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UNITED ARAB EMIRATES UNIVERSITY

PROGRAMMATION DE LA COOPERATION AVEC L'UNIVERSITE DES EMIRATS ARABES UNIS DAND LE DOMAINE DE L'ELEVAGE CAMELIN

Mission aux Emirats Arabes Unis

26 septembre au 3 octobre 2009

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Novembre 2009

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RÉSUMÉ :

Première mission faisant suite à la thèse de Rabiha SEBOUSSI (voir rapports précédents), elle avait pour but de relancer la coopération sur les camélidés en vertu de la convention signée en 2003 entre le CIRAD et l'Université des EAU. Outre la valorisation de la thèse (actuellement 6 publications dans des revues à facteur d'impact, 2 publications dans des revues nationales, 5 communications, 1 ouvrage de vulgarisation et 2 publications encore en cours), la coopération peut se poursuivre sur le thème de la composition et de la transformation du lait de chamelle (proposition de thèse et de masters en partenariat avec l'INRA et l'Université de Montpellier II) et de la nutrition (proposition de deux protocoles expérimentaux avec l'Université d'Al-Ain), la participation à des cours et des visites d'étudiants émiriens à Agropolis.

Par ailleurs, le Pr AL-HADRAMI (Doyen du *College of Food and agriculture*), et moi-même étant respectivement Président et Vice-Président de l'ISOCARD (*International Society of camelid research and Development*), les discussions dans ce cadre ont porté sur l'organisation de la conférence ISOCARD 2012, la création effective du camelpedia wiki et le partenariat entre *Emirates Journal of food and Agriculture* et *Journal of Camelid Sciences*, journal officiel de l'ISOCARD.

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RESUME

Première mission faisant suite à la thèse de Rabiha SEBOUSSI (voir rapports précédents), elle avait pour but de relancer la coopération sur les camélidés en vertu de la convention signée en 2003 entre le CIRAD et l'Université des EAU.

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REMERCIEMENTS

Je réitère mes sincères remerciements au collège « Food and Agriculture » et son doyen le Dr ALHADRAMI, pour son accueil et sa motivation toujours aussi forte. Je tiens à remercier également M. Didier GAZAGNADOU, au Service de Coopération de l'Ambassade de France pour son appui indispensable à la poursuite de cette fructueuse coopération scientifique.

INTRODUCTION

Cette mission était la première faisant suite à la thèse de Rabiha SEBOUSSI depuis sa soutenance en juin 2008. Soucieux de poursuivre la coopération scientifique fructueuse en termes de publications et de projets communs, le Service de Coopération Culturelle et Technique a insisté pour que cette mission puisse se réaliser afin d'établir un nouveau programme de coopération.

Au cours de la mission il a été essentiellement question de l'intensification des échanges entre le CIRAD et l'Université d'Al-Ain et de la gestion de l'ISOCARD (*International Society of Camelid Research and development*) dont le professeur Al-Hadrami, doyen du « College of Food and Agriculture » (CFA) assure désormais la présidence, moi-même en assurant la vice-présidence.

Seront abordés dans le présent rapport, l'état des lieux de la valorisation de la thèse de R. SEBOUSSI, les projets d'échanges scientifiques, et la gestion de l'ISOCARD.

1 - VALORISATION DE LA THESE DE R. SEBOUSSI

La qualité du travail de thèse de Rabiha SEBOUSSI est incontestable et a donné lieu à plusieurs publications dont on trouvera la liste exhaustive ci-après. L'importance de la base de données acquises au cours des expérimentations passées a permis également une méta-analyse très instructive débouchant sur un article de synthèse qui pourrait être traduit en arabe et publié aux Emirats par l'Université. On en trouvera un exemplaire en partie annexe. Pour mémoire, la liste des publications issues de ce travail est la suivante :

1-1. Revues à facteurs d'impact

1. FAYE B., SEBOUSSI R., ASKAR M., 2005. *Trace elements and heavy metals in healthy camel blood of United arab Emirates.* J. Camel Res. Pract., 12, 1-6
2. SEBOUSSI R., FAYE B., ALHADRAMI G., ASKAR M., IBRAHIM W., HASSAN K., MAHJOUB B., 2008. *Effect of different selenium supplementation levels on selenium status in camel.* Biol. Trace Elem. Res., 123, 124-138. On line on http://www.springerlink.com/content/12331434458t652n/
3. SEBOUSSI R., FAYE B., ASKAR M., HASSAN K., ALHADRAMI G., 2009. *Effect of selenium supplementation on blood status and milk, urine and fecal excretion in pregnant and lactating camel.* Biol. Trace Elem. Res., 128, 45- 57. DOI 10.1007/s12011-008-8251-3
4. SEBOUSSI R., FAYE B., ALHADRAMI G., ASKAR M., IBRAHIM W., MAHJOUB B., HASSAN K., MOUSTAFA T., ELKHOULY A., 2009. *Selenium distribution in camel blood and organs after different level of dietary selenium supplementation.* Biol Trace Elem. Res., DOI 10.1007/s12011-009-8410-1
5. SEBOUSSI R., FAYE B., ALHADRAMI G., ASKAR M., BENGOUMI M., ELKHOULY A., 2009. *Chronic selenosis in camels.* J. Camel Pract. Res., 16(1), 1-14.
6. FAYE B., SEBOUSSI R., 2009. Selenium in camel – A review. Nutrients. 1, 30-49. DOI: 10.3390/nu1010030

1-2. Revues à comité de lecture

7. SEBOUSSI R., FAYE B., ALHADRAMI G., 2004. *Facteurs de variation de quelques éléments trace (sélénium, cuivre, zinc) et d'enzymes témoins de la souffrance musculaire (CPK, ALT et AST) dans le sérum du dromadaire (Camelus dromedarius) aux Emirats Arabes Unis.* Rev. Elev. Med. Vét. Pays Trop., 57 (1-2), 87-9
8. FAYE B., SEBOUSSI R., 2008. *Experimental selenium intoxication in camel.* Veterinariya, 3, 18- 29

1-3. Congrès internationaux

9. FAYE B., SEBOUSSI R., ASKAR M., 2008. *Trace elements and heavy metals status in Arabian camel.* . Proc. of . Intern. Workshop, « Impact of pollution on animal products". Almaty (Kazakhstan), 27-30 September 2007, B. Faye and Y. Sinyavskiy (Eds), 97-106.

10. FAYE B., SEBOUSSI R., ALHADRAMI G., 2009. *Maternal transfer of selenium by blood and milk in camel.* Proc. of the 2nd conference of ISOCARD, Djerba (Tunisia), 12-14 march 2009, abstr. 156, p. 126

11. SEBOUSSI R., ALHADRAMI G., **FAYE B.,** ASFOUR T., ELKHOULY A., ALBELKHALEK T., ALMASRI J., 2006. *Effect of age, sex, breed and physiological status on selenium (Se), copper (Cu), zinc (Zn) and enzymes indicators of muscular fatigue in dromedrary (Camelus dromedarius).* Proc. 1st Intl Conf. of the International Society of Camelid Research and Development (ISOCARD), Al-Ain, 15-17 April 2006, United Arab Emirates, Poster n°66.

12. SEBOUSSI R., ALHADRAMI G., ASKAR M., **FAYE B.,** 2008. *Effect of excess selenium on dromedary camel in the United Arab Emirates.* Proc. of . Intern. Workshop, « Impact of pollution on animal products". Almaty (Kazakhstan), 27-30 September 2007, B. Faye and Y. Sinyavskiy (Eds), 143-146.

13. SEBOUSSI R., ALHADRAMI G., ASKAR M., **FAYE B**., 2009. *Selenium review in dromedary camels (Camelus dromedarius): selenium status, GSH-Px activity, hair and organs distribution and excretion.* Proc. of the 2nd conference of ISOCARD, Djerba (Tunisia), 12-14 march 2009, abstr. 157, p. 126

1-4. Autres articles prévus

14. FAYE B., SEBOUSSI R., 2009. *Variability of vitamin E concentration in camel plasma.* Soumis dans J. Camel Pract. And Res.

15. FAYE B., SEBOUSSI R., AL-HADRAMI G., 2009. *Meta-analysis of the interactions between oral selenium supplementation and haematological, biochemical and mineral parameters in camel blood.* En cours de redaction.

1-5. Ouvrage soumis pour publication aux EAU (cf. annexe)

16. FAYE B., SEBOUSSI R., AL-HADRAMI G., 2009. *Selenium in camel*

2- PROGRAMME DE COOPERATION

2-1. Supervision d'un nouveau travail de thèse sur le lait de chamelle

Le Pr. Louis LALEYE est intéressé pour démarrer un travail de thèse sur la technologie de transformation du lait de chamelle dont les propriétés physico-chimiques ne permettent pas une application identique aux technologies mises en œuvre pour le lait de vache. Un projet de thèse avait déjà été proposé en collaboration avec des collègues de l'Université de Montpellier (S. MARCHESSEAU), de l'INRA- Rennes (F. GAUCHERON) et de Supagro (G. LOISEAU) tous intéressés par le lait de chamelle comme modèle biologique d'étude.

Le projet comprenait des recherches sur le comportement des protéines du lait au chauffage, le devenir des micelles de protéines, les interactions avec les minéraux, la bio-activité des lactoprotéines. Une séance de travail avec le Pr. LALEYE a permis de mieux préciser les questions de recherche en adéquation avec la demande des industriels locaux. Les crédits fonctionnement sont disponibles ainsi que la bourse de l'étudiant pour les travaux en place aux Emirats. Il reste à identifier un bon candidat émirien, ce qui ressort des partenaires de l'Université. Un candidat est pressenti mais il convient de voir si son dossier est éligible (reconnaissance du master) pour une inscription à l'Université de Montpellier. Le projet global étant ambitieux, il est suggéré qu'il soit composé de 4 parties :

- Un PhD (inscrit à Montpellier II) sur les caractéristiques physico-chimiques du lait de chamelle, ses caractéristiques rhéologiques, et l'analyse de la fraction protéique
- Un master (français ou étranger) de l'IRC ou de Montpellier II (stage aux EAU) sur le prétraitement des laits par des techniques physiques ou enzymatiques ou des modifications de formulation
- Un master (émirien) du CFA (stage en France) sur la microbiologie des laits fermentés (souches de bactéries lactiques du lait fermenté)
- Une activité de laboratoire dans l'unité du Pr. LALEYE sur les essais technologiques

2-2. Proposition d'un protocole expérimental sur l'alimentation en minéraux et vitamines des dromadaires de course

Le dromadaire de course est un ruminant polygastrique, mais l'alimentation des champions, basée sur des rations riches en concentrés et en protéines de haute valeur, en fait un animal dont le régime alimentaire s'apparente à celui d'un monogastrique avec un risque important de déséquilibre minéral et vitaminique et surtout d'acidose gastrique. De plus, pour éviter ces désagréments, les producteurs distribuent largement des grandes quantités de suppléments sur la base des besoins des bovins souvent peu adaptés aux dromadaires.
Enfin, le dromadaire est très sensible aux maladies cutanées, ce qui est une contrainte majeure pour le bien-être et l'apparence des animaux utilisés notamment dans les « concours de beauté ». Par ailleurs, les suivis sanguins des animaux de course sont nombreux, mais leur interprétation reste difficile.

Sur cet ensemble de points, deux protocoles expérimentaux sont donc proposés (Annexe 2) :

1. Variabilité nycthémérale des éléments traces dans le sang circulant des dromadaires de course

2. Absorption du zinc et interactions avec les vitamines tégumentaires pour l'intégrité de la peau

Un document de projet sera soumis au Ministère de l'Agriculture et à l'Université pour obtenir les fonds nécessaires

2-3. Echange d'étudiants

Le doyen du CFA, le Pr. AL-HADRAMI a avancé l'idée d'une visite en France de 3-4 étudiants du secteur agro-alimentaire de l'Université (niveau master). Une telle visite pourrait se tenir dans le cadre d'Agropolis International (Montpellier) et comprendre des visites d'installations du dispositif agro-alimentaire français ainsi que quelques conférences dans ce domaine.

Dans le sens inverse, et pour alimenter les travaux de recherche proposés dans le paragraphe précédent, il est suggéré l'envoi d'un étudiant (français ou étranger) du master EPSED (Elevage dans les Pays du Sud –Environnement –Développement) codirigé par le CIRAD et Supagro-Montpellier. Il s'agit d'un stage de 4-5 mois réalisé entre avril et août de chaque année sur un thème pratique (enquête de terrain, expérimentation) faisant l'objet d'un mémoire.

Pour ces échanges, des appuis administratifs et financiers (billet d'avion pour l'étudiant français, voire séjour pour les étudiants émiriens) sont sollicités auprès de l'ambassade.

2-4. Participation à des cours

Une participation à des cours en tant que professeur visitant est envisagée pour ce qui me concerne au cours du cursus des étudiants en master au CFA. Le Pr. AL-HADRAMI étudie actuellement les modalités d'organisation d'une telle participation et sur quelle durée.

3 - L'ISOCARD

Issue de la conférence internationale tenue à Al-Ain en avril 2006, l'ISOCARD a tenu sa seconde conférence à Djerba (Tunisie) en mars 2009. Le Pr. AL-HADRAMI et moi-même étant toujours membres du Comité exécutif, nous avons en charge de la poursuite des activités de l'association. En résumé, les points de discussion ont été les suivants :

- préparation d'un partenariat entre *Emirates Journal of Food and Agriculture* (EJFA) et *Journal of Camelid Sciences*, la revue de l'ISOCARD. Un mémorandum a été discuté lors de la présente mission. L'objectif est de publier au moins deux numéros par an (publication en ligne et sur papier).
- Concrétisation d'une encyclopédie en ligne consacrée aux camélidés (*camelpedia wiki*) sur le site de l'ISOCARD (www.isocard.org) dont l'armature a été établie lors de mon séjour
- Préparation du questionnaire (*application form*) destiné aux candidats pour l'organisation de la prochaine conférence de l'ISOCARD en 2012 (quatre candidats pressentis : Maroc, Libye, Egypte et Oman)
- Représentation de l'ISOCARD auprès des conférences consacrées aux camélidés au Kenya en 2010 à Marsabit, en Grande-Bretagne en 2011 à Londres.

4 - CONCLUSION

La bonne valorisation de la thèse de R. SEBOUSSI encourage nos partenaires à poursuivre notre coopération. Pour la suite, il est donc proposé les actions suivantes qui pourraient être appuyées à partir de 2010.

- ➔ **une mission d'appui pour l'encadrement d'un nouveau thésard et d'un étudiant du master EPSED de Montpellier**
- ➔ **Un stage d'un mois en France pour un nouveau thésard (à identifier)**
- ➔ **un appui à trois ou quatre étudiants émiriens pour une visite des installations de recherche et dans le domaine agro-alimentaire en France et en particulier autour de Montpellier**
- ➔ **un appui administratif pour un stage d'un étudiant du master EPSED (billet d'avion)**

Conformément à nos discussions, l'appui du COCAC pourrait se faire par le biais d'une subvention globale au CIRAD.

ANNEXES

ANNEXE 1 - Calendrier de la mission et personnalité rencontrée

ANNEXE 2 - Protocoles de recherche proposés en nutrition

ANNEXE 3 - Revue de synthèse pour publication par l'Université

ANNEXE 1

Calendrier de la mission et personnalités rencontrées

Calendrier de la mission et personnalités rencontrées

Samedi 26 septembre

- Départ de Montpellier
- Arrivée à Al-Ain via Dubaï

Dimanche 27 septembre

- Entretien avec le Pr AL-HADRAMI, Doyen de la faculté, à l'Université
- Rédaction d'un travail de synthèse

Lundi 28 septembre

- Fin de la rédaction
- Entretien avec Louis LALEYE (Université d'Al-Ain) : projet de thèse

Mardi 29 septembre

- Réunion de travail sur l'édition de *Journal of camelid Sciences*, la revue de l'ISOCARD
- Rédaction d'un article sur les interactions sélénium * paramètres sanguins

Mercredi 30 septembre

- Départ pour Abu-Dhabi
  - Entretien avec M. Didier Gazagnadou, Conseiller de Coopération au COCAC d'Abu-Dhabi, et M. Etienne CAZIN (adjoint)
- Retour sur Al-Ain
  - Rédaction de l'article sélénium * paramètres sanguins

Jeudi 1er octobre

- Entretien avec le Pr. LALEYE : rédaction d'un projet sur le lait de chamelle
- Rédaction de l'article sur les interactions sélénium * paramètres sanguins

Vendredi 2 octobre

- Départ pour Dubaï
  - Entretien avec le Dr J.P. Girot (Genomix)
  - Rédaction du rapport de mission

Samedi 3 octobre

- Départ pour Montpellier via Paris

ANNEXE 2

Protocoles de recherche proposés en nutrition

Protocoles de recherche proposés en nutrition

EXPERIMENTAL PROTOCOL

Proposal for studying the interactions between zinc, copper, iron and vitamin A in racing camel

Context

Racing camel as other camels is a ruminating animal but according to the diet distributed to the champions, its feeding is closed to monogastric animals with high risk of mineral or vitamin imbalance, specific deficiencies, acidosis or other metabolic disorders. Elsewhere, to avoid such troubles, the camel owners are accustomed to distribute a large quantity of supplements on the basis of cattle requirements without taken in account the possible interaction between those elements; moreover, camel are very sensitive to skin diseases which is a main constraints for the animal welfare and the appearance of the animals which are of considerable importance in camel beauty exhibition. Elsewhere, the blood monitoring of the racing camel which is common lead to some difficulties for interpretation of the variability observed on animals within short period.

In that sense, metabolism of zinc, copper, iron and vitamin A which play an important role in skin and hair protection must be deepen in order to understand the eventual specificity of the camel and in a second step, to propose a convenient supplementation to the animals.

Experimental design 1: Nycthemeral variability of trace elements in blood of racing camel

- Animals: 6 adult camels (same age) will be monitored for 48 hours with blood sampling every 3 hours (i.e. 17 sampling), 3 times at 2 weeks interval. The animals must be in good health.
- Design: The camel will be shared into 2 groups: (i) 3 camels will receive basal diet, (ii) 3 camels will received the same diet for the first week, a different diet for the second week (enriched in mineral supplement) and a different also the third week (enriched in protein). Blood sampling must be achieved before food distribution in the morning

group	control	treated
Day 1-2	Basal diet	Basal diet
Day 15-16	Basal diet	Basal diet + minerals
Day 29-30	Basal diet	Basal diet + protein

- Analysis: Copper, zinc, iron, carotene and vitamin A in blood and in the diet

Experimental design 2: Absorption rate of zinc and interactions with vitamins for skin integrity

- Animals: at least 12 adult camels (approximatively same age) in good health without skin lesions will be monitored for 2 months
- Design: the animals were divided into three groups. The control group will receive a basal diet enriched in copper (zinc antagonist) in order to get zinc deficient feeding. The zinc supplemented group will receive zinc bolus (for example Zincosel ©) at the beginning of the trial. The zinc + vitamin supplemented group will received the same bolus + vitamin supplementation including vitamin A, B1 and B6, faeces and urine
- Sampling: blood (every week), skin and hair (day 0, 30 and 60), faeces and urine (continual 72 hours sampling of faeces and urine at the beginning, the middle and the end of trial)
- Analysis: trace element and circulating vitamin in blood. Zinc analysis in camel skin by laser induced-breakdown (Sun et al., 2000). Analysis of zinc in hair. Study of the correlation between zinc absorption and zinc in blood, hair and skin.

Reference

Qing Sun, Michael Tran, Benjamin W. Smith, James D. Winefordner, 2000. *Zinc analysis in human skin by laser induced-breakdown Spectroscopy*, Talanta 52: 293–300

ANNEXE 3

Revue de synthèse pour publication par l'Université

Revue de synthèse pour publication par l'Université

Selenium in camel

Bernard Faye, Rabiha SEBOUSSI, Ghaleb Alhadrami

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Abstract

Requirements for trace minerals in camel, particularly selenium, are not well-known. In Emirates, selenium supplementation using a pharmaceutical form or commercial mineral mixture is common practice in racing camels to address the cardiomyopathy often attributed to selenium deficiency. This supplementation is often empirical and based on estimated needs for cattle. Nowadays the use of selenium in animal foodstuff is commonplace and further investigation of its metabolism (ingestion, dynamic of storage-destocking, excretion) in camels is warranted. The present document aimed to synthesize all the experimental research (comparative selenium status in cow and camel, response to different levels of supplementation at different physiological stages, excretion maternal transfer, experimental toxicosis) and field observations (deficiency, supplementation practices) undertaken in camels in Emirates and around the world.

The results underline the particularity of the unique metabolic profile of the camel and lead to practical recommendations for supplementation in camels, highlighting its relative sensitivity to excess Se intake at lower levels than in cattle. The maximal tolerable dose is 8mg and the recommended dose range from 2 to 4 mg.

Keywords: **selenium, camel, requirements, deficiency, toxicity, excretion**

1. INTRODUCTION

Camels have some physiological peculiarities in their trace element metabolism due to their adaptation to arid conditions and poor mineral feeding resources (FAYE and BENGOUMI, 1994 and 2002). Some studies concerning trace elements such as copper, zinc, iron, manganese in camel have shown specific responses of this species to mineral supplementation or deficiencies. In general, the camel metabolism seems to anticipate mineral under-nutrition periods of its life with different mechanisms: increase of the absorption capacity in scarcity periods (copper, zinc) (Faye et al., 1999), higher storage capacity (copper) (FAYE and BENGOUMI, 1997), tolerance for minerals and electrolytes in excess (calcium, phosphorus, sodium) (EL-KHASMI et al., 2001, BENGOUMI and Faye, 2002), maintenance of enzymatic activity in deficient period (caeruloplasmin, superoxide-dismutase) (BENGOUMI et al., 1998a, ESSAMADI et al., 1998).

The adaptation to desert means an addition of small metabolic improvements which provide no comparative advantage when they are considered one by one, but give a full meaning to the reputation of this species when they are considered as a whole. This probably explains why the camel is able to survive under desert conditions characterized by low nutritive forages, scarcity of water, extreme climatic conditions.

Concerning selenium, there is little evidence to date of clinical deficiencies or toxicities, and up to recently, few available data on selenium requirements and metabolism in this species. However, new findings on selenium metabolism in dromedary camel have been recently reported (SEBOUSSI et al, 2008a, 2008b, 2009a, 2009b, 2009c, FAYE and SEBOUSSI, 2008 and 2009). The present document aims at giving a progress report on current knowledge concerning the status of selenium and its metabolism in the dromedary based on all data available in the scientific literature. Finally, recommendations will be done for the camel farmers and nutritionists according to the current results.

2. NORMAL SELENIUM LEVEL IN CAMEL BLOOD OR SERUM

The mean concentration of blood/serum selenium reported in the literature for large animals was around 100 ng/mL, value considered as sufficient for the maintenance of suitable metabolic functions (Maas et al., 1990). In the dromedary from Morocco, HAMLIRI et al., (1990) observed in whole blood, values varying according to age and sex, between 109.1 and 117.8 ng/mL being thus similar to those reported in sheep in the same area. Similar figures were recorded by Liu et al., (1994) in China with concentrations varying from 97 to 112 ng/mL. In Sudan, Abdel RAHIM (2005) reported values in whole blood varying between 25 and 53 ng/mL. Without specifying if it was whole blood or serum, Ma (1995) reported higher values on Bactrian camel: 274 to 288 ng/mL. The analytical method used could explain the observed differences, but the details of analysis procedures were not given in all the publications.

Serum concentrations approached these last figures: 281ng/mL on average in sera coming from the Sultanate of Oman (FAYE, unpublished data), but sampled on camels suspected of selenium unbalance. In Morocco, in dromedaries receiving probably a low Se basal diet, the plasma selenium concentration was quite lower, about 21 ng/mL (BENGOUMI et al., 1998b). Recently, in male adult camels in healthy conditions from Iran, the selenium concentration reported in serum was 12.6 ng/mL only (NAFIZI et al., 2009). In Saudi Arabia, serum Se values reported in young camels at the slaughterhouse varied between 5.3 and 131 ng/mL with 30% of samples higher than 100 ng/mL (BARRI and AL-SULTAN, 2007). In the United Arab Emirates (UAE), the

mean value was 200 ± 90 ng/mL in animals with no Se supplementation (SEBOUSSI et al., 2004). In recent experiments with different levels of Se supplementation, selenium content in serum for non-supplemented animals was on average 137.6 ± 18.7 ng/mL in non-pregnant, non-lactating camels (Seboussi et al., 2008a), 109.3 ± 33.1 ng/mL in pregnant females, and 103.4 ± 28.7 ng/mL at milking period (SEBOUSSI et al., 2009a).

The variability was thus high and the range between 12 and 200 ng/mL with an average of 100 ng/mL. However, in most of the reported values, the selenium status of the diet was unknown even if Se supplementation was not distributed to the animals. In some countries, the basal diet could be very low in natural selenium, or, at reverse, in high quantity under form with a high absorption rate. Also, the analytical procedures, as mentioned above, were not described in all the cases and could change between authors. In the experiments achieved in Emirates on adult females mentioned above, the pregnant (SEBOUSSI et al., 2009a) and non pregnant females (SEBOUSSI et al., 2008a) received 1.8 mg Se in the basal diet (6 kg of Rhodes grass -*Chloris gayana*- hay and 2 kg of concentrates), without any specific selenium supplementation. So, with approximatively 2mg Se per day brought by the normal diet, the level of Se in camel blood and serum is comparable to the other species level.

3. SELENIUM DEFICIENCY

For a long time, selenium deficiency has been suspected to occur in camels kept in zoological parks affected by cardiopathy or myopathy (Finlayson et al., 1971; WISNER and SCHOTKE, 1975; DECKER and McDermid, 1977) but no clinical descriptions and laboratory analysis have been made in these reports to confirm the role of selenium. In China also, LIU et al., (1994) suspected selenium deficiency in cases of sway-back in Bactrian camel. However, selenium deficiency with characteristic clinical signs has been recently reported in Emirates. Selenium deficiencies affect generally young animals and are responsible for white muscle disease, a degenerative muscle disease affecting muscle including the heart. Indeed, the most important lesions are degenerative myocarditis and discoloration of skeletal muscle. In the UAE, soils and feedstuffs are generally considered deficient in selenium, and **many cases of degenerative myocarditis (Figure 1a and b) are observed with histological lesions similar to those in cattle** (EL-KHOULY et al., 2001; SEBOUSSI et al., 2004).

Figure 1a. Degenerative myocarditis lesions in the heart of a one-month old camel calf (*Photo: R. SEBOUSSI*)

When the skeletal muscles are affected, symptoms vary from mild stiffness to obvious pain upon walking, to an inability to stand. Camel calves may tremble in pain when held in a standing position. When the problem occurs in newborns, they are born weak and unable to rise. Sudden exercise may trigger the condition in older camel calves. When the disease affects the heart, the animal shows signs similar to pneumonia, including difficult breathing, fever with an elevated heart and respiratory rates.

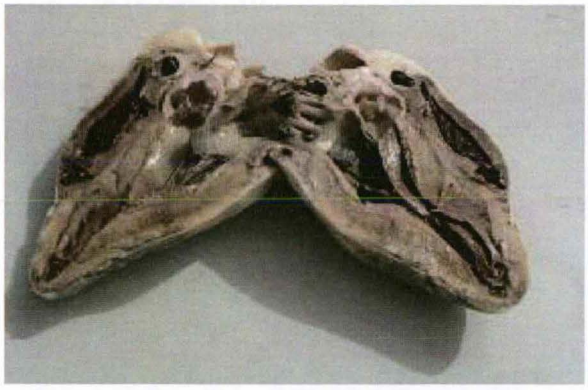


Figure 1b. Degenerative myocarditis lesions in the heart of a one-month old camel calf (*Photo: R. SEBOUSSI*)

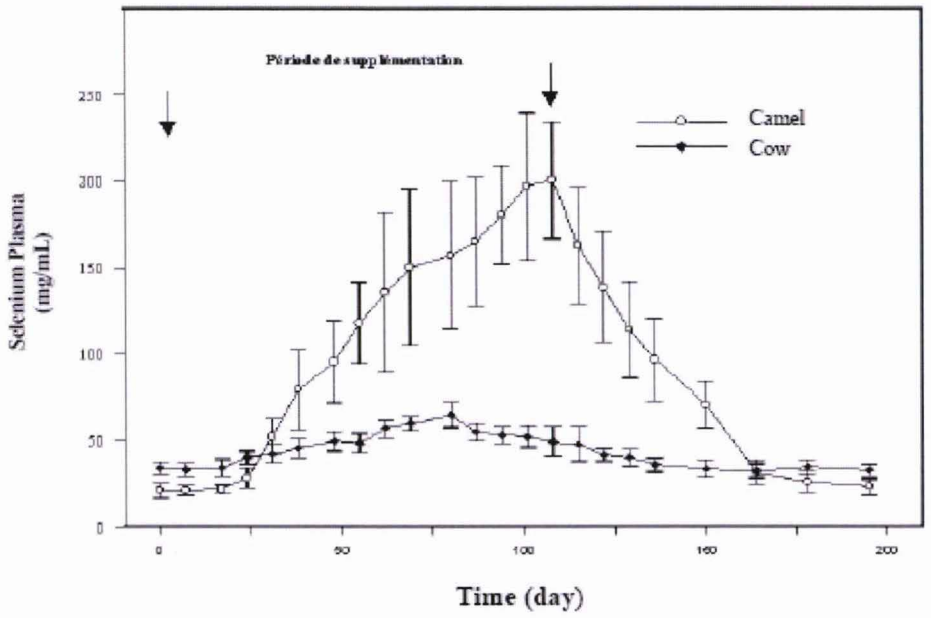
In sick animals, between 2 and 12 weeks old, remarkable signs of anemia were observed with reduction of hemoglobin concentration, slight decline of PCV% and total erythrocytes comparing to the normal levels reported in apparently healthy animals of the same age (EL-KHOULY et al., 2001) but these findings were not observed in three seleno-deficient adult camels from Saudi Arabia (EL-QARAWI et al., 2001). The histopathological findings showed alterations of the cardiac tissue with focal areas of non-inflammatory coagulative necrosis. The necrotic areas showed swollen myocardial fibers with granular cytoplasm and loss of striation. This was accompanied by severe blood vessels congestion, edema and lymphocytic infiltration. Calcium salt deposition was observed all over the necrotic area and fibrous area also (EL-KHOULY et al., 2001).

In sheep, selenium deficiency is diagnosed at blood level below 50 ng/mL. The serum selenium level in deficient camels was also obviously below this limit: in camel calves, the average level of Se serum in diseased cases was below 35 ng/mL (EL-KHOULY et al., 2001) and between 0.8 and 3.7 ng/mL in 3-yr animals (EL-QARAWI et al., 2001).

4. EFFECT OF SE SUPPLEMENTATION ON SE STATUS IN CAMEL

Few papers relate the impact of selenium complementation on the mineral status of camel and, generally, the doses applied for selenium deficiency control were those recommended for cattle. To our knowledge, the first trial achieved to assess the effect of selenium supplementation on the plasma selenium status was reported by BENGOUMI et al., (1998b). In this experiment, the selenium status of camels was compared with that of cattle with similar weight and receiving daily 2 mg Se *per os* under sodium selenite form for two months. The results showed a sharper increase of plasma selenium occurring in camels (10 times the plasma level before supplementation) compared to cows (twice the starting level) (figure 2). As the magnitude of the decrease of plasma selenium concentration after stopping supplementation was similar to the previous increase, it was supposed that plasma (or serum) selenium concentration in camel was an extremely sensitive indicator of selenium intake. The fast selenium depletion at the end of the supplementation period seemed also to indicate **a better efficiency of selenium absorption and excretion in camel compared to cow**.

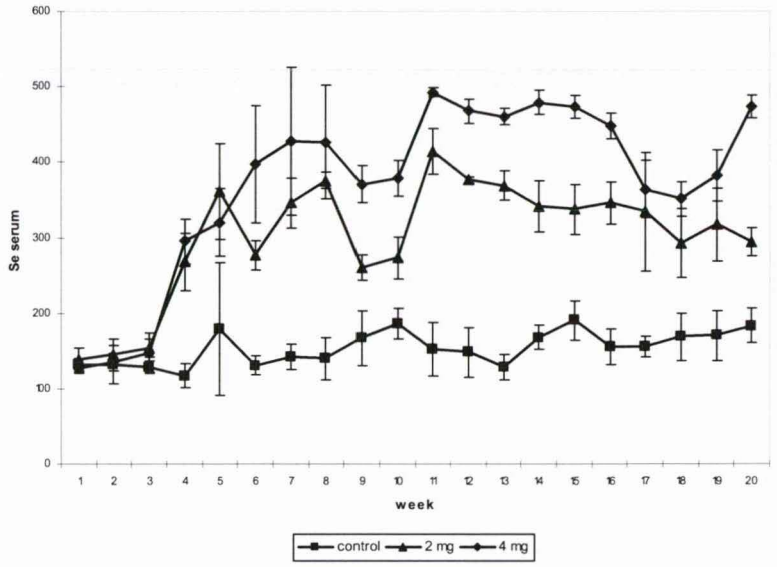
Figure 2. Comparative change in plasma selenium concentration in cow (•) and dromedary camel (o) receiving 2 mg/day selenium under sodium selenite form (BENGOUMI et al., 1998b).



In selenodeficient camels with muscular dystrophy, AL-QARAWI et al. (2001) gave an oral treatment involving selenium – vitamin E (Bo-SE, Schering – Plough Animal health, 2.19 mg sodium selenite + 50 mg vitamin E) by IM injection at a dose rate of 0.5 mg/kg body weight for two consecutive days. Following treatment, selenium concentration rose from on average 2.3 ng/mL up to 23.7 ng/mL, i.e. with a similar trend to that observed by BENGOUMI et al., (1998b). Indeed, the selenium concentration was multiplied also by 10 after supplementation.

In several studies on the effect of oral selenium supplementation (SEBOUSSI et al., 2008a, 2009a, 2009b, 2009c Faye and SEBOUSSI, 2008), different levels of supplementation were tested up to the toxic limit, from 2 up to 16 mg/day under sodium selenite form. In the first experiment, 12 non-pregnant and non-lactating female camels shared into three groups received, after a two-week adaptation period, an oral Se supplementation (0, 2 and 4 mg, respectively) under sodium selenite form for three months. Feed intakes were assessed daily, blood samples and body weight were taken weekly, feces and urine samples were collected every two weeks up to one month after the end of the supplementation period. The Se concentration in serum had increased significantly in supplemented groups (figure 3). The maximum level was observed in the period of supplementation in the camel receiving 4 mg (492.5 ng/mL), which was 4-fold compared to the value at the beginning of the trial (126 to 138.5 ng/mL depending on the groups).

Figure 3. Change in serum Se (in ng/mL) concentration in non-pregnant and non-lactating camels according to the Se supplementation level: 0 (■), 2 (✠) and 4 (♈) mg daily by period*treatment (reproduced from [9], with permission)



In the second experiment, 12 pregnant females, shared into two groups, received 0 and 2 mg Se respectively under sodium selenite form at the end of their gestation (last three months) and at the beginning of their lactation up to one month. The supplementation was stopped after one month of lactation. As for the previous experiment, feces and urine samples were collected every two weeks. The mean value of selenium content in serum was significantly higher in supplemented group (2 mg) and was three-fold higher than the concentration compared to the control group (305.9 ± 103.3 ng/mL and 109.3 ± 33.1 ng/mL respectively). The maximum level was observed two weeks before calving in the group receiving 2 mg (638.7 ng/mL). The selenium level at parturition was still significantly higher in the treated group in spite of a slight decrease around the calving period (Figure 4). On average, serum Se concentration in control and treated groups was significantly higher after parturition (121.6 and 349.7 ng/mL, respectively) than before (97.4 and 272.7 ng/mL respectively) in spite of stopping supplementation in the treated group.

In the third experiment, eight young female camels shared into four groups of two 2-y old ones received a basal diet enriched with 0, 2, 4 and 8 mg selenium under sodium selenite form for 64 days. On average, the mean values of selenium in the serum was 176.3 ± 18.0 ng/mL in the control group, 382.7 ± 107.6 ng/mL in the group receiving 2 mg Se, 519.8 ± 168.4 ng/mL in the group receiving 4 mg Se and 533.4 ± 158.6 ng/mL in group receiving 8 mg Se daily. The weekly change showed a significant increase ($P<0.001$) from week 2 up to the end of the experiment in the three supplemented groups compared to the control one (Figure 5). There was no difference between the groups receiving 4 and 8 mg Se. The maximum value (657.3 ng/mL) was observed in the group 3 at week 9 and the minimum at the beginning of the experiment (124.1 ng/mL in group 3 also).

Figure 4. Biweekly changes (mean and S.E) in serum selenium concentration (in ng/mL) in camels before and after parturition according to the selenium supplementation level, 0 mg/day (✠) and 2 mg/day (♈)

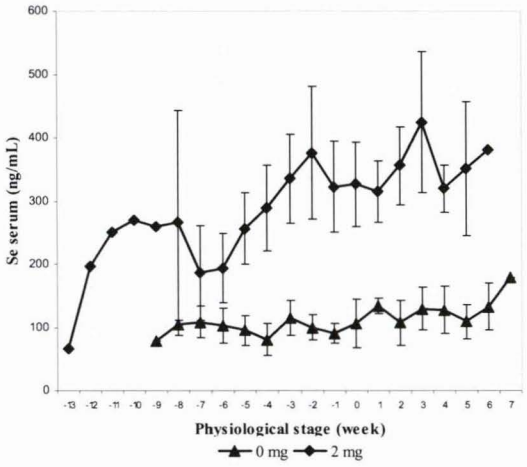
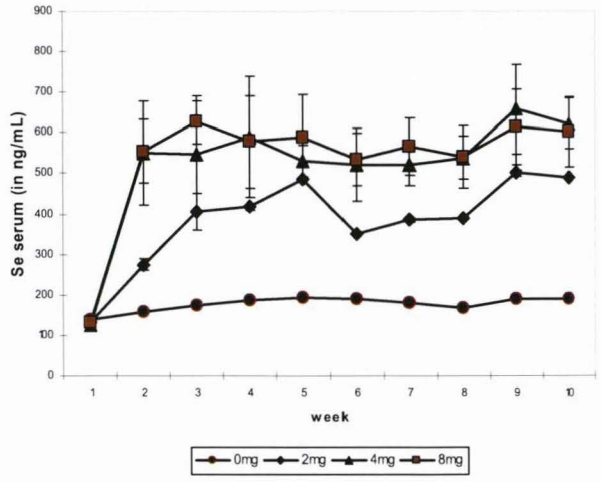


Figure 5. Weekly change in serum selenium according to Se supplementation in the basal diet of camels, 0 mg/day (●), 2 mg/day (♈), 4 mg/day (✠) and 8 mg/day (■)



In the fourth experiment, the quantity of supplied selenium throughout the trial (90 days) was for each group of four camels, 2-yr old, respectively 8 mg (i.e. 17.44 mg sodium selenite), 12 mg (i.e. 26.16 mg sodium selenite) and 16 mg (i.e. 34.88mg sodium selenite) daily. Selenium supplementation was stopped immediately at the time of apparition of chronic selenosis and hepatoprotector was given to prevent death. Camels returned to normal good health gradually. On average the mean value of selenium in serum was 358.3 ± 210.8 ng/mL (n=69) and varied between 16.3 and 899.8 ng/mL. The mean values of selenium in serum were 321.2 ± 140.5 ng/mL in group 1 (8 mg Se), 443.2 ± 231.1 ng/mL in group 2 (12 mg Se) and 298.04 ± 212.13 ng/mL in group 3 receiving 16 mg Se daily. The bi-weekly change showed a significant increase (P>0.001) from fortnight 2 up to the end of the experiment for groups 1 and 2 and up to fortnight 3 for group 3 with a value of 767.15 ng/mL. Serum Se concentration decreased significantly in fortnight 4 in group 3 up to the end of the trial to reach a value of 129.86 ng/mL when Se supplementation was stopped when selenosis symptoms appeared (Figure 6). The maximum observed value was 899.87 ng/mL.

A meta-analysis of the data including oral supplementation from 0 to 16 mg/day (only values after at least one week of supplementation were taken into account) showed clear linear relationships up to 4 mg, then a slight increase with a plateau after 12 mg/day (***Figure 7).***

Figure 6. Changes in serum Se concentrations according to the selenium supplementation level in camels (Mean and S.E) at 8 (•), 12 (✠) and 16 mg/day (♈). The * points to the Se supplementation stopping in group 3

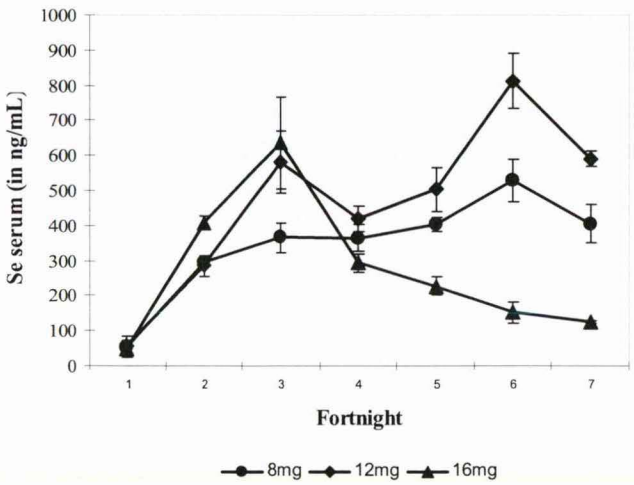
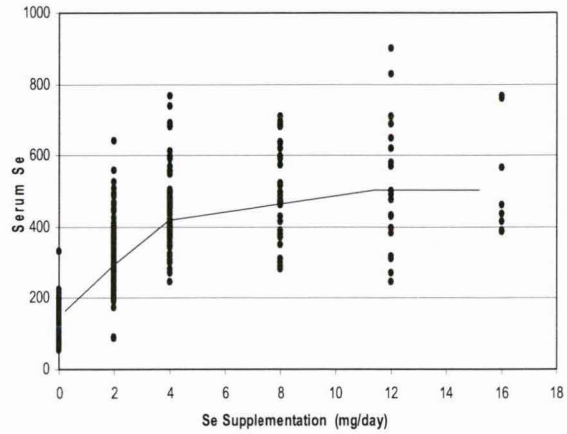


Figure 7. Change in camel serum selenium according to the level of oral supplementation

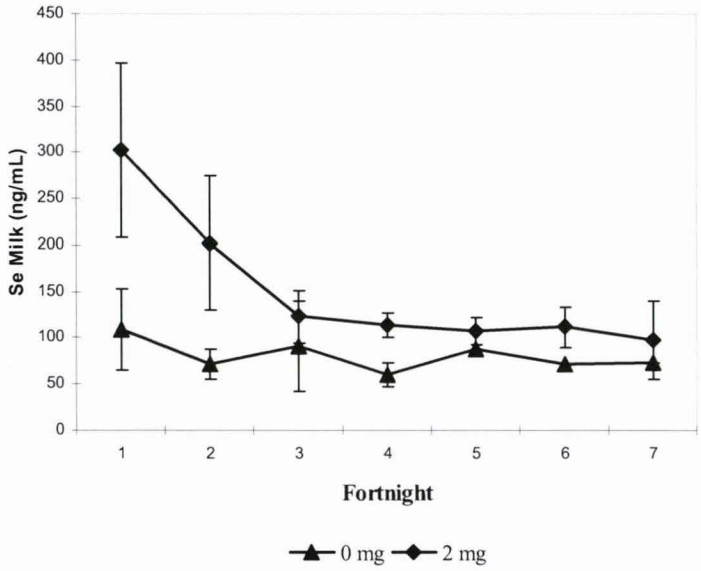


In the previous experiment on lactating camels (SEBOUSSI et al., 2009a), Se was determined in camel calves after birth and in milk. Se supplementation involved dams only. Se serum concentrations in camel calves at parturition were 106.3 ± 26.5 and 273.2 ± 48.0 ng/mL, in the control (0 mg/day) and treated groups (2mg/day), respectively. This significant difference ($P < 0.001$) was maintained for the entire milking period: 103.4 ± 28.7 and 248 ± 14.1 ng/mL in the control and treated groups, respectively.

In milk, the Se concentration varied from 39.5 to 482.6 ng/mL with an average of 86.4 ± 39.1 ng/mL in the control group and 167.1 ± 97.3 ng/mL in the treated group. At birth, Se concentration in colostrum was three-fold higher in the treated group: mean value 302 ± 94.60 *vs* 108.2 ± 43.9 ng/mL ($P < 0.001$). In both groups, Se milk concentration decreased and after the second milk sampling, no significant difference was observed (Figure 8). By considering Se concentration in colostrum and the status of the mothers and of their camel calves at parturition, positive correlations were observed with serum Se in mothers ($r = 0.659$; $P < 0.05$) and in calves ($r = 0.689$; $P < 0.05$).

The reported values of selenium in camel milk are quite scarce. AL-AWADI and SRIKUNAR (2001) reported a much lower value (13.9 ± 2.4 ng/mL) than SEBOUSSI et al., (2009a) but the former did not mention the lactation stage. In a meta-analysis performed on cattle's data (CEBALLOS et al;, 2009), it has been considered that the selenium increase in milk was on average 12.6 ng/mL only after oral Se supplementation at a dose of less than 3 mg/day under selenite form. In comparison, the apparent good efficiency of Se transfer in camel milk has to be confirmed.

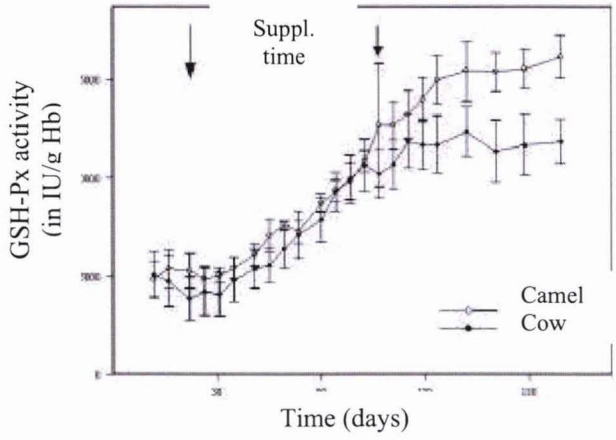
Figure 8. Biweekly changes (mean and S.E) in milk selenium concentration (in ng/mL) in she-camels for the three first months of lactation according to the selenium supplementation level: 0 mg/day (✠) and 2 mg/day (♈)



5. CORRELATIONS OF SELENIUM WITH GSH-PX ACTIVITY AND VITAMIN E

For its transport in blood, selenium is linked to specific protein (selenoprotein) including glutathione peroxidase (GSH-Px). In the comparative study of BENGOUMI et al., (1998b), the increase of GSH-Px activity was similar in camels and cows for the supplementation period with a higher correlation in camels (r = 0.94) than in cows (r = 0.68). As for other species, **GSH-Px is a good indicator of the Se status of camel.** However, after the end of the supplementation, GSH-Px activity continued to increase in camels' blood while it was stable in cows' (*Figure 9*). A similar figure was observed by SEBOUSSI et al. (2008a).

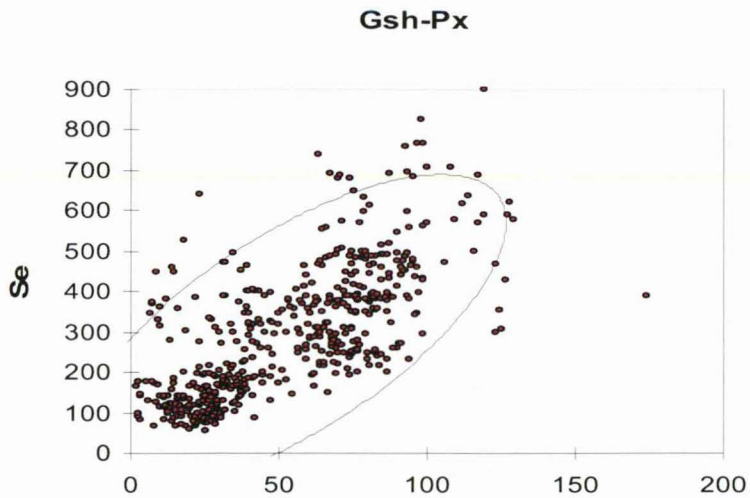
Figure 9. Comparative change in GSH-Px activity in cows (•) and dromedary camels (o) receiving 2 mg/day selenium under sodium selenite form



This increase could be explained by the maintenance of the biosynthesis induction in the camel erythrocytes from the selenium probably stored in the erythrocytes, and a longer plasmatic half-life of GSH-Px compared to those of cattle. In fact, the erythrocyte GSH-Px activity being closely related to the half-life to the red blood cells, the enzymatic activity was higher in camels than in cows when selenium was depleted because of the longer survival of camel erythrocytes (Yagil et al., 1994).

The linear relationship between erythrocyte GSH-Px and whole blood Se concentration was described in cames by several authors (Hamliri et al., 1990; ABDEL-RAHIM, 2005; SEBOUSSI et al., 2008a, 2009a, 2009b, Barri and Al-Sultan, 2007) but with variable correlation coefficients (Figure 10).

Figure 10. Relationship between Se serum and GSH-Px in camels according to data meta-analysis of SEBOUSSI et al., (2008a, b, 2009a, b and c) (r =0.699 ; P< 0.001)



Vitamin E is an essential component in the reproduction processes and performance of farm animals and acts in synergy with selenium (Se), especially in order to prevent white muscle disease (WMD) due to a severe deficiency. In the literature on camel from the UAE, the mean values were 1.13 ± 0.61 µg/mL (non-lactating and non pregnant), 1.12 ± 0.81 µg/mL (pregnant), 1.20 ± 0.80 µg/mL (lactating), 0.82 ± 1.06 µg/ml (new-born), 0.56 ± 0.22 µg/mL (young 2-y old) and 0.68 ± 0.36 µg/mL (Se intoxicated young camels) (Faye and SEBOUSSI 2009b). These results were quite similar to those described in young camels from Sudan (0.3 to 1.65 µg/mL) Al-Senaidy (1996). Similar results were reported by Corbera et al., (2003) and Mousa et al. (2006). In all the cases where serum Se and vitamin E were analyzed, **no correlation was observed**. In case of Se intoxication, a tendency to the decrease of vitamin E in intoxicated animals with clinical signs was observed but no significant correlation was reported with serum Se concentration (SEBOUSSI et al 2008b, 2009 c and d).

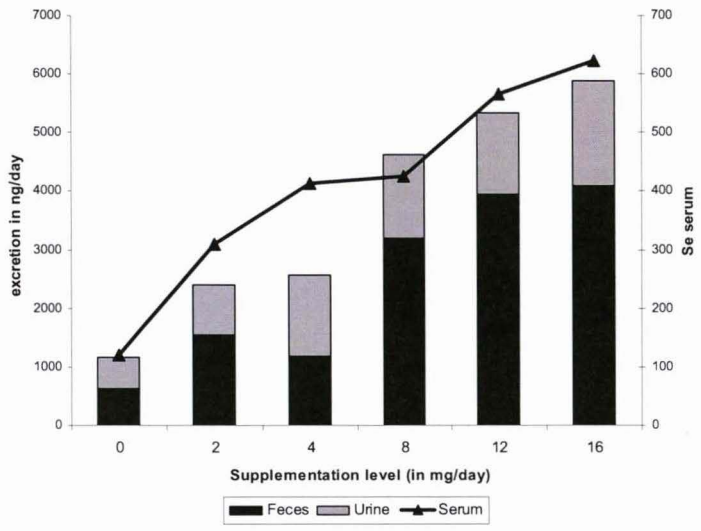
6. SE EXCRETION

Very few data are available on fecal and urinary Se excretion in camel. According to the different trials achieved in Emirates with variable levels of Se supplementation in the diet, Se fecal excretion increased slowly up to 4 mg Se in the diet, then highly from 8 mg daily supplementation up to 16 mg (*Figure 11*). The total fecal excretion varied from 637.9 ng/day in non-supplemented camels up to 4084.4 ng/day in camels

receiving 16 mg Se/day in the diet. The total fecal excretion was comparable to urinary excretion when administering up to 4 mg supplementation, but the main part of Se excretion after 8 mg of supplementation was of fecal origin. The total urinary excretion varied from 518.5 ng/day (control groups) up to 1795.9 ng/day (16 mg Se supplemented group). **Forty-five percent of the excreted Se was from urine in non-supplemented animals *vs* 26-30% only in highly supplemented camels**. Moreover, Se concentration in serum was highly correlated with Se concentration in urine and with fecal concentration and total Se fecal excretion but not with total urinary excretion.

Similar change in Se excretion was observed in cattle (JUNIPER et al., 2006). When the dietary intake was increased from 0.15 to 0.40 mg/kg DM in cattle, the selenium concentration in feces and urine increased significantly from 370 to 780 ng/g DM and from 20 to 180 ng/mL, respectively (Juniper et al., 2006)], close to the results from SEBOUSSI et al. (2009a): on average 225 and 817 ng/g in the control and the treated group, respectively. But, contrary to this former observation achieved in dairy cattle, no linear effect was observed in camel.

Figure 11. Fecal and urine excretion of selenium according to the level of supplementation (data meta-analysis of SEBOUSSI et al., 2008a, b, 2009a, b and c).



The urinary selenium concentration is considered to be a sensitive indicator of sodium selenite consumption more than nutritional requirements (LENG et al., 2000). Elsewhere, the camel is well-known for its water metabolism and its ability to excrete a more concentrate urine although the watering was *ad libitum* in the mentioned trials. The high Se urinary concentration, particularly in camels receiving Se in their diet, compared to cattle, seems to demonstrate a peculiar sensitivity to Se supplementation and a specific role of kidney in Se regulation.

7. SE STORAGE IN ORGANS

Selenium determination in organs has rarely been reported because it is of no clinical interest. In the wool of Bactrian camel from China, LIU *et al.*, (1994) reported values between 140 and 190 µg/kg depending on their physiological status. Similar results have been published by Ma (1995): 190 to 210 µg/kg. These values corresponded to camels

receiving 2mg Se supplementation in the experiment of SEBOUSSI et al., 2009c (163.6 µg/kg). In lambs, the wool Se concentration varied between 500 and 2500 µg/kg depending on the dietary Se level (CRISTALDI et al., 2005). Part of the selenium ingested is involved in hair amino acids synthesis. It was suggested that a level of selenium content should be higher than 120 ppm in cow and calf hair to avoid nutritional myopathy. Season, color of hair, age and sex, affect the selenium content in hair. The selenium concentration was higher in winter than in summer and in dark color hair than in light color hair. Hair appeared as the most sensitive organ to Se supplementation as it was reported on lamb (Lomba et al., 1973) and cattle (Perry et al., 1976). However, as with other minerals, the selenium concentration in hair is of limited interest (Combs and Combs, 1986).

In the experiment of SEBOUSSI et al (2009c), the highest total quantity of selenium was observed on average in order, in the liver (2727 µg), the kidney (807 µg), the lung (443 µg) and the heart (160 µg). Of course a high quantity was also observed in muscle (2513 µg). On average, whatever the Se supplementation level, the kidney (1129 µg/kg), the liver (921 µg/kg), the hair (545 µg/kg), the forelimb muscle (421 µg/kg), the hind limb muscle (351 µg/kg) and the lung (308 µg/kg) had the highest Se concentrations. The total quantity was higher in supplemented groups but, except in hair, liver, kidney and muscle, the quantity was not clearly linked to the Se supplementation level. By giving an index of 100 for concentrations in animals receiving 0 mg Se supplementation, the main organs where selenium was stored were hair, liver and muscles, and to a lesser extent lung, ovary and pancreas (*Figure 12*). In Bactrian camel, one reference only was available for selenium concentration in organs (Lu et al., 1995). In this study, kidney (3100 to 3900 µg/kg), liver, and heart (1100 to 1500 µg/kg), muscle and brain (620 to 640µg/kg) were the organs with the highest Se concentrations. These values, except those of the liver, appeared much higher than those found by SEBOUSSI et al. (2009b and c).

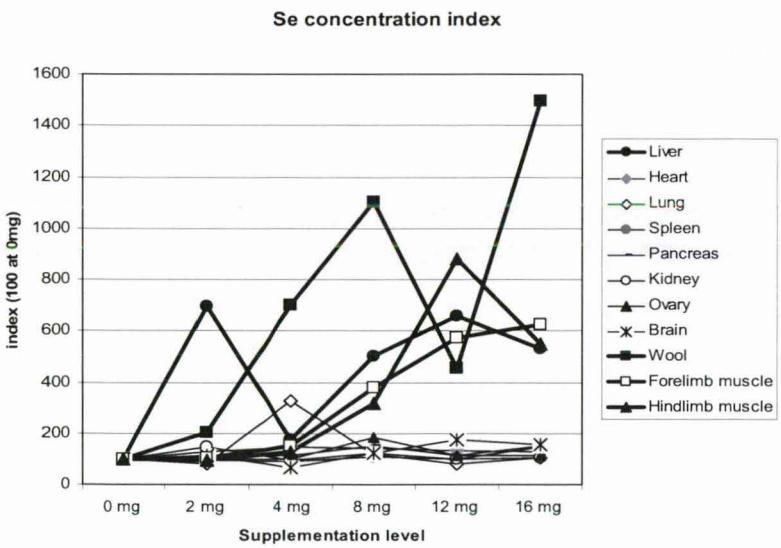


Figure 12.
Selenium concentration index in the different camel organs according to the Se supplementation level in the diet. The index 100 corresponds to Se concentration for 0 mg supplementation (data meta-analysis of SEBOUSSI et al, 2008b, 2009b and c).

In the selenium tolerance trial carried out in lamb (CRISTALDI et al, 2005), the liver had the highest Se concentration (up to 2000 µg/kg), followed by the kidney (around 1000 µg/kg). In another study on sheep, selenium was found in the highest concentrations in the kidney, followed by the liver, pancreas, heart and skeletal muscle (COMBS and COMBS, 1986). No linear trend of liver Se concentrations according to the Se supplementation level was observed in lamb (CRISTALDI et al., 2005). In calves receiving 3 ppm dietary Se treatment, Se concentrations were 4740 µg/kg in liver, 3420 in kidney, 1380 in heart and 340 in muscle (JENKINS and HIDIROGLOU, 1986). Contrary to

CRISTALDI *et al.* (2005), a regular increase of Se concentration with dietary Se level was observed by these authors. According to them, **the kidney was the major organ involved in the storage of selenium at low Se supplementation, but at high intakes, the liver became the target organ** (Jenkins and Hidiroglou, 1986; CLARK and STRUKLE, 1996). Similar figures could be observed with camel (SEBOUSSI et al., 2009b and c).

However, considering the weight of whole carcass and of the different organs in camel, the total quantity of selenium in a camel of 200 kg carcass weight was around 100 mg with 90% in the muscle, 5.5% in the blood and 2.5% in the liver. Less than 1% was stored in the kidney.

8. SE TOXICITY

At our knowledge, only experimental selenosis has been reported (FAYE and SEBOUSI, 2008, Seboussi et al., 2009 b). The first clinical disturbs appeared with a selenium supplementation of 8 mg/day. The first physiological symptoms were an increase of the respiratory rate, pulse rate, and internal temperature up to 40 °C.

The **clinical signs** occurred within two weeks with hair discoloration, followed by alopecia (*Figure 13*), more severe in animals receiving a higher quantity of selenium (up to 16 mg/day). Enlargement of the inferior cervical lymph node was seen in all intoxicated animals (figure 14). Camels tended to sit alone. Urinary excretion increased and dark watery diarrhea was also observed. Loss of appetite, thus loss of weight and weakness appeared. Tears with pale mucous were present as well as evidence of impaired vision. Dyspneic respiration and pain at auscultation appeared and camels adopted the sternal decubitus position and tended to rest their neck extended (Figure 15). Salivation occurred and finally camels showed no desire to eat and drink. The tail was elevated. Fissured pads appeared in all groups but more pronounced in groups receiving 12 and 16 mg. Consequently, camels had difficult walking (Figure 16).

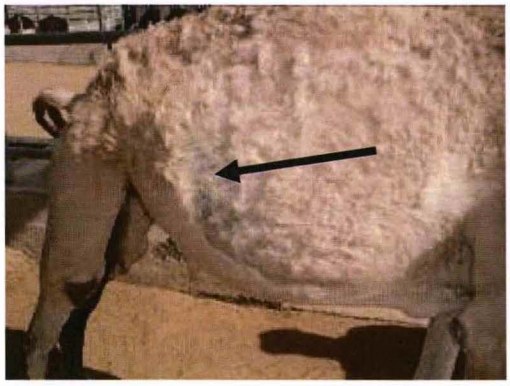
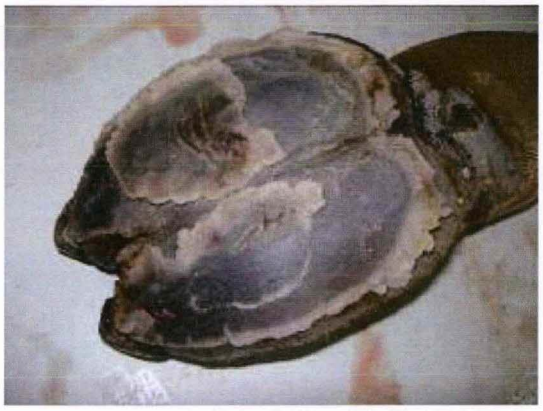


Figure 13. Alopecia on the abdominal part of a young camel receiving 16 mg Se/day (*Photo R. SEBOUSSI*)

Figure 14.
Enlargement of the inferior cervical lymph node in intoxicated animals. (*Photo R. SEBOUSSI*)

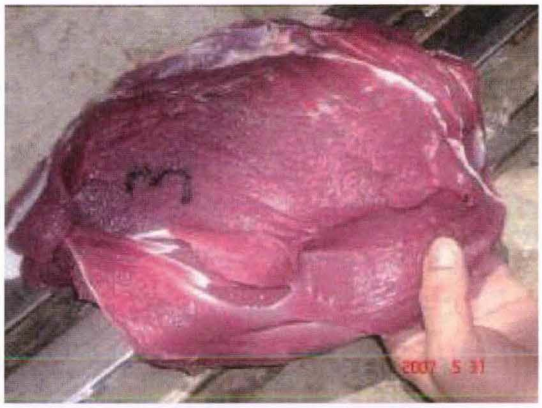
Figure 15. Camel in sternal decubitus position with neck extended on ground. (*Photo R. SEBOUSSI*)

Figure 16. Fissured pads with necrosis on foot of camel receiving 16 mg Se/day (*Photo R. SEBOUSSI*).



After slaughtering, intoxicated camels showed paleness in all abdominal muscles (Figure 17), paleness of diaphragm and intercostal muscles, hydrothorax (figure 18), pulmonary emphysema. The texture of the liver and lung was not uniform. Heart, liver and kidney were congested and necrosed. In addition to prior lesions, camels showed a flap heart with necrosis and congestion (Figure 19). However, the heart was partially white (fibrosis), congested and necrosed in camel receiving 8 mg. Hepatomegaly (*Figure 20*) was observed in all animals, while pancreas was atrophied. Brain edema was also observed (*Figure 21*).

Figure 17. Discoloration of the red muscle in Se intoxicated camel receiving 16 mg Se daily (*Photo R. SEBOUSSI*)



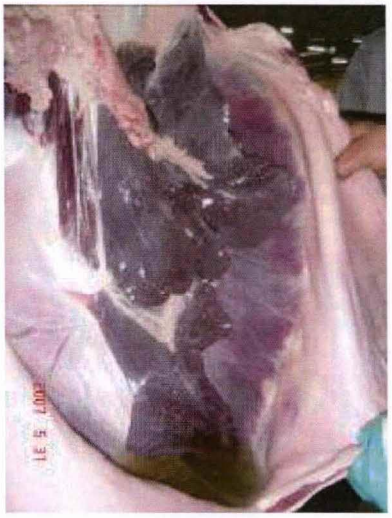


Figure 18. Hydrothorax in intoxicated animals with 16 mg Se daily (*Photo R. SEBOUSSI*)

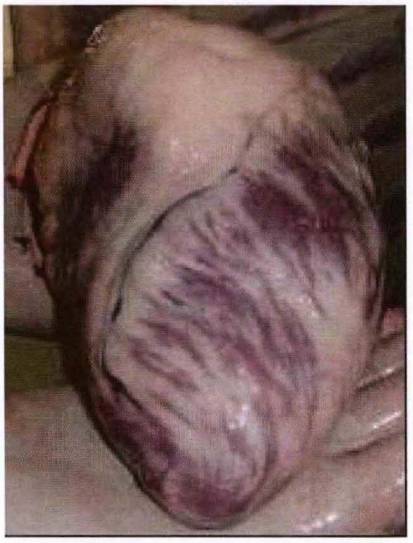


Figure 19. Heart discoloration and congestion in camel receiving 8 mg selenium per day for 45 days (*Photo R. SEBOUSSI*)

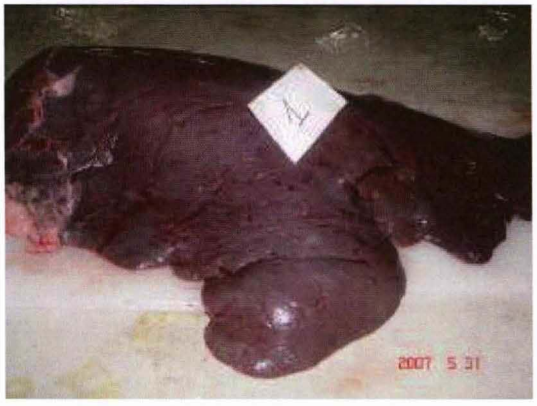


Figure 20. Hepatomegaly and liver congestion in intoxicated animals with 12 mg Se daily (*Photo R. SEBOUSSI*)

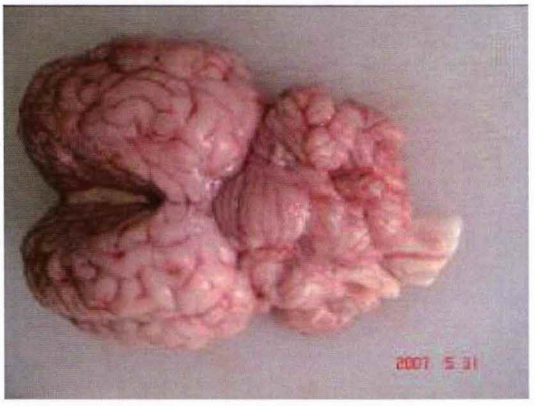


Figure 21. Brain edema in Se intoxicated camel with 16mg Se daily (*Photo R. SEBOUSSI*)

Histopathology lesions involved all the organs, and the lesions increased with the quantity of selenium in the diet. Kidneys showed eosinophilic granulated material in diluted Bowman's space and convoluted tubules in addition to degenerative changes in epithelial lining cells (*Figure 22*). The heart showed proliferation of Purkinje fibers, capillaries congestion in Purkinje fiber tissues and sub-endocardial tissues, degenerative changes in myofibers (*Figure 23*). The cardiac tissues showed edematous fluid between more eosinophilic thick myocardial fibers (*Figure 24*). Vacuolar degenerative changes were observed all over the hepatic cells of the hepatic lobules, as well as congestion in central hepatic vein and hepatic sinusoids (Figure 25). In addition, focal areas of muscular hyalinization (non–inflammatory) and edema were observed in intercostal and diaphragm muscles (*Figure 26*). In addition focal coagulative necrosis areas appeared in pancreatic acinis (*Figure 27*). Hyaline degeneration of myofibers and edema was also observed in shoulder and intercostal muscles. The brain showed perivascular edema.

Figure 22. Congestion of bllod capillaries of kidney medulla and glomerular, and epithelial degenescence (*Photo EL-KHOULY).*

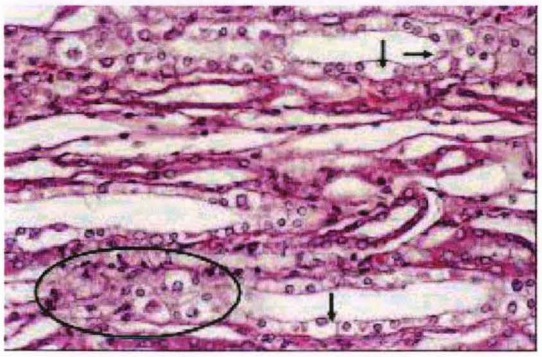


Figure 23. Proliferation of Purkinje fibers, capillaries congestion in Purkinje fiber tissues in heart (*Photo EL-KHOULY).*

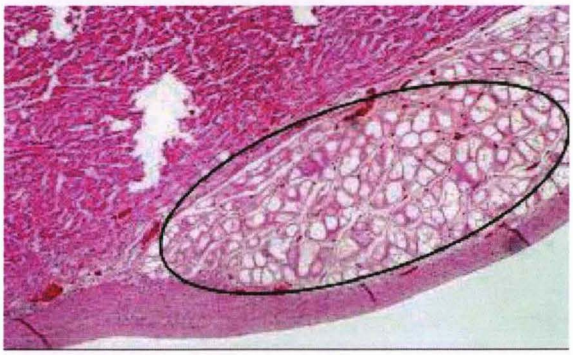


Figure 24. Congestion of the capillary vessels in the camel heart in camel receiving 12 mg Se/day (*Photo EL-KHOULY).*

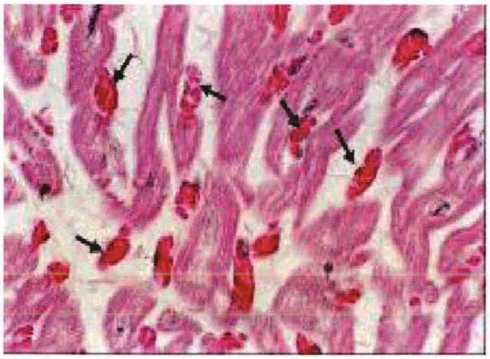
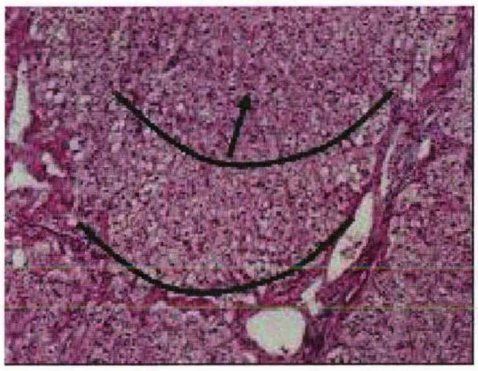


Figure 25. Hepatic cells degene-rescence around the portal vein in camel receiving 16 mg Se / day (*Photo EL-KHOULY).*



The clinical symptoms observed in camel were in accordance with previous signs reported in chronic poisoning in other species (CASTEEL et al., 1985; HARRISSON et al., 1983). When selenosis injury occurred, the selenium accumulated mainly in the circulatory and respiratory system as well as in the organs of elimination (BEATH, 1982). These findings were in accordance with lesions observed in the heart, lung, liver, kidney and urinary bladder. After the liver, the kidney, particularly the cortex, retained the highest concentration followed by the glandular tissues, especially the pancreas and pituitary. This explained the high Se level in kidney, the lesions occurring in cortex and medulla, the degenerative changes and necrosis found in the current study. The gross and histologic lesions reported in camel were comparable to those observed in lambs (TIWARY et al., 2006) and suggest that the heart, as target organ of selenium intoxication, failed, leading to pulmonary edema and hydrothorax (LAMBOURNE and Mason, 1969).

The foot lesions with the necrosis of keratonocytes were comparable to those observed in alkali disease (chronic selenosis) in cattle (O'TOOLE and RAISBECK, 1995) and horse (RAISBECK et al., 1993) in spite of the lack of hooves in camel.

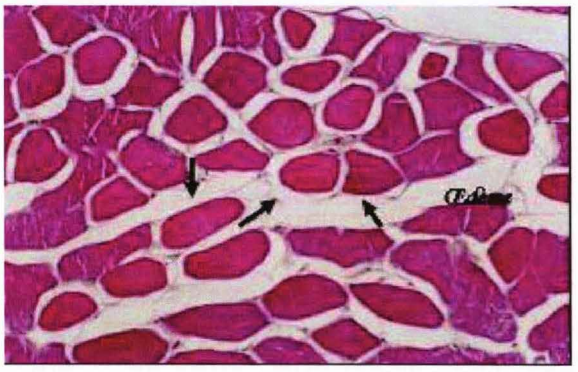


Figure 26. Edema and hyaline degenerative fiber (non-inflammatory) in intercostal muscle (*photo EL-KHOULY)*

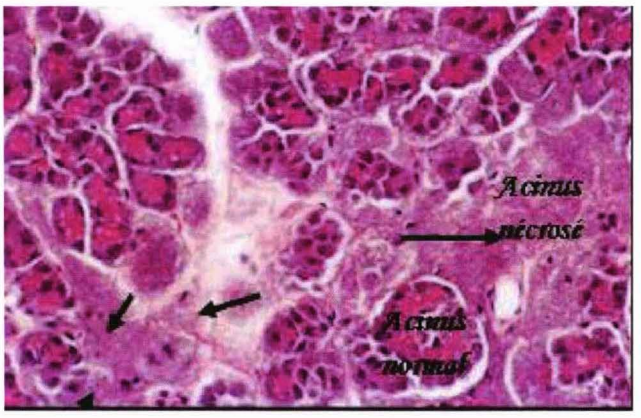


Figure 27. Focal coagulative necrosis areas in pancreatic acinis (*photo EL-KHOULY)*

Selenium deficiencies in animal, including camel, can result also in damages to the liver, heart, kidney and skeletal muscles (EL-KHOULY et al., 2001; HAMMOND et al., 1980). So, comparable necropsy lesions were reported on Se deficiency and toxicity. The lack or the excess of selenium seems to lead to similar cell damage.

According to the recommendations for beef cattle (NRC, 2000), the minimum level of selenium in the diet that causes chronic selenosis in most animal species is 4-5 mg/kg of dry matter (DM) and the minimum level needed to prevent deficiency is 0.02 – 0.05 mg/kg DM. In the experimental selenosis described by SEBOUSSI et al., (2009b), the first symptoms appeared with a diet containing 2.5 mg/kg DM only. Chronic Se poisoning is not limited to grazing livestock and can occur from consumption of high Se intake in feed. For example, in the UAE, camels' owners supplement their animals to avoid deficiency

with a commercial salt mixture and pharmaceutical form by drench or injection. However, no data on camel selenosis has been reported. The question of the **poisoning threshold** in camel has not been clearly determined. Oral ingestion of 1 to 2.2 mg of Se/kg life weight (LW) as sodium selenite has caused appreciable mortality in lambs up to 14 weeks of age (Gabbedy, 1970), but individual susceptibility to selenosis could be highly variable. Tiwary *et al.* (2006) did not observe lamb mortality with an oral sodium selenite up to 4 mg/kg LW. For other authors, the oral median lethal dose (LD50) of sodium selenite has been reported to be 1.9 ±1.2 mg of Se/kg LW (MUTH and BINNS, 1964; LAMBOURNE and MASON, 1969; CARAVAGGI et al, 1970; BLODGETT and BEVILL, 1987; YAEGER et al., 1998). A daily intake of 0.25 mg/kg LW is considered as toxic for sheep and cattle (MUTH and BINNS, 1964). Selenium poisoning was observed with diet containing 44 mg/kg DM for horses and 11 mg/kg DM for pig (MUTH and BINNS, 1964). Typical lesions of chronic selenium toxicosis were observed on young cattle receiving more than 5 mg/kg DM for 120 days (O'Toole and Raisbeck, 1995).

These levels listed previously are higher than the dietary levels in the studies performed on camel (SEBOUSSI et al, 2008b, 2009b; Faye and SEBOUSSI, 2008), i.e. 0.051 to 0.095 mg/kg LW, which seems to show a high sensitivity of camel species to Se toxicosis. The levels of selenium requirement and toxicity could be very close. For example, in intoxicated lambs with 4 mg/kg LW under sodium selenite form (four times higher than the camels receiving 16 mg Se daily in the trial of Seboussi et al., (2009a), the serum Se increased up to 274 ng/mL only (TIWARY et al., 2006), compared to 767 ng/mL observed in camel (SEBOUSSI et al., 2009b).

After one month supplementation with 12 ppm Se in the diet, pregnant cattle showed Se values in serum above 420 ng/mL (Yaeger et al., 1998). Higher values up to 1500 ng/mL were reported on large animals grazing on seleniferous pastures (RAISBECK et al., (1993). In lambs, with a diet containing 10 ppm of selenium (YAEGER et al., 1998), no toxicity was observed after one year and the selenium values reached 0.39 ppm in serum (390 ng/mL) after 12 weeks (comparatively to the results of SEBOUSSI et al., (2009a): after 90 days, 519 ± 97 ng/mL for groups receiving 3.5 ppm Se in the diet only).

9. INTERACTIONS WITH MINERALS, HEMATOLOGICAL AND BIOCHEMICAL PARAMETERS

In most of the studies including other mineral parameters as **copper, zinc or iron**, a negative correlation was observed with selenium concentration in blood (SEBOUSSI et al., 2004, 2008a and 2009a). However, this relationship was not observed in case of Se intoxication (SEBOUSSI et al, 2009b and c). There was probably interaction between selenium (Se^{--}) and copper or zinc (Cu^{++} or Zn^{++}). For example, in rat, zinc induced a decrease in the excretion of selenium in urine (CHMIELNICKA et al., 1988) and interaction at molecular level was described in human (MARET, 2000). But in other cases, no interaction was observed (HANSEN and KRISTENSEN, 1980). According to the result of the meta-analysis of several experiments (SEBOUSSI et al., 2008a, 2009a, b and c), the iron concentration in serum decreased significantly from 8 mg Se supplementation in the diet, as well as zinc concentration and in a less extend, copper concentration (*Figure 28*). The Se intoxication being linked to anemia and inflammation process of organs, the changes in trace element concentration above 8 mg supplementation could explained the drastic decrease of iron and zinc in serum. As copper concentration in blood generally increased in case of inflammation, the slight rise again from 12 mg Se supplementation could be linked to the damage of target organs.

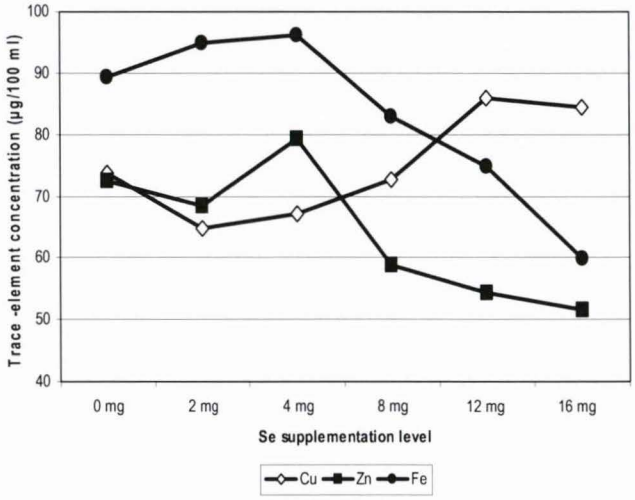
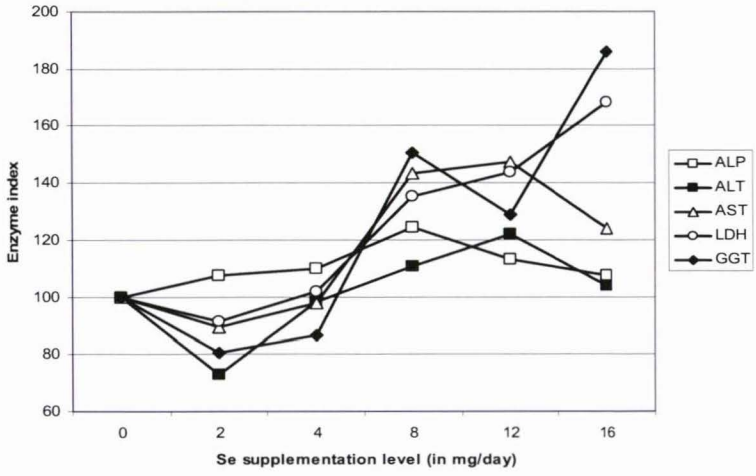


Figure 28. Changes in trace element concentration in camel serum according to the Se supplementation level (data meta-analysis of SEBOUSSI et al., 2008a, b, 2009a, b and c)

The correlations with **enzymes** Alcaline phospasphatase (ALP), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Lactate deshydrogenase (LDH), Creatine PhosphoKinase (CPK), Gamma Glutamyl Transferase (GGT) were not constant. In some cases, a negative correlation was observed between the enzymes of cellular suffering (AST, LDH and ALT) and Selenium, parameter of the cellular integrity (SEBOUSSI et al., 2004, 2008a, 2009a). One could make the assumption that those enzymes, indicators of the cellular suffering, were all the more high as the concentration in Se was weak. However, all data concerning those enzymes in the cited papers were in the range of normal values in camel (BENGOUMI et al., 1998c). At reverse, in case of Se intoxication, the cellular damage (liver, kidney, heart) increased the indicators of cellular suffering (LDH, AST, ALT) above the normal range for camel, and a positive correlation was observed (SEBOUSSI et al., 2009c) . However, by considering the meta-analysis of SEBOUSSI'S experiments, it was clear that all enzymes tended to increase with the Se supplementation level (figure 29). Surprisingly, the CPK did not increase in spite of its role as indicator of muscular dystrophy. Similar observation was reported on calves receiving high dietary Se (JENKINS & HIDIROGLOU, 1986).

Figure 29.
Changes of the enzyme index (index 100 for 0 mg Se supplementation) according to the Se supplementation level in adult camel (data meta-analysis of SEBOUSSI et al., 2008a, b, 2009a, b and c)



A negative correlation with **albumin and total protein** was observed sometimes in camel (SEBOUSSI et al, 2009a and b). Usually, after intestinal absorption, selenium forwarded by red blood cell to be reduced before passing by in plasma where it was bound to the groupings thiols of proteins, notably albumin (VITOUX et al., 1996). Probably, when selenium concentration increased in serum, the part bound to proteins would be

less important than the free selenium. This phenomenon could be more important in camel than other species and could explain the relative high concentration observed in camel after Se supplementation. However, regarding results of the meta-analysis of SEBOUSSI'S experiments, a significant decrease of all biochemical parameters from 8 mg Se supplementation was observed and a drastic decrease of bilirubin linked to the anaemia in intoxicated animals (*Figure 30*).

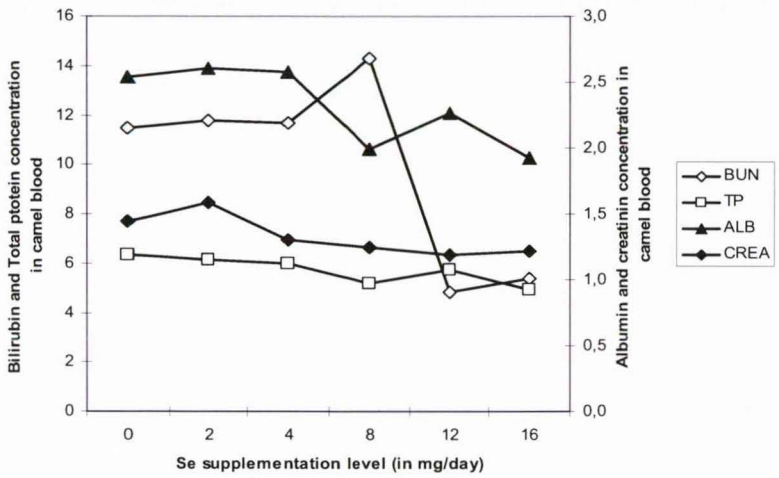


Figure 30. Changes in biochemical parameters of camel blood according to the Se supplementation level in the diet (data meta-analysis of SEBOUSSI et al., 2008a, b, 2009a, b and c)

Lymphocyte cell membranes are especially susceptible to free radical damage (BJORNSTEDT et al., 1996). So, the negative correlation with lymphocytes observed on camels could be explained by the interferences between selenium level in organism and cellular events responsible for an immune response. Elevated Se has been shown to promote peroxidative damage in *in vitro* and *in vivo* systems. The negative relationship between selenium and PCV or Hb in case of Se intoxication was sometimes observed in camel (SEBOUSSI et al., 2009c) but the anemia was not a constant symptom of selenosis in herbivorous (JENKINS and HIDIROGLOU, 1986; RAISBECK *et al.*, 1996). However, the meta-analysis of the data of SEBOUSSI et al (2008a, 2009a, b and c), showed a significant decrease of **PCV and Hb** ($P < 0.0001$) with the increase of Se supplementation level (*Figure 31*).

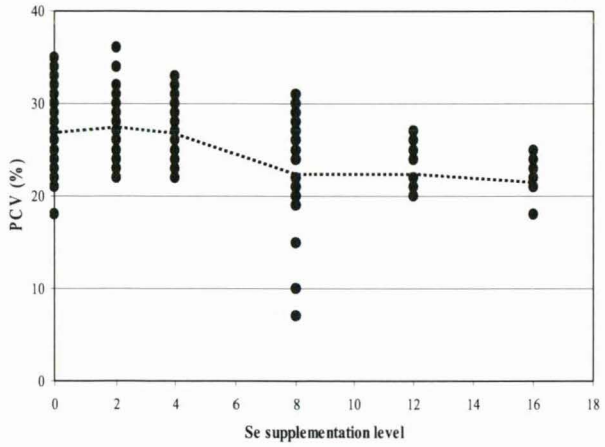


Figure 31. Change in PCV according to the Se supplementation level in mg/day (data meta-analysis of SEBOUSSI et al., 2008a, b, 2009a, b and c).

10. CONCLUSION

The metabolism of selenium in camel is quite comparable to that of the other herbivorous with similar diseases in case of deficiency or toxicosis, comparable values in serum and organs and comparable way of excretion. However, some specificities could be observed: **the richness of camel milk in selenium**, **the role of fecal excretion in case of intoxication**, **the apparent sensitivity to toxicity**, and **the high concentration in blood with high Se supplementation**. The analysis of the interactions with other blood parameters indicated the beginning of cellular suffering and significant changes from a 8mg Se supplementation per day. According to dietary Se supply and mean weight of the animal, selenosis appeared with 0.05 mg/kg LW Se supply only. Severe intoxication occurred with 16 mg Se supplementation, i.e. 0.10 mg/kg LW. These values were 5 times lower than those for sheep and cattle. **Based on these results, it seems essential to limit Se supplementation in camel at 0.01-0.02 mg/kg LW, i.e. approximately 4-8 mg per day for adult animals or 0.5-1 ppm in the diet**.

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