RELATION TO DIET

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CATARACTS IN VISCACHAS

Abstract: An increased incidence of cataract and fatty liver in plains viscachas (*Lagostomus maximus*) was noted for many years at the Zurich Zoo. Based on elevated serum fructosamine and glucose, diabetes mellitus was diagnosed, and these parameters normalized when the diet of the animals was changed from a low-fiber to a high-fiber diet. Here, we evaluate 177 necropsy reports from before and after the diet change for the incidence of cataracts and fatty liver. Sixteen of 56 animals (29%) that were born before the diet change developed cataract. In contrast, only two of 121 animals (1.65%) that were born after the diet change developed cataracts. The prevalence of cataract and fatty liver in animals born after the diet change (1% and 0%, respectively) was significantly lower than in animals born before (9% and 6%, respectively) the diet change. The results suggest that the plains viscachas at Zurich Zoo were affected by an alimentary induced diabetes mellitus (type 2) before the diet was changed.

Key words: plains viscacha, *Lagostomus maximus*, rodent, pathology, diabetes mellitus, cataract, diet
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INTRODUCTION

Plains viscachas (*Lagostomus maximus*; Rodentia, Chinchillidae) are crepuscular rodents, which inhabit the pampas grasslands of Paraguay, Bolivia, and Argentina. In the wild, the diet of plains viscachas consists mainly of a wide variety of grasses, forbs and shrubs.² Viscachas are hindgut fermenters with a voluminous caecum¹⁴ which use coprophagy as a digestive strategy⁶. These animals are rare in captivity, but have been kept at Zurich Zoo, Switzerland, since 1964. A survey of pathology reports of plains viscachas at this institution by Rübel et al.²⁸ revealed an increased incidence of bilateral cataracts and fatty livers. These findings raised the suspicion that viscachas might be susceptible to diet-induced diabetes mellitus. Diabetes with cataracts has been described previously in other rodents. For example, nutritionally induced type 2 diabetes with possible cataract formation was described in the fat sand rat (*Psammomys obesus*),³¹ a genetic component seems to be involved because different genetic lines of *Psammomys obesus* can be generated that differ in their proneness to diet-induced diabetes.¹⁶,¹⁸ A diet-induced diabetes mellitus with cataract formation is described also in degus (*Octodon degus*)⁷, while a genetic etiology is suspected in Chinese hamsters (*Cricetulus griseus*)¹² and chinchillas (*Chinchilla lanigera*)¹⁰. Additionally, viruses associated with a type 1 diabetes-like syndrome have been described in several rodent species e.g. mouse (*Mus musculus*), rat (*Rattus norvegicus*), degu and hamster.³⁶ Throughout these reports it is acknowledged that a high energy diet increases the likelihood to develop diabetes mellitus in predisposed animals.

After the problem in plains viscachas was recognized, several measures were initiated.

1. Free-ranging plains viscachas were captured in Argentina in order to generate blood and urine reference values which could be used for monitoring.³⁵

2. Mineral concentrations in serum and whole blood of free-ranging and captive viscachas were compared; the results did not suggest a mineral implication in the formation of cataracts.¹¹
3. The diet was changed to contain a higher proportion of roughage and a lower proportion of easily digestible carbohydrates such as fruits, grains, and bread.\textsuperscript{34}

4. A group of animals was bled 3, 6 and 9 months before and after the initial diet change. Elevated blood parameters indicative for diabetes dropped within the reference values after the diet change in the adult animals.\textsuperscript{34} It was suggested that these findings were consistent with (but not necessarily proof of) a non-insulin dependent (type 2) diabetes mellitus.

However, if this diet change was to be considered effectual, one should expect a decrease in pathological findings associated with diabetes, such as cataract and fatty liver.

In the 8 years following the diet change a total of 121 animals born after the change had died, allowing a comparison of necropsy findings from animals with a low-fiber diet with findings from those examined prior to the change. The current report presents these results.

\textbf{MATERIALS AND METHODS}

Between 5\textsuperscript{th} May 1992 and 4\textsuperscript{th} May 2008 Zurich Zoo housed 234 plains viscachas. During this period 177 individuals died and all available medical records, necropsy reports and the stocklist were evaluated. The animals had been housed at three different locations: two indoor enclosures and one outdoor enclosure where the viscachas were kept together with vicunas (\textit{Vicugna vicugna}) and lesser rheas (\textit{Pterocnemia pennata}). The diet offered to the animals was identical for all locations, with the exception that animals kept with the vicunas and rheas could have had incidental access to the food intended for these other species.

The low fiber diet (diet 1) consisted of a formerly common zoo diet for herbivores: apples, carrots, lettuce, bread, a commercial mineral/vitamin supplement (Multiforsa M21, Multiforsa AG, 6312 Steinhausen, Switzerland), rye grass hay and local browse branches offered ad
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libitum. However, the apples, carrots and bread were fed in such amounts that no additional
hay intake would have been necessary to meet the theoretical energy requirements. On
average, each animal received 0.3 kg carrots, 0.3 kg apples, 0.1 kg bread and mineral/vitamin
supplement per day. Based on published data it was estimated that this diet contained (in dry
matter DM) 9.0 % crude protein, 5.2 % crude fiber 1.0% ether extracts, 3.9% crude ash and
80.8 % nitrogen-free extracts (estimated without intake of additional hay or branches).

On 5th May 2000 the diet was changed to a high-fiber diet based on reports mentioned above
on the occurrence of diabetes in other rodent species. The diet contained a 1:1 rye grass hay
and straw mixture, a vitamin/mineral supplement (Multiforsa M21, Multiforsa AG, 6312
Steinhausen, Switzerland) and a pelleted feed, based on alpine meadow hay (PRE ALPIN
Lepo, Agrobs GmbH, 82541 Degerndorf, Germany). The actual intake of this diet was
measured during three days. It contained (in DM): 7.5 % crude protein, 39.2 % crude fiber,
1.7 % ether extracts, 5.6 % crude ash and 46.1 % nitrogen-free extracts. The digestible energy
(DE) content of the original ration was estimated at 12.5 MJ/kg DM, that of the new ration at
10.3 MJ/kg DM by using data on nutrient content, data on nutrient digestibility measured in
viscachas on a mixed diet, and the factorial estimation of DE for rabbits.

Necropsy reports from 5th May 1992 - 4th May 2008 were used, allowing a comparison of
animals born before and after the diet change of 5th May 2000. The reports were analyzed in
particular for the occurrence of cataracts and fatty degeneration of the liver. All pathologic
examinations were carried out at the Institute of Veterinary Pathology of the Vetsuisse
Faculty of the University of Zurich. The first mention of ocular cloudiness in the medical
record was used to determine the onset of cataract development. It was not possible to allocate
animals from post mortem records reliably to one of the three locations which meant that
potential correlations with pathological findings due to location could not be determined. The
objective of the analyses presented in this report was to determine whether there were
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significant differences in cataract occurrence and nutritional related diseases between the
different time periods.

All statistical analyses were performed using a statistics program (Statistica™ 7.1, StatSoft®
Inc., Tulsa, Oklahoma 74104, USA). Statistical significance was set at p<0.05. Animals born
before and after the diet change were regarded as two independent groups. The categorical
outcome of cataracts in the two diet groups was examined using contingency tables. The
comparison of average age of death between the groups was tested by two-sample t-test. Due
to the small number of cataracts in the new diet group the Mann-Whitney U test was applied
to see whether cataract development was age-related. A p value < 0.05 was accepted as level
of significance.

RESULTS

One hundred and seventy seven plains viscachas died at Zurich Zoo between 5th May
1992 and 4th May 2008. The major findings were bite lesions (n=64, 36%). Forty (62.5%) of
the 64 animals with bite lesions died perinatally. Cataracts were found in 10% (n=18) of the
cases (Table 1). Cataract was bilateral in 16 cases, one case was unilateral, and in one case it
could not be reconstructed from the report whether the noted state was uni- or bilateral. The
one viscacha with unilateral cataract showed cystic degeneration of the left lens, and focal
ablation of the retina was suspected. It was born before the diet change. In three animals the
cataract was described as cortical and in three cases calcification of the lens was diagnosed
(Table 3). Two animals with cataract were originally free-ranging plains viscachas from
Argentina. Sixteen of 56 (29%) animals that were born before the diet change and only two of
121 animals (1.65%) born after the diet change developed cataracts (Table 2) - a significant
difference ($\chi^2 = 30.37, p < 0.001$). Post mortem examination revealed fatty liver in 6%
(n=11), 6 of which (55%) also had cataract; all were born before the diet change. Here too,
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There was a significant reduction of fatty liver occurrence in the high-fiber diet group ($x^2 = 25.34, p < 0.001$). The age distribution was different between the two groups that were compared. While diet 1 (low-fiber diet) was fed, 44% of plains viscachas that died were younger than one year, while during the time of diet 2, 81% of plains viscachas were younger than one year. However, medical record analysis documented that cataract development was independent of age (Mann Whitney U test: $U = 12, p = 0.57$). The onset of cataract was recorded in 11 viscachas that were born in the zoo. Five animals developed cataract under one year, in 4 the cataract was noticed between the age of 2 – 4 years. One animal was older than 6 years, and the two wild caught viscachas developed cataract after more than 7 years in captivity.

DISCUSSION

The findings support the conclusion that the change of the dietary regime from a ration with a high proportion of fruits, grains, and bread to a diet with a high proportion of roughage led to a reduction of cataract and fatty liver in the captive plains viscachas at Zurich Zoo. Cataract formation in plains viscachas appears to indicate a diabetic status. This interpretation is supported by elevated fructosamine, glucose, and cholesterol values recorded in animals before the diet change,

$^{34}$ and implies that cataract is caused by diabetes. This is in accord with literature reports in other rodents e.g. degus$^{7}$, sand rats$^{31}$, and in rats with streptozotocin induced diabetes$^{38}$. Other etiologies are possible for cataract, such as inheritance, toxins or medication, injury of the lens or inflammation of the eye. In dogs and humans an age-related cataract is known, and additionally, dietary deficiencies can cause cataract.$^{8}$ To our knowledge, nothing but the diet was changed in the husbandry of the viscachas at Zurich Zoo, making other causes unlikely.
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In diabetic animals, fatty degeneration of the liver occurs as the result of lipid accumulation in the liver due to increased lipid mobilisation.\textsuperscript{24} Fatty infiltration of the liver is also reported in sand rats which are fed a normal laboratory chow that induces diabetes mellitus type 2 in this species.\textsuperscript{23} Reasons for the finding that fatty livers and cataract appeared to rather occur alternatively, and less so together, in animals before the diet change, cannot be elucidated. We can only speculate that susceptibility to fatty liver and cataract formation could differ between individuals, or between different stages during diabetes.

Cataract formation in diabetic animals is caused by abnormally elevated glucose concentration in the lens. The enzyme aldose-reductase converts glucose to sorbitol, which increases the osmotic pressure in the lens; this causes water to flow in, and results in clouding.\textsuperscript{4} Cataract formation in plains viscachas might be accelerated by an increased aldose-reductase activity in the lens similar to degus with type 2 diabetes mellitus.\textsuperscript{7} Diabetic cataract is always bilateral. In the animal with unilateral cataract, a trauma-related etiology was suspected. In three cases cataract was described as cortical and in three cases calcification of the lens was diagnosed; in the other viscachas the localization of cataracts was not specified. Dystrophic calcification is associated with hypermature cataracts.\textsuperscript{8} Dogs fed a 30% galactose diet develop cataract, that approaches from initial accentuations of the sutures to cortical opacities.\textsuperscript{30} In humans, senescent cortical cataracts are generally associated with derangement of electrolyte and water balance.\textsuperscript{8}

Further nutritional etiologies for cataract are described in a review of Bunce et al.\textsuperscript{4} i.e. a riboflavin deficient diet for carnivores such as young cats (\textit{Felis catus}),\textsuperscript{13} chinook salmons (\textit{Oncorhynchus tshawytscha})\textsuperscript{15}, rainbow trout (\textit{Oncorhynchus mykiss})\textsuperscript{17} and for omnivores like young rats (\textit{Rattus norvegicus})\textsuperscript{9} and young pigs (\textit{Sus scrofa domestica}).\textsuperscript{25} Prenatal deprivation of vitamin E can also lead to cataract formation in the omnivorous turkey (\textit{Meleagris gallopavo})\textsuperscript{4} and the rat\textsuperscript{4}. Cataract is not described in conjunction with vitamin C deficiency, but scorbutic guinea pigs (\textit{Cavia porcellus}) which were fed a high-galactose diet
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developed cataract sooner than their ascorbate-supplemented littermates.\textsuperscript{4} Deficiencies of some essential aminoacids may also have a cataractogenic effect. Deficiency of phenylalanine\textsuperscript{21}, tryptophan\textsuperscript{4}, and histidine\textsuperscript{21} causes cataract in the omnivorous rat. In the kitten\textsuperscript{26} and atlantic salmon\textsuperscript{3} (\textit{Salmo salar}) (carnivores) histidine deprivation leads to cataract. Cataract is caused by methionine deficiency in fingerling atlantic salmon\textsuperscript{4}.

Some mineral deficiencies also cause cataract, e.g. in association with hypocalcemia due to renal failure or hypoparathyroidism in dogs.\textsuperscript{8} Nutritional hypocalcemia with resultant cataract is described in rabbits\textsuperscript{4} and rats\textsuperscript{4}. Zinc deficiency is known to cause cataract in rainbow trout\textsuperscript{20} and chinook salmon\textsuperscript{27}. Cataract also appears in the second and third generation of rats on a selenium deficient diet,\textsuperscript{4} while selenium excess too may cause cataract when administered as selenite in young rats.\textsuperscript{32} While a comparison of the mineral status of serum from free-ranging and the captive Zurich viscachas\textsuperscript{11} had revealed a generally higher mineral status in selenium, copper and zinc, these elevated levels were considered unlikely to be involved in cataract formation, because the elevated level in the captive animals were still well below reported toxic levels.

The diets were not analyzed for the various nutrients mentioned above. Mineral and vitamin deficiencies are improbable because the viscachas were supplemented with the same mineral/vitamin supplement (Multiforsa M21, Multiforsa AG, 6312 Steinhausen, Switzerland) before and after the diet change. Plains viscachas use coprophagy as a digestive strategy.\textsuperscript{6} Therefore, it is improbable that they suffer from a particular amino acid deficiency on mixed diets. One may assume that they are capable of using microbial protein as an additional source of amino acids similarly to other coprophagic animals like rabbits; rabbits can meet their amino acid needs on quite simple diets based on forage and grain by-products.\textsuperscript{5} Additionally, diet 1 had a 1.5% higher crude protein content than diet 2. In spite of the multiple possibilities, it may be concluded that diet induced diabetes is the most probable cause of cataract development in the plains viscachas of Zurich Zoo.
The authors suppose that plains viscachas develop a diabetes mellitus type 2 on a formerly common zoo diet for herbivores, because they are adapted to the barren pampas grassland and therefore also adapted to less energetic food - similar to fat sand rats (*Psammomys obesus*) which are adjusted to desert life and develop diabetes mellitus type 2 on a regular laboratory diet. The new diet for the viscachas with a high proportion of roughage was considered to be more similar to their natural diet.

Nevertheless a genetic predisposition could be present, similar to sand rats where a diabetic prone and a diabetic resistant line was isolated or similar to humans where the incidence of diabetes mellitus type 2 is increased in so-called thrifty genotypes in some populations. However, even in the case of a genetic predisposition, the diet change should be considered as an appropriate (though symptomatic) prophylactic measure.

Although both fatty liver and initial stages of sugar cataracts are reversible, diabetic cataracts may become irreversible after a prolonged period without appropriate treatment in humans; therefore, animals born before and animals born after the diet change were analyzed separately. One of the two animals with cataract that were born after the diet may have consumed high-energy dietary components (eg. carrots) of the vicunas that were housed in the same enclosure. The other animal lived in an indoor enclosure without access for visitors. It is unknown why it developed cataract, because it should theoretically never have received high-energy feed and suggests that cataract may have developed for other than dietary reasons.

**Acknowledgement**

We thank the keepers at Zurich Zoo, for their professional care and management of the animals and especially G. Hürlimann for additional information.
LITERATURE CITED


Cataracts in Viscachas


**TABLE 1.** Pathology findings in plains viscachas (*Lagostomus maximus*), which died between 5th May 1992 and 4th May 2008. More than one finding was possible.

<table>
<thead>
<tr>
<th>Condition</th>
<th>5th May 1992 - 4th May 2008</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total No of death</td>
<td>177 (100%)</td>
</tr>
<tr>
<td>Bite lesions</td>
<td>64 (36.2%)</td>
</tr>
<tr>
<td>Euthanasia</td>
<td>34 (19.2%)</td>
</tr>
<tr>
<td>Cataract</td>
<td>18 (10.2%)</td>
</tr>
<tr>
<td>Used as fodder</td>
<td>14 (7.9%)</td>
</tr>
<tr>
<td>Fatty liver</td>
<td>11 (6.2%)</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>7 (3.9%)</td>
</tr>
<tr>
<td>Neonatal, has breathed</td>
<td>6 (3.4%)</td>
</tr>
<tr>
<td>Cachexy</td>
<td>6 (3.4%)</td>
</tr>
<tr>
<td>Abscess/Phlegmon</td>
<td>5 (2.8%)</td>
</tr>
<tr>
<td>Nephritis</td>
<td>5 (2.8%)</td>
</tr>
<tr>
<td>Difficulty at birth and aftereffects</td>
<td>4 (2.3%)</td>
</tr>
<tr>
<td>Graphidium (tichostrongylid)</td>
<td>4 (2.3%)</td>
</tr>
<tr>
<td>Glucosuria</td>
<td>3 (1.7%)</td>
</tr>
<tr>
<td>Abnormalities</td>
<td>3 (1.7%)</td>
</tr>
<tr>
<td>Yersiniosis</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Sepsis</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Hepatitis</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Enteritis</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Colon prolapse</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Endometritis/metritis</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Intrauterine asphyxia</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Inadequate lung expansion</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>2 (1.1%)</td>
</tr>
<tr>
<td>Other (individual cases)</td>
<td>18 (10.2%)</td>
</tr>
</tbody>
</table>
TABLE 2. Occurrence of cataract and fatty liver in plains viscachas (*Lagostomus maximus*) born before and after the diet change from 5th May 2000 as well as occurrence of cataract and fatty liver in the entire analysed period from 5th May 1992 - 4th May 2008.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No of animals</td>
<td>56 (100%)</td>
<td>121 (100%)</td>
<td>177 (100%)</td>
</tr>
<tr>
<td>Cataract*</td>
<td>16 (29%)</td>
<td>2 (1.65%)</td>
<td>18 (10%)</td>
</tr>
<tr>
<td>Fatty liver</td>
<td>11 (20%)</td>
<td>0 (0%)</td>
<td>11 (6%)</td>
</tr>
</tbody>
</table>

* Cataract was bilateral in 16 cases, one case was unilateral and in one case it is unknown if cataract was uni- or bilateral.
TABLE 3. Pathologic findings of the eyes of all viscachas with cataract. In 16 cases cataract was bilateral, one case was unilateral and in one cases it is unknown, whether cataract was uni- or bilateral.

<table>
<thead>
<tr>
<th>Date of death</th>
<th>Viscachas with cataract born before diet change (5th May 1992 - 4th May 2000)</th>
</tr>
</thead>
<tbody>
<tr>
<td>07.11.92</td>
<td>Pronounced, bilateral cataract</td>
</tr>
<tr>
<td>24.11.92</td>
<td>Cataract bilateral, rupture of the lens capsule. In one eye synechia of the Iris and the lens. Irregular orientation and swelling of lens fibres; Morgagni bodies</td>
</tr>
<tr>
<td>24.11.92</td>
<td>Bilateral cataract. Irregular orientation and swelling of lens fibres; Morgagni bodies</td>
</tr>
<tr>
<td>07.12.92</td>
<td>Cortical cataract, bilateral. Cortical lens fibres degenerated; Focal proliferation and fibrous metaplasia of the epithelium of the lens.</td>
</tr>
<tr>
<td>13.04.95</td>
<td>Bilateral cataract</td>
</tr>
<tr>
<td>22.12.97</td>
<td>Bilateral cataract</td>
</tr>
<tr>
<td>13.07.98</td>
<td>Cataracta calcarea totalis, bilateral</td>
</tr>
<tr>
<td>27.01.99</td>
<td>Bilateral cataract</td>
</tr>
<tr>
<td>27.01.99</td>
<td>Bilateral cataract</td>
</tr>
<tr>
<td>16.03.99</td>
<td>End stage cataract, bilateral; multiple foci of calcification in the sclera</td>
</tr>
<tr>
<td>19.02.00</td>
<td>Cystic degeneration of the left lens stroma, focal intravital retina ablation suspected.</td>
</tr>
<tr>
<td>15.05.00</td>
<td>Cortical cataract, bilateral</td>
</tr>
<tr>
<td>26.05.00</td>
<td>Pronounced bilateral cataract, with partial calcification of the lense</td>
</tr>
<tr>
<td>13.11.01</td>
<td>Cataract, bilateral</td>
</tr>
<tr>
<td>30.08.01</td>
<td>Cataract (unknown if uni- or bilateral)</td>
</tr>
<tr>
<td>29.10.02</td>
<td>Bilateral cataract</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date of death</th>
<th>Viscachas with cataract born after diet change (5th May 2000 - 4th May 2008)</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.01.05</td>
<td>Bilateral cataract</td>
</tr>
<tr>
<td>07.03.05</td>
<td>Pronounced cortical cataract, bilateral</td>
</tr>
</tbody>
</table>