FATTY LIVER SYNDROME IN CAPTIVE BUSTARDS: CLINICAL, PATHOLOGICAL AND EPIDEMIOLOGICAL FINDINGS

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Running title: FATTY LIVER IN CAPTIVE BUSTARDS

SUMMARY

Clinical, pathological, and epidemiological findings are presented on fatty liver syndrome mainly in houbara bustards (Chlamydotis undulata macqueenii) but also in some other bustard species. Of 72 houbara bustards, 34 (47%) had fatty liver diagnosed post-mortem. Males and females were equally susceptible, and both adults and juvenile birds were affected. Bustards with fatty liver had significantly greater abdominal fat reserves than unaffected birds. Other predisposing factors included poor husbandry, translocation between aviaries, handling, and capture paresis.

INTRODUCTION

Fatty liver syndromes, loosely defined as the accumulation of more lipid in the hepatocytes than would normally be expected, have been described in many species, including cattle, goats (Black *et al*., 1988), sheep (Richards & Harrison, 1981), other ruminants, dogs (Jubb *et al*., 1993), cats (Thornburg *et al*., 1982), and horses (Jeffcott & Field, 1985). Fatty liver has been reported in a number of avian species including domestic fowl (Butler, 1976), geese, turkey (Gazdinski *et al.*, 1994), duck, various psittacines (Murphy, 1992; Baker, 1980), merlins (Cooper & Forbes, 1983; Forbes & Cooper, 1991), and other species (Wadsworth *et al*., 1984).

The houbara bustard (Chlamydotis undulata) is found as three subspecies across North Africa and into Asia. The exact status of houbara bustard populations is unknown, but they have declined in number over most of their range during the last few decades, due to the combined effects of intensive farming practices, pesticide use, industrial development, over-hunting and disturbance (Collar, 1979, 1980; Cramp & Simmons, 1987). The houbara bustard has been defined by the International Union for the Conservation of Nature and Natural Resources (IUCN) as "vulnerable"; a category which includes populations that are seriously depleted, or decreasing due to over-exploitation or other factors, and which are at risk of becoming "endangered" if causal factors continue unchecked (IUCN, 1994).

The study described here was initiated after finding fatty liver disease during post-mortem examination of houbara bustards under the care of the National Avian Research Center (NARC), United Arab Emirates (UAE). The NARC is a scientific and conservation organisation concerned primarily with the conservation of the houbara bustard, but also involved in captive breeding programmes for other bustard species. Fatty liver was first confirmed microscopically during examination of livers from seven houbara bustards from a private farm in the UAE, five of which had features typical of fatty change (Nicholls, unpublished data). A survey of post-mortem findings in bustards in the UAE (Bailey *et al*., in press) suggested that fatty liver syndrome was present in 37% of houbara bustards, although not all of these were examined histologically.

MATERIALS AND METHODS

Findings from previous post-mortem examinations of captive birds were reviewed. Formalin-fixed tissues were retrieved from the NARC tissue archive. All available bustard livers were retrieved, paired with kidneys where possible, from birds examined post-mortem between August 1992 and March 1995. The fixed tissues were sent to the Central Veterinary Research Laboratory, Dubai, for routine processing to paraffin wax, sectioning, and staining with haematoxylin and eosin. Sections were examined microscopically, and the pathologist was unaware of each section's origin. Each liver was graded on an integer scale from 0 (no significant fatty change) to 3 (marked fatty change).

For the purposes of this study, livers scored as Grade 0 or 1 were not regarded as having fatty liver disease, and livers scored as Grade 2 or 3 were defined as having fatty liver disease. When available, kidneys were examined microscopically, and graded on a similar scale.

Analyses were performed to investigate correlations between fatty liver disease and species, age, sex, body weight, body condition score (estimated by pectoral muscle palpation, using a value of 1 for emaciated, 2 for poor, 3 for fair, 4 for good, and 5 for obese), abdominal fat weight, time since translocation, and management details. The results were analysed using either the chi-squared test or the t-test (Swinscow, 1983). Blood samples for biochemistry analysis were collected from ten houbara bustards within a week prior to death. The details of methods for blood collection and analysis are given elsewhere (D'Aloia *at al.*, 1996). Briefly, each bird was manually restrained whilst 0.5 ml of blood was withdrawn from a brachial vein, using 3 ml disposable syringes and 23 x 5/8 inch disposable needles. Blood was allowed either to clot for serum collection, or lithium heparin was used as an anticoagulant prior to centrifugation to separate plasma. A Kodak Ektachem DT II system (Eastman Kodak Co., U. S. A.) and a Dimension Photoanalyser (Dupont, U. K.) were used for blood analysis.

RESULTS

The sex distribution of each group is given in Table 1, and it can be seen that the sex ratios were close for all the species examined except the black bustard (Eupodotis afra), of which there was only one (a male, with liver score of 0 and kidney score of 0). The black bustard data will not be discussed further.

Tables 2 and 3 show the organ fatty scores and prevalence by sex respectively for each species. These differences in disease incidence between the species were not found to be statistically significant.

The relationships between abdominal fat depot, body condition, body weight, and fatty liver disease are illustrated in Table 4. In bustards with fatty liver, the mean weight of abdominal fat was greater than in those without fatty liver. Mean pectoral score was not correlated with presence or absence of fatty liver. The mean body weight of birds with fatty liver was greater than those without fatty liver, although the difference was not statistically significant. It should be noted that the mean body weights of affected and unaffected birds were both lower than clinically normal birds now in the NARC collection.

The correlation of gross appearance of the liver at post-mortem examination with subsequent microscopic diagnosis was poor, and 20% of livers diagnosed as fatty (score 2 or 3) by microscopy were not recognised as fatty by gross examination post-mortem. Additionally, 8% of livers were diagnosed as fatty in appearance at post-mortem inspection, but were not found to have prominent fatty change by microscopic examination. Of the eight kidneys diagnosed microscopically as having

significant fatty change, none was diagnosed grossly as being fatty. Two kidneys thought to be fatty by visual inspection post-mortem were not found to have significant microscopic changes.

Analysis of fatty liver score by bird origin was undertaken to investigate correlation of husbandry system with prevalence of fatty liver. Three groups of houbara bustards were examined, including two captive sources and one imported source (Table 5). There was a greater prevalence of fatty liver in birds from Source 3 (captive) than Source 1 (imported). For rufous-crested bustards (Eupodotis ruficrista) there were birds from captive, imported, and unknown sources (Table 5). The only three cases of fatty liver in this species were in the captive group, although the data set is too small for meaningful analysis. Although three of the 10 kori bustards (Ardeotis kori) did have fatty liver, all these birds were in the same captive group, and so a comparative analysis was not possible.

Fatty liver was seen in approximately one fifth of juvenile (under one year) rufous-crested bustards and kori bustards, although only two juvenile houbara bustards were examined, neither of which had fatty liver. Fatty kidney was not present in the small number of juvenile birds examined.

A histogram depicting the incidence of fatty liver with respect to time after movement from Source 3 (private farm) to NARC aviaries is shown in Figure 1. It can be seen that there was a high incidence of fatty liver within 0 - 3 months after movement. Of the twenty birds that died in this period, fifteen died within the first week after movement.

A further analysis looked at the incidence of fatty liver with respect to other conditions diagnosed post-mortem or clinically. The results are summarised in Table 6. For houbara bustards, it can be seen that the number of birds in each group suffering simultaneous multiple diseases was similar. Capture paresia and a history of recent handling were more common in the birds with fatty liver. No clear pattern was seen in the rufous-crested bustards, although those birds with fatty liver appeared to have less concurrent disease than those without fatty liver. It should be noted that the two species were under different management, which could explain the differences in disease prevalence.

The clinical biochemistry of houbara bustards with fatty liver is summarised in Table 7. All data (except the reference values) are for birds in which fatty liver was confirmed by histological examinations post-mortem. Glucose, total protein, albumin, alanine aminotransferase (ALT), and alkaline phosphatase were lower in birds with fatty liver. Aspartate aminotransferase (AST) levels were increased, and lactate dehydrogenase (LDH) and creatine kinase (CK) appeared high in this study, although normal values were not available for this species.

DISCUSSION

These findings indicate that fatty liver disease can be an important condition of bustards in captivity. In order to manage this condition it is important to try and identify causal factors, or birds which are more at risk. Fatty liver may have a multifactorial pathogenesis, and this is well reviewed by Butler

(1976). There are some notable differences between the fat metabolism of birds and that of mammals, which are discussed by Murphy (1992) and Squires & Leeson (1988).

Signalment

The apparent greater prevalence of fatty liver in houbara bustards compared to rufous-crested bustards must be interpreted with caution, since each species may have been under different management or have had different origins. Fatty liver syndromes in commercially managed poultry flocks may show sex biases for purely managemental reasons, such as the fatty liver-haemorrhagic syndrome seen in commercial egg-laying flocks (Pearce & Balnave, 1978). Since hormonal causes are also postulated in fatty liver syndromes (Stake *et al*., 1981) it would not be unreasonable to expect a difference in incidence between the sexes. The analysis of incidence by sex in this study showed no such sex-associated risk. The small number of juveniles (birds under one year) in this study makes a realistic assessment of age-related risk impossible, however, the presence of fatty liver in some juvenile birds means that young birds certainly are at risk from fatty liver disease. In summary, fatty liver can occur in houbara, rufous-crested, and kori bustards. Females and males appear equally susceptible to fatty liver, and juvenile birds as well as adults may suffer from the disease.

Body condition

Since fatty liver can be seen in animals in good condition, often following reduced appetite or increased energy demand, it is important to see whether affected bustards were in good, average, or poor body condition. Although male bustards with fatty liver disease at post-mortem examination had a slightly higher mean body weight than bustards with no fatty liver, both groups were underweight compared with the mean body weight for healthy male bustards (1570 g, range 1290 - 1720 g) (Bailey, unpublished data). The same findings were true for female houbara bustards, since healthy female birds have a mean body weight of 1050 g, range 950 - 1170 g, (Bailey, unpublished data). Body condition assessment by palpation of pectoral muscle scoring was not found to be a useful predictor of fatty liver disease. A limited survey of abdominal fat weights in free-living houbara, killed during hunting, found that two females had no significant fat, whilst a third had 65 g (5.3 % body weight) of abdominal fat. Two males were available and had abdominal fat depots of 0 g and 30 g (1.6% body weight) (Bailey, unpublished data). The data set from free-living bustards is too small to gain a reliable impression of normal abdominal fat weights, especially since there may be cyclic variations in body weight associated with the breeding season (Jacquet, 1995), but these preliminary impressions suggest that abdominal fat weights might be a useful indicator of risk for fatty liver disease, with those birds having large abdominal fat depots being more at risk than those with smaller abdominal fat depots. This would be consistent with a proposed pathogenesis involving overload of the liver by sudden release of fat from the depots of birds in good condition, due to intercurrent disease or reduced feed intake for other reasons. It would also be consistent with

a diet that is high in fat but low in nutrients such as protein, biotin or choline, which are needed for fat metabolism in the liver (Bain *et al*., 1988). Since these values were recorded after death, it would be useful to know the weights and condition scores of birds before the onset of disease. This would involve regular weighing of birds, with a retrospective analysis of weight changes in birds which died from fatty liver and other diseases. The current data show the weights of affected birds at death, and so it is difficult to know whether fatty liver disease is associated with periods of weight loss or weight gain.

Source and management of birds

Husbandry conditions such as diet and housing are known to affect the incidence of fatty liver disease in other avian species (Blair *et al*., 1975; Pearson & Butler, 1978). The data presented in Table 5 suggest that the source, and presumably the management, of birds can affect the risk of developing fatty liver disease. The significance of the different incidence at Al Ain Zoo compared with the private farm should be assessed alongside available data on differences in management, such as diet and housing, between the two sites. At the private farm the birds were housed at a high stocking density (in large aviaries), in poorly ventilated and dusty conditions, with irregular feeding and variable diet composition (including minced red meat, bread, and chopped fruit). Many birds at the farm were found to be in poor body condition when initially examined. There may have been competition for food due to the small number of feeding bowls, and occasionally birds went without food for short periods due to lapses in management. Husbandry at Al Ain Zoo, before NARC management, involved a high stocking density (in small, poorly ventilated rooms), and likely overfeeding on a diet of minced red meat, bread, alfalfa and chopped fruit (Anderson, 1995). These birds were found to be in good body condition. Intermittent periods of poor food supply, especially if certain nutrients are inadequate, is a likely predisposing factor for fatty liver disease, and diet is certainly an important factor in the pathogenesis of fatty liver disease (Blair *et al*., 1975). Lead toxicosis in birds eating flakes of paint in aviaries at the private farm has been noted (Bailey *et al*., 1995) and lead has been reported as a cause of fatty liver disease in poultry (Murphy, 1992). Of livers available from the eleven captive birds which died in Al Ain Zoo (source 2), both deaths in 1992 (before NARC took over management of the flock) were fatty liver positive, three of seven were positive in 1993 (the period of change of management), and neither of the two birds dying in 1994 (under NARC management with specially formulated pelleted diet, vitamin supplements, small group size and natural style aviaries) had fatty liver. For the rufous-crested bustards, fatty liver was seen only in captive birds. This might suggest that captive conditions do predispose to the development of fatty liver disease in these birds, although the data set is limited.

Capture, transport and translocation

Figure 1 shows that relocation was associated with a period of high mortality in the first few weeks, with a decreasing incidence over the subsequent months. The birds were manually caught and transported in cardboard boxes by car from a private farm (source 3) to NARC aviaries at Al Ain Zoo. The whole process could last from 3 - 5 hours. Many birds suffered from capture paresia on the day of translocation, with many deaths occurring in the first week (some with no previous signs). Moreover, there were a large number of cases of fatty liver in these deaths. This suggests that capture, transport, translocation and their associated stresses are associated with increased mortality (all causes) and prevelance of fatty liver disease.

Association with other disease

The data in Table 6 show that many birds had multiple conditions diagnosed at post-mortem examination, although this did not appear to be a predisposing factor by itself for fatty liver. Capture paresia, and a history of recent handling (but no observed paresia), appeared to predispose to fatty liver disease in houbara bustards, perhaps due to an associated reduction in food intake. No causal relationship can be assumed, since fatty liver could have predisposed the birds to other diseases.

Clinical biochemistry findings

The results are summarised in Table 7. Normal blood values for houbara bustards (two sub-species) are given elsewhere (D'Aloia *et al*., 1996). Although glucose was reduced in houbara bustards with fatty liver disease, serum was separated from clotted samples 6 - 12 hours after collection, and frozen for up to 12 months before analysis. This may have allowed glucose levels to fall during the post-collection interval. Although low blood glucose is seen in fatty liver disease (Austin & Scott, 1991), in this case the low glucose could also have been due to the method of collection. The low total protein and albumin are consistent with liver failure, or may reflect poor nutrition. Aspartate aminotransferase (AST) levels were increased, consistent with fatty liver and other liver diseases (Bogin *et al*., 1984; Murphy, 1992). Alanine aminotransferase (ALT) levels were lower than in normal birds, but have been regarded as being of controversial diagnostic value in some species (Lumeij & Westerhof, 1987) and may not be raised in turkeys with liver damage (McDougald & Hansen, 1970). LDH has been reported as being increased in fatty liver disease (Bogin *et al*., 1984; Murphy, 1992), although it is not specific for liver disease and has been suggested to be a rather poor indicator of liver damage on other avian species (Lumeij, 1993). LDH and CK appeared high in this study, although normal values were not available for this species. Increased phosphorus levels have been recorded in laying hens with fatty liver syndrome (Miles & Harms, 1981), although the increase found in this study was not significant. Potassium levels were low, but again this could have been due to movement from plasma to erythrocytes after collection (Lumeij, 1993).

Possible methods of diagnosis of fatty liver disease.

Clinical signs of fatty liver disease include depression (Murphy, 1992), sudden death (Cooper & Forbes, 1983), reduced growth, lethargy, aphagia, recumbency, sometimes paralysis (Austin & Scott, 1991), or simply increased mortality / sudden death (Gazdinski *et al*., 1994). In these birds, although other clinical diseases were often present, it was not possible to attribute any specific signs to fatty liver disease. Therefore, other means of diagnosis may be needed. The predisposing factors described above, such as capture, handling and poor husbandry, could be used to identify groups of birds at increased risk of fatty liver disease. More specific tests might include blood sampling (to look for signs of liver disease), possibly including serum bile acids (Flammer, 1994), endoscopy and / or liver biopsy (Murphy, 1992; Lothrop *et al*., 1986). Increased liver size might be detected either endoscopically or radiographically. Otherwise, diagnosis can be confirmed post-mortem, although it should be noted that gross examination without histology was often unreliable as a means of identifying fatty liver, being wrong in either direction.

Prevention

Preventive measures might include reducing the stress of capture and transport. The addition of vitamin E to the feed as a preventive or therapeutic measure has also been recommended for commercial poultry (Gazdinski *et al*., 1994), although this would depend on what the current levels of vitamin E and selenium in the current feed are found to be. Preventive treatment with vitamin E (for example by administration via the drinking water) might be a useful measure prior to capture and transport. Avoidance of heat stress and prolonged periods of recumbency in transport boxes might reduce the level of capture paresia, and associated fatty liver disease. The levels of dietary components such as choline, biotin, vitamin E, and selenium might also be analysed to check for possible deficiencies (Butler, 1976; Squires & Leeson, 1988).

ACKNOWLEDGEMENTS

The authors would like to thank the Crown Prince of Abu Dhabi, H. H. Sheikh Kalifa bin Zayed Al Nahyan, president of the National Avian Research Center, H. H. Sheikh Mohammed bin Zayed Al Nahyan, Chairman of the Board, and Mr M. Al Bowardi, Managing Director, for their support of this work. We also thank Dr. J. Naldo and Mr. A. Azur for technical assistance, Dr. B. Nothelfer, Dr. U. Wernery and Dr. Khalig at the Central Veterinary Research Laboratory, Dubai, UAE, for preparing the histology sections, and Professor J. E. Cooper for reviewing the manuscript.

REFERENCES

ANDERSON, S. (1995). *Bustard micro-nutrient review*. NARC External Report No. 4. National Avian Research Center, Abu Dhabi, United Arab Emirates.

AUSTIN, R. E., & SCOTT, M. L. (1991) Nutritional Diseases, in: B. W. Calnek (Ed) *Diseases of Poultry,* 9th edn, pp. 59-61. (London, Wolfe Publishing Ltd).

BAILEY, T. A., NICHOLLS, P. K., SAMOUR, J. H., NALDO, J., WERNERY, U., & HOWLETT, J. (1996). Post-mortem findings of bustards in the United Arab Emirates. *Avian Diseases,* **40** In Press.

BAILEY, T. A., SAMOUR, J. H., NALDO, J. & HOWLETT, J, C. (1995) Lead toxicosis in a flock of houbara bustards. *Veterinary Record,* **137**, 193 - 194.

BAIN, S. D., NEWBREY, J. W., & WATKINS, B. A. (1988). Biotin deficiency may alter tibiotarsal bone growth and modelling in broiler chicks. *Poultry Science*, **67**, 590 - 595.

BAKER, J. R. (1980). A survey of the causes of mortality in budgerigars (Melopsittacus undulatus). *Veterinary Record*, **106**, 10 - 12.

BLACK, H., HUTTON, J. B., SUTHERLAND, R. J., JAMES, M. P.(1988). White liver disease in goats. *New Zealand Veterinary Journal*, **36**, 15 -17.

BLAIR, R., WHITEHEAD, C. C., & TEAGUE, P. W. (1975). The effect of dietary fat and protein levels, form and cereal type on fatty liver and kidney syndrome in chicks. *Research in Veterinary Science,* **18**, 76 - 81.

BOGIN, E., AVIDAR, Y., MEROM, M., ISRAELI, B., MALKINSON, M., SOBACK, S., & KUDLER, Y. (1984). Biochemical changes associated with fatty liver in geese. *Avian Pathology*, **13**, 683 - 701.

BUTLER, E. J. (1976). Fatty liver diseases in the domestic fowl: A review. *Avian Pathology*, **5**, 1 - 4 .

COLLAR, N. J. (1979). The world status of the houbara bustard: a preliminary review. In: *Symposium papers on the Great bustard Otis tarda (Sofia, Bulgaria, May 26, 1978) and the houbara bustard Chlamydotis undulata (Athens, Greece, May 24, 1979).* FISG/CIC/Game Conservancy, pp. 1 - 12.

COLLAR, N. J. (1980). Bustards in decline. *British Birds,* **73**, 178 - 199.

COOPER, J. E., & FORBES, N. (1983). A fatty liver-kidney syndrome of merlins. *Veterinary Record*, **112**, 182 - 183.

CRAMP, I. S., and SIMMONS, K. E. L. (1987). *Handbook of the Birds of Europe, the Middle East and North Africa. The Birds of the Western Palearctic.* Vol. 2. Hawks to Bustards pp. 636 - 668. Oxford University Press, Oxford, U.K.

D'ALOIA, M. -A., SAMOUR, J. H., HOWLETT, J. C., BAILEY, T. A., & NALDO, J. (1996). Normal blood chemistry of the houbara bustard (Chlamydotis undulata). *Avian Pathology*, **25**, 167 - 173.

FLAMMER, K. (1994). Serum bile acids in Psittacine birds. *Proceedings of the Association of Avian Veterinarians*, pp. 9 - 12.

FORBES, N., & COOPER, J. E. (1991). Diseases and pathology of the European Merlin (Falco columbarius). In: *Biology and Conservation of Small Falcons, Proc. Hawk and Owl Trust Conference,* pp. 100 - 116.

GAZDINSKI, P., SQUIRES, E. J., & JULIAN, R. J. (1994). Hepatic lipidosis in turkeys. *Avian Diseases*, **38**, 379 - 384.

I.U.C.N. (1994). *IUCN Red List of Threatened Animals*. International Union for the Conservation of Nature and Natural Resources, Cambridge, U.K. 1994, p. 44.

JACQUET, J.-M. (1995). Food intake in the houbara bustard: relationship with body weight and ambient temperature. NARC Internal Research Report. National Avian Research Center, Abu Dhabi, United Arab Emirates.

JEFFCOTT, L. B., & FIELD, J. R. (1985). Current concepts of hyperlipaemia in horses and ponies. *Veterinary Record*, **116**, 461 - 466.

JUBB, K. V. F., KENNEDY, P. C., & PALMER, N. (Eds.) (1993). *Pathology of the Domestic Animals*. 4th Ed, Vol 2. Ch 2. The Liver and Biliary System pp. 330-337. Academic Press Inc., London.

LOTHROP, C., HARRISON, G. J., SCHULZ, D., & UTTERBRIDGE, T. (1986). Miscellaneous Diseases, in: HARRISON & HARRISON (Eds) *Clinical Avian Medicine and Surgery*, p. 526. (Philadelphia, W. B. Saunders Co.).

LUMEIJ, J. T. (1993). Avian plasma chemistry and health and disease. *Proceedings of the American Association of Avian Veterinarians*, pp. 20 - 27.

LUMEIJ, J. T., & WESTERHOF, I. (1987). Blood chemistry for the diagnosis of hepatobiliary disease in birds; a review. *The Veterinary Quarterly*, **9**, 255 - 261.

MCDOUGALD, L. R., & HANSEN, M. F. (1970). Histomona meleagridis. Effect on plasma enzymes in chickens and turkeys. *Experimental Parasitology,* **27**, 229 - 235.

MILES, R. D., & HARMS, R. H. (1981). An observation of abnormally high calcium and phosphorus levels in laying hens with fatty liver syndrome. *Poultry Science*, **60**, 485 - 486.

MURPHY, J. (1992). Psittacine fatty liver syndrome. *Proceedings of the American Association of Avian Veterinarians*, pp. 78 - 82. New Orleans.

PEARCE, J., & BALNAVE, D. (1978). A review of biotin deficiency and the fatty liver and kidney syndrome in poultry. *British Veterinary Journal*, **134**, 598 - 609.

PEARSON, A. W., & BUTLER, E. J. (1978). Environmental temperature as a factor in the etiology of fatty liver hemorrhagic syndrome in the fowl. *Research in Veterinary Science*, **25**, 133 - 138.

RICHARDS, R. B., & HARRISON, M. R. (1981). White liver disease in lambs. *Australian Veterinary Journal*, **57**, 565 - 568.

SQUIRES, E. J., & LEESON, S. (1988). Aetiology of fatty liver syndrome in laying hens. *British Veterinary Journal*, **144**, 602 - 609.

STAKE, P. E., FREDRICKSON, T. N., & BOURDEAU, C. A. (1981). Induction of fatty liver-haemorrhagic syndrome in laying hens by exogenous beta-estradiol. *Avian Diseases*, **25**, 410 - 422.

SWINSCOW, T. D. V. (1983). *Statistics at Square One*. 8th Ed. pp. 33 - 53. (London, British Medical Journal).

THORNBURG, L. P., SIMPSON, S., & DIGILIO, K. (1982). Fatty liver syndrome in cats. *Journal of the American Animal Hospital Association*, **18**, 397 - 400.

WADSWORTH, P. F., JONES, D. M., & PUGSLEY, S. L. (1984). Fatty liver syndrome in birds at the Zoological Society of London. *Avian Pathology*, **13**, 231 - 239.

Table 1. Sex distribution of livers examined.

Species	No. males	No. females	No. unknown sex
Houbara bustard	37 (35)[1]	33 (33)	2 (0)
Rufous-crested bustard	7 (6)	9 (9)	7 (4)
Kori bustard	3 (3)	3 (4)	4 (4)
Black bustard	1 (1)	0 (0)	0 (0)

[1] Figures in brackets represent number of kidneys examined in each group.

Table 2. Organ fatty scores by species.

Score	Houbara		Rufous-crested		Kori	
	Liver	Kidney	Liver	Kidney	Liver	Kidney
0	14	42	11	15	3	6
1	24	17	9	4	4	5
2	22	7	3	-	3	-
3	12	2	-	-	-	-
Total	72	68	23	19	10	11

Table 3. Fatty liver and kidney prevalence by sex.

Diagnosis	Houbara		Rufous-crested			Kori		
	M	F	M	F	U	M	F	U
Fatty liver	18	15	0	1	2	1	1	1
Fatty kidney	4	5	-	-	-	-	-	-

M = male, F = female, U = unknown

Table 4. Fatty liver prevalence and abdominal fat weight, pectoral muscle condition score, and body weight (houbara bustards).

	Sex	Fatty liver disease			No fatty liver disease			
		Mean	SD	N	Mean	SD	N	P
Abd. fat (g)	Male	41	28	12	7	4.3	8	0.005
	Female	31	8.1	9	5	14	7	0.03
Muscle score	Male	2.0	0.82	13	2.8	1.1	17	0.04
	Female	3.0	0.76	8	2.1	1.2	17	0.05
Body wt. (g)	Male	1341	289	15	1255	213	16	0.34
	Female	925	162	12	842	146	15	0.17

Table 5. Prevalence of fatty liver and kidney by source.

Species	Organ	score	S1	N	S2	N	S3	N
Houbara	Liver	0 - 1	21	29	6	11	11[1]	32
		2 - 3	8		5		21	
	Kidney	0 - 1	25	30	8	9	26	29
		2 - 3	5		1		3	
Rufous	Liver	0 - 1	5	5	13	16	2	2
		2 - 3	0		3		0	
	Kidney	0 - 1	5	5	12	12	2	2
		2 - 3	0		0		0	

[1] Of these eleven birds, eight had liver scores of 1, indicating some fatty change.

S1 = Imported houbara and rufous-crested bustards.

S2 = Captive houbara (Al Ain Zoo) and rufous-crested bustards.

S3 = Captive houbara (private farm) bustards, rufous-crested bustards of unknown source.

Table 6. Fatty liver disease and other conditions diagnosed post-mortem.

	Houbara bustards		Rufous-crested bustards	
Disease	Fatty liver	Non-fatty	Fatty liver	Non-fatty
multiple diseases	25	26	1	6
capture paresis	12	1	-	-
handling	5	1	0	4
aspergillosis	24	22	-	-
trauma	16	16	2	7
endoparasites	7	11	0	3
trichomoniasis	2	4	0	2
paramyxovirus	1	5	0	4
lameness	4	8	0	1
bacterial infection	-	-	2	3
enteritis	-	-	0	3
heartworm	-	-	0	4
other	8	1	0	1

Table 7. Biochemical findings in houbara bustards with fatty liver disease

value	fatty liver mean +/- SEM	n	normal mean +/- SEM	n
Glucose (mg/dl)	195 +/-17	8	289+/-7	38
Uric acid (mg/dl)	11 +\- 1.4	6	9.8+/-0.4	36
Creatinine (mmol/l)	0.27 +/-0.06	6	0.34+/-0.02	35
Bilirubin (mg/dl)	0.57 +/-0.14	7	0.5+/-0.2	35
Total protein (g/dl)	2.9+/-0.2	9	3.6+/-0.1	37
Albumin (g/dl)	1.0+/-0.1	8	1.4+/-0.3	38
Globulin (g/dl)	1.9+/-0.2	7	2.1+/-0.6	36
AST (IU/l)	486+/-86	6	373+/-13	37
ALT (IU/l)	19+\-6	4	36+/-2.4	31
Alkaline phosphatase (IU/l)	48+/-5	7	137+/-13	38
Lactate dehydrogenase (IU/l)	938+/-4	7	-	-
Creatine kinase (IU/l)	494+/-181	7	-	-
Carbon dioxide (mmol/l)	31+/-5	4	26+/-0.8	37
Magnesium (mg/dl)	3.6+/-0.8	5	2.6+/-0.1	38
Phosphorus (mg/dl)	5.1+/-0.7	10	4.1+/-0.4	38
Calcium (mg/dl)	8.7+/-0.6	10	9.6+/-0.4	38
Potassium (mmol/l)	2.9+/-0.8	5	3.9+/-0.1	35
Sodium (mmol/l)	130+/-33	5	151+/-2	38
Chloride (mmol/l)	116+/-7	4	115+/-1	34

Table 7. Biochemical findings in houbara bustards with fatty liver disease

value	1 - 7 days mean +/- SEM	n	1 - 20 months mean +/- SEM	n	normal mean +/- SEM	n
Glucose (mg/dl)	195 +/-17	8	173+/-27	5	289+/-7	38
Uric acid (mg/dl)	11 +\- 1.4	6	9.8+/-1.3	5	9.8+/-0.4	36
Creatinine (mmol/l)	0.27 +/-0.06	6	0.17+/-0.7	3	0.34+/-0.02	35
Bilirubin (mg/dl)	0.57 +/-0.14	7	0.23+/-0.13	2	0.5+/-0.2	35
Total protein (g/dl)	2.9+/-0.2	9	2.5+/-0.3	7	3.6+/-0.1	37
Albumin (g/dl)	1.0+/-0.1	8	0.5+/-0.0	2	1.4+/-0.3	38
Globulin (g/dl)	1.9+/-0.2	7	-	-	2.1+/-0.6	36
AST (IU/l)	486+/-86	6	525+/-57	8	373+/-13	37
ALT (IU/l)	19+\-6	4	13	1	36+/-2.4	31
Alkaline phosphatase (IU/l)	48+/-5	7	84+/-19	4	137+/-13	38
Lactate dehydrogenase (IU/l)	938+/-4	7	826+/-116	6	-	-
Creatine kinase (IU/l)	494+/-181	7	595+/-484	6	-	-
Carbon dioxide (mmol/l)	31+/-5	4	37	1	26+/-0.8	37
Magnesium (mg/dl)	3.6+/-0.8	5	3.8	1	2.6+/-0.1	38
Phosphorus (mg/dl)	5.1+/-0.7	10	5.1+/-0.8	9	4.1+/-0.4	38
Calcium (mg/dl)	8.7+/-0.6	10	7.9+/-0.9	9	9.6+/-0.4	38
Potassium (mmol/l)	2.9+/-0.8	5	1.6	1	3.9+/-0.1	35
Sodium (mmol/l)	130+/-33	5	332	1	151+/-2	38
Chloride (mmol/l)	116+/-7	4	138	1	115+/-1	34