

## Clinical effects of zinc deficiency in Iraqi camels

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### Abstract

Clinical signs and parameters in comparison with Zn levels have been studied in Iraqi camels in Al-Najaf slaughter house. Among 1562 examined camels 40 camels from different ages and both sex with skin lesion (dry scaly) as a deficient and other 40 camel apparently healthy as control. Affected animals showing sign of parakeratosis 90%, alopecia with 52.5%, pal mucus membrane 30%, emaciation 22.5%, diarrhea 7.5%, abnormal hoof formation 5% and excessive lacremation 5%. No significant differences has been detected in body temperature, whereas respiratory and heart rate significantly increased at ( $p<0.05$ ) when compared with control. Serum zinc analyzed by Atomic Absorption Spectrophotometers, the result revealed normal Zn value in Iraqi camel was ( $62.9\pm 3.5\text{mg/dl}$ ) but results of diseased camel revealed significant decrease in serum zinc ( $32.4\pm 2.1\text{mg/dl}$ ).

### التأثيرات السريرية لنقص الزنك في الجمال العراقية

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### الخلاصة

تم دراسة العلامات والمؤشرات السريرية ومقارنتها بمستوى الزنك في الجمال العراقية في مجزرة النجف الاشرف. من خلال فحص 1562 جمل من مختلف الأعمار ومن كلا الجنسين تم تعيين 40 حيوان يحمل آفة جلدية (جافة ومتقشرة) كحالات مرضية، ومقارنتها مع 40 حيوان بحالة صحية جيدة ظاهرياً ولا تحمل أي مؤشرات مرضية كعينة سيطرة. أظهرت الحيوانات المصابة 90% حالات فرط التقرن، 52.5% حالات تساقط الشعر، شحوب الأغشية المخاطية 30%، هزال 22.5% حالات إسهال 7.5%، حالات تشوه في الخف 5%، حالات تدمع العينين 5%. لم يسجل فرق إحصائي ( $P>0.05$ ) في درجات الحرارة للحيوانات المصابة مقارنة مع عينات حيوانات السيطرة، بينما سجل فرق إحصائي ( $P<0.05$ ) في معدلات النبض والتنفس. تم تحليل مصل الدم للحيوانات المصابة وحيوانات السيطرة باستخدام جهاز امتصاص الطيف الذري وأظهرت النتائج إن معدل الزنك الطبيعي في الجمال العراقية هو ( $62.9\pm 3.5$  ملغم/ د.ل) ولكن الجمال المريضة أظهرت فرق إحصائي ( $P<0.05$ ) وكانت نسبة الزنك فيها ( $32.4\pm 2.1$  ملغم/ د.ل).

### Introduction

Minerals are very crucial for animal health and productivity by playing an important role in many physiological activities and their deficiency causes a variety of pathological problems and metabolic defects (1). The level of nutrition and mineral intakes are known to affect the production and reproductive ability of male and female camels (2, 3). Zinc has a catalytic, coactive, or structural role in a wide variety of enzymes that regulate many biological processes and consequently animal health and productivity (4). Its presence is of particularly important in rapidly dividing cells including those of epidermis (5). Zinc deficiency results in failure of keratinization, which leads to parakeratosis, loss and failure of growth of hair, lesions of coronary bands(6). This probably reflects the importance of zinc in protein synthesis (7) In Germany, skin lesions have occurred in alpacas and llamas with low Zn and copper

status (8). Several investigators reported that there are indications of Zn deficiency or border-line of deficiency in camels, (9) found that serum Zn level are <50 mg/100 ml in zoo camels in France. (10) found a low mean serum Zn level (41 mg /dl) with a range of 37-46 mg/100 ml in the camels in the United Arab Emirates together with high infertility in females. These result are confirmed by finding a low Zn in pastures 1.2-21.8 mg /kg DM which is far below Agricultural Research Council ARC recommendation for cattle (30 mg/ kg DM), (11). Zinc deficiency is comparatively rare in camels in field, it is clinically evidenced by Skin disorders and reduced performance and has been reported under natural conditions. These are manifested by parakeratosis, anorexia, growth failure, impaired reproduction functions (12). Very few researches have found a relationship between Zn deficiency and skin lesion in camelids, therefore the current work aimed to present the clinical signs and biochemical values of zinc deficiency in Iraqi camel.

### Material and Methods

Eighty camels of different age and both sex were selected as 40 apparently healthy as control and 40 diseased animals which exhibited signs of loss of hair, thickening and scaling of the skin, emaciation, all examined camels was negative for gastrointestinal ,blood and external parasites. Blood sample collected from each animals from the jugular vein in vacutainer tubes without anticoagulant, after centrifugation at 1.500×g for 10 min, the collected serum were kept at -20°C until analysis by Atomic Absorption Spectrophotometer (PYE Unicam SPg atomic absorption spectrophotometer). Method after diluted with deionizer water at 1:1 ratio, Zinc level are reading in ppm. After using all previous procedures concerned, contents of mineral was to be determined must be looked to divert a value. Atomic Absorption apparatus in solution state was used to reveal actual value. The following equation was used:

$$Gm = \frac{r \text{ (mg/ lit)} \times vV \times 100}{10^6 Wt} \times 100$$

Where:

r: value determination by atomic absorption in solution condition by ppm.

V: prepared volume of tested sample(100 ml).

Wt: weigh t of sample per gram

- **Statistical analysis:** The significance of variation in the values of Zn deficient and control camels with different clinical parameters were analyzed statistically using One way analysis of variance (SPSS Program) according to (13).

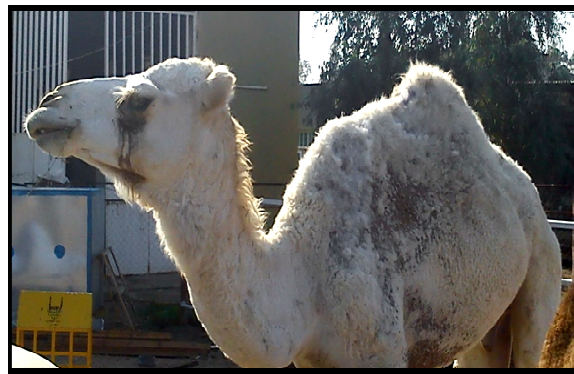
### Results and Discussions

Diseased camels with Zn deficiency showed (Table 1) signs of dry scaly skin in 36 animals 90% which may be bilateral which appear in 24 animals 60%, and symmetrical in 12 cases 30%, affecting lesion manifested in the different side of body (head, neck, breast, shoulder), typical gross hyperkeratotic lesion are observed in Fig.(1). Also they manifested in limbs, folds near the joint (knee, fetlock) with thick cracked areas, some times with blood oozing, with no pain, no itching. Hair loss or alopecia was almost observed in this study with 21 animals 52.5% in (neck, legs, shoulders, breast, flanks), (Fig. 2). These results are in agreement with (14, 15), whose reported that dry scaly lesion characterized by hair loss, thickening of the skin with tightly adhering crusts in llama and alpacas observed in South America and with (8, 16) observed the same lesion in ruminants. Zn is critical for activities of enzymes and transcription factors involved in epithelial cell proliferation and development. The result showed nine anorexia and emaciated camels with 9 animals 22.5% of the deficient group (Fig. 2). Zinc deficiency in animals is usually associated with anorexia and depressed food intake possibly by inhibiting the release of neuropeptide Y that is required for receptors activation that may be the reason for emaciation in Zn deficient animals of this study. Zinc is essential for the taste perception because taste is mediated through a salivary zinc dependent polypeptide (gustin)

therefore low salivary Zn levels invariably leads to a reduction of taste, hence to a greatly reduced appetite and emaciation, Similar result finding and reported in camels by (12, 17, 18). Zn deficiency in animals result in growth failure and feed intake and loss of hair and skin lesion and decreased in feed consumption and efficiency of utilization in addition to retardation of metabolic function responsive for growth and maturation (19). (20) started that zinc deficiency and energy malnutrition in mice are characterized by reduced growth and reduce lymphocyte number and increase susceptibility to infection. The infection may be the causes of lacremation of the camel in present study with two animals 5% (Fig. 2). Pale mucus membrane observed in 12 30% of diseased camels which indicated to anemia occur in affected animal resulting in decrease of RBC, due to role of zinc deficiency which lead to impairment of cell replication and protein synthesis and generation of blood cells (21). Macrocytic normochromic anemia were mentioned by (22) in buffalo calve and sheep affected with Zn deficiency. (23) attributed this to fact that Zn deficiency in the rat increases the fragility of red cell subjected to hypotonic stress and due to oxidative damage to the membranes. Growth failure and hoof deformation record in the present study in two cases with 5% from the deficient cases (Fig. 3). (24, 25) have been reported that Zn deficiency may causes skeleton abnormalities because Zinc is a growth factor and its deficiency adversely affected growth in many animals species and humans in as much as zinc need for protein and DNA synthesis and for cell division, the effect of Zn on protein synthesis may be attributable to it vital role in nucleic acid metabolism in addition to role of zinc in growth hormone (GH) metabolism and it's deficiency may also affect bone metabolism and gonadal function (5, 26, 27). Diarrhea was observed in 3 deficient camels (7.5%) the diarrhea can be attributed to zinc deficiency impairing the regeneration of epithelial cells and their brush border to replace those that have sloughed in the intestine, resulting in a malabsorptive diarrhea (28). Zinc deficiency also results in decreased activity of zinc dependent digestive enzyme from the liver, which results in a maldigestive diarrhea (23).



**Fig. (1)** He camel with 3 years old showing advance lesions, as shown in knee joint were characterized by fissured area.



**Fig. (2)** She camel 6.5 years old showing loss of hair (alopecia) and emaciation with lacremation



**Fig. (3)** He camel with two years old suffering from zinc deficiency showing hoof deformation

**Table (1) Clinical signs of camels affected with Zinc deficiency**

Clinical sign	No. of cases (n=40)	Percentage of cases%
Parakeratosis	36	90
Hair loss (alopecia)	21	52.5
Decreased growth rate	15	37
Pale mucus membrane	12	30
Emaciation (anorexia)	9	22.5
Diarrhea	3	7.5
Abnormal hoof formation	2	5
Excessive lacremation	2	5

**Table (2) Clinical parameters of deficient& control camels**

Parameter	Body temperature °c	Respiratory rate/ min	Heart rate/min	Zn mg/dl
Control	36.2±0.4 <sup>a</sup>	11.6±0.4 <sup>a</sup>	40.5±0.5 <sup>a</sup>	62.9±3.5 <sup>a</sup>
Deficient	36.4±0.5 <sup>a</sup>	15.1±1.3 <sup>b</sup>	50.3±0.2 <sup>b</sup>	32.4±2.1 <sup>b</sup>

<sup>a,b</sup> Values with different superscript within a column differ significantly (P<0.05)

No significant difference was detected in body temperature, whereas respiratory and heart rates were significantly increased (P<0.05) in diseased animals in comparison with control (Table 2). Due to the role of Zinc as an integral part of the carbonic Anhydrase enzyme, which present in high concentration in red blood cells, this enzyme is responsible for rapid combination of carbon dioxide with water in the red blood cells of the peripheral capillary blood and for rapid release of carbon dioxide from the pulmonary capillary blood into the alveoli and that will explain why the Zn deficiency causes respiratory elevated, Studies in various species, including rodents, domestic fowl, ruminants have found that dietary zinc deficiency significantly reduces red blood cell carbonic Anhydrase activity which may impair respiratory function (29). This may cause increased respiratory rate which was detected in diseased camels in present study. Increased rate of respiration in present study may be occur due to hypoxia (anemic hypoxia) caused by decreased in Hb concentration, affecting oxygen transportation to tissue, as the body may receive in a adequate of oxygen which result in panting in animals (8). The heart act as an automaton, responding to the demand of the tissue from oxygen (30), so the heart rate elevated in both groups to reaper the oxygen deficient from the tissue due to zinc deficiency. (22) observed that was significant increase in respiratory and heart rate in hypozincemia in Iraqi cattle. Camels appear to maintain Zn levels at lower values than other domestic ruminants <60 mg/dl. (31) have explained this low level by camel adaptation to extreme thermal conditions and nutritional stresses; stress causes increase in zinc dependent enzymes requirement so then causing increase in intestinal absorption and liver uptake of zinc. This value may came from the low of Zn concentration in the general nutrition of Iraqi camels particularly in the region of the study, this lack of Zn in the forage or wild plant came from the low Zn concentration of Iraqi soil. (32) published that widespread Zn deficiency in wheat is found in Iraq. (33) and (34) found that the available Zn concentrations in the soils of Iraq were the lowest of the 30 countries studied and that zinc would seem to be a major problem. The meandiethyl triamine penta-acetic acid (DTPA). The risk of Zn deficiency may increased when the pH of Iraqi soil in grassing area raised above 6.5 (34). Zn deficiency may occur due to present of some antagonist minerals like Calcium in high concentration in the wild plant that the animals depended on it in feed or present of excessive dietary sulphur, Calcium is know to block zinc uptake by animals even in sufficient diet (35) and (36), Some legumes contain less zinc than grasses grown on the same soil and Zn concentration decreases with aging of plant (37). The majority of camels live in desert and subdesertic areas where feeding resource are generally scattered and poor. Camels breeders don't usually provide mineral supplementation,

except during salt cure in some subsaharian region, so it would not be surprising to observe trace element deficiency (38). (11) reported low zinc value in soil and pasture in Sudan despite the fact that the major animal population in the country is dependent on natural grazing. Zinc deficiency- responsive dermatoses has been described, but the true role of Zn deficiency is debated and the differences in nutrient metabolism between camelids and other ruminants responsible for unique nutritional disease concerns with Zn (39).

### References

1. Deen, A.; Bhati, A. & Sahani, M. 2004. Trace mineral profiles of camels blood and sera. J. Camel Pract. Res., 11: 135-136.
2. Ali, A.; Tharwat, M. & Al-Sobayil, F. A. 2010. Hormonal, biochemical and hematological profiles in female camels (*Camelus dromedaries*) affected with reproductive disorders. Anim. Reprod. Sci., 118:372-376.
3. El-Bahrawy, K. A. & El-Hassanein, E. E. 2011. Seasonal variation of some blood and seminal plasma biochemical parameters of male Dromedary camel. Am-Euras. J. Agric. Environ. Sci., 10:354-360.
4. Vallee, B. L. & Falchuk, K. H. 1993. The biochemical basis of zinc physiology. Physio. Rev., 73: 79-118.
5. Nishi. Y. 1996. Zinc and growth. J. of Am. Coll. of Nut.,15:340-344.
6. Oberleas, D. & Harland, B. F. 2008. Treatment of zinc deficiency without zinc fortification. J. of Zhjiang Uni. Sci., B.9(3):192-196.
7. Meglia, G. E.; Holtenius, K.; Petersson, L.; Ohagen, P. & Waller, K. P. 2008. Prediction of vitamin A, vitamin E, selenium and zinc status of preparturient dairy cows using blood sampling during the mid dry period. Acta Veterinaria Scandinavica, 45:119-128.
8. Radostitis, O. M.; Blood, D. C.; Gay, C. C. & Hinchliff, K. W. 2010. Veterinary Medicine: A text book of the diseases of cattle, sheep, goat and horses, 10<sup>th</sup>, Ed., W. B. Sounder Co. PP.1730-1733.
9. Faye, B. & Bengoumi, M. 1998. Trace element metabolism in camel. Proc. Third Ann. For animal production under arid conditions, 1, 9-35, UAE University, Al-Ain.
10. Abdalla, O. M.; Wasfi, I. A. & Gadir, F. A. 1988. The Arabian race camel normal parameters. I. Haemogram, enzymes and minerals. Comp. Bioch. Physiol. 90A: 237-239.
11. Abu-Damir, H. 1998. Mineral deficiencies, Toxicities and imbalance in the camel (*Camelus dermoedarius*): a review. Vet. Bulletin 68: 1103-1119.
12. Al-Ani, F. K. 2004. Camel management and diseases. 1<sup>st</sup>. ed. Dar Ammar Book Publisher, Jordan.
13. Leech, N. L.; Barrett, K. C. & Morgan, G. A. 2007. SPSS for intermediate statistics: use and interpretation. 1<sup>st</sup> ed., Lawrence Erlbaum Asso. USA. PP. 20-51.
14. Fowler, M. E. 1998. Medicine and surgery of south American Camelids. Iowa State University Press, Ames. USA.
15. Rosychuk, R. A. W. 1994. Llama dermatology. Vet. Clinics N. A., Food Anim. Pract. 10(2): 228-239. Vav Saun Small Rum. Res., 61.
16. Clauss, M.; Lendl, C.; Schramel, P. & Streich, W. J. 2004. Skin lesion in alpacas and llamas with low zinc and copper status-a preliminary report. Vet. J., 167(3): 302-305.
17. Zong-ping, L.; Zhuo, M. & You-Jia, Z. 1994. Studies on the relationship between sway disease of Bactrian camels and copper status in Gansu Province. Vet. Res. Comm., 18:251-260.
18. Akgul, A.; Agaoglu, Z. T.; Kaya, A. & Sahin, T. 2000. The relationship between the syndromes of wool eating and alopecia in Akkaraman and Morkaraman sheep fed corn silage and blood changes (Haematological, biochemical and trace elements). J. Vet. Med., 56: 16-25.
19. Miller, W. J. 1979. Mineral and trace element nutrition of dairy cattle in: Animal feeding and nutrition. Ed, Millar, W. J., PP. 42-149. Academic press. New York. NY.

20. Fraker, P. J.; King, L. E.; Laakko, T. & Vollmer, T. L. 2000. The dynamic link between the integrity of the immune system and zinc status. *J. of Nut.*, 130:1399s-1406s.
21. O'Dell, B. L.; Hardwick, B. C.; Reynolds, G. & Sarage, J. E. 1997. Connective tissue detects in chicks resulting from copper deficiency. *Pro. So. Exp. Biol. Med.*, 108: 402-405.
22. Al-Saad, K. M.; Al-Sadi, H. I. & Abdulla, O. A. 2011. Zinc deficiency (hypozinacemia) in local Iraqi cattle. *Res. Opin. in Animal & Vet. Sci. University of Mosul, Iraq.*
23. Bettger, W. J. 1993. Zinc and Selenium, site-specific versus general antioxidantation. *Can. J. Physiol. Pharmacol.*, 71 (9):721-724.
24. Murray, E. F. 2010. In collaboration with P. Walter Bravo. *Medicine and Surgery of Camelids*. 3<sup>rd</sup> Ed.
25. Campbell, M. H. & Miller, J. K. 1998. Effect of supplement dietary vitamin E and zinc on reproductive performance of dairy cows and heifers fed excess iron. *J. Dairy Sci.*, 81:2693-2699.
26. Prasad, A. S. 1983. Zinc deficiency in human subject *Prog. Clin. Biol. Res.*, 129:1-33.
27. Prasad, A. S. 1985. Clinical manifestation of zinc deficiency. *Anu. Rev. Nutr.*, 5:34-63.
28. Watson, D. W. & Sodeman, W. A. 1979. The small intestine in pathologic physiology: Mechanisms of disease, eds. Sodeman, W. A., Sodeman, T. M., PP. 824-849, WB savnders co. Philadelphia, P.A.
29. Lukaski, H. C. 2005. Low dietary zinc decreases erythrocyte carbonic Anhydrase activities and impairs cardiorespiratory function in men during exercise. *Am. J. Clin. Nut.*, 81(5):151-159.
30. Guyton, A. C. & Hall, J. E. 2006. *Textbook of Medical Physiology*, eleventh edition. P. 879.
31. Ghosal, A. K. & Shekawati, V. S. 1992. Observations on serum trace elements levels (zinc, copper and iron) in camel (*Camelus dromedaries*) in the arid tracts of Thar Desert in India.
32. Brian, J. A. 2008. Zinc in soils and crop nutrition. 2<sup>nd</sup> ed. by international zinc association (IZA), International fertilizer industry association (IFA), Brussels, Belgium and Paris, France.
33. FAO. 2004. FAOSTAT Production Statistics, Food and Agriculture Organization, Rome.
34. Timsina, J. & Connor, D. J. 2001. Productivity and management of rice-wheat cropping systems: issues and challenges. *Field Crops Res.*, 69:93-132.
35. Singer, L. J.; Herron, A. & Altman, N. 2000. Zinc responsive dermatopathy in goat: Two field cases. *Contemp. Top. Lab. Anim. Sci.*, 39(4): 32-35.
36. Nelson, D. R.; Wolff, W. A.; Blodgett, P. J.; Luecke, B.; Ely, W. & Zachary, J. F. 1984. Zinc deficiency in sheep and goat: there filled cases. *J. Am. Vet. Med. Assoc.*, 184(12):1480-1485.
37. Arrayet, J. L.; Oberbauer, A. M.; Fmula, T. R.; Garnett, I.; Oltjen, J. W.; Imhoof, J.; Kehrli, M. E. & Graham, T. W. 2002. Growth of Holstein calves from birth to 90 days: The influence of dietary zinc and BLAD status. *J. Anim. Sci.*, 80: 545-552.
38. Faye, B.; Saint-Martin, G.; Cherrier, R. & Ruffa, A. 1992. The influence of high dietary protein, energy and mineral intake on deficiency.
39. Robert, J. V. 2005. Nutritional disease, Rickets; Hepatic liposis in llama and alpacas. Pennsylvania State University, Dep. Of Vet. Sci. USA.