## SERUM CHEMISTRY AND MINERAL CONTENT OF NORMAL AND DEFORMED TARSOMETATARSAL OSTRICH (*STRUTHIO CAMELUS*) CHICKS

## M.O. Fahmy; H.H. Khalifa<sup>\*</sup> And N.S. Ibrahim.

Biolog. Applic. Dep., Nuclear Rese. Cent., Atomic Energy Authority, P.O Box 13759\* Facu. of Agric, Anim. Production Dept., Al-Azhar Univ., Cairo, Egypt.

### Received: 18/10/2006 Accepted: 29/11/2006

**Abstract:** To study leg deformation as a major factor affecting ostrich mortality, tarsometatarsus bones and blood samples were collected from 30 non-deformed and 30 deformed ostrich chicks aged from 2-10 weeks. Blood samples and tarsometatarsas bones were collected from the Ostrich Production Farm of the Atomic Energy Authority, Cairo, Egypt and analyzed for minerals content (Ca, P, Mg, Zn and Mn). Whereas, the following biochemical parameters were determined in the sera: total proteins, albumen, globulin, albumin/globulin ratio (A/G ratio), urea and alkaline phosphatase. The obtained results indicated that:

- 1. Chick mortality during the first 8 weeks after hatch ranged between 10 to 25%. Mortality due to leg deformation represented 5 to 15% of total chicks and 50 to 100% of dead chicks.
- 2. Mortality due to leg deformation (as percentage from total mortality) tended to increase with age indicating that leg deformities were the most important cause of post-hatch chick culling or mortality.
- 3. The levels of calcium, manganese and zinc in deformed tarsometatarsus bones were significantly lower than that in the normal ones.
- 4. Phosphorus and magnesium levels were significantly higher in the deformed tarsometatarsus bones than in the normal ones. As a result, the Ca/P and Ca/Mg ratios were significantly lower in deformed than in normal tarsometatarsus bones.
- 5. The mean serum calcium, manganese, Ca/P ratio, Ca/Mg ratio, and A/G ratio were significantly lower in ostrich chicks with deformed than with normal tarsometatarsus bones.
- 6. The mean serum alkaline phospatase, urea, total protein, albumin, globulin, phosphorus and zinc were significantly higher in the deformed tarsometatarsus chicks than in normal ones.

7. No significant differences were found between mean serum magnesium of ostrich chicks with deformed and normal tarsometatarsus bones.

## **INTRODUCTION**

The ostrich (*Struthio camelus*) is a flightless bird belonging to the ratite family and known as the largest living bird. Ostrich is native to semiarid and desert areas of Africa, they have been raised intensively in South Africa for more than 100 years and reared commercially mainly to produce usable products, including meat, hides, feathers, and eggs (Kreibich and Sommer, 1995).

The investment base of the world ostrich industry has increased dramatically over the last ten years. The most important challenges which have been widely felt to cause considerable financial lose to ostrich farmers and chick wastage is leg disorder. Leg deformities were reported to be the most causes of ostrich chick wastage (Bezuidenhout et al., 1994; More, 1996 and Mushi et al., 1999). Juan and Baowei (2004) summarized the causes, types and prevention measurements of leg disease in young ostrich. They concluded that the reasons of leg diseases in ostriches are complicated, it dues to multi-factors (leg diseases caused by: viruses, bacteria, vitamin and mineral deficiency and mechanical factors). As the industry develops, additional efforts must be made to address the causes and solutions to these inevitable causes of chick wastage.

Lack of knowledge on the specific nutritional needs of breeding ostriches has possibly been a major contributing factor in their historically poor breeding performance. Brand et al (2002b) stated that successful intensive commercial farming of ostriches (Struthio camelus domesticus) requires an adequate knowledge of the nutritional needs of the birds and of the components used in formulating diets. Although South Africa has had a well-established ostrich industry for over a century, information on ostrich nutrition, in particular specific nutritional requirements at different stages of production, is still sparse. Nutritional information extrapolated from the poultry industry has been used widely, but often proved unsatisfactory for ostriches and has resulted in several nutrition-related problems, especially in young, growing ostriches.

Brand et al. (1999) mentioned that nutritionists still do not agree about the nutritional standards that should be used for ostriches and ostrich diets have been formulated on the basis of poultry, turkey or pig requirements. As a result, the recommendations and composition of diets for ostriches still vary considerably. Consequently, Brand et al. (2002a) reported that a diverse recommendations regarding the protein content in starter (14.6–22%), grower (15.0–21.8%), finisher (12.0–15.6%), as well as maintenance (12.0–17.8%) and breeding diets (16.0–22.0%) are still found, while recommendations for the energy requirements of breeding birds, for example, vary between 7.9 and 10.6 MJ ME. Tully and Shane (1998) revealed that blood chemistry profiles are extremely important in the health management of ostrich.

The aim of the present study is to prove that leg deformation is mainly due to deficiency in minerals related to bone formation rather than to mechanical factors which is due to lack of knowledge on the specific nutritional needs of breeding and growing ostriches.

## **MATERIALS AND METHODS**

The present study was carried out in Ostrich Production Farm, Nuclear Research Center, Atomic Energy Authority, Cairo, Egypt. Data were collected during the period from August 2001 till October 2003. Vitamin and mineral composition of grower and breeder rations are postulated in Table (1).

## 1- Collection and preparation of tarsometatarsus bones for mineral detection:

Thirty normal and thirty abnormal tarsometatarsus bones for chicks aged from 2 to 8 weeks were collected, cleaned of all adhering tissues, rinsed with distilled water and dried over night at 100 °C. Normal bones were collected from normal growth chicks died from reasons other than leg deformation. A portion of each bone was grinded using steel mixture then 1 g was subjected to ashing according to method described by Charles <u>et al.</u>, (1984). Calcium, Mg, Zn and Mn were determined by using Atomic Absorption Spectrophotometer (Buck Scientific, Model 210 VGP). While, P content was determined using the calorimetric technique of Goldenberg and Fernandez (1966).

#### 2- Collection of blood samples:

Blood samples were collected from the right jugular vein of normal and deformed chicks at similar ages (2 to 8 weeks of age) through a clean dry needle 21 G into 10-ml test tube. Blood samples were left at room temperature for 30-60 minutes then centrifuged for 5 minutes at 4000 r.p.m. Serum was separated and transferred into 1.5 ml Apondorf tubes and stored at -20  $^{\circ}$ C until analysis.

### Serum biochemical parameters:

Serum total protein and albumin were measured calorimetrically

according to Henry (1964) and by using Albumin-Kit (Bio-Merieux Vitek Inc. USA). The concentration of globulins in each sample was obtained by subtracting the albumin values from the total protein concentration. A/G ratio was determined by dividing the value of albumin on the value of globulin in the serum. Serum urea was determined according to Patton and Crouch (1977). Alkaline phosphate was determined calorimetrically using TECO kit, (TECO, Diagnostic, California, U.S.A).

#### Serum minerals content:

Serum inorganic phosphorus was determined colorimetrically by phosphorus kit (BioAdwic, El-Nasr Co. Egypt, A.E.P). Total calcium, manganese, magnesium and zinc contents in blood serum were determined in five times serum dilution for trace elements and in 50 times for macro elements, by using Atomic Absorption Spectrophotometer (Buck Scientific, Model 210).

1994).				
Nutrient	Measure /ton	0-6 months	6 months – slaughter	Breeder
Vitamin A	IU	12000000	900000	15000000
Vitamin D3	IU	3000000	2000000	25000000
Vitamin E	IU	40000	10000	30000
Vitamin K3	gm	3	2	3
Vitamin B1	gm	3	1	2
Vitamin B2	gm	8	5	8
Niacin	gm	60	50	45
Ca Pantothenic acid	gm	14	8	18
Vitamin B12	mg	100	10	100
Vitamin B6	gm	4	3	4
Choline chloride	gm	500	150	500
Folic acid	gm	2	1	1
Biotin	mg	200	10	100
Mg	gm	50	-	40
Mn	gm	120	80	120
Zn	gm	80	50	90
Cu	gm	15	15	15
Ι	gm	0.5	1	1
Со	gm	0.1	0.3	0.1
Iron	gm	35	20	35
Se	gm	0.3	0.15	0.3
Ca	gm	12-15	9-10	20-25
Avail P	gm	4-4.5	3.2-3.6	3.5-4.0

Table (1): Vitamin, trace element and macromineral recommended for<br/>South African ostrich rations (Cilliers and van Schlakwyk,<br/>1994).

**Mortality rate:** Mortality rate (as percent) was recorded weekly up to eight weeks of age and calcified to mortality due to tibiotarsal rotation or to other reasons.

## Statistical analysis:

Data were analyzed using statistical analysis system software (SAS, 1988). Student t test (Procedure TTEST of SAS) was used to test the significance between deformed and normal groups.

### **RESULTS AND DISCUSSION**

#### **Post-hatch chick mortality:**

Chick mortality during the first 8 weeks after hatch ranged between 10 to 25%. Mortality due to leg deformation represented 5 to 15% of total chicks and 50 to 100% of dead chicks (Table, 2). The overall average of chick mortality due to leg deformation was 10% (8 out of 80 chicks) which represent 66.7% of the total chicks died during this period (8 out of 12). Table (2) reveal that from 4 to 8 weeks of age mortality rate ranged between 10-15 %, however, mortality due to leg deformation (as percentage from total mortality) tended to increase by age. In South Africa, Allwright, (1996) found that the typical chick mortality is reported to be 40% or 50% up to 3 months of age and 10% from 3-6 months of age (Smith <u>et al.</u>, 1995). While, Verwoerd <u>et al.</u>, (1998) reported that typical mortality at 1 week of age is 10-20%, and 10-30% at 3 months. From 3 to 12 months of age, mortality is typically 5%. Mortality rate in the present study is in agreement with that found by Verwoerd <u>et al.</u> (1998) and Bezuidenhout (1999) but it is lower than that reported by Allwright, (1996).

Leg deformities were reported to be the most important cause of ostrich chick wastage (More, 1996 and Mushi et al., 1999) and its incidence reached peak levels in 2-3 weeks old. Meanwhile, the lowest incidence was recorded at 10 weeks of age and no cases were observed thereafter. Bezuidenhout and Burger (1993) stated that pelvic appendicular skeletal abnormalities and lateral rotation of tibiotarsus (affected ostrich chicks between 2 weeks and 6 month of age) have a significant contribution to mortalities. The peak incidence of leg deformities was found to be around 4 to 7 weeks of age (Foggin, 1992). Morrow et al., (1997) added that many ostrich chicks developed swelling of the hock joint at 6 to 12 weeks old. The swelling was localized to the proximal extremity of the tarsometatarsus. However, the birds became unthrifty and stunted with torsional and angular deformities of the tibiotarsal and tarsometatarsal bones. Furthermore, Squire and More (1998) stated that a deficiency in one or more of the Ca, P, Mg, Mn and Zn may contribute to limb deformities as a result of poor mineralization.

The causes of mortality due to other factors during the early posthatch period were explained by Shivaprasad (1993) who found that mortality of ostrich chicks was sometimes as high as 60-100% as a result of leg deformities, gastro-intestinal diseases, yolk sac infections, liver diseases, haemopoietic disorders and respiratory diseases.

### Bone mineral content of normal and deformed tarsometatarsus:

The bone-mineral content in normal and deformed tarsometatarsus bones of the ostrich chicks are presented in (Table, 3). The levels of calcium, manganese and zinc in deformed tarsometatarsus bones were significantly lower than that in the normal ones. Meanwhile, phosphorus and magnesium levels were significantly higher in the deformed tarsometatarsus bones than in the normal ones. As a result, the Ca/P and Ca/Mg ratios were significantly lower in deformed than in normal tarsometatarsus bones. The lower Ca content in deformed tarsometatarsus bones is in consistent with the previous results that Ca deficiency may cause limb deformity (Levy et al., 1986; Huchzermeyer, 1994 and Mushi et al., 1999). Also, Bezuidenhout et al., (1994) stated that the bone calcium of deformed chick with tibiotarsal rotation was significantly lower compared with the values in normal chicks. The reduction in Ca content in deformed tarsometatarsus bones may be due to: low calcium intake i.e. diet deficient in Ca (Bezuidenhout and Burger, 1993; Rath et al., 2000; Creedon and Cashman, 2001 and Bar et al., 2003), a decrease in Ca absorption from the intestinal lumen (Yalcin et al., 2000) or ingestion of an excess of dietary calcium (Morrow et al., 1997). Perry et al., (1991) added that high levels of phytate and cellulose fibers in the diet can interfere with Ca absorption and result in hypocalcemia and decrease bone strength. Furthermore, Heany (1998) postulated that supplemental Ca with protein may be necessary to maintain optimal Ca balance and bone health. Similarly, diets consisting of highly saturated fats can have adverse effect on bone mineralization and low fat diets can increase cancellous bone strength and bone content.

On the other hand, the significantly higher P content in the deformed tarsometatarsus bones than normal ones disagrees with results of Huchzermeyer (1994) and Mushi <u>et al.</u>, (1999) who found that deformed tibiotarsal bones had a significant reduction in P content. This may be due to imbalance between diet Ca and P as suggested by Bissinger and Huchzermeyer (1998). Chen <u>et al.</u>, (1997) showed that lameness in young ostriches was caused by phosphorus deficiency (if the diet given to the ostriches had a very high Ca or low P content) so that lameness in young birds occurred with 90.9% morbidity. Nelson <u>et al.</u>, (1990) found that Ca:P ratios from 1.6:1 to 2.94:1 did not cause leg abnormalities which coincided with the ratio in normal tarsometatarsus bones, while deformed tarsometatarsus bones had lower Ca:P ratio (1.05).

The significantly lower Mn in deformed than in normal bones agrees with Dick and Deeming (1996) and Cooper (2000) who reported that Mn deficiency has been implicated in deformed leg syndrome, slipped tendons and porosis.

The obtained results of significantly lower Zn content in deformed than in normal bones agree with that reported by Sunde (1972) who found that leg bones of many chicks appeared deformed at about 2 weeks of age (various deviation of the tarsometatarsus distal to the tarsal joint) which was similar to leg deformities of zinc deficient. Therefore, chicks fed diets deficient in zinc have abnormal leg development and deformed leg. Cooper (2000) reported that zinc deficiency may lead to limb deformities, enlarged joint, and thickening of the skin on the feet and legs. Also, Cook <u>et al.</u>, (1984) found that additional zinc to the diet was successful in decreasing the severity of leg deformities. On the other hand, Bezuidenhout <u>et al.</u>, (1994) demonstrated that there was no difference between the bone zinc content of affected and normal bird. They explained that the increase in Zn in affected birds may be due to an attempt by the body to produce more osteoid (bone matrix) in response to the bone abnormality (poor mineralization).

The significantly higher Mg% and lower Ca:Mg ratio in deformed than in normal bones may be explained by the decreased muscle activity in deformed leg birds. Christensen and Biellier (1982) stated that increased plasma Ca to Mg ratio may play a role in increased muscular activity and ionic Ca stimulates muscular contraction whereas Mg inhibits contraction.

From above-mentioned results, it can be concluded that deficient formation or poor mineralization of the osteoid matrix is associated with deficiencies in the micromineral, Zn, Ca, Mn, Ca:P ratio and Ca:Mg ratio. Meanwhile, it may be affected by high Mg content in bones.

## Serum mineral content of normal and deformed tarsometatarsus ostrich chicks:

Serum mineral content (Mean±SE) of ostrich chicks with normal and deformed tarsometatarsus bones are presented in (Table, 4). Statistical analysis indicated that the mean serum calcium, manganese, calcium to phosphorus ratio (Ca/P), and calcium to magnesium ratio (Ca/Mg) were significantly lower in ostrich chicks with deformed than with normal tarsometatarsus bones. Meanwhile, the mean serum phosphorus and zinc were significantly higher in the serum of chicks with deformed tarsometatarsus bones than in normal ones. No significant difference was found between mean serum magnesium of ostrich chicks with deformed and normal tarsometatarsus bones.

Limb deformity in ostrich chicks were found to be due to deficiencies of calcium, phosphorus, manganese, magnesium, copper and zinc (Bezuidenhout et al., 1994; Huchzermeyer, 1994; Mushi et al., 1998 and Squire and More, 1998). Guittin (1986) stated that severe rickets has been observed in ostrich chicks fed with breeder diets containing about 3% Ca. The levels of available phosphorus and the Ca:P ratio may be also important in the prevention of this disorder. The deficiency in serum Ca, Mn, Ca:P ratio and Ca:Mg ratio of deformed chicks in the present results are in agreement with the previous literature. However, the significantly higher serum P and Zn in deformed than in normal chicks in the present study are in contrast with that in literature. Squire and More (1998) stated that a deficiency in one or more of the Ca, P, Mg, Mn, Zn, and Cu may contribute to limb deformities as a result of poor mineralization, however, the elevated levels of these minerals in the serum of birds with leg deformities was a paradoxical finding. Bezuidenhout et al., (1994) found that serum Zn level was significantly elevated in the affected ostrich chicks with limb deformities compared with normal chicks which agrees with the present result. They added that Zn works as co-enzyme in the process of osteoidmatrix formation and plays an important role in mineralization of the osteoid which may explain the increase in serum Zn of deformed chicks as an attempt by the body to produce more osteoid. Also, Kaneko (1989) reported that the noticeable muscle catabolism, which was due to muscular dystrophy may be a result of the increase in the levels of plasma Zn.

#### Serum parameters of normal and deformed ostrich chicks:

Table (5) shows that mean serum concentrations of alkaline phosphatase, urea, total protein, albumen and globulin were significantly higher in deformed tarsometatarsus bones than in normal chicks. Meanwhile, albumen to globulin ratio (A/G) was significantly lower in deformed than in normal chicks.

The significantly higher serum alkaline phosphatase (Alkp) in deformed than normal chicks may be due to physiological increase in osteoclastic activity or decalcification. Levy <u>et al.</u>, (1986) showed that activity due to serum alkaline phosphates (Alkp) elevations may be due to physiological increase in osteoclastic activity. Al-Bustany <u>et al.</u>, (1998) stated that elevated plasma Alkp and loss of bone material were found in calcium deficient hens irrespective of supplemental phosphorus. Hurwitz and Griminger (1960) reported that decalcification rather than calcification of bone is associated with increased alkaline phosphatase levels.

The significant increase in blood urea in deformed chicks may be due to the catabolism of body protein. Palomeque <u>et al.</u>, (1991) reported that high blood urea in ostriches and birds may result from catabolism of body protein and dietary deficiency in essential amino acids may elevate levels of urea.

Results in Table (5) indicates that the significantly higher plasma total proteins in deformed than in normal chicks was due to significant increase in plasma albumin and globulin. The increase in serum globulin was more pronounced than that in albumin causing a significant reduction in A/G ratio. The significant increase in globulin in deformed chicks may be due to increased antibody formation because they are more susceptible to diseases. Kwak et al (1999) indicated that globulin portion mainly involved in antibody formation as well as cell membrane activities and cell division and cell proliferation.

It is worthy to note that plasma levels of Ca and P of both normal and leg deformed groups in the present study are lower than the range had been reported by Fudge (1996) (6-13 mg/dl and 3.1-7.4 mg/dl for Ca and P, respectively) for pooled ostrich samples or juvenile chicks indicating that the recommended requirements of ostrich chicks from Ca, P and may be vitamin  $D_3$  used in the present study are insufficient.

It can be concluded that, leg deformation in ostrich chicks was due to low Ca, Mn and Zn in bones and low Ca and Mn in blood plasma and further studies are needed for to investigate the adequate vitamins and minerals requirements for ostrich breeders and chicks to avoid the high incidence of leg deformation in ostrich chicks.

Age	0-1 week		1	-4	4-6		6-8		Total	
			we	eks	we	eks	we	eks		
Category	No	%	No	%	No	%	No	%	No	%
Died due to leg deformities	3	15	1	5	2	10	2	10	8	10
Died from other reasons	2	10	1	5	1	5	0	0	4	5
Alive	15	75	18	90	17	85	18	90	68	85
Total	20	100	20	100	20	100	20	100	80	100

Table (2): Prevalence of mortality and leg deformation in ostrich chicks during the first 8 weeks of age.

tarsometatarsus done mineral content.							
Minerals	Normal Tarsometatarsus	Deformed Tarsometatarsus					
Ca%	18.43±0.06 <sup>a</sup>	$9.59 \pm 0.05^{b}$					
P%	7.92±0.02 <sup>b</sup>	$9.38 \pm 0.033^{a}$					
Ca / P ratio	2.35±0.023 <sup>a</sup>	1.05±0.015 <sup>b</sup>					
Mg%	$0.094{\pm}0.002^{b}$	$0.226 \pm 0.0025^{a}$					
Ca / Mg ratio	196.6±2.7 <sup>a</sup>	42.7±2.1 <sup>b</sup>					
Mn%	$0.0033 \pm 0.0003$ <sup>a</sup>	$0.0027 \pm 0.0005^{b}$					
Zn%	0.246±0.001 <sup>a</sup>	0.169±0.001 <sup>b</sup>					

Table	(3):	Mean±SE	of	the	normal	and	deformed	ostrich	chick's
tarsometatarsus bone mineral content.									

a,b means in the same row with different superscripts are significantly different ( $P \le 0.05$ )

## Table (4): Mean±SE of serum mineral content of ostrich chicks with normal and deformed tarsometatarsus bone.

Minerals	Normal Tarsometatarsus	Deformed Tarsometatarsus
Ca%	$2.56 \pm 0.014^{a}$	$2.07 \pm 0.013^{b}$
P%	$1.8 \pm 0.019^{b}$	$2.0 \pm 0.015^{a}$
Ca / P ratio	$1.5 \pm 0.012^{a}$	$1.15 \pm 0.014$ <sup>b</sup>
Mg%	$0.513 \pm 0.004^{a}$	$0.55 \pm 0.005$ <sup>a</sup>
Ca / Mg ratio	$5.45 \pm 0.015^{a}$	$4.12 \pm 0.023^{b}$
Mn%	$0.68 \pm 0.011^{a}$	$0.41 \pm 0.012^{b}$
Zn%	$2.3 \pm 0.04^{b}$	$4.04\pm0.07^{a}$

a,b means in the same row with different superscripts are significantly different (P≤0.05)

# Table (5): Mean±SE of serum blood chemistry of ostrich chicks with normal and deformed tarsometatarsus bones.

Serum parameter	Normal	Deformed		
	Tarsometatarsus	Tarsometatarsus		
ALK.P (IU/l)	$191 \pm 3.8^{b}$	$293.2 \pm 2.6^{a}$		
Urea (mmol/l)	$3.02 \pm 0.03^{b}$	$4.1 \pm 0.025^{a}$		
T.prot (g/dl)	$3.44 \pm 0.03^{b}$	$4.2 \pm 0.02^{a}$		
Album (g/dl)	$1.86 \pm 0.02^{b}$	$1.93 \pm 0.015^{a}$		
Globu. (g/dl)	$1.58 \pm 0.03^{b}$	$2.27\pm0.02^{a}$		
A/G ratio	$1.18 \pm 0.014^{a}$	$0.85 \pm 0.02^{b}$		

a,b means in the same row with different superscripts are significantly different ( $P \le 0.05$ )

### REFERENCES

- Al-Bustany, Z., Al-Athari, A.K., and Abdul-Hassan, I.A. (1998). Plasma alkaline phosphatase and production traits in laying hens as influenced by dietary protein, strain and age. Br. Poult. Sci., 39: 568-571.
- Allwright, D. (1996). Viruses encountered in intensively reared ostriches: in Southern Africa. In: Improving our understanding of ratites in a farming environment, Deeming, D.C. (ed.), Ratite Conference, Oxford Shire, UK. pp. 27–33.
- Bar, A., Shinder, D., Yosefi, S., Vax, E., and Plavnik, I. (2003). Metabolism and requirements for calcium and phosphorus in the fast-growing chicken as affected by age. Brit. J. of Nutr., 89: 51-60.
- Bezuidenhout, A.J., (1999). THE OSTRICH: BIOLOGY, PRODUCTION AND HEALTH, Chapter, 2, Anatomy, Deeming, D.C. (ed.), CABI Publishing, pp. 15-17& 37-39.
- Bezuidenhout, A., Burger, W.P., Reyers, F. and Soley, J.T. (1994). Serum- and bone-mineral status of ostriches with tibiotarsal rotation. Onderstepoort J. Vet. Res., 61: 203-206.
- Bezuidenhout, A.J. and Burger, W.P. (1993). The incidence of tibiotarsal rotation in the ostrich (Struthio camelus). J. of the South African Vet. Assoc., 64: 159-161.
- Bissinger, K., and Huchzermeyer, F.W. (1998). Factors influencing calcium and phosphorus as well as vitamin D metabolism in ostriches on farms in Southern Africa. Proceedings of the Second International Scientific Ratite Congress, Oudtshoorn, South Africa, 21-25 September 1998, pp. 28-30.
- Brand, T.S., Brand, Z., Nel, C.J., Van Schalkwyk, S.J. & Salih, M., 1999. Ostrich nutrition: Review of recent research results. Proc. Ostrich Information, 30: 1-15.
- Brand, Z., Brand, T.S. and Brow, C.R., 2002b. The effect of dietary energy and protein levels on body condition and production of breeding male ostriches. South African Journal of Animal Science, 32: 231-239.
- Brand, T., Nel, K., Brand, Z. and van Schalkwyk, K, 2002a. Recent advantages in ostrich nutrition in South Africa: Effect of dietary energy and protein level on the performance of growing ostriches. South African Society for Animal Science: Popular-scientific

articles, Vol., 3: 1-8. (<u>http://www.sasas.co.za/Popular</u>/Popular. html)

- Charles, C., Cormic, Mc. and Cunningham, D. (1984). Forced resting by high dietary, zinc: tissue zinc accumulation and reproductive organ weigh changes. Poult. Sci., 63: 1207-1212.
- Chen, Y., Huang, Yq., Li, H., Li, Hy., Liu, Z.T., Deng, H., Huang, S.J., and Zhao, H.Q. (1997). Study on phosphorus deficiency in young ostriches. Chinese J. of Vet. Sci. and Technology, 27: 15-17.
- Christensen, V.L., and Biellier, H.V. (1982). Physiology of turkey embryos during piping and hatching, plasma total calcium, magnesium concentrations, and total calcium to magnesium ratios. Poult. Sci., 61: 1918-1923.
- Cilliers, S,C. and van Schlakwyk, 1994. Volstruisproduksie, Kleinkaroo Lanboukoeperasie, Oudtshoorn, South Africa. Sited by DISEASES OF OSTRICH AND OTHER RATITES, F.W. Huchzermeyer (ed.), Agricultural Research Council, Onderstepoort Veterinary Institute, 0110 Onderstepoort, Republic of South Africa Publisher, 1998, pp. 83.
- Cook, M.E., Sunde, M.L. Stahl, J.L., and Hanson, L.E. (1984). Zinc deficiency in pheasant chicks fed practical diets. Avian Diseases, 28: 1102-1109.
- Cooper, R.G. (2000). Management of ostrich (Struthio-camelus) chicks. World's Poult. Sci. J., 56: 33-44.
- **Creedon, A. and Cashman, K.D. (2001).** The effect of calcium intake on bone composition and bone resorption in the young growing rats. Brit. J. Nut., 86: 453-459.
- Dick, A.C.K., and Deeming, D.C. (1996). Veterinary Problems Encountered on Ostrich Farms in Great Britain. In: Improving Our Understanding of Ratites in a Farming Environment, Deeming, D.C. (ed.), Ratite Conference, Oxford Shire, UK, pp. 40-41.
- Foggin, C.M. (1992). Veterinary Problems of Ostriches. In: THE TOPAZ INTRODUCTION TO PRACTICAL OSTRICH FARMING, Chapter 8, M.G. Hallam (ed.), The Producers Association of Zimbabwe, Harare, pp. 80-82.
- Fudge, A.M. (1996). Clinical Hematology and Chemistry of Ratites. In: Ratite Management, Medicine and Surgery, Chapter 11, T.N. Tully and S.M. Shane (ed.), Krieger Publishing Company, Malabar, Florida, pp. 106.

- Goldenberg, J., and Fernandez, A. (1966). A simplified method for estimation of inorganic phosphorus in body fluids. Chlin. Chem., 12: 871 - 876.
- Guittin, P. (1986). Bow leg syndrome in ratite birds. Avicultured Magazine., 92: 70-79.
- Heany, R.P. (1998). Excess dietary protein may not adversely affect bone. J. Nutr., 128: 1054-1057.
- Henry, R.J. (1964). CLINICAL CHEMISTRY, Harper and Row Publishers, New York, pp. 181.
- Huchzermeyer, F.W. (1994). Nutritional diseases. In. OSTRICH DISEASES. Huchzermeyer, F.W., (ed.), Agricultural Research Council, Onderstepoort, pp. 48-58.
- Hurwitz, S., and Griminger, P. (1960). The response of plasma alkaline phosphatase, parathyroids, blood and bone minerals to calcium intake in the fowl. J. Nutr., 73: 61.
- Juan, G. and Baowei, C. (2004). Causes and prevention of leg diseases in young ostriches. Proceedings of International Conference on Development of Ostrich Estate (ICDOE2004), 3-5 April, 2004, Xi Aan, China, pp. 337-339.
- Kaneko, J.J. (1989). Serum proteins and disproteinaemia, In CLINICAL CHEMISTRY OF DOMESTIC ANIMALS, edited by Kaneko, J.J., 4<sup>th</sup> (ed.). San Diego: Academic Press Inc. pp. 142-165.
- Kreibich, A., and Sommer, M. (1995). Miscellaneous. In: Kreibich, A., and Sommer, M. (eds.). OSTRICH FARM MANAGEMENT, (Landwirtschafts verg, Gmbh, Munster-Hiltrup, Germany), pp. 166-169.
- Kwak, H. Austic, R.E. and Dietert, R.R. (1999). Influence of dietary arginine concentration on lymphoid organ growth in chickens. Poult. Sci., 78: 1536-1541.
- Levy, D.H., Morris, R.E., Smallwood, J.E., and Loomis, M.R. (1986). Mortality of chicks and decreased fertility and hatchability of eggs from a captive breeding pair of ostriches. J. Amer. Vet. Med. Assoc., 189: 1124-1126.
- More, S.J. (1996). The performance of farmed ostrich chicks in eastern Australia. Preventive Veterinary Medicine, 29: 91-106.
- Morrow, C.J., Browne, C.J., Donne, O. and Thorp, H.B.H. (1997).

*Hypo- phosphataemic rickets and nephrocalcinosis in ostrich chicks brooded and reared on limesonesand. Vet. Rec., 140: 531-532.* 

- Mushi, E.Z., Chabo, M.G., Isa, J.F.W., and Phuti, M.S. (1999). Limb deformities of farmed ostrich (Struthio camelus) chicks in Botswana. Tropical Animal Health and Production, 31: 397-404.
- Mushi, E.Z., Isa. J.F.W., Chabo, M.G., Nyange, J., Modisa, L. (1998). Selenium-vitamin E responsive myopathy in farmed ostriches (Struthio camelus) in Botswana. Avian Pathology, 27: 326-328.
- Nelson, T.S., Harris, G.C., Kirby, L. and Johnson, Z.B. (1990). Effects of calcium and phosphorus in the incidence of leg abnormalities in growing broilers. Poult. Sci., 69: 1496-1502.
- Palomeque, J., Pinto, D., and Viscor, G. (1991). Hematological and blood chemistry values of the Masai ostrich (Struthio camelus). J. of Wild life Diseases, 27: 34-40.
- Patton, C.J., and Crouch, S.R, (1977). Anal. Chem., 49: 464-469.
- Perry, R.W., Rowland, G.N., Foutz, T.L. and Glisson, J. R. (1991). Poultry malabsorption syndrome. III. Skeletal lesions in market age turkeys. Avian Disease., 35: 707-713.
- Rath, N.C., Huff, G.R., Huff, W.E., and Balog, J.M. (2000). Factors regulating bone maturity and strength in poultry. Poult. Sci., 79: 1024-1032.
- SAS (1988). Statistical Analysis Systems, STAT/User's Guide, Release 6.03 ed., SAS Institute, Cary NC, USA.
- Shivaprasad, H.L. (1993). Neonatal mortality in ostriches: an overview of possible causes. Proceedings of the Annual Conference of the Association of Avian Veterinarians, Nashville, 31 August 4 September, 1993, 282-293.
- Smith, W.A., Colliers, S.C., Mellet, F.D., and van Shalkwyk, S.J. (1995). Ostrich production. South African perspective. In: Lyons, T.P and Jacques, K.A. (eds.). Biotechnology in the Feed Industry, Proceedings the 11th Allteck Annual Symposium, Nottingham University Press, Nottingham, pp. 175-198.
- Squire, B.T., and S.J., More, (1998). Factors on farms in Eastern Australia associated with the development of tibiotarsal rotation of ostrich chicks. Australian Vet. J., 76: 110-117.
- Sunde, M.L. (1972). Zinc requirement for normal feathering of commercial

leghorn type pullets. Poult. Sci., 51: 1316-1322.

- **Tully, T.N., Shane, S.M. (1998).** Ratites. The Veterinary Clinic of the North America: food animal practice. W.B. Saunders Company Philadelphia.
- Verwoerd, D.J., Olivier, A.J., Henton, M.M., and Vanderwalt, M. (1998). Maintaining health and performance in the young ostrich: Applications for amannanoligo saccharide. In: Lyons, T.P., and Jaques, K.A. (eds.). Biotechnology in the feed industry, Proceedings on the 14<sup>th</sup> Alltech Annual Symposium. Nottingham University Press, Nottingham, pp., 539-551
- Yalcin, X., Zhang, L.M., Crista, G.R., Mc. Daniel and Kuhlers, D.L. (2000). Effect of divergent selection for incidence of Tibial dyschondroplasia (TD) on purebred and crossbred performance, %TD incidence and calcium and phosphorus plasma concentrations. Br.. Poult. Sci., 41: 562-565.

الملخص العربى

التركيب الكميائى لمصل الدم والمحتوى المعدنى للعظام الرسغمشطية فى كتاكيت النعام الطبيعية والمشوهه

محمد أسامة فهمي، هشام حسين خليفة\*، نشأت سعيد ابراهيم

قسم التطبيقات البيولوجية مركز البحوث النووية هيئة الطاقة الذرية

\* جامعة الآز هر كلية الزراعة قسم الآنتاج الحيواني مدينة نصر - القاهرة

لدراسة تشوه الأرجل في كتاكيت النعام كعامل أساسى مؤثر على نفوق النعام تم جمع عظام ساق الآرجل وعينات الدم من عدد ٣٠ كتكوت مشوهة الأرجل ٣٠ كتكوت طبيعى يتراوح أعمارها من ٢-١٠ أسابيع وذلك من مزارع إنتاج النعام فى هيئة الطاقة الذرية – القاهرة – جمهورية مصر العربية. تم تحليل محتوى العظام من العناصر المعدنية الآتية: الكالسيوم - الفوسفور -الماغنسيوم - المنجنيز - الزنك وتم تقدير بعض المقاييس الكيميائية على المصل فى كلتا المجموعتين من البروتين الكلى – الآلبيومين – الجلوبيولين - نسبة الآلبيومين الى الجلوبيولين - الفوسفاتيز القاعدى.

أهم النتائج التي تم التوصل إليها:

 تراوحت نسبة نفوق الكتاكيت خلال أول ٨ أسابيع بعد الفقس بين ١٠-٢٥ % ويرجع هذا النفوق إلى تشوهات الأرجل والذي يمثل ٥-١٠% من اجمالي الكتاكيت ومن ٥٠-١٠٠% من اجمالي الكتاكيت النافقة .

- ٢. يزداد النفوق الراجع إلى تشوه الأرجل كنسبة من اجمالى النافق بزيادة العمر مما يدل على أن تشوه الأرجل كان من أهم اسباب فقد واستبعاد الكتاكيت بعد الفقس.
- . كانت مستويات الكالسيوم والمنجنيز والزنك في العظمة الرسغمشطية المشوهة أقل معنويا من عظمة الساق الطبيعية.
- ٤. كانت مستويات الماغنسيوم و الفوسفور في العظمة الرسغمشطية المشوهة أكثر معنويا من الطبيعية وكانت نسبة الكالسيوم إلى الفوسفور ونسبة الكالسيوم إلى الماغنسيوم في العظام المبيعية.
- حان متوسط مستوى الكالسيوم والمنجنيز و نسبة الكالسيوم إلى الفوسفور ونسبة الكالسيوم إلى الماغنسيوم ونسبة الألبيومين الى الجلوبيولين فى مصل الدم أقل معنويا في كتاكيت النعام المشوهة الارجل عنها فى الطبيعية.
- ٢. كان متوسط تركيز مصل الدم من الفوسفاتيز القاعدى و اليوريا والبروتين الكلى والالبيومين والجلوبيولين و الفوسفور و الزنك أكثر معنويا في الكتاكيت المشوهة الارجل بالمقارنة بالكتاكيت الطبيعية.
- ٧. لم يكن هناك أي اختلافات معنوية لمستوى الماغنسيوم في مصل دم كتاكيت النعام ذات العظام الرسغمشطية المشوهة والطبيعية.